# *The* AUSTRALIAN AND NEW ZEALAND COLLEGE of PERFUSIONISTS GAZETTE

### DECEMBER 2016

www.anzcp.org



# **TABLE OF CONTENTS**

- Page 2 Letter from the Editor, Molly Oldeen
- Page 3 Letter from the President, Jon Van Den Berg
- Page 4 ABCP Board Report, Mark Mennen

### Perfusion News

- Page 4 Changing of the Guard at Green Lane Clinical Pefusion, Tim Willcox
- Page 6The "Bubble" Man Hands Over His Reins,<br/>Perfusion Team at Auckland City Hospital
- Page 7 Around the Pump Room Princess Margaret Hospital for Children, Rae Kelly
- Page 8 Around the Pump Room Waikato Hospital, Jack Bhana
- Page 8 Pediatric Observation at Lady Cilento Children's Hospital, Majid Arammanesh
- Page 10 Open Day at Lady Cilento Children's Hospital, Govindasamy Maheshkumar
- Page 13 Around the Pump Room...but not my own!, Casey Edwards
- Page 14 Open Heart International Bolivia, Rona Steel

Conference Corner

- Page 16 Annual Scientific Meeting, Susan Donovan
- Page 18 Inaugural Australasian Simulation and Perfusion Meeting (ASaP), Jane Ottens & Darryl McMillan
- Page 20 ELSO and Pediatric AmSECT, Fellow of Pediatric Perfusion, Molly Oldeen

### Opinion

- Page 23 King of Hearts Book Review, Martin Gill
- **Original Articles**
- Page 24 Is Serum Lactate a Predictor of Outcome for Paediatric VA ECMO?, Clarke Thuys
- Page 29 The Association between Intraoperative Glycaemic Change and Mortality is Modulated by Pre-Existing Hyperglycaemia in Cardiac Surgery, Richard Newland & Rob Baker
- Page 32 An Evaluation of Haemoglobin Measurement Devices for Cardiac Surgery, Casey Edwards
- Page 39 2016 Annual Scientific Meeting Award Winners
- Page 41 Abstracts from the 33rd Annual Scientific Meeting
- Page 57 Calendar of Events

# We care about cardiac outcomes



Terumo Australia Pty Ltd Macquarie Park NSW 2113 Australia Mount Albert Auckland 1025 New Zealand E: cs\_australia@terumo.co.jp Orders: au\_orders@terumo.co.jp

T: 1800 837 866 T: 0800 66 77 57

© 2016 Terumo Australia Pty Ltd CVS161011D859V1 | PBD00188



# *The* AUSTRALIAN AND NEW ZEALAND COLLEGE *of* PERFUSIONISTS **GAZETTE**

### www.anzcp.org

### **EDITOR**

### Molly Oldeen, CCP

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

# **ASSOCIATE EDITORS**

### Carla Zazulak, CCP

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

### Jane Ottens, CCP

Ashford Hospital Ashford SA Email: jane.ottens@acha.org.au

### SUBSCRIPTIONS

Subscription is only available through membership of the ANZCP. Please visit www.anzcp.org for the latest membership rates, terms and conditions. Note that all monies quoted are in Australian dollars and are inclusive of GST. New Zealand Members: fees for Australian Members less GST. One year free subscription is available to student perfusionists enrolled in the ABCP course. For further information on membership/ subscription, please contact the ANZCP Secretary: c/- PO Box 921, Parkville, Victoria 3025, Australia.

### ADDRESS FOR SUBSCRIPTION

Change of address, including both the old and new addresses of the member/subscription should be forwarded to the ANZCP Secretary at least one month in advance.

### ADVERTISING

To advertise in the Australian and New Zealand College of Perfusionists Gazette contact the Gazette Editor. Tel. 07 3068 3692, email. gazette@anzcp.org.

### EDITORIAL CORRESPONDENCE

Editorial correspondence should be addressed to the Gazette Editor, gazette@anzcp.org (postal address as above).

**The ANZCP Gazette** is the official publication of the Australian and New Zealand College of Perfusionists. Copyright 2007 by the ANZCP Inc. All rights are reserved. No part of this publication may be reproduced or transmitted in any form or by any means. Permission for reprinting should be obtained from the Gazette Editor.

### DISCLAIMER

All views expressed are that of an individual or institution and are not necessarily the expressed views of the Editors, the ANZCP Executive Committee or Members of the ANZCP.

### **EDITORIAL POLICIES**

Manuscripts are accepted for consideration on the condition that the Editorial Committee has the final decision on publication. The Editorial Committee reserves the right to reject or edit any material submitted for publication without reason.

**Preparing material for publication:** Preferred delivery method is by e-mail using document attachment in Microsoft Word to the editorial correspondence address. Authors who do not have access to e-mail please mail material. Correspondence should be on A4 paper type written with double-spaced lines. Please do not fold mail; use an A4 sized envelope. Please supply direct contact numbers and address for the corresponding author. Material should contain (1) Title, (2) Subject, (3) Author(s), (4) Co-author(s), and (5) Institution(s). British/ Australian English is the preferred language.

This edition of the Gazette has been proudly supported by LivaNova.

# *A* MESSAGE FROM THE EDITOR

# by Molly Oldeen, CCP.

The Gazette is a useful tool to spread knowledge and information to fellow members of the Australian and New Zealand College of Perfusion. In addition to the educational benefit, I hope it continues to inspire others to provide their opinions and experiences, as well as motivate them to conduct research.

We begin this edition with a special contribution that includes two articles about a well-known Life Member of the College, and one considered a grandfather of perfusion, Tim Willcox. After recently stepping down as Chief Perfusionist at Auckland City Hospital, Tim has submitted an article to reflect on his time in that role. In addition, the team has included a collection of accomplishments and photos to congratulate him and show their appreciation for his strong leadership over the years.

To follow, we have a variety of Around the Pump Room pieces, complete with a spotlight on the Lady Cilento Children's Hospital in Brisbane. Clinical site visit articles describe student's experiences visiting the unit. More site visits should be encouraged not only for students, but certified clinical perfusionists as well. In addition to student visits at LCCH, we also have a highlight of the Open Day held recently. Families and their children were invited to visit the hospital, tour the operating rooms and ICUs, as well as obtain some hands on experience with equipment and instruments. We have an assortment of conference reviews from the Annual Scientific Meeting, ELSO, as well as the inaugural Australasian Simulation and Perfusion Meeting. For those unable to attend the most recent Annual Scientific meeting held in Townsville, this edition contains the submitted abstracts. The conference award winners have been listed as well and deserve their congratulations.

I want to thank those who contributed articles. Success in the field of perfusion is highly due to collaboration in these types of formats. Volunteer roles for perfusionists, such as being a member of the college executive committees, presenting at conferences, as well as writing articles for the Gazette, are invaluable and greatly appreciated. For the Gazette specifically, we provide gratitude and even incentive in the form of the two awards given out at the Annual Scientific Meeting each year. Congratulations again to Martin Gill and Cynthia Riddell.

I look forward to my continued role as Editor, feeling supported by the authors, the Associate Editors, Carla and Jane, and of course, our corporate sponsors.

Molly Oldeen The Gazette Editor gazette@anzcp.org

# ANZCP STRUCTURE

### The Executive

Jon Van Den Berg – President Mark Ambrose – Vice President Matt Sheminant – Secretary Kuljeet Farrar – Treasurer Killian O'Shaughnessy – Registrar Charles McDonald – Member

### The Board

Mark Mennen-Chairman Chris Morley – Member Andrew Lahanas – Member Vincent Rajkumar – Secretary Sarah Varghese - Member

### Sub-Committees

Alison Horton – Registration Committee Jane Ottens – Simulation Committee Tim Willcox – PIRS Editor Jack Bhana - Webmaster

# A MESSAGE FROM THE PRESIDENT

# by Jon Van den Berg, CCP.

Undoubtedly everyone who attended the Townsville Annual Scientific Meeting would agree to its success. On behalf of the College thank you to our hosts Helen, Susan and Monique from the Townsville Perfusion Unit for your significant contribution.

The Executive would like to acknowledge the contribution made by outgoing Treasurer Carla Zazulak who made everything run so smooth and Mark Mennen who steps down from the ABCP. We welcome Kuljeet Farrar as our new Treasurer.

In my first term as ANZCP President I have focused on being in touch with the various groups who make up our organisation. Looking at our strengths and how we might advance our college and profession to the future. Clearly our strengths are derived from many individuals who generously contribute to our Profession.

# Website Redevelopment.

The College website refurbishment has been a priority this year. Our new webmaster Jack Bhana has made excellent progress in establishing a totally new site through webbuilder Hayley Marie design. There are a number of key upgrades especially the new interface and the addition of the e-commerce payment and receipt system. PIRS has also been revamped in collaboration with Tim Willcox. The development of the site will be ongoing.

# Perfusion Training Board Planning Meeting, Melbourne.

The meeting focused on devising a plan to update Perfusion course modules. The key outcomes, all modules will now follow a consistent format. Each module will be allocated to a supervisor who will review, update from time to time and mark each module. Supervisors did not have to be Board members.

# Simulation.

The inaugural ANZCP simulation meeting took place at the Royal North Shore Hospital in May this year. The two day course could only be described as a great success. The College has owned the Orpheus simulator for quite some years however this was the first organised training meeting.

# Perfusion Survey.

Some eighty five responses were received from Australasian Perfusionists. Outcomes from the survey were presented by Arnika Van den Berg at the Townsville ASM. Information will soon be posted on the College website. Thank you to all who participated. There were a good number of respondents who offered to participate in the profile survey, from this group we were able to contact randomly selected individuals for interview.

# Developing a future strategy.

# Training and Recertification

Simulation has gained increased recognition by the Public and Professional Groups. Consideration should be given to promoting simulation within the recertification process. Initially this could be in the form of attracting higher education points, then a compulsory component in the recertification cycle.

# **Increased Membership**

Ideally ANZCP should represent all Clinical Perfusionist's working locally regardless of which board certification they may hold. Increasing membership supports stronger representation to Government and Regulatory Bodies.

# **Regulation.**

In the interest of Patient Safety ANZCP is actively promoting regulation of Perfusion practice through the Health Practitioners Competency Assurance Act in New Zealand and the self-regulation model through Allied Health Professionals Australia. Regulation will ensure standards of practice and individuals are maintained, improved to provide best patient outcomes.

In closing I would like to thank fellow members of the ANZCP Executive for their input to a productive year.

### Jon Van den Berg CCP

President ANZCP jon.vandenberg@waikatodhb.health.nz

# AUSTRALASIAN BOARD OF CARDIOVASCULAR PERFUSION

# CHANGING OF THE GUARD AT GREEN LANE CLINICAL PERFUSION

# by Tim Willcox FANZCP, Perfusionist Ordinaire

by Mark Mennen, Chairman ABCP

At this year's AGM the board sought to pass a resolution to add an additional member to the board. The additional position is that of course coordinator and reflects the additional workload of the board with the Autotransfusion course and the return of the Diploma course. Prior to the Swinburne Masters course, the Board had seven members and was only responsible for the Diploma course, as we were yet to commence recertification or the Autotransfusion course. While we have had a great deal of assistance from individuals who are not elected members of the board, we are keen to have this effort recognised in a formal manner.

Once again the focus of the board is the continuing improvement of the Diploma Course. We are now taking a midyear student intake and also moving the midyear exam to September to make an even six months between exams to better align with students completing the diploma at that time.

We currently have 18 students enrolled in the Diploma Course (11 Australia, 4 New Zealand, 2 Singapore, and 1 Hong Kong). We expect around six of those to sit the certification exams in February along with two Swinburne graduates. I encourage all students and supervisors to be active in the reviewing of course content and delivery. The feedback of those on the receiving end of the material is extremely valuable.

The Autotransfusion Course has continued to receive strong interest/enrolments at each of the three intakes and has been completed by well over 200 individuals. Thanks to Jessica Ozdirik for graciously stepping up to assist in the delivery and coordination of the course.

Thirty-seven Perfusionists were due to recertify this with three individuals retiring and only two late returns, one of whom was on maternity leave. This is the best response in some years so thank you to all. As stated in the last board report, any member whose certification has lapsed and wishes to recertify, please feel free to contact the board as we are keen to accommodate where possible.

As my time with the Board has come to an end, I would like to thank all those who have contributed so much of their time, particularly Clarke Thuys, Arthur Preovolos, Andrew Lahanas, Vincent Rajkumar, Chris Morley, Sara Varghese, Jessica Ozdirik, and Martin Bennet. On November 16th, I stepped down from the role of Chief Perfusionist after about three and a half decades in that role. This was a conscious decision made last January and confirmed 6 months ago - so my team have had time to absorb what they might well have thought was a pipe dream.

Perfusion is simultaneously undergoing a formal review of the way the department can meet future needs and like most of these exercises in DHBs this is typically running behind schedule but hopefully will be completed by the new year. Until the appointment process for the chief perfusionist position is competed (there is local and overseas interest) Jude Clark will be acting Chief.

I am thus now a carefree clinical perfusionist to have more time in the OR which I enjoy, be more involved with our three trainees' teaching, have more time for research interests, be available to mentor colleagues where there is interest and continue running the Perfusion Downunder Meeting and the Perfusion Incident Reporting System as well as my involvement with Simon Mitchell at the University.

It's been a fascinating ride and the changes and gains from those heady days at Green Lane in the 70s have been remarkable and exciting. Starting a career in perfusion with Sir Brian, David Cole, Eve Seelye, Marie Simpson, Basil Fergus, Ed Harris John Neutze, Trevor Agnew and those that followed as mentors and friends was a unique opportunity for a youngster.

I am deeply indebted to the perfusionists both past and present who have given me outstanding support in developing the service to a point where we have re engaged in training and at the other end have a number of perfusionists pursuing post graduate studies. As well there is a very solid team of very experienced perfusionists to take perfusion forward.

Externally my friendship and association with Prof David Stump from Wake Forest university, Prof John Murkin from London Ontario through the Key West

Outcomes group, Prof Hilary Grocott from Duke, now at St Boniface in Winnipeg, Prof Merry and last but not least Prof Rob Baker from Flinders has been a profound influence in my role in the Green Lane CTSU, any success I may have achieved and to my love of the job.

So, a new era for both myself and the unit. Change is exciting.





# 

# **TCM**5 monitor

# Smarter safer transcutaneous monitoring

Offering non-invasive monitoring of patient  $tcpCO_2$  during anaesthesia and recovery



Smart meets sensible

Phone: 1800 247 254 Email: marketing@radiometer.com.au

www.radiometer.com.au

# *THE "BUBBLE" MAN* HANDS OVER HIS REINS

# by The Perfusion Team at Auckland City Hospital

After a 35 year illustrious reign Tim Willcox has decided to step down from his position as Chief Perfusionist at Auckland City Hospital.

Tim Willcox moved from Rotorua to Auckland in 1970 and in his words "to avoid starvation got a job as an orderly at Auckland Hospital". He was seconded to Green Lane to train as an anaesthetic tech however a position came available in the then "bypass unit" and Tim never looked back. As Tim quotes in his work bio "They were heady days with enormous challenges – having Sir Brian Barratt-Boyes as the boss was a marvellous experience. The kit then was rudimentary - low level alarms were a novelty. Through a series of staff changes (bloodless coups) I got the charge position in 1976".

What started as 4 perfusionists & an assistant in a tiny box of an office (with smoke constantly pouring out) has now grown to a staff of 21 with ballroom facilities.

Through the years Tim has been known to be a colourful, vibrant personality focused on occasion to a mixture of bad habits such as 2 pies & chips for lunch, smoking, drinking red wine and dying his hair in the perfusion tub causing frequent hair colour changes. The older Tim is now health focused, has dry times, exercises and eats veges, although often done so with his fingers or a second hand dirty fork.

On a more serious note Tim has made a significant contribution to the perfusion profession. His research in "bubble studies" has changed perfusion practices worldwide, earned numerous awards and resulted in Tim been invited to present all over the world.

Tim has served 3 years as president of the ANZCP, 4 years on the Board and was awarded a life membership in 2012. He is continuing to develop the PDU meeting he helped establish in 2005 and has an ongoing interest in research. Tim will remain an invaluable mentor in the department and we look forward to having him on the floor more.

Tim should be classed as a grandfather of perfusion after having served 44 years (prisoners serve less for murder). We wish him well as he takes on a better work life balance heading toward his golden years, spending time with family (7 rabbits included) and pursuing his recently found passion of tramping with Brig.



# AROUND THE PUMP ROOM

# Rae Kelly, Director of Perfusion, Princess Margaret Hospital for Children.

I thought I would give you some insight to Paediatric Cardiac Services at Princess Margaret Hospital in Western Australia.

Cardiac surgery in Western Australia commenced in the 1970's at Royal Perth Hospital. With very few of the original people around from that era I cannot guarantee the exact timeline for some of the dates.

I believe that the first cases were closed cardiac operations. Sadly, no one can remember the name of the Surgeon who started the programme.

Mr Trevor Nicholls and Mr Martin Carter took over Paediatric Cardiac surgery at Royal Perth Hospital including open cases and I believe Gordon Tozer was the keen Clinical Perfusionist at the time. Trevor provided the Cardiac Services for Children in May 1986 when they moved from the Royal Perth Hospital to Princess Margaret Hospital.

Royal Children's Hospital in Melbourne continued to provide care for patients with more complex congenital cardiac lesions.

When Trevor retired there was no paediatric cardiac surgery in Western Australia. The team from Melbourne Children's Hospital took on the service in 1997 visiting Perth 2-3 times per year until Mr David Andrews finished his fellowship at the end of 1999. Late 1999 I went to Sydney to learn about Paediatric Perfusion and later joined David Andrews back in Perth in early 2000.

In early 2000 David Andrews commenced as a full time Cardiac Consultant at Princess Margaret Hospital with Perfusion provided by myself as a secondment from Royal Perth Hospital. On the 1st of July 2010 Princess Margaret Hospital took the decision to further develop the cardiac surgical service and employ 2 full-time Clinical Perfusionists. Nigel and I took up those positions.

Nigel joined us at Royal Perth Hospital in 2004 from Great Ormond Street Hospital and was very keen to delve back into



the land of Paediatric Perfusion.

I myself worked at Royal Perth Hospital as a Cardiac Nurse and started my interest in Perfusion as a secondment to see if this profession was for me. This was around 1996 where I spent 3 months finding my feet. I loved it and have never looked back.

In the early part of 2017 Princess Margaret Hospital will be moving sight to QEII and Princess Margaret Hospital will have a name change to Perth Children's Hospital.

We have been fortunate to early adopt one of two new fully mast mounted S5 Sorin Heart Lung Machines. We have the Connect data management system but until we move to the new hospital we will continue to use the existing data management system.

Below is a picture of our pump with the two blank monitors being the Surgeon's camera and the hemodynamic monitoring.

We use the NIRS Foresight Elite Monitoring on all our cases which downloads to the DMS and to the Surgeons monitor throughout each and every case.

This week we have had Sohail and Nicola from LivaNova helping us while we transition from the S3 to the S5. We absolutely love the S5 and we are both looking forward to installing them in the new Perth Children's Hospital in 2017.

We continue to develop our ECMO service which includes a bi-annual ECMO nurse specialist education program. The threshold for instituting ECMO as a recognised therapy (rather that a last ditch rescue attempt) has greatly lessened and as such our ECMO run numbers are increasing year on year.

We are also very lucky to be supported by our hospital and David Andrews to allow us to attend cardiac missions with Open Heart International. Both Nigel and I have visited Cambodia, Rwanda, Tanzania and PNG and as many of you know these trip can be challenging yet overwhelmingly rewarding both professionally and on a more personal level. We have also had the opportunity to work in Brisbane at the Mater as an exchange programme where both Nigel and myself have spent 2 weeks using the S5 and the different environment and staff that each unit works under.

We have enormous opportunities that are funded such as conferences both local, interstate and internationally. If anyone is thinking to shift to Perth we have a great city with the best weather.

I look forward to our next chapter when the big move occurs early next year.

# AROUND THE PUMP ROOM

# Jack Bhana CCP, Waikato Hospital

We are a medium sized unit located in Hamilton, heart of the Waikato region famed for the Lord of the Rings movie set and richest Dairy Farming land in NZ. Located some one and a half hours drive south from Auckland.

Our unit is currently funded to perform 640 adult cases annually. We conduct support services for all adult heart lung procedures, Minimally Invasive AVR's, ECMO/VAD, IABP, Cell saving, HIPEC, Hybrid Cath Lab procedures, Hybrid surgical theatre, TAVI's, Lead Extractions and hybrid procedures, including major Aortic Arch reconstruction. We operate a closed venous bag system and a hardshell reservoir. We look forward to upgrading our three Heartlung machines and heater-cooler units in the New Year.

We serve a catchment area of 50,000 sq Kms, approximately 800,000 people with five main Regional Hospitals. We have five Cardiothoracic Surgeons and one Fellow.

Our perfusion staffing is currently set at four full time FTE's. British, American, and two Kiwi's.

Having taken on the role as webmaster for the new ANZCP website and with Jon Van Den Berg as College President our nonclinical time has become well occupied.

# PAEDIATRIC OBSERVATION AT LADY CILENTO CHILDREN'S HOSPITAL

# Majid Arammanesh, St George Hospital

At St George Hospital, Sydney we mainly provide Adult Cardiac surgery. I was keen to visit a paediatric centre and observe different aspects of Paediatric perfusion.

I was given the opportunity to visit Lady Cilento Children's Hospital, Brisbane.

This centre has been designed to provide specialist Paediatric services and Cardiac Surgery since November 2014.

I was warmly welcomed by the team of Perfusionists, Surgeons, Theatre Nurses and PICU staff.

Considering, Carla Zazulak, Director of Paediatric Perfusion Department and team of Perfusionists were busy at times, they still patiently managed to answer my questions and were forthcoming with advice to make me feel supported and part of the team.

During my week at Lady Cilento Children's Hospital, I learned so many things about Paediatrics Cardiac Surgery and specific perfusion techniques.

This included:

I observed all aspects of Paediatric cardiac surgery.

I learned how to set up a new paediatric circuit and went through the check list alongside a senior Perfusionist.

I understood the importance of delicate preparation of paediatric prime solution, PH adjustment and electrolyte balance. I was impressed by the team effort for preserving blood to the last drop, continuous cell salvage and auto-transfusion.

I realised the critical role of continuous Zero Balance Ultra Filtration (ZBUF) and Modified Ultra Filtration (MUF) in Paediatric Cardiac Surgery.

I understood the concept and set up system for Vacuum Assist Venous Drainage (VAVD).

Also, I had the chance to experience firsthand preparation and initiation of neonatal VV-ECMO and retrieval from adjacent Hospital to Lady Cilento Children's Hospital.

It was invaluable experience.

I would like to thank Carla, Anthony, Mahesh and Nicole who made my journey such a fulfilling and pleasant experience.





# Asia-Pacific ELSO Conference 2017 ECMO...and Beyond

# The future of Mechanical Support

# October 12-14 Gold Coast Convention & Exhibition Centre IMMEDIATELY FOLLOWING ANZICS/ACCCN ASM 2017

The 2017 APELSO conference will welcome global leaders and innovators in the field of ECMO, VAD, and total artificial hearts.

A multi-disciplinary team approach is essential to ensure that our patients thrive, not just survive

This event is endorsed by:



Hear from world experts and innovators on the role of ECMO both now and into the future, and engage in discussions on how to move forward in this exciting area of research and clinical management. Discussions and debates will draw on experiences from clinicians, allied health professionals, engineers, scientists and researchers.

# Get Hands-On

- State of the art, high fidelity ECMO simulation workshops, run by an expert team from Hong Kong
- Mechanical support workshops hosted by a team from Hôpital Pitié-Salpétrière Paris, one of Europe's largest cardiothoracic centres

Experience an Australian-first interactive learning opportunity. Learn in a truly hands-on environment, focussing on circuit composition, cannulation & induction of ECPR.

This is your chance to stand side by side with, and learn directly from, global leaders as they demonstrate and teach the practical skills needed to ensure optimal patient outcomes.

An amazing opportunity like this only has a limited number of spaces, to ensure a truly interactive learning experience - so don't miss out!

REGISTER YOUR INTEREST NOW: www.apelso.com

# SAVE THE DATE

TANZCP

Critical Care

Associate Professor Daniel Brodie MD Director, Medical Intensive Care & Medical Critical Care Service, Columbia University Medical Center



Professor John F Fraser Director of Critical Care Research Group, The Prince Charles Hospital QLD

Associate Professor Andreas Schibler Paediatric Intensive Care Staff Specialist. Director of Paediatric Critical Care Research Group. Lady Cilento Children's Hospital



Professor Alain Combes MD PhD Professor of Intensive Care Medicine, University of Paris Head of ICU, Institut de Cardiologie, Höpital Pitié-Salpétrière, Paris, France

Associate Professor Graeme MacLaren Director of Cardiothoracic ICU, National University Hospital, Singapore



Professor Robert H Bartlett MD Professor Emeritus, Section of General Surgery, Division of Acute Surgery University of Michigan Medical School

Associate Professor Vin Pellegrino Care Specialist & Head of ECMO Clinical Services The Alfred Hospita



urism and Event

**Q**ueensland





Critical Care

Senior Inte

THE COMMON GOOD

# OPEN DAY AT LADY CILENTO CHILDREN'S HOSPITAL

# Govindasamy Maheshkumar, Pediatric Perfusionist, LCCH, Brisbane, Australia

One of our Consultant Cardiac Surgeon Dr. Prem Venugopal, perceived the idea of inviting the kids from the Cardiac Service at Lady Cilento Children's Hospital (LCCH), along with their parents to give them a "behind the scenes look" at our cardiac services at LCCH.

The event was advertised on the hospital and heartkids.org websites, requesting the interested parents to register for the same. The event was well received by parents and kids with more than expected attendees (over 100 people incl. children) to grace the 'first ever Open Day occasion' in Queensland.

Open Day was well supported by staff volunteers and generously sponsored by multinational companies who make & supply equipments and disposable heart care products.

The event kicked off with registration and morning tea for the visitors.

The morning session started with presentations about the history of the cardiac service, staff perspectives about working with the heart kids, exciting developments in the field of cardiac research at LCCH and more.

After light lunch, attendees were divided into 4 groups for the tour of the hospital with stop over at the Cardiac Operating Room and Cath Lab. The kids were even dressed up like surgeons and were allowed to try the interactive simulations and a chance to touch and feel the products & devices and then meet the companies who make them. This created an inspiration & motivation for the kids who aspire to be a surgeon one day!

The programme closed with a Q & A session by parents and doctors, which ended with a very positive, constructive feedback and suggestions from the parents.



# 

Hynes Convention Center/Sheraton Boston Boston, Massachusetts AmSECT



AMERICAN ASSOCIATION FOR THORACIC SURGERY A Century of Modeling Excellence

In Collaboration with AATS-AMERICAN ASSOCIATION FOR THORACIC SURGERY

Australian and New Zealand College of Perfusionists 3rd INTRAOPERATIVE

# MANAGEMENT OF BLOOD

5th and 6th May 2017

Amora Hotel, Sydney, New South Wales

# Key Note Speakers:

**Dr Jonathan. H. Waters** Magee Womens Hospital of UPMC Pittsburgh. USA

Professor James Isbister A M University of Sydney

# **Dr Bruce Cartwright**

Staff Specialist Anaesthetist RPAH Clinical Lecturer, University of Sydney



# **Dr Kerry Gunn**

Consultant Anaesthetists Auckland City Hospital

# Associate Prof Rob Baker

Perfusionist Flinders Medical Centre Major Sponsor

NOV.



# AROUND THE PUMP ROOM... BUT NOT MY OWN!

# by Casey Edwards

Following the recent ANZCP ASM in Townsville, I was fortunate to spend a week with Carla, Tony and Mahesh at the Lady Cilento Children's Hospital, Brisbane. As part of obtaining our CCP students must observe 10 paediatric cardiac surgical cases, which was the purpose of my visit.

The hospital itself is stunning. Having been opened in 2014, the modern facility is bright, fresh and spacious. It sports numerous rooftop gardens plus ample training and professional development facilities.

Needless to say, visiting Lady Cilento was an exceptionally valuable learning experience. I appreciated the opportunity to see the use of paediatric-specific equipment. Observing other perfusionists in theatre and discussing their case management strategies highlighted that in our profession, different approaches may be used to achieve a common goal. I have taken away a number of ideas, perfusion techniques and strategies from LCCH I intend to research and hopefully implement into my own practice. I enjoyed the opportunity to set up and prime an unfamiliar perfusion circuit, which tested my understanding of circuit dynamics. Above all, I admired the collaborative way that the Lady Cilento perfusion team works together, and the incredible dedication they show in providing the best possible care for their little patients. I was also fortunate to spend a day with Charles McDonald and the Prince Charles Hospital perfusion team. Observing adult cardiac surgery was much more within the realm of my comfort zone. However, the differences in practice I observed and the discussions I had at Prince Charles further inspired me to reflect on my own practice and strive to do better. I admired the diverse case mix that their perfusion team is able to support, plus the organization involved in undertaking 6 cardiac cases per day. I was also amazed by the incredible on-site research facilities and look forward to seeing the ideas and products being developed at Prince Charles Hospital come to fruition.

Above all, my trip to Brisbane highlighted what an incredibly unique and supportive profession we are a part of. I felt so welcome at both institutions and every perfusionist I spoke to was selfless with their time and knowledge. I would encourage fellow and future trainees to visit other units. Not only did I learn an incredible amount of useful knowledge, I retuned home feeling excited about perfusion and grateful to be a part of such a collaborative, supportive professional community. Many thanks to Carla, Tony, Mahesh, Charles and the Prince Charles team.



# OHI TRIP TO BOLIVIA

# by Rona Steel, September 2016

I was thrilled to be invited to accompany Open Heart International (OHI) on their first trip to Bolivia! Bolivia is in eastern Europe- isn't it?? Alas, off to South America we went in September this year for a week of open heart surgery at the Univalle Hospital in the city of Cochabamba. The trip was partially funded by Rotary, one of the project partners.

To my surprise it was a clean, relatively modern city. Women in high heels walking along the pavements outside glass-fronted shops selling fitness gear. Also to my surprise, the variety and quality of food and wine available was exceptional.

The Univalle hospital is a privately owned hospital, although even the government hospital are user pay, and already engages in open heart procedures- approximately 20 cases per year. It had a modern cath lab and clean wards with more than adequate monitoring and anaesthetic equipment. The local perfusion and anaesthetic staff were very experienced and warmly welcomed us and were keen to hear of our suggested improvements.



Our mission was to educate and share experience with the Bolivian clinicians, as well as operate on low soscioeconomic patients who would otherswise have no access to such surgery. In Bolivia, the poor primarily include villagers and farmers from the high country. Most of Bolivia is at very high altitude and people living at this altitude have adapted using various compensatory measures to their physiology. They have very high hemoglobin levels (one patient had a preop Hct of 61%), higher O2 carrying capacity as well, larger alveolar area, shorter heights and higher nitric oxide levels. These compensatory mechanisms were advantageous for their perfusion run- but not so for the other specialties involved in their treatment. We performed 9 open heart procedures over the 5 days and none of them were straight forward. Each 'simple' valve case ended up requiring additional reconstructions and remodelling once we opened up. Dr Nicholson did an amazing job, as did the entire theatre team!!!



The complex physiology and anatomy made the cases challenging. On top of this, the old local Stockert heart lung machine (HLM) also proved exceptionally challenging. It was personally owned by one of the local cardiac surgeons.

Understandably, there were no 'mod cons':

- CDI
- In line monitoring/ venous saturations
- Electronic data capture
- Vacuum assist
- UPS

Actually, there were no 'highly desirables' either:

- Temperature monitoring
- Air detection of any sort
- Low level devices
- Constant reliable power source



In fact, there were also no 'basics' like:

- Cardioplegia pump
- Lighting (it was so dark I couldn't see the colour of the venous blood)
- Any pressure monitoring (at all)

• A recirculation line in the circuit (which is surprisingly

- handy upon reflection)
- An oxygen analyzer
- Reliable raceway (it was quite worn & occlusion impossible)
- An ACT machine

• An arterial filter holder- it was sitting sideways

• A heater cooler (just a heater)

• Tubing clamps that successfully clamped the tubing

But apart from these minor considerations, it was just like home!! I had brought an old sphygmo gauge for pressure monitoring, some spare 2 litre blood bags and an ACT machine from home just in case. These proved invaluable. I compensated for the lack of other apparatus with hypervigilance, my i-phone torch and lots of adrenaline. Furthermore, ashamedly my Spanish was non-existent and we communicated as best we could, often with many hand gestures and smiles.



The mountain people with regurgitant valves had massive circulating volumes

which was great for transfusion requirements and low level safety but proved challenging when I had to store these great volumes rapidly. The 2L bags successfully stored the extra volume. Shame there was only a small luered port in which to remove the flooding volume. I rapidly secured other overflow options- like the tray from the circuit packaging. Luckily it was never required.



These 2L bags were also great to collect the residual pump blood into at the end of the case for return back to the patient. This was a new feature for the locals. We also discussed prime recipes, occlusions, blood cardioplegia via a pump and the importance of measuring ACT's (especially with only 5000U heparin was given in the prime for these patients with massive circulating volumes).

The circuit they used was a single use, sterile 'standard 'Bolivian circuit made by Terumo. Suggestions on circuit modifications were not really possible as all Bolivian centres use the same circuit. Any changes would have to benefit all Bolivian centres and communication between centres is difficult.

It was a blast from the past to arrest the heart using cold crystalloid cardioplegia (made by hand) given by the anaesthetist via a giving set. Well done to the muscles used and ingenuity of the giving set design.



In conclusion, we had a fantastic team of hard working, fun, resourceful professionals, each a maestro in their field and I enjoyed the experience immensely. It challenged my skills, I witnessed extreme physiology and I enjoyed sharing ideas and techniques with the local perfusionists. Furthermore, we were incredibly well looked after during our entire stay.

After the week in Cochabamba, I took the long way home and trekked in Peru, saw Macchu Piccu and stopped over in Bueno Aires. A wonderful trip!



# by Susan Donovan

The 33rd ANZCP ASM was held in Townsville on 27th to 29th October this year at the Southbank conference Centre. The conference was well attended by delegates and speakers from Australia, New Zealand, India, Japan and the USA.

We would like to sincerely thank Hideshi Itoh and Phil Scott for travelling the long distances from Japan and the USA respectively to present at our conference. We would also like to acknowledge our invited speakers Dr Anand Iyer and Dr Pankaj Saxena. Both of these speakers are well regarded surgeons from the Townsville Hospital and took time out of there busy schedules to present at our conference. Dr Saxena was instrumental in organizing a live stream from USA so that his co author James Neal could present and participate in discussions on their topic.

Our keynote speakers provided us with high quality and interesting presentations on a range of subjects from Paedeatric surgery, ECMO, surgical techniques, minimizing primes, prime additives and blood physiology during cardiac surgery. Additionally a very important topic on the minds of many perfusionists was addressed by Darryl McMillan with a session on HCUs and NTM.

We were very pleased that Geoff Dobson who presented at the 2015 ASM was able to do an encore presentation on developments of his research with ALM and his work with the US Special Forces involving ultra small volume therapy for hypotensive resuscitation.

The format of the conference included an around the pump room session and short poster style presentations which were included to encourage new and nervous speakers an opportunity to be involved. We also had five trainee perfusionists present as part of their course requirements. These papers were of a very high standard. The caliber and enthusiasm of the trainees is an indication that the future of perfusion is in good hands. Social activities are an integral part of any conference. Our two main functions included a Friday night dinner held at the Picnic Bay Surf Life Saving Club. This informal function provided time for attendees to meet and greet and renew old acquaintances. The venue showcased Townsville's beautiful Strand and the weather was perfect.

The more formal Gala event was held at the Brewery which has historical value. The building was constructed between 1886 and 1888 and served as a postal and telephone office before being used as the Military Communications headquarters in WW2 and then back to being a post office until 2001 when it was transformed into Brewery incorporating a bar, restaurant and function room. The event was well attended and a good time was had by all.

Congratulations to the award winners:

Terumo Award	Annette Mazzone			
Medtronic Encouragement award	Richard Newland			
Liva Nova Sid Yarrow Award	Casey Edwards			
ANZCP Meritorious Award	Clarke Thuys			
Gazette Award Martin Gill & Cynthia Riddell				
Education Scholarship Emmers	on Sgammotta			

And last but not least on behalf of the ANZCP and the organizing committee we would like to thank our corporate sponsors for their ongoing support. The amount of time and effort required to attend our conferences is significant. The trade displays were educational and interactive. Congratulations to Terumo for being awarded the best trade display.





# AUSTRALASIAN BOARD OF CARDIOVASCULAR PERFUSION AUSTRALASIAN AUSTRALAS

The ABCP invites all healthcare workers interested in Autotransfusion to enrol in the 2017 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 2017 course commencement dates:

20 February (applications close 13 Feb)
22 May (applications close 15 May)
21 August (applications close 14 Aug)

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

# HIGH AND LOW FIDELITΥ "HANDS-ON" WORKSHOPS PERFUSION APPLICATIONS AND TECHNIQUES

Jane Ottens and Darryl McMillan



The inaugral ASaP Meeting, Australasian Simulation and Perfusion, was held this year in May 2016, at the Kollings Institute of Medical Research, adjacent to Royal North Shore. Hospital, Sydney.

Regular simulation workshops have not occurred for many years within the College. The ANZCP was one of the leaders in perfusion simulation, when we first initiated workshops for students many years ago. Simulator days attached to the ASM were trialled for a number of years, but due to lack of numbers this was ceased. Simulation is a learning tool, that should be incorporated into all of our continuing education, and as the College owns an Orpheus simulator, it was decided to re instate a simulation workshop, to run alternate years to the ANZCP IMob meeting (Interoperative Management of Blood)

The meeting program was developed to target all practicing perfusion groups, with the aim to either provide new or refresh knowledge on perfusion techniques and applications.

- Perfusion Trainees
- New Graduates
- Senior Perfusionists

A conscious effort to steer away from the standard crises management normally associated with simulation by the organisers, to focus more on perfusion techniques and application.

We had the pleasure of Mr Mark Bearss, currently Medtronic's principle specialists in perfusion simulation, help with our planning development as well as joining as an instructor.

The instructors for this meeting included Mark Ambrose, Sarah Armarego, Monique Brouwer, Ray Miraziz, Keiron Potger, Arthur Preovolos, Vincent Rajkumar, Brain Wright, Darryl McMillan and Jane Ottens. We would like to gratefully thank everyone involved for their enormous amount of their personal time working on their topics and scenarios and input into the workshops.

Although we use the term "instructors" for those running the simulation stations, their role was more as a facilitator or catalysts for the exchange of information. Each participant was encouraged to add to each scenario with opinions/ options used in their units, so to share knowledge. The instructors also all took away information as well.

Decades ago, if we trained as perfusionists, we would visit

units routinely to see what was new/ how to implement a new technique into clinical practice. Now with the internet, we can "Google" how to undertake any procedure that we need to do. While this is an amazing "tool", the face to face interaction between us all and what we do is so invaluable. We can all pick up ideas of how to (and not to) do things by sharing what we all do day to day – which we all discovered ,there are so many different ways we all undertake the same technique!

The meeting was limited to 25 - 30 participants and this year, with attendees from Australia, New Zealand and a number of perfusionists from Vietnam. The experienced of the attendees ranged from student to senior practicing perfusionists, who all rotated through 3 high fidelity stations with heart lung machines/ ECMO, and 1 low fidelity stations.

All groups experienced every scenario on each day, providing 9.5 hrs of hands on simulation over the 2 days.

### Day 1

- Vacuum assisted Drainage
- Perfusion for thoracic aortic surgery
- ECMO VV
- Heparin management

### Day2

- Prime reduction RAP (and various technique to undertake this) circuit size
- Failure to wean from CPB options
- ECMO VA
- Cerebral Oximetry

We would like to take this opportunity to thank our major sponsor Medtronic Australasia who support, generosity and encouragement in helping us to create this meeting has been limitless. In particular Annika Wright who assistance and wizardry was of great assistance and help throughout the planning of ASaP. The generosity of LivaNova, Maquet Australia, Terumo Australia ,Covidien, Device Technologies and Ecomed who without any reservation or remuneration offered their supported to this meeting, suppling all the equipment we require to make ASaP happen.

The support from all the companies was unbelievable from the moment the meeting concept was pitched to the corporate sector -any request made was met with nothing but enthusiasm and positive responses. And last our thank you to the Kolling Institute of Medical Research at the Royal North Shore Campus. The logistics to undertake this type of workshop is enormous and a big thank you to Royal Northshore hospital and the Team, for helping with last minute requests for equipment, to ensure the two days ran smoothly. A debrief from the meeting, along with looking at all feedback, occurred in September. We discussed what worked, what we would /could do differently and how we can improve to continually make what is simulated, to be relevant to current day perfusion practice. We are also looking at increased ABCP points gained for attendance, due to the intensive hands on nature of the workshop.

We look forward to ASaP in 2018, and hope you will all support this workshop and this will become a regular addition to perfusion calendar



Getting ready for the simulation sessions



Scenarios run in theatre like conditions



Group discussions and input from all, made the sessions valuable to all who attended.



Each room had different equipment and set ups

























# ELSO 2016

# Molly Oldeen, CCP

The September 2016 Extracorporeal Life Support Organization (ELSO) meeting had the benefit of being combined with the Pediatric American Society of Extracorporeal Technology (AmSECT) meeting. It is always well attended with participants from around the world. Attendance reached a record high this year with specialties ranging from perfusionists, surgeons, intensivists, nurses, respiratory therapists, and more. As a prior attendee of the ELSO/Pediatric AmSECT meeting in Ann Arbor, Michigan in 2014, I have seen the advantages of combining the meetings multiple times now.

The conference venue was at the Manchester Grand Hyatt in downtown San Diego, California, located directly on the bay waterfront at one of the most popular locations in Southern California.

The preconference symposium was well attended and included topics such as cannulation techniques, and decisions to cannulate in unique and difficult situations. This was presented alongside best and worst case ECMO scenarios. The welcome session included talks from some of the top leaders in the field, Heidi Dalton and Robert Bartlett. Session breaks took place in a large conference room that hosted submitted abstract posters, as well as the usual vendor tables.

Simultaneous sessions on Saturday included breakouts, such as one specific to Perfusion, CPB, ECMO and VADs (which I attended). There was a nice combination of quality improvement projects (protamine administration consistency), perfusion strategies for special techniques such as ABO incompatible heart transplant, management of MAPCAs on CPB, as well as an informational session on Rotem v Thromboelastography. The pediatric session concluded with presentations of Fellow of Pediatric Perfusion awards, of which I was a lucky recipient. The simultaneous sessions ended with the presentations of awards. This includes ELSO Center of Excellence designation, presented to qualifying programs, as well as best abstract awards.

The conference ended Sunday morning with notable presentations on important topics such as anticoagulation on ECMO using Anti-Xa as the sole indicator, the new EOS membrane oxygenator, as well as usage of blood products on ECMO. In addition, there was a heartfelt session with families, where the patients and clinicians were able share their personal experiences.

One of the more relevant presentations in my opinion was study of the relatively new EOS membrane oxygenator for ECMO. When product availability becomes uncertain at times due to backorders or manufacturer issues, it is convenient to know other options, and how they compare to ones currently in use. I personally appreciated this talk because the study design was simple but very informative, and could inspire more centers to engage in this type of research.

Another useful presentation discussed the primary usage of Anti-Xa as a measure of anticoagulation on ECMO patients. There was a discussion on the variability in ACT measurement device results, as well as the lack of correlation between ACTs and Anti-Xas. With a high variability of tests used to measure anticoagulation, this was quite applicable. There is the possibility that heparin and its monitoring may not be as applicable in the future, due to heparin alternatives that some centers are looking into.



From a technology and organizational standpoint, the ELSO conference is always impressive with the consistent use of the CrowdCompass mobile phone application that allowed access to both the conference program, presentation slides, conference attendees and contact information, as well as presentation evaluations. Access to the application is available both during the conference and for a set period of time afterwards.

Overall, it was an excellent meeting packed with distinctive and educational presentations. The social events provided entertainment as well, and consisted of the usual opportunities for networking, collaboration, and catching up with colleagues within the perfusion and ECMO community.

# Fellow of Pediatric Perfusion Designation

As I mentioned during the conference summary, a session of the yearly Pediatric AmSECT portion of the conference gives out awards to those designated as a "Fellow of Pediatric Perfusion", also known as FPP. This designation has been awarded to just over 100 pediatric perfusionists so far. It reflects a dedication to the field of pediatrics, and a commitment to strive to improve the quality and consistency of our patient care.

Applicants are required to be certified by the American Board of Cardiovascular Perfusion, or equivalent programs, such as the European Board of Cardiovascular Perfusion, the Society of Clinical Perfusion Scientists of Great Britain and Ireland, and the Australian Board of Cardiovascular Perfusion.

Other requirements include a variety of items, such as a case log documenting 200 cases (40% of which are patients under 10kgs). It also requires letters of recommendation from a surgeon, anesthetist, and chief perfusionist. An updated resume and personal statement are necessary as well. FPP designation is an ongoing responsibility. Future requirements to maintain this status may involve conference participation/presentations, as well as other obligations. Although award winners are not necessarily required to attend the conference, it is highly recommended, as there is a presentation ceremony to provide the deserved congratulations.

For more information and a complete list of requirements, visit the AmSECT website, amsect.org.

# **Fellowship Pledge:**

Recognizing that the American Society of ExtraCorporeal Technology seeks to exemplify and develop the highest traditions of our profession, I hereby pledge myself, as a condition of Fellowship within the Society, to live in strict accordance with its principles and regulations.

I pledge to pursue the practice of pediatric perfusion with honesty and integrity and to place the rights of my patient above all else. I promise to deal with each patient, as I would wish to be dealt with if I were in the patient's position. I also promise to further my knowledge through research and education, and to share my knowledge and experience with others in an effort to advance the practice of pediatric perfusion.

Upon my honor, I declare that I will advance my knowledge and skills, will respect my colleagues, and I will seek their counsel when in doubt about my own abilities. In turn, I will willingly help my colleagues when requested.

Finally, I solemnly pledge myself to cooperate in advancing and extending the art and science of pediatric perfusion by my Fellowship in the American Society of ExtraCorporeal Technology.





# **INSPIRE**<sup>™</sup> Optimized perfusion

# Because no two of your patients are the same.

INSPIRE™ is part of the integrated **HeartLink™ System** and offers a low (DOV) Dynamic Operating Volume to reduce hemodilution and (AKI) Acute Kidney Injury occurrence.



Superior handling of gaseous micro emboli (GME) lowers the risk of cognitive dysfunctions.

www.livanova.com



# Book Review

# KING OF HEARTS: THE TRUE STORY OF THE MAVERICK WHO PIONEERED OPEN HEART SURGERY

# Reviewed by Martin Gill

# G. Wayne Miller. 352 pp. New York, New York: Times Books, 2000. ISBN 0-8129-3003-7

Cardiac surgery to repair intracardiac defects is largely considered to be routine in all but the most exceptional of cases today. Of course, it has not always been this way. Only as far back as the early 1950's successful repair of an atrial septal defect in a safe and repeatable manner was fanciful. Due to the skill, bravery and perseverance of a very select few pioneers open heart surgery moved from experimentation in the animal lab to the hospital operating theatre (although some may argue that a period of experimentation in the hospital operating theatre was another phase in this process). Arguably the greatest of these pioneers was the charismatic and polarising C. Walton Lillehei. It is his tremendous story G. Wayne Miller recounts in this book.

Miller, in a somewhat dramatized fashion, relays in tremendous detail key aspects of Lillehei's professional and personal life, although it is Lillehei's obsession with the theory of blood diversion away from the heart, in order to repair congenital heart defects, that is the common thread underpinning much of this book.

Whilst researching and writing this book Miller apparently spent a great deal of time with Lillehei, his patients and their families, as well as colleagues. This is certainly apparent to the reader who can one moment bear witness to the rigors of laboratory research with all of its successes and failures, and the next moment be sharing in a parents heart ache at the tribulations and frustrations of post-operative cardiac care in the 1950's.

As a perfusionist reading this book one is absolutely absorbed at how Lillehei dismissed John Gibbons screen oxygenator as overly complex and instead persevered with cross circulation until he could come up with a viable alternative. Indeed, the tremendously detailed description of the cross circulation procedure from a technical, professional and personal perspective is fascinating.

The most thrilling part of the story is when a chance meeting with Richard DeWall eventually resulted in the construction and, eventually, implementation into clinical practice of a cheap and ready to use heart lung machine incorporating a bubble oxygenator. This achievement above all others must surely be the greatest innovation in making heart surgery available beyond the walls of all but the most venerable institutions.

Perhaps not covered in perfusion texts, but given weight within this book is Lillehei's involvement in the development of the cardiac pacemaker and heart valve technology. These are two other areas that have benefited millions of patients worldwide. I found it astonishing how one person had such involvement and influence in so many aspects of cardiac surgery and research whilst still teaching and carrying out a very busy clinical workload (and a very busy social life- another 'part of the man' recounted within the pages of this book).

As with any great teacher, it is often the legacy they leave that is a true measure of the individual, Lillehei is no exception. Over the years, Lillehei influenced hundreds of trainees who returned to their institutions and implemented many of Lillehei's techniques and innovations, thus improving the quality of life of millions of children and their families.

Not all of Lillehei's life is of an upward trajectory and Miller does not shy away from covering the fall from grace of this gifted surgeon and innovator. The same attention to detail is given to covering the tax fraud allegations that dogged the later years of Lillehei as was given to his prior successes.

A story of this magnitude deserves a happy ending, and Miller does not disappoint. A box of tissues may be required for an emotional finale that would not be out of place in a Hollywood movie.

This book is written for a wide audience, and not just medical professionals. I am certain that this will not detract from a truly amazing story recounting the life of an even more amazing individual, C Walton Lillehei.

This book is available in paperback, or if you are like me and do not often get the time to pick up an actual book, the audio book is available on iTunes.



# ARTICLE IS SERUM LACTATE A PREDICTOR OF OUTCOME FOR PAEDIATRIC VA ECMO?

# by Clarke Thuys

# Introduction

Serum lactate level and/or clearance have been studied as a predictor of outcome for ECMO support for adults [1] [2] [3], neonates [4] [34] and paediatric patients [5] [6] [34]. There are also numerous studies examining lactate level or rate of lactate clearance as a predictor of outcome for patients being treated for injury, cardiac arrest, sepsis and septic shock [7] [8] [9] [10] [11] [12] [13] [14] [15] [16] [17] [18] [19] [20] [21] [22] [23] [24] [25] [26]. The study by Hannan in a series of 8 patients [27] showed persistent elevated or rising lactate levels after initiation of ECMO was indicative of fatal outcome.

The aim of this retrospective study was to determine whether the lactate level at 12 hours, 24 hours or 48 hours after the initiation of VA ECMO was a predictor of outcome for any paediatric patient, and more specifically a patient placed on ECMO for support during cardiac arrest. Lactate values were stratified as: Normal lactate <2 mmol/L, intermediate lactate >2<4 mmol/L and high lactate ≥4 mmol/L, [24] [8].

# Methods

All patient were supported using Maquet Rotaflow systems and a range of oxygenators consisting of Maquet Quadrox D, Medos Hilite 2400, Medos Hilite 7000 and Dideco Lilliput 2. The systems were either blood primed or clear primed according to standard RCH ECLS protocols. Lactate levels were measured using a Siemans Rapidlab 1265 Blood Gas Analyser. Time frames for lactate data collection were not rigidly adhered to and the peak level was determined to be the highest level measured 2 hours either side of the initiation of support; allowing for increased lactate due to washout on support, or the emergent nature of initiation of support not allowing sampling. The 12 hour sample was taken in the 11-13 hour time window while the 24 hour and 48 hour samples were taken at 22-26 hours and 46-50 hours respectively. If more than one sample was taken in this period the one taken closest to the nominated sample time was used. Patients were excluded only if the peak lactate was not measured within the 4 hour time frame, there were no subsequent lactate measurements due to the withdrawal of support, or underwent cardiopulmonary bypass within the first 12 hours of support. Patients converted from centrifugal VAD to ECMO were not excluded. Analysis used the categories of values rather than the absolute values because of the highly skewed nature of some of the data. Log transformation did not adequately resolve the skewness or kurtosis of the raw data. Fisher's Exact Test was used to compare categorical variables.

# Results

At the Royal Children's Hospital regular measurement of lactate for ECLS patients was started in 2005. Analysis of data from 263 patients supported with veno-arterial ECMO between 2005 and 2012 showed that 70.0% had a high level peak lactate, 18.7% had a peak lactate at the intermediate level while only 12.7% had a peak lactate that was considered normal. After 12 hours of support these percentages had changed to 21.6%, 34.3% and 44.1% respectively.

217/267 (81%) patients were successfully weaned from support and 160/257 (60%) were discharged from hospital, 50/267 (19%) patients had support withdrawn for a variety of reasons.

	Survivors (median,) n = 159	Non Survivors (median,) n = 104	р
Age (days)	54	43	.931
BSA (m <sup>2</sup> )	0.229	0.22	.618

Looking at the actual lactate level rather than a categorised level was possible for peak lactate because it was normally distributed. The results showed patients who survived to discharge had significantly lower peak lactate.

	Mean (SD)	Median (min, quartiles, max)	n
Survivor	6.92* (5.15)	6.1 (0.7, 2.90, 9.32, 29.8)	161
Non survivor	9.41* (5.96)	8.8 (0.8, 4.15, 12.85, 25.9)	106

\*p = 0.0006

Lactate level	Peak	12 Hours	24 Hours	48 hours
<2	11.3%	44.1%	56.3%	66.2%
≥2<4	18.7%	34.3%	33.7%	24.4%
>1	74 40/	04.00/	10.00/	0.40/

Percentage of patients in each lactate group over the first 48 hours of ECMO.

Actual numbers in each group varied as some patients were not supported for 48 hours, or were missing data for any of the times frames following the peak value.

Lactate level	Peak	12 Hours	24 Hours	48 hours
<2	30	117	144	141
≥2<4	50	74	82	52
≥4	187	53	24	20

Numbers per group per timeframe

Peak	Discharged	Not discharged	% Discharged
<2	19	11	63.3*#
≥2<4	36	14	72.0*^
≥4	106	81	56.7#^

No significant difference between the groups. \*p = 0.4615, #p = 0.5544, ^p = 0.0529

12 hrs	Discharged	Not discharged	% Discharged
<2	76	31	71.0*#
≥2<4	46	37	55.4* ^
≥4	23	30	43.4# ^

Significant differences in survival to discharge across the categories looking only at this time point. \*p = 0.0446, #p = 0.0012,  $^p = 0.2815$ 

24 hrs	Discharged	Not discharged	% Discharged
≤2	93	41	69.4*#
>2<4	44	38	53.6* ^
≥4	9	15	37.5# ^

Significant differences in survival to discharge across the categories looking only at this time point. \*p = 0.0069, #p = 0.0049, ^p = 0.2456

48 hrs	Discharged	Not discharged	% Discharged
≤2	89	52	63.1*#
>2<4	46	37	54.7*^
~1	44	0	

No significant difference between the groups. \*p = 0.2621, #p = 0.6231, ^p = 1.00

12hrs change from peak Lactate	Discharged	Not discharged	% discharged
<2 No Change	20	7	74% *
≥2<4 No Change	8	4	67% #
≥4 No Change	22	29	43% * #
≥2<4 - <2	19	10	67% ∞
≥4 - <2	43	18	73% *
≥4 - ≥2<4	31	28	52% * ∞
<2 - ≥2<4	2	1	67%
<2 - ≥4	1	1	50%
Normalised	82	35	70.1%
Not normalised	64	63	50.4%

\* p = 0.0162, # p = 0.2017,  $\mathfrak{s}$  p = 0.2641,  $\infty$  p = 0.2641

Lactate clearance at 12 hours shows no difference except when comparing <2 'no change' to  $\geq$ 4 'no change'. Looking further there is significant difference in survival if you compare patients who had normalised lactate at 12 hours compared to all the others; p= 0.0018.

24hrs change from peak Lactate	Discharged	Not discharged	% discharged
<2 peak: no change	18	7	70% *
≥2<4: no change	3	0	83% #
≥4: no change	9	14	36% * #
≥2<4 - <2	26	14	67% ∞
≥4 - <2	55	24	70% *
≥4 - ≥2<4	31	32	51% * ∞
<2 - ≥2<4	2	3	25%
<2 - >4	0	1	0%
Normalised	99	45	68.7%
Not normalised	45	49	47.9%

\*p = 0.0377, # p = 0.0691,  $\infty$  p = 0.1141, **s** p = 0.0259

The same clearance results are seen at 24 hours. Survival was better in patients with normalised lactates compared to those who had not normalised in this timeframe p = 0.0018.

48hrs change from peak Lactate	Discharged	Not discharged	% discharged
≤2: no change	15	10	60% *
>2<4: no change	5	1	83% *# ¤
≥4: no change	10	9	53% #
≥2<4 - <2	21	10	68% ∞
≥4 - <2	55	34	62% *
≥4 - ≥2<4	21	20	51% ∞ *¤
<2 - ≥2<4	1	0	100%
<2 - ≥4	1	0	100%
Normalised	91	54	62.7%
Not normalised	36	30	54.5%

\*p = 0.3826, # p = 0.3449,  $\infty$  p = 0.2277, **2** p = 0.3383,  $\square$  p = 0.244

But at 48 hours once again, as with the static measurements there are no statistically significant differences in survival to discharge based on lactate clearance. Looking at the larger cohorts that finished up with a normalised lactate level, all had survival rates equal to or greater than 60% while those who failed to normalise had an average survival of 54% or less, but even this did not reach significance with p = 0.2897.

### **Discussion:**

The first task in this analysis is to determine whether we are seeing lactic acidosis or hyperlactaemia. Hyperlactaemia is defined as increased levels of lactate in the blood, without the evidence of lactic acidosis or shock. demonstrating lactic acidosis. Lactic acidosis, on the other hand, is associated with major metabolic dysregulation, tissue hypoperfusion, the effects of certain drugs or toxins, and congenital abnormalities in carbohydrate metabolism.

There are many etiologies for elevated lactate. The most common reasons for the use of VA ECMO in these patients were sepsis and septic shock, cardiogenic shock, and cardiac arrest or imminent cardiac arrest.

Septic shock is often associated with macrocirculatory dysfunction causing arterial hypotension, as well as microcirculatory dysfunction, and decreased oxygen and nutrient extraction by peripheral tissues.

The utility of lactate in cardiogenic shock has not been evaluated extensively but studies in patients with myocardial dysfunction resulting in shock after cardiac surgery found profoundly elevated lactate levels in this setting. Investigators found that the elevation was primarily related to increased tissue lactate production and not decreased clearance. [28]

The study by Attana showed patients in cardiogenic shock following ST-elevation myocardial infarction with ineffective lactate clearance (<10%) had a lower survival rate. [21]

A confounding issue with increased lactate following cardiopulmonary bypass can be the use can be the use of adrenaline as an inotrope. In some patients this practise can lead to the development of lactic acidosis. This action is most likely a beta-mediated effect, and is associated with increased whole-body and lower limb blood flow and decreased wholebody and transfemoral oxygen extraction. The phenomenon does not appear to be related to decreased tissue perfusion and does not have the poor outlook of lactic acidosis associated with shock. [29] Once the inotrope is stopped the lactic acidosis clears. As our ECMO protocol mandates the cessation of inotropes once adequate support has been established this may account for some of the reduction in lactate seen in patients receiving adrenaline either as continued inotropic support, or by bolus for cardiac arrest or imminent arrest. Dead bowel or gut, necrotizing fasciitis, and a multitude of toxicological causes can be dismissed as the causes of increased lactate in this patient population.

Nolan in 2008 showed that etiologies of persistently elevated lactate in the post-arrest period may include systemic inflammatory response and ongoing tissue hypoxia, myocardial stunning causing cardiogenic shock, an uncorrected underlying etiology of the original arrest, microcirculatory dysfunction, and mitochondrial injury and dysfunction. [30] There are conflicting reports about the efficacy of lactate as a predictor of survival in children supported by ECMO. Kumar's study [31] found lactate level at 24 hrs greater than 4 was associated with hospital mortality, but pre-support levels were not predictive of outcome. Cheung [4] showed plasma lactate levels at admission, at peak, and 12 hr after starting ECMO were significantly elevated in the early deaths (vs. the 2 groups of survivors). Shah [32] showed that high lactate at the time of initiation of ECMO support correlated strongly with non-survival as did Demondion [33], although his paper looked at adult patients. Buijs [34] showed in pediatric patients that the magnitude and trend over time of arterial lactate levels, but not the duration of lactate derangement predict mortality. He also showed the value of arterial lactate for predicting outcome in neonatal ECMO patients is limited. The Kolovos [35] study showed highest lactate as a predictor in the first 48 hours but lowest lactate was not predictive.

There is also some conjecture as to whether single time point lactate or lactate clearance (serial lactates) is a better predictor of outcome. Husain [13] reported effective lactate clearance and single time point level are associated with decreased mortality in surgical intensive care patients not supported with ECMO. Buijs [34] demonstrated that either can predictive of outcome while Li [36] with a series of adult patients showed the value of lactate clearance in the first 12 hours of support as a predictor of mortality. Park [37] showed that serial points over 24 hours were a strong predictor of outcome in adult patients postcardiotomy.

### Conclusion

Survivors to discharge had significantly lower peak lactate levels. Single point lactate, serial lactates and lactate clearance at 12 hours and 24 hours after initiation of ECMO appear to be predictive of outcome. Normalisation of lactate is the common factor across these indices with the rate of survival to discharge significantly better for those who normalise their lactate within 24 hours. At 48 hours the game changes and lactate is no longer predictive of outcome. The patient cohort might be too wide to pick up differences across reasons for support, such as ECPR patients compared to postcardiotomy patients. Categorisation of age might also be worth investigating although this would reduce the numbers in each group. The aim of this study was to determine whether lactate could be a simple indicator of survival in what are generally very complex situations. The result is inconclusive.



Royal Children's Hospital ECMO Circuit with Rotaflow pump and Medos oxygenator.

### REFERENCES

- W.-J. Ko, C.-Y. Lin, R. Chen, S.-S. Wang, F.-Y. Lin and Y.-S. Chen, "Extracorporeal Membrane Oxygenation Support for Adult Postcardiotomy Cardiogenic Shock," Ann Thorac Surg, pp. 538-45, 2002.
- [2] F. Bakhtiary, H. Keller, S. Dogan, O. Dzemali, F. Oezaslan, D. Meininger, H. Ackermann, B. Zwissler, P. Kleine and A. Moritz, "Venoarterial extracorporeal membrane oxygenation for treatment of cardiogenic shock: Clinical experiances in 45 adult patients.," JTCVS, pp. 382-, 2008.
- [3] A. J. Rastan, A. Dege, M. Mohr, N. Doll, V. Falk, T. Walther and F. W. Mohr, "Early and late outcomes of 517 consecutive adult patients treated with erxtracorporeal membrane oxygenation for refractory postcardiogenic shock.," Journal of Thoracic and Cardiovascular Surgery, vol. 139, pp. 302-311, 2010.
- [4] Cheung, P.-Y. Etches, P. Weardon, M. Reynolds, A. Finer, N. Robertosn and Charlene, "Use of plasma lactate to predict early mortality and adverse outcome after neonatal extracorporeal membrane oxygenation: A prospective cohort in early childhood.," Crit Care Med, vol. 30, no. 9, pp. 2135-2139, 2002.
- [5] B. Alsoufi, A. Awan, C. Manhliot, A. Guechef, Z. Al-Halees, M. Al-Ahmadi, B. W. McCrindle and A. Kalloghlian, "Results of rapid-response extracorporeal cardiopulmonary resuscitation in children with refractory cardiac arrest following cardiac surgery," European Journal of Cardio-Thoracic Surgery, vol. 45, pp. 268-275, 2014.
- [6] S.-C. Huang, E.-T. Wu, Y.-S. Chen, C.-I. Chang, I.-S. Chiu, S.-S. Wang, F.-Y. Lin and W.-J. Ko, "Extracorporeal membrane oxygenation rescue for cardiopulmonary resuscitation in pediatric patients," Pediatric Critical Care, vol. 36, no. 5, pp. 1607-1613, 2008.
- [7] H. B. Nguyen, E. Rivers, B. Knoblich, G. Jacobsen, A. Muzzin, J. Ressler and M. Tomlanovich, "Early lactate clearance is associated with improved outcome in severe sepsis and septic shock.," Crit Care Med, pp. 1637-1642, 2004.
- [8] S. Trzeciak, P. Dellinger, M. Chansky, R. Arnold, C. Schorr, B. Milcarek, S. Hollenberg and J. Parrillo, "Serum lactate as a

predictor of mortality in patients with infection.," Intensive Care Medicine, pp. 970-977, 2007.

- [9] B. M. Fuller and R. P. Dellinger, "Lactate as a hemodynamic marker in the critically ill.," Current Opinion in Critical Care, vol. 18, pp. 267-272, 2012.
- [10] D. Abramson, T. Scalea, R. Hitchcock, S. Trooskin, S. Henry and J. Greenspan, "Lactate clearance and survival following injury.," J Trauma, pp. 588-589, 1993.
- [11] R. Arnold, N. Shapiro, A. Jones, C. Schorr, J. Pope, E. Casner and e. al, "Emergency Medicine Shock Research Network (EMShockNet) Investigators: Multicenter study of early lactate clearance s a determinant of survival in patients with presumed sepsis," Shock, pp. 35-39, 2009.
- [12] P. Cardinal Fernandez, E. Olano, C. Acosta and e. al, "Prognostic value of lactate clearance in the first 6 hours of intensive medicine course.," Med Intensiva, pp. 166-170, 2009.
- [13] F. Husain, M. Martin, P. Mullinex and e. al, "Serum lactate and base deficit as predictors of mortality and morbidity.," Am J Surg, pp. 485-491, 2003.
- [14] T. Jansen, J. van Bommel, F. Schoonderbeek and e. al, "LACTATE study group: Early lactate-guided therapy in intensive care unit patients: A multicenter, open-label, randomized controlled trial.," Am J Respir Crit Care Med, pp. 752-761, 2010.
- [15] A. Jones, N. Shapiro, S. Trzeciak and e. al, "Emergency Medicine Shock Research Network (EMShockNET) Investigators: Lactate clearance vs central venous oxygen saturation as goals of early sepsis therapy: A randomized clinical trial.," JAMA, pp. 739-746, 2010.
- [16] P. Marty, A. Roquilly and F. e. a. Vallee, "Lactate clearance for death prediction in severe sepsis or septic shock patients during the first 24 hours in Intensive Care Unit: An observational study.," Ann Intensive Care, 2013.
- [17] J. McNelis, C. Marini, J. A and e. al, "Prolonged lactate clearance is associated with increased mortalityi in the surgical intensive care unit.," Am J Surg, pp. 481-485, 2001.
- [18] J. Vincent, P. Dufaye, J. Berre, M. Leeman, J. Degaute and R. Kahn, "Serial lactate determinations during circulatory shock.," Critical Care Medicine, pp. 449-451, 1983.
- [19] L. Kliegal, H. Losert, M. Holzer, A. Zeiner, C. Havel and A. Laggner, "Serial lactate derminations for prediction of outcome after cardiac arrest.," Medicine (Baltimore), pp. 274-279, 2004.
- [20] M. Puskarich, S. Trzeciak, N. Shapiro, A. Albers, A. Heffner, K. J and A. Jones, "Whole blood lactate kinetics in patients undergoing quantitative resuscitation for severe sepsis and septic shock.," Chest, pp. 1548-1553, 2013.
- [21] P. Attana, C. Lazzeri, M. Chiostri, C. Picariello, G. Gensini and V. S., "Lactate clearance in cardiogenic shock following ST elevation myocardial infarction: A Pilot Study," Acute Cardiac Care, pp. 20-26, 2012.
- [22] M. Suistomaa, E. Ruokenon, A. Kari and J. Takala, "Time-pattern of lactate and lactate to pyruvate ratio in the first 24 hours of intensive care emergency admissions.," Shock, pp. 8-12, 2000.
- [23] O. Kruse, N. Grunnet and C. Barfod, "Blood lactate as a predictor for in-hospital mortality in patients admitted acutely to hospital: a systematic review.," Scandinavian Journal of Trauma, Resuscitation and emergency Medicine, 2011.
- [24] M. Mikkelsen, A. Miltiades, D. Gaieski, M. Goyal, B. Fuchs, V. Shah, S. Bellamy and J. Christie, "Serum lactate is associated with

mortality in severe sepsis independent of organ failure and shock.," Crit Care Med, pp. 1670-1677, 2009.

- [25] A. Nichol, M. Bailey, M. Egi, V. Pettila, C. French, E. Stachowski, M. Reade, D. Cooper and R. Bellomo, "Dynamic lactate indices as predictors of outcome in critically ill patients.," Critical Care, 2011.
- [26] A. Rishu, R. Khan, H. Al-Dorzi, H. Tamim, H. Al-Qahtani, G. Al-Ghamdi and A. Y., "Even mild hyperlactatemia is associated with increased mortality in critically ill patients," Critical Care, 2013.
- [27] R. Y. Hannan, J. White, J. Ojito, A. Rossi and R. Burke, "Patterns of Lactate Values after Congenital Heart Surgery and Timing of Cardiopulmonary Support.," Annals of Thoracic Surgery, vol. 80, pp. 1468-74, 2005.
- [28] R. Chiolero, J. Revelly and X. Leverve, "Effects of Cardiogenic Shock on Lactate and Glucose Metabolism After Heart Surgery," Critical Care Medicine, pp. 3784-3791, 2000.
- [29] R. Totaro and R. Raper, "Epinephrine-induced lactic acidosis following cardiopulmonary bypass.," Critical Care Medicine, pp. 1693-1699, 1997.
- [30] J. Nolan, R. Neumar and C. Adrie, "Post-cardiac arrest epidemiology, pathophysiology, treatment and prognostication.," Resustation, pp. 350-379, 2008.
- [31] S. Kumar, D. Zurakowski, H. Dalton, S. Talwar, A. Allard-Picou, L. Duebner, P. Sinha and A. Moulick, "Extracorporeal membrane oxygenation in post-cardiotomy patients: factors influencing outcome.," Journal of Thoracic and Cardiovascular Surgery, pp. 330-336e2, 2010.
- [32] S. Shah, V. Shankar, K. Churchwell, M. Taylor, B. Scott, R. Bartilson, D. Byrne, K. Christian and D. Drinkwater, "Clinical Outcomes of 84 Children with Congential Heart Disease Managed with Extracorporeal Membrane Oxygenation after Cardiac Surgery," ASAIO Journal, pp. 504-507, 2005.
- [33] P. Demondion, L. Fournel, J.-L. Golmard, M. Niculescu, A. Pavie and P. Leprince, "Predictors of 30-day mortality and outcome in cases of myocardial infarction with cardiogenic shock treated by extracorporeal life support," European Journal of Cardiothoracic Surgery, pp. 47-54, 2014.
- [34] E. Buijs, R. Houmes, D. Rizopoulos, E. Wildschut, I. Reiss, C. Ince and D. Tibboel, "Arterial lactate for predicting mortality in children requiring extracorporeal membrane oxygenation.," Minerva Anestesiologica, vol. 80, no. 12, pp. 1282-1293, 2014.
- [35] N. Kolovos, S. Bratton, F. Moler, E. Bove, R. Ohye, R. Bartlett and T. Kulik, "Outcome of Pediatric Patients Treated with Extracorporeal Life Support After Cardiac Surgery," Annals of Thoracic Surgery, pp. 1435-1442, 2003.
- [36] C.-L. Li, H. Wang, M. Jia, N. Ma, X. Meng and X.-T. Hou, "The early dynamic behavoiur of lactate is linked to mortality in postcardiotomy patients with extracorporeal membrane oxygenation support: A retrospective observational study.," Journal of Thoracic and Cardiovascular Surgery, pp. 1445-1450, 2015.
- [37] S. Park, S.-P. Kim, J. Kim, S.-H. Jung, S. Choo, C. Chung and J. Lee, "Blood lactate level during extracorporeal life support as a surrogate marker for survival.," Journal of Thoracic and Cardiovascular Surgery, pp. 714-720, 2014.
- [38] R. Thiagarajan, P. Laussen, P. Rycus, R. H. Bartlett and S. L. Bratton, "Extracorporeal Membrane Oxygenation to Aid Cardiopulmonary Resuscitation in Infants and Children," Circulation, vol. 116, pp. 1693-1700, 2007.



# XTRA<sup>®</sup> Innovative, intuitive and powerful ATS system

# The complete solution for blood management and neurological protection.

LivaNova XTRA<sup>®</sup> autotransfusion device is part of the integrated **HeartLink<sup>™</sup> System**. It delivers washed, fresh and vital autologous red blood cells, significantly decreasing the inflammatory response.





# THE ASSOCIATION BETWEEN INTRAOPERATIVE GLYCAEMIC CHANGE AND MORTALITY IS MODULATED BY PRE-EXISTING HYPERGLYCAEMIA IN CARDIAC SURGERY.

Richard Newland BSc CCP, Rob Baker PhD CCP. Flinders Medical Centre and Flinders University, on behalf of the Australian and New Zealand Collaborative Perfusion Registry

# Introduction

Hyperglycaemia occurs frequently in patients with and without diabetes who are undergoing cardiac surgery, particularly with the use of cardiopulmonary bypass and has been associated with increased mortality and morbidity (1, 2).

Recent evidence has shown that targeting tight glucose control (80-110 mg/dl) in ICU patients with pre-existing glycaemia is not associated with similar benefits as compared to patients without glycaemia (3). These findings suggest that patients suffering from chronic glycaemia have developed a certain tolerance to hyperglycaemia. The mechanism for this adaptation may be attributed to the process of expression of glucose transporters in different cell types (4, 5). Aiming for tight perioperative glucose control in patients that have adapted to chronic hyperglycaemia may result in an acute derangement of glycaemic homeostasis, with insufficient cellular glucose uptake as a consequence, which could be harmful (6).

Glycolated haemoglobin (HbA1c) is used as an indicator of the average level of glycaemia over the previous 2-3 months and has been shown to be frequently elevated in patients, with or without known diabetes, undergoing cardiac surgery (7) and has been shown to tightly correlate with average blood glucose levels (8) and therefore may be useful in determining the optimal target for perioperative blood glucose control.

Although the association between glycaemia and mortality has been reported to be modulated in critically ill patients with pre-existing hyperglycaemia (9, 10), what is not known is whether there is a difference in ability to tolerate a reduction in intraoperative blood glucose in patients with pre-existing hyperglycaemia. We evaluated whether the association between intraoperative glycaemic change and mortality is modulated by pre-existing hyperglycaemia.

### Methods

Institutional ethics review board approval was obtained at all centres for participation in the Australian and New Zealand Collaborative Perfusion Registry in conjunction with approval from the National Health and Medical Research Council (386.15). Data from 3,026 adult patients adult patients undergoing isolated coronary artery bypass grafting (CABG), valve repair or replacement (or both), and valve/ CABG procedures with CPB was collected from March 2007 – February 2016 in 3 Australian centres routinely collecting HbA1c measurements; The Royal Hobart Hospital, Hobart, Flinders Medical Centre, Adelaide, and Westmead Hospital, Sydney. Data were collected prospectively using the Australian and New Zealand Collaborative Perfusion Registry as previously described (11, 12), which integrates intraoperative data from the Data Management System (LivaNova, London, England), CONNECT (LivaNova, London, England), and JOCAP (Maquet, Wayne, NJ) heart lung machine data collection software, including patient and blood gas monitoring systems. All perfusion-related factors and temperature measurements were collected electronically in a continuous manner at 20- to 60-second intervals. Clinical data definitions were based on those reported by the Australian and New Zealand Society of Cardiothoracic Surgeons database (13). Pre-existing hyperglycaemia was defined as a preoperative HbAlc >7%.

A glycaemic ratio (GR) for each patient was calculated by dividing the minimum CPB glucose by the chronic average blood glucose level (AG). To convert HbA1c levels to AG, we used the following equation:  $AG(mmol) = 1.59 \times HbA1c - 2.59$  (8).

Stata SE, version 14.0 (StataCorp LP, College Station, TX) was used for the statistical analyses. Cut off points in the relationship between GR and mortality were assessed visually using LOWESS plots and the influence of having GR above or below the cut off points on mortality was determined using separate multivariate models for patients with or without pre-existing hyperglycaemia. Preoperative and intraoperative characteristics and the number of perioperative red blood cell transfusions were evaluated for univariate association with mortality, in addition to the year of operation to evaluate temporal variation. Variables found to be significant predictors were included in the multivariate models, clustered by centre. Patient preoperative and intraoperative characteristics were compared in patients above or below the cut off points for the GR. Group differences were considered statistically significant with a p value of <0.05. p values were calculated for continuous variables using the Wilcoxon-Mann-Whitney U test and the chi-squared test for categorical variables.

### Results

Pre-existing hyperglycaemia was identified in 21% of patients. LOWESS plots identified GR>1 for normoglycaemic patients (figure 1) and GR <0.5 for patients with pre-existing hyperglycaemia (figure 2) as having an increase in the likelihood for mortality.



Figure 1. LOWESS smoothing plot showing the relationship between the glycaemic ratio and probability of mortality in normoglycaemic patients. An increase in the probability for mortality is seen for GR>1.



Figure 2. LOWESS smoothing plot showing the relationship between the glycaemic ratio and probability of mortality in patients with preexisting hyperglycaemia. An increase in the probability for mortality is seen for GR<0.5.

Patient preoperative, intraoperative characteristics and outcomes for normoglycaemic patients are compared according to having a GR above or below the cut off in Table 1, and for patients with pre-existing hyperglycaemia in Table 2.

Table 1. Normoglycaemic patients	Glycaemic ratio <1	Glycaemic ratio >1	р
<u>N</u> N	1716	668	
Preoperative characteristics			
Female	473 (27.6%)	180 (27.0%)	0.80
Diabetes	341 (19.9%) 62 (2.7%)	181 (27.1%)	< 0.001
Chronic pulmonary disease	376 (21.9%)	113 (16.9%)	0.20
Congestive heart failure	695 (40.5%)	213 (31.9%)	<0.001
Pulmonary hypertension	1072 (62.7%)	284 (42.9%)	<0.001
Hypertension	1235 (72.0%)	469 (70.2%)	0.39
Smoking history	1112 (64.8%)	428 (64.2%)	0.77
Redo	113 (6.6%)	52 (7.8%)	0.32
Emergency	15 (0.9%)	31 (4.6%)	< 0.001
Myocardial infarction	584 (34 1%)	203 (30.4%)	0.40
Age, median (IQR)	66 (56, 75)	66 (57, 74)	0.62
Preoperative creatinine, median (IQR)	87 (75, 104)	84.5 (73, 103)	0.023
Euroscore, median (IQR)	3.32 (1.61, 6.24)	3.41 (1.7, 6.705)	0.33
Intraoperative characteristics			
CPB duration, median (IQR)	81 (64, 107)	92 (70.5, 121)	<0.001
Valve	468 (27.3%)	161 (24.1%)	0.12
Valve/CABG	182 (10.6%)	89 (13.3%)	0.062
CABG	846 (49.3%)	307 (46.0%)	0.14
Lowest psopharyngeal temp, median (IOR)	220 (12.0%)	337(321342)	<0.010
Average CPB MAP median (IQR)	61 (56, 66)	61 (55 66)	0.23
Lowest CPB DO2i, median (IQR)	258 (223.5, 290)	260 (223, 297)	0.35
Lowest CPB Hb, median (IQR)	88 (76, 100)	87 (73, 98)	0.002
Any blood product transfusion	834 (48.7%)	314 (47.0%)	0.47
RBC units transfused, median (IQR)	0 (0, 2)	0 (0, 2)	0.72
Postoperative outcome			
Death	36 (2.1%)	26 (3.9%)	0.021
Stroke	28 (1.6%)	11 (1.7%)	1.00
Ventilation >48hrs	101 (6.0%)	60 (9.1%) 70 (10.5%)	0.011
Reoperation	91 (5.3%)	44 (6 6%)	0.021
Combined morbidity	258 (15.2%)	133 (20.1%)	0.005
Table 2. Patients with are existing hyperglycaemia		Observation of the	
Table 2. Patients with pre-existing hyperglycaemia	Glycaemic ratio >.5	Glycaemic ratio <.5	p
Table 2. Patients with pre-existing hyperglycaemia	Glycaemic ratio >.5 527	Glycaemic ratio <.5 115	р
Table 2. Patients with pre-existing hyperglycaemia N Preoperative characteristics Female	Glycaemic ratio >.5 527 160 (30.5%)	Glycaemic ratio <.5 115 39 (33.9%)	
Table 2. Patients with pre-existing hyperglycaemia N Preoperative characteristics Female Diabetes	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%)	Glycaemic ratio <.5 115 39 (33.9%) 102 (88.7%)	<i>p</i> 0.50 0.071
Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%)	Glycaemic ratio <.5 115 39 (33.9%) 102 (88.7%) 7 (6.1%)	<i>p</i> 0.50 0.071 1.00
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%)	Glycaemic ratio <.5 115 39 (33.9%) 102 (88.7%) 7 (6.1%) 19 (16.5%)	<i>p</i> 0.50 0.071 1.00 0.44
Table 2. Patients with pre-existing hyperglycaemia           N           Preoperative characteristics           Female           Diabetes           Severe Left ventricular dysfunction           Chronic pulmonary disease           Congestive heart failure	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%)	Glycaemic ratio <.5 115 39 (33.9%) 102 (88.7%) 7 (6.1%) 19 (16.5%) 40 (34.8%)	<i>p</i> 0.50 0.071 1.00 0.44 0.32
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%)	Głycaemic ratio <.5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           24 (64.7%)	p 0.50 0.071 1.00 0.44 0.32 0.61
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension	Glycaemic ratio >.5           527           160 (30.5%)           494 (93.7%)           34 (6.5%)           107 (20.3%)           157 (29.8%)           236 (45.6%)           458 (86.9%)           244 (5.5%)	Głycaemic ratio <.5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (24.8%)           56 (48.7%)           94 (81.7%)           70 0.0 (24.17%)	р 0.50 0.071 1.00 0.44 0.32 0.61 0.18
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Pende	Glycaemic ratio >.5           527           160 (30.5%)           494 (93.7%)           34 (6.5%)           107 (20.3%)           157 (29.8%)           236 (45.6%)           458 (86.9%)           344 (65.3%)           26 (45.6%)	Glycaemic ratio <.5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1720)	p 0.50 0.071 1.00 0.44 0.32 0.61 0.18 0.66 0.20
Table 2. Patients with pre-existing hyperglycaemia N Preoperative characteristics Female Diabetes Severe Left ventricular dysfunction Chronic pulmonary disease Congestive heart failure Pulmonary hypertension Hypertension Smoking history Redo Emergency	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 344 (65.3%) 24 (4.6%) 40 (8%)	Glycaemic ratio <.5 115 39 (33.9%) 102 (88.7%) 7 (6.1%) 19 (16.5%) 40 (34.8%) 56 (48.7%) 94 (61.7%) 78 (67.8%) 2 (1.7%) 1 (0 %)	<i>p</i> 0.50 0.071 1.00 0.44 0.32 0.61 0.18 0.66 0.20 1.00
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 54 (10.2%)	Glycaemic ratio <.5 115 39 (33.9%) 102 (88.7%) 7 (6.1%) 19 (16.5%) 40 (34.8%) 56 (48.7%) 94 (81.7%) 78 (67.8%) 2 (1.7%) 1 (0.9%) 10 (8.7%)	<i>p</i> 0.50 0.071 1.00 0.44 0.32 0.61 0.18 0.66 0.20 1.00 0.73
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease         Myocardial infarction	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (46%) 4 (0.8%) 54 (10.2%) 246 (46.7%)	Glycaemic ratio <.5 115 39 (33.9%) 102 (88.7%) 7 (6.1%) 19 (16.5%) 40 (34.8%) 56 (48.7%) 94 (81.7%) 94 (81.7%) 78 (67.8%) 2 (1.7%) 1 (0.9%) 10 (8.7%) 66 (57.4%)	P 0.50 0.071 1.00 0.44 0.32 0.61 0.18 0.66 0.20 1.00 0.73 0.040
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease         Myocardial infarction         Age, median (IQR)	Glycaemic ratio >.5           527           160 (30.5%)           494 (93.7%)           34 (6.5%)           107 (20.3%)           157 (29.8%)           236 (45.6%)           458 (86.9%)           344 (65.3%)           24 (4.6%)           4 (0.8%)           54 (10.2%)           246 (46.7%)           64 (57, 71)	Glycaemic ratio <.5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           1 (0.9%)           10 (8.7%)           66 (57.4%)           55 (50.68)	p           0.50           0.071           1.00           0.44           0.32           0.61           0.18           0.66           0.20           1.00           0.73           0.040           <0.001
Table 2. Patients with pre-existing hyperglycaemia N Preoperative characteristics Female Diabetes Severe Left ventricular dysfunction Chronic pulmonary disease Congestive heart failure Pulmonary hypertension Hypertension Smoking history Redo Emergency Cerebrovascular disease Myocardial infarction Age, median (IQR)	Glycaemic ratio >.5           527           160 (30.5%)           494 (93.7%)           34 (6.5%)           107 (20.3%)           157 (29.8%)           236 (45.6%)           344 (65.3%)           24 (4.6%)           4 (0.8%)           54 (10.2%)           246 (46.7%)           64 (57.7 1)           85 (71, 106)	Glycaemic ratio <.5 115 39 (33.9%) 102 (88.7%) 7 (6.1%) 19 (16.5%) 40 (34.8%) 56 (48.7%) 94 (81.7%) 78 (67.8%) 2 (1.7%) 10 (8.7%) 66 (57.4%) 59 (50.68) 89 (72, 121)	p           0.50           0.071           1.00           0.44           0.32           0.61           0.66           0.20           1.00           0.73           0.040           <0.001
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease         Myocardial infarction         Age, median (IQR)         Euroscore, median (IQR)	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57,71) 85 (71, 106) 2.73 (1.59, 5.37)	Glycaemic ratio < 5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           10 (8.7%)           66 (57.4%)           59 (50.68)           89 (72.121)           2.59 (1.51, 4.83)	р 0.50 0.071 1.00 0.44 0.32 0.61 0.18 0.66 0.20 1.00 0.73 0.040 <0.001 0.11 0.24
Table 2. Patients with pre-existing hyperglycaemia           N           Preoperative characteristics           Female           Diabetes           Severe Left ventricular dysfunction           Chronic pulmonary disease           Congestive heart failure           Pulmonary hypertension           Hypertension           Smoking history           Redo           Emergency           Cerebrovascular disease           Myocardial infarction           Age, median (IQR)           Preoperative creatinine, median (IQR)           Intraoperative characteristics	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 3344 (65.3%) 24 (46%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57, 71) 85 (71, 106) 2.73 (1.59, 5.37)	Głycaemic ratio <.5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           1 (0.9%)           10 (8.7%)           66 (57.4%)           59 (50.68)           89 (72, 121)           2.59 (1.51, 4.83)	p           0.50           0.071           1.00           0.44           0.32           0.61           0.18           0.66           0.20           1.00           0.73           0.040           <0.001
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease         Mycoardial infarction         Age, median (IQR)         Preoperative creatinine, median (IQR)         Intraoperative characteristics         CPB duration, median (IQR)	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (46%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57,71) 85 (71, 106) 2.73 (1.59, 5.37) 82 (60, 108)	Głycaemic ratio <.5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           94 (81.7%)           95 (6.48,7%)           10 (3.7%)           66 (57.4%)           59 (50.68)           80 (72, 121)           2.59 (1.51, 4.83)           80 (64, 109)	P 0.50 0.071 1.00 0.44 0.32 0.61 0.73 0.66 0.20 1.00 0.73 0.040 <0.001 0.11 0.24
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease         Myocardial infarction         Age, median (IQR)         Preoperative creatinine, median (IQR)         Intraoperative characteristics         CPB duration, median (IQR)         Valve         Volve CABC	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57,71) 85 (71,106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%)	Glycaemic ratio <.5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (24.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           1 (0.9%)           10 (8.7%)           66 (57.4%)           59 (50.68)           80 (72, 121)           2.59 (1.51.4.83)           10 (8.7%)           10 (8.7%)	P 0.50 0.071 1.00 0.44 0.32 0.61 0.18 0.66 0.20 1.00 0.73 0.040 <0.001 0.11 0.24 0.42 0.65 1.00
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease         Mycoardial infarction         Age, median (IQR)         Preoperative characteristics         CPB duration, median (IQR)         Valve         Valve         Cable	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57.71) 85 (71, 106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%)	Glycaemic ratio <.5 115 39 (33.9%) 102 (88.7%) 7 (6.1%) 19 (16.5%) 40 (34.8%) 56 (48.7%) 94 (81.7%) 78 (67.8%) 2 (1.7%) 10 (8.7%) 66 (57.4%) 59 (50.68) 89 (72, 121) 2.59 (1.51, 4.83) 80 (64, 109) 17 (14.8%) 10 (8.7%) 86 (73%)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.73           0.040           <0.001
Table 2. Patients with pre-existing hyperglycaemia           N           Preoperative characteristics           Female           Diabetes           Severe Left ventricular dysfunction           Chronic pulmonary disease           Congestive heart failure           Pulmonary hypertension           Hypertension           Smoking history           Redo           Emergency           Cerebrovascular disease           Myocardial infarction           Age, median (IQR)           Furgoerative characteristics           CPB duration, median (IQR)           Valve           Valve CABG           CABG           Other	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 24 (4.6%) 4 (0.8%) 24 (4.6%) 6 4 (57,71) 85 (71,106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 373 (70.8%) 37 (70.8%)	Glycaemic ratio < 5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           10 (8.7%)           66 (57.4%)           59 (50.68)           89 (72, 121)           2.59 (1.51, 4.83)           10 (8.7%)           80 (64, 109)           17 (14.8%)           10 (8.7%)           85 (73.9%)           3 (2.6%)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.73           0.040           0.73           0.040           0.73           0.040           0.73           0.42           0.65           1.00           0.51
Table 2. Patients with pre-existing hyperglycaemia           N           Preoperative characteristics           Female           Diabetes           Severe Left ventricular dysfunction           Chronic pulmonary disease           Congestive heart failure           Pulmonary hypertension           Hypertension           Smoking history           Redo           Emergency           Cerebrovascular disease           Myocardial infarction           Age, median (IQR)           Intraoperative characteristics           CPB duration, median (IQR)           Valve           Valve           CABG           Chard           Lowest nasopharyngeal temp, median (IQR)	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 226 (45.6%) 458 (86.9%) 334 (65.3%) 24 (4.6%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57, 71) 85 (71, 106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 37 (7.0%) 33.9 (33.2, 34.4)	Głycaemic ratio < 5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           1 (0.9%)           10 (8.7%)           66 (57.4%)           59 (50.68)           89 (72, 121)           2.59 (1.51, 4.83)           10 (64, 109)           17 (14.8%)           10 (8.7%)           85 (73.9%)           3 (2.6%)           33.8 (32.55, 34.2)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.73           0.001           0.73           0.001           0.73           0.040           <0.01
Table 2. Patients with pre-existing hyperglycaemia           N           Preoperative characteristics           Female           Diabetes           Severe Left ventricular dysfunction           Chronic pulmonary disease           Congestive heart failure           Pulmonary hypertension           Hypertension           Smoking history           Redo           Emergency           Cerebrovascular disease           Mycoardial infarction           Age, median (IQR)           Purtacoerative characteristics           CPB duration, median (IQR)           Valve           Valve           Valve CABG           CABG           Other           Lowest nasopharyngeal temp, median (IQR)	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (46%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57,71) 85 (71,106) 2.73 (1.59,5.37) 82 (60,108) 68 (12.9%) 373 (70.8%) 37 (70.9%) 33.9 (33.2, 34.4) 61 (56, 66)	Głycaemic ratio <.5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           1 (0.9%)           10 (8.7%)           66 (57.4%)           59 (50.68)           80 (64, 109)           17 (14.8%)           10 (8.7%)           85 (73.9%)           3 (2.5%, 34.2)           33.8 (32.55, 34.2)           66 (55.5)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.73           0.60           0.001           0.73           0.40           <0.01
Table 2. Patients with pre-existing hyperglycaemia           N           Preoperative characteristics           Female           Diabetes           Severe Left ventricular dysfunction           Chronic pulmonary disease           Congestive heart failure           Pulmonary hypertension           Hypertension           Smoking history           Redo           Emergency           Cerebrovascular disease           Myocardial infarction           Age, median (IQR)           Intraoperative creatinine, median (IQR)           CPB duration, median (IQR)           Valve           Valve      <	Glycaemic ratio >.5 527 527 160 (30.5%) 434 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57.71) 85 (71, 106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 373 (70.8%) 373 (7	Glycaemic ratio < 5 115 39 (33.9%) 102 (88.7%) 7 (6.1%) 19 (16.5%) 40 (34.8%) 56 (48.7%) 94 (81.7%) 78 (67.8%) 2 (1.7%) 10 (8.7%) 66 (57.4%) 59 (50.68) 88 (72, 121) 2.59 (1.51, 4.83) 10 (8.7%) 80 (64, 109) 17 (14.8%) 10 (8.7%) 33 (32.55, 34.2) 61 (56, 65) 243 (217, 275)	P           0.50           0.071           1.00           0.44           0.32           0.66           0.20           1.00           0.73           0.040           <0.001
Table 2. Patients with pre-existing hyperglycaemia           N           Preoperative characteristics           Female           Diabetes           Severe Left ventricular dysfunction           Chronic pulmonary disease           Congestive heart failure           Pulmonary hypertension           Mypertension           Smoking history           Redo           Emergency           Cerebrovascular disease           Myocardial infarction           Age, median (IQR)           Intraoperative characteristics           CPB duration, median (IQR)           Valve           Valve           Valve           Valve           Unsepharyngeal temp, median (IQR)           Lowest CAB Do-ja, median (IQR)           Lowest CPB Hb, median (IQR)	Glycaemic ratio >.5 527 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 24 (4.6%) 4 (0.8%) 24 (46.7%) 64 (57,71) 85 (71,106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 373 (70.8%) 373 (70.8%) 373 (70.8%) 33.9 (33.2, 34.4) 61 (56, 66) 243 (210, 277) 84 (73, 96)	Glycaemic ratio < 5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           13 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           94 (81.7%)           94 (81.7%)           94 (81.7%)           95 (67.8%)           2 (1.7%)           10 (8.7%)           66 (57.4%)           59 (50.68)           80 (54, 109)           17 (14.8%)           10 (8.7%)           80 (54, 109)           17 (14.8%)           10 (8.7%)           33.8 (32.55, 34.2)           61 (56.65)           243 (217, 275)           80 (70, 94)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.73           0.040           <0.01
Table 2. Patients with pre-existing hyperglycaemia           N           Preoperative characteristics           Female           Diabetes           Severe Left ventricular dysfunction           Chronic pulmonary disease           Congestive heart failure           Pulmonary hypertension           Hypertension           Smoking history           Redo           Emergency           Cerebrovascular disease           Myocardial infarction           Age, median (IQR)           Preoperative creatinine, median (IQR)           Euroscore, median (IQR)           Intraoperative characteristics           CPB duration, median (IQR)           Valve           Valve CABG           CAG           Other           Lowest nasopharyngeal temp, median (IQR)           Lowest CPB DoJ, median (IQR)           Lowest CPB DD, median (IQR)	Glycaemic ratio >.5 527 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (66.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 4 (0.8%) 4 (0.8%) 24 (4.6%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57, 71) 85 (71, 106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 373 (70.8%) 373 (70.8%) 373 (70.8%) 373 (70.8%) 373 (70.8%) 373 (70.9%) 33.9 (33.2, 34.4) 61 (56, 66) 243 (210, 277) 84 (73, 96) 261 (49.5%)	Glycaemic ratio < 5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           10 (8.7%)           66 (57.4%)           59 (50.68)           89 (72, 121)           2.59 (15.1, 4.83)           10 (8.7%)           85 (73.9%)           3 (2.6%)           33.8 (32.55, 34.2)           61 (56.65)           24 (217, 275)           24 (217, 275)           80 (70, 94)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.20           1.00           0.73           0.040           <0.01
Table 2. Patients with pre-existing hyperglycaemia           N           Preoperative characteristics           Female           Diabetes           Severe Left ventricular dysfunction           Chronic pulmonary disease           Congestive heart failure           Pulmonary hypertension           Hypertension           Smoking history           Redo           Emergency           Cerebrovascular disease           Mycoardial infarction           Age, median (IQR)           Intraoperative creatinine, median (IQR)           Euroscore, median (IQR)           Valve           Valve           Valve           CABG           CABG           Chard           Lowest nasopharyngeal temp, median (IQR)           Lowest CPB DO <sub>2</sub> , median (IQR)           Lowest CPB DO <sub>2</sub> , median (IQR)           Lowest CPB DO <sub>2</sub> , median (IQR)           Lowest CPB Ho, median (IQR)           Lowest CPB DO <sub>2</sub> , median (IQR)           Lowest CPB Ho, m	Glycaemic ratio >.5 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 334 (65.3%) 24 (4.6%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57, 71) 85 (71, 106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 373 (70.8%) 37 (70.9%) 373 (70.8%) 37 (70.8%) 37 (70.8%) 37 (70.8%) 37 (70.9%) 33.9 (43.2, 34.4) 61 (56, 66) 243 (210, 277) 84 (73, 96) 261 (49.5%) 0 (0, 2)	Glycaemic ratio <.5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           1 (0.9%)           10 (8.7%)           66 (57.4%)           59 (50.68)           89 (72, 121)           2.59 (1.51, 4.83)           10 (8.7%)           80 (64, 109)           17 (14.8%)           10 (8.7%)           85 (73.9%)           38 (32.55, 34.2)           61 (56.65)           243 (217, 275)           80 (70, 94)           74 (64.3%)           1 (0, 2)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.73           0.001           0.73           0.001           0.73           0.040           <0.01
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease         Myocardial infarction         Age, median (IQR)         Intraoperative creatinine, median (IQR)         Luracore, median (IQR)         Valve         Valve         Valve         Valve         Valve CABG         CABG         Other         Lowest CPB Do_i, median (IQR)         Lowest CPB Hab, median (IQR)         Average CPB MAP, median (IQR)         Lowest CPB Hab, median (IQR)         Any blood product transfusion         RBC units transfused, median (IQR)         Postiperative outcome	Glycaemic ratio >.5 527 160 (30.5%) 434 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57.71) 85 (71, 106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 373 (70.8%) 373 (70.8%) 373 (70.9%) 33.9 (33.2, 34.4) 61 (56.66) 243 (210, 277) 84 (73, 96) 261 (49.5%) 0 (0, 2)	Glycaemic ratio < 5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (24.8%)           56 (48.7%)           94 (61.7%)           78 (67.8%)           2 (1.7%)           78 (67.8%)           2 (1.7%)           10 (8.7%)           66 (57.4%)           59 (50.68)           89 (72, 121)           2.59 (1.51, 4.83)           10 (8.7%)           80 (64, 109)           17 (14.8%)           10 (8.7%)           85 (73.9%)           3 (2.6%)           33.8 (32.55, 34.2)           61 (56.65)           243 (217, 275)           80 (70.94)           74 (64.3%)           1 (0, 2)	P           0.50           0.071           1.00           0.44           0.32           0.66           0.20           1.00           0.73           0.040           <0.011
Table 2. Patients with pre-existing hyperglycaemia           N           Preoperative characteristics           Female           Diabetes           Severe Left ventricular dysfunction           Chronic pulmonary disease           Congestive heart failure           Pulmonary hypertension           Mypertension           Smoking history           Redo           Emergency           Cerebrovascular disease           Myocardial infarction           Age, median (IQR)           Preoperative characteristics           CPB duration, median (IQR)           Urave CABG           CABG           Other           Lowest CPB DD-j., median (IQR)           RBC units transfused, median (IQR)           Postoperative outcome           Death	Glycaemic ratio >.5 527 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57.71) 85 (71, 106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 373 (70.8%) 373 (70.9%) 261 (49.5%) 0 (0, 2)	Glycaemic ratio <.5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           18 (16.5%)           40 (24.8%)           56 (48.7%)           94 (81.7%)           94 (81.7%)           94 (61.7%)           10 (8.7%)           66 (57.4%)           59 (50.68)           89 (72, 121)           2.59 (1.51, 4.83)           10 (8.7%)           80 (64, 109)           17 (14.8%)           10 (8.7%)           80 (64, 109)           17 (14.8%)           10 (8.7%)           80 (64, 109)           17 (14.8%)           10 (8.7%)           80 (64, 109)           17 (14.8%)           10 (8.7%)           80 (70, 9%)           32.62.6%)           33.8 (32.55, 34.2)           61 (56, 65)           243 (217, 275)           80 (70, 94)           74 (64.3%)           1 (0, 2)           5 (4.3%)           0 (70.9%)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.18           0.66           0.20           1.00           0.73           0.040           <0.011
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease         Myocardial infarction         Age, median (IQR)         Preoperative creatinien, median (IQR)         Euroscore, median (IQR)         Juraoperative characteristics         CPB duration, median (IQR)         Valve         Valve         Valve CABG         CABG         Other         Lowest CPB DApi, median (IQR)         Lowest QPB Hb, median (IQR)	Glycaemic ratio >.5 527 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 4 (0.8%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57,71) 85 (71,106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 373 (70.8%) 373 (70.8%) 373 (70.8%) 373 (70.8%) 373 (70.8%) 373 (70.8%) 373 (70.8%) 373 (70.9%) 33.9 (33.2, 34.4) 61 (56, 66) 243 (210, 277) 84 (73, 96) 261 (49.5%) 0 (0, 2) 9 (1.7%) 4 (0.8%)	Glycaemic ratio < 5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           10 (8.7%)           66 (57.4%)           59 (50.68)           89 (72.121)           2.59 (1.51, 4.83)           00 (84, 109)           17 (14.8%)           10 (8.7%)           80 (64, 109)           17 (14.8%)           10 (8.7%)           33.8 (32.55, 34.2)           61 (56.65)           243 (217, 275)           80 (70, 94)           74 (64.3%)           1 (0, 2)           5 (4.3%)           0 (0.0%)           11 (P 6%)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.73           0.040           0.73           0.040           0.73           0.44           0.73           0.040           0.73           0.41           0.24           0.55           1.00           0.57           0.688           0.030           0.660           0.53           0.043           0.044           0.15           1.00           0.57
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease         Myocardial infarction         Age, median (IQR)         Preoperative creatinine, median (IQR)         Euroscore, median (IQR)         Intraoperative characteristics         CPB duration, median (IQR)         Valve         Valve         Valve CABG         CABG         Other         Lowest cPB DAP, median (IQR)         Lowest CPB DAP, median (IQR)         Lowest CPB DAP, median (IQR)         Average CPB MAP, median (IQR)         Lowest CPB DAP, median (IQR)         Lowest CPB DB, median (IQR)         Lowest CPB DB, median (IQR)         Devest CPB DB, median (IQR)         Devest CPB DB, median (IQR)         Devest DPB AP, median (IQR)         Lowest CPB DB, median (IQR)         Desath<	Glycaemic ratio >.5 527 527 160 (30.5%) 494 (93.7%) 34 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 458 (86.9%) 324 (65.3%) 24 (4.6%) 4 (0.8%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57, 71) 85 (71, 106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 373 (70.8%) 377 (70.8%) 377 (70.8%) 377 (70.9%) 33.9 (53.2, 34.4) 61 (56, 66) 243 (210, 277) 84 (73, 96) 261 (49.5%) 0 (0, 2) 9 (1.7%) 4 (0.8%) 41 (7.8%) 53 (10.1%)	Glycaemic ratio < 5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           7 (6.1%)           19 (16.5%)           40 (34.8%)           56 (48.7%)           94 (81.7%)           78 (67.8%)           2 (1.7%)           10 (8.7%)           66 (57.4%)           59 (50.68)           89 (72,121)           2.59 (1.51,4.83)           10 (8.7%)           80 (64,109)           17 (14.8%)           10 (8.7%)           85 (73.9%)           31.8 (25.55,34.2)           61 (56.65)           243 (217,275)           80 (70,0.94)           74 (64.3%)           1 (0.2)           5 (4.3%)           1 (0.2)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.20           1.00           0.73           0.040           <0.01
Table 2. Patients with pre-existing hyperglycaemia         N         Preoperative characteristics         Female         Diabetes         Severe Left ventricular dysfunction         Chronic pulmonary disease         Congestive heart failure         Pulmonary hypertension         Hypertension         Smoking history         Redo         Emergency         Cerebrovascular disease         Myocardial infarction         Age, median (IQR)         Intraoperative creatinine, median (IQR)         Intraoperative characteristics         CPB duration, median (IQR)         Valve         Valve         Valve CABG         Other         Lowest CPB MAP, median (IQR)         Lowest CPB MAP, median (IQR)         Lowest CPB Hb, median (IQR)         Lowest CPB Hb, median (IQR)         Lowest CPB Hb, median (IQR)         Any blood product transfusion         RBC units transfused, median (IQR)         Postoperative outcome         Death         Stroke         Ventilation >48hrs         ARF         Reoperatio	Glycaemic ratio >.5 527 527 160 (30.5%) 434 (6.5%) 107 (20.3%) 157 (29.8%) 236 (45.6%) 4458 (86.9%) 344 (65.3%) 24 (4.6%) 4 (0.8%) 54 (10.2%) 246 (46.7%) 64 (57,71) 85 (71, 106) 2.73 (1.59, 5.37) 82 (60, 108) 68 (12.9%) 49 (9.3%) 37 (7.0%) 3.39 (33.2, 34.4) 61 (56, 66) 243 (210, 277) 84 (73, 96) 261 (49.5%) 0 (0, 2) 9 (1.7%) 4 (0.8%) 41 (7.8%) 53 (10.1%) 28 (49.%)	Glycaemic ratio < 5           115           39 (33.9%)           102 (88.7%)           7 (6.1%)           19 (16.5%)           40 (24.8%)           56 (48.7%)           94 (61.7%)           78 (67.8%)           2 (1.7%)           10 (8.7%)           96 (57.4%)           59 (50.68)           89 (72, 121)           2.59 (1.51, 4.83)           10 (8.7%)           80 (64, 109)           17 (14.8%)           10 (8.7%)           85 (73.9%)           3 (2.6%)           33 (2.55, 34.2)           61 (56.65)           243 (217, 275)           80 (70.94)           74 (4.3%)           1 (0.2)	P           0.50           0.071           1.00           0.44           0.32           0.61           0.18           0.66           0.20           1.00           0.73           0.040           <0.011

In multivariate analysis, GR >1 was an independent predictor of mortality in normoglycaemic patients (OR, 1.3; 95% CI, 1.05-1.62; p=0.017) and GR <0.5 in patients with pre-existing hyperglycaemia (OR, 5.23; 95% CI, 1.54-17.73; p=0.008). Results of the multivariate analysis in normoglycaemic patients are shown in figure 3, and those with pre-existing hyperglycaemia in figure 4.



Figure 3. Odds ratios for each variable included in the multivariate model for normoglycaemic patients.



Figure 4. Odds ratios for each variable included in the multivariate model for normoglycaemic patients.

# Conclusions

The association between relative glycaemic change and mortality for cardiac surgical patients appears to be modulated by pre-existing hyperglycaemia. These preliminary results suggest that intraoperative hyperglycaemia should be managed with consideration of the preoperative HbA1c. In patients with pre-existing hyperglycaemia, reducing glucose levels >50% may be detrimental.

### REFERENCES

- 1. Knapik P, Nadziakiewicz P, Urbanska E, et al. Cardiopulmonary bypass increases postoperative glycemia and insulin consumption after coronary surgery. Ann Thorac Surg 2009; 87: 1859–1865.
- Giakoumidakis K, Nenekidis I, Brokalaki H. The correlation between peri-operative hyperglycemia and mortality in cardiac surgery patients: a systematic review. Eur J Cardiovasc Nurs 2012; 11(1):105-13.
- Krinsley JS, Egi M, Kiss A, et al. Diabetic status and the relation of the three domains of glycemic control to mortality in critically ill patients: an international multicenter cohort study. Crit Care 2013; 17(2):R37.
- 4. Brosius FC, Heilig CW. Glucose transporters in diabetic nephropathy. Pediatr Nephrol 2005; 20(4):447-51.
- Kahn BB, Rosen AS, Bak JF, et al. Expression of GLUT1 and GLUT4 glucose transporters in skeletal muscle of humans with insulin-dependent diabetes mellitus: regulatory effects of metabolic factors. J Clin Endocrinol Metab 1992; 74(5):1101-9.
- Thiessen S, Vanhorebeek I, Van den Berghe G. Glycemic control and outcome related to cardiopulmonary bypass. Best Pract Res Clin Anaesthesiol. 2015; 29(2):177-87.
- 7. Engoren M, Habib R, Zacharias A, et al. The prevalence of elevated hemoglobin A1c in patients undergoing coronary artery bypass surgery. J Cardiothorac Surg 2008; 3:63.
- 8. Nathan D, Kuenen J, Borg R, et al. Translating the A1c assay into estimated average glucose values. Diabetes Care 2008; 31:1473-8.
- 9. Plummer MP, Bellomo R, Cousins CE, et al. Dysglycaemia in the critically ill and the interaction of chronic and acute glycaemia with mortality. Intensive Care Med 2014; 40:973–980.
- Egi M, Bellomo R, Stachowski E, et al. The interaction of chronic and acute glycemia with mortality in critically ill patients with diabetes. Crit care med 2011; 39(1):105-11
- Newland R, Baker RA, Stanley R, Place K, Willcox TW. The Perfusion Downunder Collaborative Database project. J Extra Corpor Technol 2008; 40:159–65.
- Baker RA, Newland RF, Fenton C, McDonald M, Willcox TW, Merry AF. Developing a benchmarking process in perfusion: a report of the Perfusion Downunder Collaboration. J Extra Corpor Technol 2012; 44:26–33.
- 13. Dinh DT, Tran L, Chand V, et al on behalf of the Australasian Society of Cardiac and Thoracic Surgeons (ASCTS) Victorian Cardiac Surgery Database Project Steering Committee. Cardiac surgery in Victorian public hospitals, 2009–10. Public report. Department of Health, Melbourne, Victoria, Australia. Available at http://anzscts-database.org/sites/default/files/ SurgVicHosp2009-10.pdf. Accessed February 29, 2016.

# AN EVALUATION OF HAEMOGLOBIN MEASUREMENT DEVICES FOR CARDIAC SURGERY

by Casey Edwards, Trainee Cardiac Perfusioninst, The Canberra Hospital Student Research Project, Submitted for review 27th September 2016

**Introduction:** Allogeneic blood transfusions expose recipients to numerous health risks but may be unavoidable as a result of haemodilution on cardiopulmonary bypass. Haemoglobin levels below an institution-specific threshold result in erythrocyte administration. It is therefore essential that haemoglobin-measuring instruments are accurate to ensure appropriate transfusion decisions. To measure haemoglobin during cardiac surgery, the Canberra Hospital has the I-stat, Hemocue and Gem4000 (which literature suggests is the most accurate device). This study investigates the variance in haemoglobin readings between these instruments to infer their suitability for use in the clinical setting.

**Methods:** 100 blood samples were taken from 34 patients during cardiopulmonary bypass and haemoglobin was concurrently tested on the I-stat, Hemocue and Gem4000.

**Results:** Assuming the Gem4000 to be correct, the mean bias of the I-stat was  $-4.04\mu g/dL +/- 5.46$  and the Hemocue  $-0.82\mu g/dL +/- 5.94$ . Given the relatively large standard deviations observed for both devices, limits of agreement indicate that a patient's observed haemoglobin must be  $81\mu g/dL$  (I-stat) or  $82\mu g/dL$  (Hemocue) for 95% certainty that true haemoglobin is above  $70\mu g/dL$ , our institutional transfusion threshold. Concurrently running samples on the I-stat and Hemocue then choosing the higher haemoglobin reading was the most accurate estimation of true haemoglobin, with a mean bias of  $0.22\mu g/dL +/- 5.37$ .

**Conclusion:** When a patient's haemoglobin is expected to be below 81µg/dL, the Gem4000 is the most suitable instrument to avoid inappropriate transfusions and testing duplication. In circumstances where the Gem4000 is inaccessible, concurrent I-stat and Hemocue testing with higher-value selection is the optimal strategy.

**Clinical Significance:** Our haemoglobin measuring protocol has changed. Sample duplication has reduced so transfusion decisions can be made more quickly and the appropriate haemoglobin measurement device can be selected for individual patients.

### Introduction

In the current era of perfusion, blood management is at the forefront of our practice. In fact, blood has been described as "the most precious and personal substance in the world" (1). Recent literature in the realm of cardiac surgery and perfusion has encouraged clinicians to conserve autologous blood and aim to avoid allogeneic blood product transfusions where possible (2,3,4,5,6). However in some circumstances, erythrocyte transfusion during cardiopulmonary bypass becomes essential to achieve adequate oxygen delivery to tissues. Central to the decision of whether to transfuse allogeneic blood is measurement of a patient's haemoglobin concentration (7).

A key function of erythrocytes is transportation of the respiratory gases: oxygen (O2) and carbon dioxide (CO2) (8). Haemoglobin is the protein molecule within erythrocytes responsible for binding O2 in lung capillaries. O2 is released into tissues in exchange for CO2 to facilitate aerobic metabolism (8). If a patient's haemoglobin concentration falls below a critical level during cardiac surgery via haemodilution or hemorrhage, oxygen delivery and carbon dioxide removal from tissues is compromised. Consequently, an affected patient may suffer tissue hypoxia and end organ damage (8,9). Low haemoglobin concentrations during cardiac surgery have been associated with higher mortality, increased troponin release, impaired glomerular filtration rate, acute kidney injury, longer duration of mechanical ventilation and longer length of hospital stay (9). To avoid these complications, healthcare professionals may elect to transfuse allogeneic erythrocytes if sufficiently low haemoglobin is observed during cardiac surgery.

The transfusion of red blood cells is also associated with risks including the transmission of viruses, bacteria and parasites, immunological reactions both acute and delayed, alloimmunisation, autoimmunization, transfusion related acute lung injury and transfusion associated circulatory overload (10). Evidently, both red blood cell transfusion and severe acute anaemia have the potential to cause harm to a patient. Perfusionists must therefore evaluate the associated risks of transfusion versus low haemoglobin for a given patient and decide whether or not to administer a transfusion on bypass (9,10).

Individual cardiac surgery units may adopt a 'transfusion trigger' protocol to standardize blood transfusion decisions. Usually a transfusion trigger involves a set haemoglobin (Hb) concentration at which to transfuse (11). Therefore, being able to accurately and rapidly measure patients' haemoglobin is critical. There are multiple haemoglobin measurement devices available to purchase. This study investigates three such devices: the Gem Premier 4000 blood gas analyser, the i-STAT blood gas analyser and the Hemocue haemoglobin analyser.

The i-STAT is point of care blood gas device is produced by Abbott Laboratories. It is capable of analysing multiple parameters simultaneously, including pH, pCO2, pO2, electrolytes, metabolites, haemoglobin and haematocrit (12). To operate the device, 65-100µl of blood is introduced to the capillary of a cartridge containing multiple analytes specific to the desired testing parameters. The cartridge is then inserted into the analyser and a report of results is generated in approximately 120 seconds (13). To determine haematocrit, the i-STAT measures the conductivity of blood within the cartridge between two electrodes. The device relies on the principle that blood haematocrit is inversely related to conductivity of the sample. After deducing haematocrit, the central processing unit of the i-STAT corrects the result for electrolyte concentration within the sample and calculates haemoglobin by multiplying the haematocrit by 0.34 (14).

Quality control of the i-STAT is done daily with a cartridge simulator and monthly with a fluid of known electrolyte concentrations. Each batch of cartridges also undergoes quality control before use (13). Documentation of correct quality control is available for the particular i-STAT used in this study. The device was set on 'haemodilution' mode, which improves its analysis of samples from patients undergoing cardiopulmonary bypass. The advantages of the i-STAT include its variety of testing parameters, its speed and its portability. However experientially, the device appears to produce low estimates of haemoglobin. The extent of this underestimation is to be investigated in this study.

The Hemocue is a small, light portable point of care device (15). Its sole function is the quantification of haemoglobin from whole blood samples. 10µl of blood is required for analysis. Blood is introduced to the instrument via a cuvette containing sodium deoxychrolate to haemolyse erythrocytes, sodium nitrite to convert haemoglobin to methaemoglobin and sodium azide to convert methaemoglobin to haemoglobinazide (13). The blood-filled cuvette is inserted into the holder of the instrument and a hemoglobin reading is produced within 45 seconds. The Hemocue uses the absorbance of light at 570 and 880nm plus an inbuilt algorithm to calculate haemoglobin based on the sample's absorbance (13).

The Hemocue is calibrated and quality controlled manually using a reference cuvette. The Hemocue device used in this study has a documented quality control history in accordance with its instruction manual. The device's advantages are its speed in producing a result, its simplicity to operate and its portability. However, the fact that it does not measure other blood gas variables is a key disadvantage as compared with the other two devices in this study.

The Gem Premier 4000 (Gem4000) is a bench top blood gas analyser. It is the bulkiest, heaviest, slowest and the most expensive of the three devices being examined for the purpose of this study. It measures the largest variety of blood gases, electroytes and metabolites as well as haemoglobin and hematocrit (13). The system uses co-oximetry to measure haemoglobin. Co-oximetry involves spectrophotometry, but at a more sophisticated level than the Hemocue. The absorption of several dozens of wavelengths or light is used to distinguish oxyhaemoglobin, deoxyhaemoglobin, carboxyhaemoglobin and methaemoglobin (13). Quality control is auto-regulated by the machine itself. Gem4000's 'IQM' (internal quality management) software completes all necessary system testing at fixed time intervals, promoting consistency and preventing human-error in the quality-control process. Users can download an historical log of quality management.

The Gem4000 is the most accurate machine available within the theatre complex of The Canberra Hospital (16). Cooximetry has been experimentally validated as an accurate method of haemoglobin quantification. With respect to the Gem4000 specifically, its analytical performance has been validated for all parameters in terms of accuracy and the reliability. The device fulfills the requirements for laboratory use (16). For this reason, the Gem4000 has been designated the 'gold standard' haemoglobin measurement device for the purpose of this study. In reality, the International Council for Standardization in Hematology defines the true gold standard for haemoglobin analysis to be the haemiglobincyanide method (17). However, a device utilizing this method is not available to staff in cardiac surgery at The Canberra Hospital, so our designated gold standard has been elected based on its correlation with the hemiglobinocyanide method (18).

With respect to the accuracy of the other two devices: Hemocue and i-STAT, there is a lack of consensus in the literature. In 2013, Skelton et al. (19) compared the Hemocue to a cooximetry device and a flow cytometer typically found in hospital haematology laboratories. It was found that the HemoCue and laboratory test gave similar results but that the co-oximeter's readings varied significantly. However, the same research team reports the converse result in a previous paper. Their research in 2010 concluded that there was a strong correlation between the co-oximeter-derived Hb and laboratory measurements (20). Similarly, a study in critically ill patients using the same three instruments showed that the laboratory test correlated better with the co-oximeter than the Hemocue (21). The i-STAT has been repeatedly reported to underestimate haemoglobin (14, 23, 24, 25). In fact, Ng et al. (2014) have recommended that clinicians avoid using haemoglobin readings from the i-STAT alone for clinical decision-making when considering blood transfusion (23).

The Canberra Hospital currently relies upon the i-STAT system for routine Hb monitoring at approximately 20-minute intervals. Should a patient's Hb read 70mg/dL or lower, the Hemocue is generally employed as a "second opinion" to further delineate whether red blood cells should be transfused. We have observed that results from the two systems often vary despite having analysed the same blood sample. Historically it has been common for us to analyse a sample on both devices and record the higher of the two numbers, particularly if it involves avoiding a blood transfusion. If significant variation is observed, the sample may also be analysed on the Gem4000 device. With three different estimates of Hb obtained, we are most likely to use the Gem4000 result when deciding whether to transfuse. However, running three samples in succession is time consuming. For patients who are in need of a blood transfusion, testing duplication increases their time spent in a hypoxic state. Testing duplication is also a wasteful process. Disposables for the i-STAT and Hemocue plus reagents within the Gem4000 are used in the process, which comes at a cost to the hospital.

Therefore, the aim of this study is to investigate the accuracy and reliability of haemoglobin measurements from the i-STAT and Hemocue point of care devices (POCDs) in comparison to the Gem4000 bench top co-oximeter, to better understand the extent to which the systems differ and evaluate which system is the optimal choice for our clinical use.

Based on the available literature, it is hypothesized that there will be a significant difference between the benchtop co-oximeter and POCDs. Clinical experience with the devices suggests that the Gem4000 will produce the highest haemoglobin results, the Hemocue will read lower than the Gem, but the I-stat will have the lowest haemoglobin readings of all three devices.

# Materials and Methods

### Patient population

Blood was drawn from 34 patients undergoing cardiac surgery at the Canberra Hospital, totalling 100 samples. The number of samples taken per patient ranged from 2 to 6. This variation was due to differences in case length and the frequency with which the practicing perfusionist wished to run arterial blood gases. There were no exclusion criteria for this study. Patient data was not recorded for the purposes of this study, instead each patient was allocated a number between 1 and 34.

### Sampling and analysis protocol

Samples were taken at nonspecific time points throughout cardiac surgical cases when the perfusionist elected to run an arterial blood gas. At a sampling time, 2mL of blood was withdrawn using a syringe from the sampling manifold of the Medtronic Fusion oxygenator reservoir and analysed on three haemoglobin-measuring platforms concurrently: an i-STAT POCD, a Hemocue POCD, and a Gem4000 bench-top blood gas machine. Each device was run as per its operating instructions. The haemoglobin result reported by each device was recorded for subsequent statistical analysis.

### Results

For the purpose of statistical analysis, all tests in this study assume the Gem4000's reading to be the 'true' haemoglobin, against which the other devices' readings are compared. The collected haemoglobin data was first analysed using a 1-way ANOVA with blocking (where the sample was designated the block). This was done in order to quantify the mean difference in haemoglobin estimation between machines, as well as the variance. The results of this analysis are presented in table 1:

Table 1: Pairwise comparisons of three haemoglobin measurement devices using a one-way ANOVA

(I) test	(J) test	Mean difference (ا-یا)	Std. error	df
HbGem4000	HbHemocue	0.820	0.603	198
	Hblstat	4.040*	0.603	198
HbHemocue	HbGem4000	-0.820	0.603	198
	Hblstat	3.220*	0.603	198
Hblstat	HbGem4000	-4.040*	0.603	198
	HbHemocue	-3.220*	0.603	198

\* = The mean difference is significant at the 0.05 level.

Table 1 shows that the mean difference in haemoglobin

readings between the Gem4000 and the Hemocue was -0.82µg/dL, a statistically insignificant difference. This result indicates that on average, when analysing the same whole blood sample, the Hemocue will interpret haemoglobin 0.82mg/dL lower than the Gem4000.

The difference between the Gem4000 and the i-STAT was statistically significant, with a mean difference in haemoglobin reading of -4.04 $\mu$ g/dL. This result indicates that, the i-STAT interprets haemoglobin 4.04  $\mu$ g/dL lower on average than the Gem4000 given the same whole blood sample.

To further delineate the magnitude of the difference between the machines, Bland Altmann plots were generated, firstly between the i-STAT and Gem4000 (see figure 1).

Figure 1: Bland Altmann plot depicting the difference against mean for i-STAT and Gem4000 haemoglobin data with associated descriptive statistics



### **DESCRIPTIVE STATISTICS**

	N	Minimum	Maximum	Mean	Standard Deviation
i-STAT vs. Gem4000	100	-8.00	21.00	4.04	5.46

Figure 1: A Bland-Altman plot of N=100 haemoglobin values as determined by the Gem4000 versus the i-STAT collected from 34 subjects undergoing cardiopulmonary bypass. The vertical axis shows the observed differences between haemoglobin when measured by the two devices. The horizontal axis depicts the mean of the 2 measures. Each dot represents 1 individual Gem400Hb – i-STATHb value. The plot displays the mean bias in haemoglobin reading by the Gem4000 as a solid red line ( $4.04\pm5.46$ ), and 95% confidence intervals as red dashed lines. The solid black lines denotes zero, the point at which the bias line would appear if the instruments read haemoglobin identically.

Figure 1 demonstrates that on average, the Gem4000 interprets haemoglobin 4.04 units higher than the i-STAT with a standard deviation of 5.46. The 95% confidence intervals calculated at 1.96 standard deviations either side of the mean are -6.66 and 14.77, as depicted by the red dashed lines in figure 1. The range of variation between the machines is summarized in the descriptive statistics section, under 'minimum' and 'maximum'. Within the 100 samples collected, the Gem4000 read from a mimimum of 8 units lower to 21 units higher than the i-STAT.

The same style of analysis was completed to compare the Gem4000 and the Hemocue. These results appear in Figure 2.

Figure 2: Bland Altmann plot depicting the difference against mean for Hemocue and Gem4000 haemoglobin data with associated descriptive statistics



**DESCRIPTIVE STATISTICS** 

	N	Minimum	Maximum	Mean	Standard Deviation
i-STAT vs. Gem4000	100	-30.00	24.00	0.82	5.94

Figure 2. A Bland-Altman plot of N=100 haemoglobin values as determined by the Gem400 versus the Hemocue collected from 34 subjects undergoing cardiopulmonary bypass. The vertical axis shows the observed differences between haemoglobin when measured by the two devices. The horizontal axis depicts the mean of the 2 measures. Each dot represents one individual Gem400Hb – Hemocue value. The plot displays the mean bias in haemoglobin reading by the Gem4000 as a solid red line  $(0.82\pm5.94 \,\mu\text{g/dL})$ , and 95% confidence intervals as red dashed lines. The solid black line denotes zero, the point at which the bias line would appear if the instruments read hemoglobin identically.

From figure 2 it is evident that the Gem4000 interprets haemoglobin 0.82 units higher than the Hemocue on average, with a standard deviation of 5.94. Therefore, the Hemocue reads more closely to the Gem4000 than does the i-STAT. The 95% confidence intervals were calculated to be -10.82 and 12.46, as depicted by the red dashed lines in figure 1. With respect to the range of variation between the machines, the Gem4000 read from a minimum of 30 units lower to 24 units higher than the Hemocue.

The third Bland Altmann plot (figure 3) does not compare two devices directly as figures 1 and 2 have done. It has been previously stated that running concurrent samples on the i-STAT and Hemocue then making transfusion decisions based on the higher of the two numbers has historically been common practice at The Canberra Hospital. Figure 3 evaluates the validity of this method by comparing the Gem4000 results to the higher result obtained from a POCD for the same sample.

Figure 3: Bland Altmann plot depicting the difference against mean for higher POCD value (Hemocue or i-STAT) and Gem4000 haemoglobin data with associated descriptive statistics



### DESCRIPTIVE STATISTICS

	N	Minimum	Maximum	Mean	Standard Deviation
Higher POCD vs. Gem4000	100	-30.00	21.00	-0.22	5.37

Figure 3. A Bland-Altman plot of N=100 haemoglobin values as determined by the Gem400 versus the higher of the POCD values (either i-STAT or Hemocue) collected from 34 subjects undergoing cardiopulmonary bypass. The vertical axis shows the observed differences in haemoglobin measured by the devices. The horizontal axis depicts the mean of the 2 measures. Each dot represents 1 individual Gem400Hb – highest POCD value. The plot displays the mean bias in haemoglobin reading by the Gem4000 as a solid red line (0.22±5.37 µg/dL), and 95% confidence intervals as red dashed lines. The solid black lines denotes zero, the point at which the bias line would appear if the instruments read hemoglobin identically.

Assuming that the Gem4000's reading to be correct, choosing the higher number from the POCDs will result in an overestimate of hemoglobin by 0.22µg/dL on average. This is the smallest mean bias observed across the 3 Bland Altman plots completed in this study. Choosing the higher value from the Hemocue and i-STAT proved to be a more accurate method than using either instrument alone. The standard deviation for this method was 5.37 with 95% confidence intervals -10.30 and 10.74. Regarding the range of variation between the machines, the Gem4000 read from a mimimum of 30 units lower to 21 units higher than the combined POCDs.

### Discussion

This study aimed to investigate the accuracy and reliability of the i-STAT and Hemocue point of case devices (POCDs), as compared with the Gem4000 bench top co-oximeter. In terms of mean accuracy, the data obtained in this study suggest that the i-STAT is the least accurate estimation of haemoglobin, the Hemocue is more accurate, but the most accurate estimation is obtained by running a sample on both machines simultaneously and recording the higher of the two results. With respect to reliability, large standard deviations and confidence intervals were observed for both devices. This suggests a high level of variance in accuracy by both POCDs, limiting clinicians' confidence in making transfusion-related decisions based on a POCD result. Using the 95% confidence interval data from figures 1, 2 and 3,"threshold haemoglobin values" can be calculated. If a haemoglobin result from a POCD is above the device's threshold value, there is a low risk of failing to transfuse based on an incorrect POCD result. Assuming a transfusion trigger of Hb =  $70\mu g/dL$ , to be 95% certain that an i-STAT haemoglobin is not less than 70µg/dL, a result of 76.66 µg/dL (i.e. 77) would have to be obtained. For the Hemocue, a result of 80.82  $\mu$ g/dL (i.e. 81) would have to be obtained. For both devices combined with higher-value selection, a value of 80.3 µg/dL (i.e. 81) would have to be obtained. Ultimately, these results suggest that if a patient's haemoglobin is expected to be less that 81 µg/dL, it is more appropriate to skip point-of-care testing and instead use the Gem4000 for analysis. Otherwise, there is a risk of failing to transfuse erythrocytes when they are clinically indicated.

Conversely, if the result of a POCD is 70µg/dL indicating an erythrocyte transfusion, the patient's true haemoglobin may be as high as 84.77µg/dL if measured on the i-STAT, 82.46 on the Hemocue or 80.74 using higher-value POCD selection. Trusting the POCDs in this situation could lead to administration of inappropriate transfusions. These results suggest that the Hemocue and i-STAT are not reliable instruments for transfusion decisions given their propensity for variation.

One key limitation affects the results of this study. Statistical analysis has assumed that the Gem4000 reads true haemoglobin. This assumption is inherently incorrect since all devices have a margin of error and the Gem4000 is not the absolute gold standard of Hb reading devices. However, this assumption was made to model the situation within cardiac surgery at the Canberra Hospital. The three devices investigated in this study are the only haemoglobin measuring instruments available and literature has indicated that the Gem4000 is the most accurate (16, 17, 18). Results from the Gem4000 were designated to be 'correct' since the device is the best estimate of haemoglobin that can be achieved in the clinical setting. As a consequence of this assumption the mean biases reported for the i-STAT and Hemocue are not their true biases, instead they are an indication their underestimation of haemoglobin compared to the Gem4000. In the planning phase of this study, efforts were made to have the samples in this study also tested independently in the hospital's hematology laboratory. However practical barriers such as cost, paperwork and inconvenience to the laboratory prevented this analysis from occurring. A further study could be done to verify the accuracy of the Gem4000 by comparing it to the Coulter counter analyser in our haematology laboratory. If the Gem4000 proved to be accurate, it would add validity to the results of this study.

Other additional research could investigate whether there is variation between batches of disposables for the POCDs, which could explain some of the variation observed in this study. A similar study method could also be completed on multiple Gem4000, i-STAT and Hemocue devices from different cardiac centres. Providing all instruments were maintained with strict adherence to quality control protocol, a broader picture of the variation between the devices could be ascertained. Introducing a fourth device such as a continuous, non-invasive blood gas monitor into the study would be an opportunity to evaluate a new technology. 'Spectrum Medical' distributes a haemoglobin-measuring device that clips onto the outside of cardiopulmonary bypass tubing. By subjecting Spectum's device to a similar method, its relative accuracy and reliability could be calculated to evaluate whether the device would be suitable for clinical use.

As a result of this study, perfusionists at The Canberra Hospital have adopted a revised protocol for haemoglobin testing. If a haemoglobin result is expected to be below 81µg/dL, point of care testing is skipped entirely and the Gem4000 is used. There are circumstances where expected haemoglobin could be less than 81µg/dL but testing on the Gem4000 is impossible, such as when the machine is already in use, during its quality control cycle, during a power outage or when there are no staff members able to transport a sample to the Gem4000. In these circumstances, the perfusionist is advised to run a sample on the i-STAT and Hemocue concurrently and record the higher of the two results. Comparison of transfusion rates pre and post implementation of this protocol may provide an insight into whether transfusions have been given too liberally or too restrictively in the past based on POCD results.

This study aimed to investigate the accuracy and reliability of haemoglobin measurements from the i-STAT, and Hemocue point of case devices (POCDs), as compared with the Gem4000 bench top co-oximeter. This aim was successfully achieved, with both devices having a negative bias as compared to the Gem4000. The i-STAT's negative bias was the largest, the Hemocue's was intermediate. The higher-value POCD selection method resulted in a slight positive bias, but proved to be the most accurate estimation of the Gem4000. All methods exhibited large variations in accuracy, as reflected in the large standard deviations and 95% confidence intervals observed. A secondary aim was to evaluate which system is the optimal choice for our clinical use. This aim was also successfully achieved with a revised haemoglobin measurement protocol having been implemented at the Canberra Hospital. As a result of this study, it is hoped that patients will benefit, as the risks of inappropriate transfusions will be reduced.

### Funding and conflict of interest statement

No funding received and no conflict of interest declared REFERENCES

- Samolys, KA. State-of-the-art blood management in cardiac surgery. Seminars in Cardiothoracic and Vascular Anesthesia 2009; 13: 118-121.
- Ferraris, VA, Ferraris, SP, Saha, SP et al. Perioperative blood transfusion and blood conservation in cardiac surgery: The Society of Thoracic Surgeons and The Society of Cardiovascular Anesthesiologists clinical practice guideline. The Annals of Thoracic Surgery 2007; 83: 27-86.
- Murphy, GJ, Reeves, BC, Rogers, CA et al. Increased Mortality, Postoperative Morbidity, and Cost After Red Blood Cell Transfusion in Patients Having Cardiac Surgery. Circulation 2007;116:2544-2552
- Surgenor, SD, Kramer, RS, Olmstead, EM et al. The association of perioperative red blood cell transfusions and decreased longterm survival after cardiac surgery. Anaesthesia and Analgesia. 2009;108: 1741-1746
- Emmert, MY, Salzberg, SP, Theusinger, OM et al. How good patient blood management leads to excellent outcomes in Jehovah's witness patients undergoing cardiac surgery. Interactive Cardiovascular and Thoracic Surgery. 2011; 12: 183-188
- Moskowitz, DM, McCullough, JN, Shander, A et al. The impact of blood conservation on outcomes in cardiac surgery: is it safe and effective? The Annals of Thoracic Surgery 2010; 90: 451-458
- 7. Carson, J, Grossman, B, Kleinman, S et al. (2012). Red Blood Cell

Transfusion: A Clinical Practice Guideline From the AABB. Annals of Internal Medicine. 2012; 157: 49-58

- 8. Jandl, JH. Blood Textbook of Hematology. Boston:Little, Brown and Company; 1996: 72-72.
- Loor, G, Li, L, Sanik, JF et al. Nadir hematocrit during cardiopulmonary bypass: end-organ dysfunction and mortality. Journal of Thoracic and Cardiovascular Surgery. 2012; 144(3): 654-662.
- Marcucci, C, Madjdpour, C, Spahn, DR. Allogeneic blood transfusions: benefit, risks and clinical indications in countries with a low or high human development index. British Medical Bulletin. 2004; 70(1): 15-28.
- Friedman, BA, Burns, TL, Schork, MA. An Analysis of Blood Transfusion of Surgical Patients by Sex: A Quest for the Transfusion Trigger. Transfusion. 1980; 20(2): 179-188
- Myers, FB, Lee, LP. Innovations in optical microfluidic technologies for point-of-care diagnostics. Lab on a Chip. 2008; 8: 2015-2031
- Ruckman, JS, A comparative study of total hemoglobin measurement technology: noninvasive pulse co-oximetry and conventional methods. Master's Theses, University of Conneticut Graduate School, Paper 75. Available from http://digitalcommons. uconn.edu/gs\_theses/75. Accessed June-July 2016.
- 14. Hopfer, Nadeau, Sundra et al. Effect of protein on hemoglobin and hematocrit assays with a conductivity-based point-of-care testing device: comparison with optical methods. Annals of Clinical Laboratory Science. 2004; 34(1): 75-82
- Lardi, A, Hirst, C, Mortimer, A et al. 'Evaluation of the HemoCue for measuring intra-operative hemoglobin concentrations: a comparison with the Coulter Max-M' Anaesthesia. 1998; 53: 349-352.
- 16. Beneteau-Burnat, B, Pernet, P Pillon, A et al. Evaluation of the GEM Premier 4000: a compact blood gas CO-Oximeter and electrolyte analyzer for point-of-care and laboratory testing. Clinical Chemistry Laboratory Methods. 2008; 46(2):271-279
- O'Reilly, M. Considerations for Evaluating the Accuracy of Hemoglobin Monitoring. Anesthesiology. 2012;117: 429-430.
- Zwart, A. Spectrophotometry of hemoglobin: various perspectives. Clinical Chemistry. 1993; 39(8): 1570-1572
- 19 Skelton, VA, Wijayasinghe, N, Sharafudeen, S, et al. Evaluation of point-of-care haemoglobin measuring devices: a comparison of radical haemoglobin measurements in obstetric patients. Anaesthesia. 2013; 68: 40–45
- 20. Macknet MR, Allard M, Applegate RL, Rook J. The accuracy of noninvasive and continuous total hemoglobin measurement by pulse co-oximetry in human subjects undergoing hemodilution. Anesthesia and Analgesia. 2010; 111: 1424–6.
- Frasca, D, Dahyot-Fizelier, C, Catherine, K et al. Accuracy of a continuous noninvasive haemoglobin monitor in intensive care unit patients. Critical Care Medicine. 2011; 39: 2277–82.
- Butwick A, Hilton G, Carvalho B. Non-invasive haemoglobin measurement in patients undergoing caesarean section. British Journal of Anaesthesia. 2012; 108:271–7.
- 23. Ng, WL, Short, TG, Gunn, KN et al. Accuracy and reliability of the i-STAT point-of-care device for the determination of haemoglobin concentration before and after major blood loss. Anaesthesia and Intensive Care. 2014; 42(4): 495-499
- Schneider, J, Dudziak, R, Westphal, K et a. The i-STAT analyzer. A new, hand-held device for the bedside determination of hematocrit, blood gases, and electrolytes. Anaesthetist. 1997; 46(8): 704-714
- 25. Papadea, C, Foster, J, Grant, S, et al. Evaluation of the i-STAT portable clinical analyzer for point-of-care blood testing in the intensive care units of a university children's hospital. Annals of Clinical and Laboratory Science. 2002; 32(3):231-243







Perfusionist Occupational Hazards

# SE ENERTY Perfusion Incident Reporting System

PIRS

# What is PIRS?

PIRS is a voluntary system for reporting perfusion related incidents and accidents, open to the international perfusion community. Confidentiality is assured by de-identification and anonymity. PIRS data will not be passed to any third party or regulatory body. For further information see <u>www.anzcp.org</u>.



# TO SUBMIT TO PIRS VISIT www.anzcp.org ...and hit the PIRS tab.

# 2016 AUSTRALIAN AND NEW ZEALAND COLLEGE of PERFUSION

# ANNUAL SCIENTIFIC MEETING AWARDS

### Terumo Award

The Terumo award is in recognition of the best scientific or clinical paper presented by a full ANZCP member.

Awarded to Annette Mazzone; Levels of Circulating and Urinary Micro RNAs are increased During Cardiac Surgery with Cardio-pulmonary Bypass.

### Medtronic Encouragement Award

To encourage the Scientific development of Australasian Perfusionists.

Awarded to Richard Newland; Intraoperative dysglycemia: Perfusion registry data on incidence, management and postoperative outcomes.

### Liva Nova Sid Yarrow Award

Awarded to the Student member of the College who has made the best presentation at the annual Scientific Meeting.

Awarded to Casey Edwards; An evaluation of Haemoglobin Measurement Devices for Cardiac Surgery

### **ANZCP** Meritorious Award

The Purpose of the ANZCP sponsored Meritorious Presentation Award is to acknowledge presentations of excellence and which are considered to be sufficiently meritorious.

Awarded to Clarke Thuys; NO! YES! Nitric Oxide and Cardiopulmonary Bypass.

# **Education Scholarship**

Awarded to the Student who achieves the highest mark in the ABCP Certification Examination.

Awarded to Emmerson Sgammotta

# Gazette Award

Awarded by the Editorial Committee for outstanding contribution to the Gazette.

Awarded to: Cynthia Riddell

Awarded to: Martin Gill

# **Best Trade Display**

Awarded to Terumo Corporation Australia

### Maquet Open Heart International Scholarship

This scholarship is awarded via application to a Junior member of ANZCP. The scholarship provides travel funding to gain experience on an Open Heart International Mission.

This year Maquet will fund Angel Johns to attend an OHI Mission. However the ANZCP Executive voted to fund a second applicant to this very worthy cause. Casey Edwards will also attend an OHI Mission.

# GETINGE GROUP

Maquet Australia Extra Corporeal Membrane Oxygenation 8 hours Clinical Workshop

# Clinical Workshop **Details**

Aimed at All Clinical Staff actively involved in patient cannulation, device priming and set up and managing a patient supported by Extra Corporeal Membrane Oxygenation (ECMO) in the Clinical environment.

Maquet is committed to the provision of up to date, high quality clinical training to improve our customers knowledge of Extracorporeal Life support (ECLS) and to improve patient outcomes. To achieve this we utilise interactive lectures, scenario based skill stations and high fidelity simulation utilising our specialised cannulation mannequin. These comprehensive training methods allow us to mirror real clinical situations and provide troubleshooting strategies to assist clinicians caring for this challenging patient group. We rely on an experienced and dedicated National Clinical Applications Team (NCAS) to deliver a comprehensive course which has so far received fantastic feedback.

Cost: \$2,500 (GST exclusive) | Maximum 12 people

Includes workbook and catering for tea breaks and lunch. For further information please email **ncas.au@maquet.com** 





This ECMO Clinical Workshop is endorsed by ACN according to our Continuing Professional Development (CPD) Endorsed Course Standards. It has been allocated 8 CPD hours according to the Nursing and Midwifery Board of Australia – Continuing Professional Development Standard.

MAQUET GETINGE GROUP The following pages contain the abstracts from the 33<sup>rd</sup> Annual Scientific Meeting Australian and New Zealand College of Perfusionists.

# RESUSCITATING PATIENTS FROM DEEP HYPOTHERMIA

# Phillip Scott, The MAYO Clinic, Rochester, USA

Resuscitating patients from deep hypothermia almost always requires the expertise of a perfusionist. Yet most perfusionists rarely, if ever at all, experience this type of patient. The management of this patient group is specific, especially upon initial discovery. Proper management of these patients moves against all natural instincts of first responders and emergency room personnel. Perfusionists should grasp the opportunity to participate in the care management of deep hypothermia victims.

This lecture employs experiential based case studies involving the care of more of more than twelve hypothermic patients. Some patients were successfully revived only to be lost the next day because advice offered was refuted. Some patients survived and were discharged within forty eight hours. Yet many patients were mishandled in the first sixty minutes of discovery and therefore became a lost cause of resuscitation efforts.

This lecture will provide laboratory values unseen by typical inpatients. The lessons learned from mistakes - such as underestimation of cerebral compression—will be discussed. Likewise, the excitement of witnessing physiological phenomenon - such as sodium/potassium pump inversion - will be shared. In the end, successful resuscitation depends on initial treatment... which in most cases...should be no treatment at all until the perfusionist is ready.

# ARCH SURGERY TECHNIQUES

Dr Anand Iyer, Dept Cardiac Surgery, Townsville Hospital

Arch surgeries have been traditionally done under deep hypothermia with circulatory arrest but a lot of centres are performing arch surgeries with mild to moderate hypothermia with refined surgical and perfusion techniques. Hybrid surgeries are also being done with increasing frequency due better branched grafts. The main purpose of this topic is to present the current trends in arch surgeries which have always been a challenging subset of cases both from the perfusion and surgical side.

# TRANSIENT LEUKOPENIA IN PMEA-COATED CIRCUITS IN PEDIATRIC PERFUSION

# Hideshi ITOH, Ph.D., Associate Professor Junshin Gakuen University, Fukuoka, JAPAN

We compared the clinical effectiveness and biocompatibility of poly-2-methoxyethyl acrylate (PMEA)-coated and heparin-coated cardiopulmonary bypass (CPB) circuits in a prospective pediatric trial.

Infants randomly received heparin-coated (n=7) or PMEA-coated (n=7) circuits in elective pediatric cardiac surgery with CPB for ventricular septum defects. Clinical and hematologic variables, respiratory indices, and hemodynamic changes were analyzed perioperatively. Demographic and clinical variables were similar in both groups.

Leukocyte counts were significantly lower 5 minutes after CPB in the PMEA group than the heparin group. Hemodynamic data showed that PMEA caused hypotension within 5 minutes of CPB. The respiratory index was significantly higher immediately after CPB and 1 hour after transfer to the intensive care unit (ICU) in the PMEA group, as were levels of C-reactive protein 24 hours after transfer to the ICU.

Our study shows that PMEA-coated circuits, unlike heparin-coated circuits, cause transient leukopenia during pediatric CPB and perhaps systemic inflammatory respiratory syndrome after pediatric CPB.

# NEONATAL CARDIOPULMONARY BYPASS FOR 1ST STAGE PALLIATIVE NORWOOD-SANO PROCEDURE IN OKAYAMA UNIVERSITY HOSPITAL

# Hideshi ITOH, Ph.D. Junshin Gakuen University, Fukuoka, JAPAN Shunji SANO, MD. Ph.D. Okayama University Hospital, Fukuoka, JAPAN

We have 49,507 cardiovascular surgeries including 10,835 congenital heart surgeries in 2013 in Japan. In our unit, we have approximately 260 pediatric cardiopulmonary bypass cases. We have 21 Norwood –SANO procedures in these five years. In this report, we will focus on our isolated cerebral perfusion for 1st stage palliative Norwood-SANO procedure.

Cardiopulmonary bypass (CPB) is established by cannulation of the ductus arteriosus and the innominate artery (INNA) via 3.0 mm PTFE graft as dual arterial cannulaes, and right atrium as venous cannulae. CPB flow is commended at a flow rate 150 to 180 ml/min/kg. The branch right and left pulmonary arteries are temporarily occluded with tourniquets and systemic cooling is initiated. At a nasopharyngeal temperature of less than 22 degrees, the descending aorta is clamped. After removal of the perfusion cannulae from the duct, all duct tissue is excised from the descending aorta. The left carotid artery and left subclavian artery are snared. Isolated cerebral perfusion (ICP) and myocardial perfusion is established by placing a clamp just distal to the innominate artery. With the heart beating, aortic arch was opened inferiorly and the back wall of the descending aorta is anastomosed to the posterior wall of the aortic arch. At this stage, cold crystalloid cardioplegic solution (20ml-kg<sup>-1</sup>) was administered over 3 minutes either from the aortic root or from a side port of the arterial cannula during temporary total circulatory arrest. The innominate artery is snared proximal to the perfusion site, and the clamp on the arch is removed. CPB is resumed for ICP through the innominate artery. The perfusion flow of ICP is 30 to 50 % of full flow to maintain INNA pressure 30 to 40 mmHg. The entire aortic arch and ascending aorta are reconstructed by direct anastomosis of the proximal main pulmonary artery. A small right ventriculotomy is made in the out flow tract for proximal anastomosis of the RV-PA shunt. CPB is reinstituted, and all snares were removed before placing of RV-PA shunt. During this anastomosis, we start rewarming. After completion of the aortic reconstruction and atrial septectomy; proximal anatomosis of the RV-PA shunt is performed with the heart beating.

Cardiopulmonary bypass we use that consisted of a hollow fiber membrane oxygenator (D100; Dideco, Modena, Italy), open hard-shell reservoir (D100; Dideco, Modena, Italy), arterial filter (FT-15; JMS, Tokyo, Japan), and roller pump for perfusion (HAS II; MERA, Tokyo, Japan). CPB circuits are heparin coating and  ${}^{3}_{16}$  x  ${}^{1}_{4}$  AV loop (JMS, Tokyo, Japan) and  ${}^{1}_{4}$  pump boot. We use a Terumo CDI 500 arterial gas and venous saturation hematocrit monitor. We perform modified ultra filtration (MUF) in all cases. After MUF, we controlled the pulmonary blood flow by clipping the RV-PA shunt. Generally, we aim for systemic oxygen saturation of approximately 80% on Fi0<sub>2</sub> 0.5.

# A REPORT OF OPEN HEART SURGERY IN A PEDIATRIC PATIENT WITH HEREDITARY SPHEROCYTOSIS AND TETRALOGY OF FALLOT

# Takaya Hirayama,<sup>1</sup> Hideshi Itoh,<sup>2</sup> Takuma Douguchi,<sup>1</sup> Shingo Kasahara,<sup>1</sup> Shunji Sanol

<sup>1</sup>Department of Cardiovascular Surgery, Okayama University Hospital, Okayama, Japan <sup>2</sup>Department of Medical Engineering, Faculty of Health Sciences, Junshin Gakuen University, Fukuoka, Japan

### Introduction

We report a case of open heart surgery with a cardiopulmonary bypass (CPB) in a pediatric patient with hereditary spherocytosis (HS) and tetralogy of Fallot (TOF).

### **Case Report**

This patient was 9 months old girl and weighted 6.5 kg. We diagnosed as having TOF by using echocardiography and HS based on reticulocyte count of 19.4% and her family history. We changed the blood pump boot to 3/8 inch and the pump head to  $\phi$  150 to inhibit hemolysis. The CPB circuit was primed with 260 cc blood. We measured levels of hemoglobin (Hb), aspartate aminotransferase (AST), lactate dehydrogenase (LDH), total bilirubin (T-Bil), and potassium (K) before operation, just after congenital intensive care unit (CICU) admission, and at 3, 6, 24, and 48 hours after operation to evaluate the degree of hemolysis. The CPB times were 70 minutes, and the aortic cross-clamp time was 47 minutes. We changed the pump boot and head, which resulted in a decrease in the number from 134 rpm to 40 rpm.

### Discussion

Open heart surgery with CPB for patients with HS is associated

with a risk of perioperative hemolysis owing to the fragility of red blood cells. As shown in Table 1, the T-Bil, AST, and LDH concentrations were increased but were decreased without continuing at 6, 24, and 48 hours after the operation, respectively. We thought that we successfully inhibited hemolysis. In addition, the intubation times were 6.83 hours, and the hospital discharge of CICU was 98 hours.

Table 1. AST, LDH, T-Bil, and K levels until 48 hours after the operation

	Before operation	Just after CICU admission		6 hours after operation	24 hours after operation	48 hours after operation
Hb (g/dl)	11.1	14.5	14.7	15.0	11.8	11.3
AST (μ/Ι)	39	148	156	159	94	51
LDH (µ/I)	313	332	363	413	420	361
T- <u>Bil</u> (mg/dl)	1.66	2.64	2.71	2.67	1.81	1.02
K (mmol/l)	4.7	3.8	3.6	4.1	3.7	4.1

# HOW ACCURATE ARE THE TEMPERATURE MEASUREMENTS OF THE ARTERIAL OUTLET ON THE INSPIRE 6M OXYGENATOR?

# Cynthia Riddell, Auckland Hospital

Many studies have highlighted the potential harmful effects of hyperthermia and rapid rewarming rates on patients, particularly when it comes to the brain. Historically, Terumo luer thermistors were used in our circuits as a control measurement of arterial blood temperature as oxygenators' measurements were deemed to be unreliable. This study thus investigates the accuracy of the arterial outlet temperature measurements of the Inspire 6M Oxygenator, which is a new generation oxygenator, by comparing it to a reference temperature and also by comparing it to Terumo luer thermistors. This is done using an in vitro model, at static temperatures as well as during a rewarming sequence, and with two different temperature points being used as reference temperatures. The temperatures measured at the arterial outlet of the oxygenators were found to be read more accurately than the ones read by the Terumo luer thermistors, with a maximum error margin of  $\pm 0.2$  at static temperatures. Both oxygenators and Terumo luer thermistors under read at all temperatures during rewarming. However the margin of errors was greater in the Terumo luer thermistors. The results of the study suggest that the temperature measurements read at the arterial outlet of the Inspire 6M oxygenator are more reliable than the Terumo luer thermistor's ones, and that both under read during rewarming. It is therefore important for the perfusionist to take that into consideration in order to prevent patients from becoming hyperthermic.

# SAVE THE DATE

# 2017 AmSECT DECLARIZED AMONGALIAN September 7-9, 2017 | Miami, FL

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.** 

# COMPARATIVE STUDY BETWEEN POINT OF CARE iSTAT ANALYZER AND ABL800 FLEX RADIOMETER IN CARDIAC SETTING

# Mitchell Bago, Perfusion Services, Melbourne

**Aim:** We evaluated the use of an iSTAT point-of care analyzer in the perioperative stage of cardiopulmonary bypass and assess if we can use this point of care system interchangeably with our reference blood gas analyzer, the ABL800 flex radiometer.

**Methods:** A total of ninety subjects were enrolled in a randomized study. Fifty patients were tested on cpb mode and forty patients tested on non-cpb mode, CG8+ cartridge were used in this study. All subjects were analyzed first on iSTAT POC analyzer with PICO heparinized syringe then subsequently by ABL800 FLEX Radiometer.

**Results:** The mean bias for hemoglobin in non-cpb mode yields a value of  $-11.22 \pm 4.2$  while on cpb mode had a range of bias from  $-2.3 \pm 5.3$ , indicating more correction factor provides lesser bias on measurement, no significant difference on other parameters in terms of bias. The linear regression analysis showed a strong correlation of most parameters except for pO<sub>2</sub> in non-cpb mode (0.64). In terms of coefficient variation, the iSTAT B.E measurement showed lower values compared to ABL800 (-114.24 vs. -74.24)/ (-115.96 vs. -54.15) and hemoglobin non-cpb mode is (23.008 vs. 16.98). The limit of agreement ranges from -13.09 to 8.49 in cpb mode hemoglobin while in non-cpb mode. The limit of agreement in this study showed a wide range forhemoglobin and pO<sub>2</sub> and could potentially change the course of management either by giving blood and increasing FiO<sub>2</sub>.

**Conclusion:** The two machines did not agree on hemoglobin and  $pO_2$ , while the remaining parameters are within acceptable range and marked as comparable. We conclude that the ABL800 FLEX Radiometer as our reference bench top blood gas analyzer cannot be replaced or interchangeably use with iSTAT point of care analyzer.

# AN EVALUATION OF HAEMOGLOBIN MEASUREMENT DEVICES FOR CARDIAC SURGERY

# Casey Edwards, The Canberra Hospital

**Purpose:** Allogeneic blood transfusions expose recipients to numerous health risks but may be unavoidable as a result of haemodilution on cardiopulmonary bypass. Haemoglobin levels below an institution-specific threshold result in erythrocyte administration. It is therefore essential that haemoglobin-measuring instruments are accurate to ensure appropriate transfusion decisions. To measure haemoglobin during cardiac surgery, the Canberra Hospital has the I-stat, Hemocue and Gem4000 (which literature suggests is the most accurate device). This study was designed to investigate the variance in haemoglobin readings between these instruments to infer their suitability for use in the clinical setting.

Methods: 100 blood samples were taken from 34 patients during cardiopulmonary bypass and haemoglobin was concurrently tested on the I-stat, Hemocue and Gem4000.

**Results:** Assuming the Gem4000 to be correct, the mean bias of the I-stat was  $-4.04\mu g/dL +/-5.46$  and the Hemocue  $-0.82\mu g/dL +/-5.94$ . Given the relatively large standard deviations observed for both devices, limits of agreement indicate that a patient's observed haemoglobin must be  $81\mu g/dL$  (I-stat) or  $82\mu g/dL$  (Hemocue) for 95% certainty that true haemoglobin is above  $70\mu g/dL$ , our institutional transfusion threshold. Concurrently running samples on the I-stat and Hemocue then choosing the higher haemoglobin reading was the most accurate estimation of true haemoglobin, with a mean bias of  $0.22\mu g/dL +/-5.37$ .

**Conclusion:** When a patient's haemoglobin is expected to be below 81µg/dL, the Gem4000 was found to be the most suitable instrument to avoid inappropriate transfusions and testing duplication. In circumstances where the Gem4000 is inaccessible, concurrent i-stat and Hemocue testing with higher-value selection becomes the optimal strategy. As a result of this research, our haemoglobin measuring protocol has changed. Sample duplication has reduced so transfusion decisions can be made more quickly and the appropriate haemoglobin measurement device can be selected for individual patients.

# DOES THROMBOELASTOGR APHY INFLUENCE BLOOD PRODUCT TRANSFUSION RATES IN CARDIAC SURGERY?

# Miss Jane Ennor, Mr James McMillan and Kyriakos Angus-Anagnostou, Perfusion Services Pty Ltd, Australia

# Abstract:

**Objectives:** Several authors of previous published studies have suggested that the use of Thromboelastography (TEG) in cardiac surgery can reduce homologous transfusions of fresh frozen plasma, platelets, cryoprecipitate and packed red blood cells. After a review of previous published studies and the availability of the various systems and technology a decision was made to purchase the Haemonetics TEG® 6s, with the aim of reducing homologous transfusion rates for patients undergoing cardiopulmonary bypass. A study was constructed to test this hypothesis.

**Method:** Data on blood product usage from a single cardiac centre was collected and analysed. Two data groups, the "Pre TEG" group and "Post TEG" group were sequentially studied and compared. The Pre TEG group consisted of all cardiac patients who underwent cardiopulmonary bypass in the cardiac theatres during January 2015 – July 2015, and the post TEG group consisted of all patients undergoing cardiopulmonary bypass August 2015 – December 2015 after introduction of the TEG system. The pre-TEG group consisted of 321 patients who were empirically treated where as the post TEG group consisted of 202 patients who were treated according to the TEG 6s transfusion guidelines. The post-TEG group data collection also included a survey for the staff member initiating the TEG to further evaluate the impact of the TEG and its influence made to changes in management of a bleeding patient. Ethics approval was sought and obtained for this study.

**Results:** There was no significant difference in the volume of red blood cells (p=0.8881), fresh frozen plasma (p=0.0829), platelets (p=0.8099) or Cryoprecipitate (p=0.1856) transfusions between the two groups (see table 1.2 & figure 1.1), unadjusted for other factors. Statistical significance was not found for any of the products transfused when the pre TEG and post TEG groups were evaluated. The post TEG group survey had an 84% response rate, where two thirds of the surveyed operators felt that the TEG guidelines changed their management of the patient's coagulation status.

**Conclusion:** The introduction of the TEG 6s system did not achieve statistical significance by reducing our homologous transfusion rates following cardiopulmonary bypass. Although our findings were not as several other authors have found, the TEG system has enabled us to pin-point the technology to identify transfusion requirements to selected products. Therefore this technology should be part of the essential equipment for cardiac procedures.

### **REFERENCES:**

- 1 Sharma, A. Et al 2014, 'Does incorporation of Thromboelastography improve bleeding prediction following adult cardiac surgery?' Blood Coagulation and Fibrinolysis, vol. 25(6):561-570
- 2 Thakur, M & Ahmed, AB 2012, 'A Review of Thromboelastography', International Journal of Perioperative Ultrasound and Applied Technologies, vol. 1(1):25-29
- 3 Haemonetics 2015, TEG® 6s User Manual, P/N115191-IE, Manual Revision: AB, pdf, Haemonetics
- 4 Johnson, P & Westbrook, A 2012, 'Coagulation Mesurement and Optimisation in Cardiac Surgery, Perioperative Considerations in Cardiac Surgery, Chapter 10, Prof. C Narin (ed.)
- 5 Görlinger, K, Dirkmann, D & Hanke, AA, 2013, Potential value of transfusion protocols in cardiac surgery, Current Opinion in Anaesthesiol, vol.26(2):230-243



# AN AUDIT OF ECMO SUPPORT AT ST VINCENT'S HOSPITAL FROM JANUARY 2009 TO JULY 2016

# Adam Roshan, St Vincent's Hospital, Sydney

**Introduction:** The first successful clinical use of ECMO was in 1971 by the Surgeon Donald Hill on an adult patient suffering from acute respiratory insufficiency. In 1972 Dr Bartlett and Dr Al Gazzaniga used this device for the first time on a child in cardiogenic shock who was supported for 36 hours. St Vincent's Hospital (SVH) had its first clinical use of ECMO in 1972 on two patients with mixed result. The turning point came in the early 2000s with the improvement of technology and more reliable equipment. The ECMO program at SVH restarted in 2004 with the successful support of two patients in respiratory failure. The complexities and complications associated with ECMO are recognised as a significant cause of morbidity and mortality. It is imperative that centres practising ECMO conduct audits to ensure application of this technology are perfected, and quality of care and outcomes meet the international registry benchmarks.

**Method:** A retrospective audit was conducted on all ECMO runs at SVH between January 2009 and July 2016. Data was collected related to demographics, indication and type of ECMO, total time on ECMO, complications and survival. An analysis of the data was performed and compared with international results.

**Results:** A total of 350 ECMO applications took place during this period. There were 121 applications of V-V ECMO, 206 of V-A ECMO and 24 for right heart support (V-PA ECMO). Within in the V-A ECMO group, there were 39 applications of ECMO for cardiopulmonary arrest. ECMO insertion was carried out on patients with a diverse range of ages (12-85 years). Of these, 60% were male with a mean age of 49.1 years and 40% were females with a mean age of 41.8 years. The mean total time for ECMO support was 7.7 days (range 0.04 - 65.1 days). The longest mean duration of ECMO support by type was on V-V ECMO with 11.1 days (range 0.75 - 65.1 days), whilst V-A ECMO had the lowest time per application of 5.8 days (range 0.04 - 26.4 days). V-PA ECMO support had a mean time of 7.4 days (range 1.8 - 23 days). From the 350 ECMO applications, 78% of patients were successfully weaned off ECMO, and 62% survived to discharge. Our highest survival rate from weaning was 92% in the V-PA ECMO group. Mortality rates of 50% or greater were seen in ECMO patients developing DIC, infection, arrhythmias, haemolysis free Hb and those requiring oxygenator changeout.

**Conclusions:** In Summary, ECMO is a valuable tool with a greater application in a wider setting. With advancements in equipment, techniques and improved skills of managing ECMO has allowed us to reach previously unobtainable outcomes. Our audit illustrates that our survival rates have exceeded the world benchmark for adults during this period. Notably, this audit has illustrated areas where we need to focus our attention, one being limb ischaemia and causation of the higher rate of DIC and pneumothoraces.

# THE AUSTRALASIAN PERFUSION WORKFORCE 2016

# Arnika Van den Berg, MSc Psychology, BSc Neuroscience, Dip Grad Psychology

This presentation discusses the outcome of the 2016 Australasian Perfusion Workforce Survey, which uses principles of Organisational Psychology to better understand current and future trends of Perfusion as a profession in Australasia.

Questions addressed include the age and years of experience of practicing perfusionists, how might this impact on future training requirements, number of units that qualify as potential training sites, the average staff to caseload ratio, how many Perfusionists have local certification, and how many of the workforce are members of the ANZCP.

The second chapter of this presentation discusses key knowledge, skills, attributes, and other characteristics identified from interviews with experienced Perfusionists using the worker-oriented (KSAO) job analysis model. These findings are useful in identifying individuals more likely to be successful as Trainee Perfusionists, and attributes desired for those seeking promotion to Chief/Charge Perfusion roles.





# "One-fits-all" washing chamber

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



### Sensors

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



# Unique height adjustment of corpus

for user convenience



### Efficient data management with bar code scanner and standard USB interface

For more information please contact your Account Manager Vicki McLean m: 0400 087 839 e: vicki.mclean@fresenius-kabi.com

Fresenius Kabi Australia Pty Limited Level 2, 2 Woodland Way Mount Kuring-gai NSW 2080 Telephone: 1300 732 001 www.fresenius-kabi.com.au PM2016.128

# **FRESENIUS KABI** caring for life

CATSmart

# CARDIOPULMONARY BYPASS AND ACUTE KIDNEY INJURY: A REVIEW

Mazzone, A.L., Baker, R.A. and Gleadle, J.M.

The incidence of acute kidney injury (AKI) is a frequent and serious complication of cardiac surgery. In 2013, 95% of cardiac surgical procedures performed in Australia and New Zealand utilised cardiopulmonary bypass (CPB). Acute kidney injury following CPB is well known yet the perioperative factors contributing to its development are incompletely understood. Acute kidney injury following CPB has significant implications on both short and long term outcomes. The techniques for conducting CPB have evolved moving towards evidence-based practice however there is still no generally accepted definition of optimal perfusion and its conduct. This presentation will examine the current incidence of AKI following cardiac surgery and the short and longer-term effects of AKI on morbidity and mortality. The main focus is to discuss the perioperative risk factors related to CPB and their contribution to the development of AKI. Off pump cardiac surgery, the role of remote ischaemic preconditioning on AKI and outcomes in patients with chronic renal failure undergoing cardiac surgery will also be discussed.

# USING HYDRODYNAMICS TO MINIMIZE CIRCUIT PRIMES

# Phillip Scott, The MAYO Clinic, Rochester, USA

Many of the beliefs upheld by practicing perfusionists today encompassing circuit dynamics stem from their training as well as established "comfort" margins. But many of the beliefs upheld by clinical perfusionists today have been established upon a cracked foundation. In fact, some beliefs regarding blood damage within an extracorporeal circuit have never truly been scientifically studied. There is a new frontier regarding cardiopulmonary bypass circuit (CPB) design in an effort to minimize hemodilution and there are many opportunities for improving our practice.

This lecture will expound voids in our current belief system regarding blood hydrodynamics and discuss pertinent issues such as: proportional sizing when utilizing dual venous cannulation, the true limits for line pressures of an extracorporeal circuit without inducing blood damage, understanding critical velocities and pressure drops for varied tubing diameters, and how roller pumps are less hemolytic than centrifugal pumps. Lessons learned from neonate CPB and ECMO demonstrate that modern day adult CPB practice has considerable room for advancement amid efforts of reducing hemodilution in CPB patients to elevate the DO2 index.

# CANNULATION: A SURGEON'S PERSPECTIVE

Pankaj Saxena, FRACS, PhD., Associate Professor, James Cook University; Cardiothoracic Surgeon, The Townsville Hospital, Townsville, Australia.

**Abstract:** Cannulation for institution of cardiopulmonary bypass represents a critical part of cardiac surgery. We have presented our approach to standard cannulation for cardiac surgery and a number of variations that are encountered to manage different scenarios during different cardiac surgical procedures. We have also outlined our approach in the settings when problems occur during institution or conduct of cardiopulmonary bypass. We have emphasized the need to maintain an effective communication with the Perfusion team during any cardiac surgical operation to achieve the best outcome.

# ADDRESSING MYOCARDIAL PROTECTION AND THE TRAUMA OF CARDIAC SURGERY: A GLOBAL CHALLENGE.

# Geoffrey P Dobson PhD FAHA, Heart, Trauma and Sepsis Laboratory, College of Medicine and Dentistry, James Cook University, Townsville, Qld 4811 Australia, geoffrey.dobson@jcu.edu.au

For over 50 years cardiac surgery has been performed with extraordinary success using cardiopulmonary bypass (CPB). However, CBP is associated with a systemic inflammatory response and coagulopathy, which may be further exacerbated by cardioplegia type, ischemia-reperfusion injury, hypothermia, fluid loading and the trauma of surgery itself. The patient's response to surgery is affected by the type and duration of surgery, anesthesia, age, gender, ethnicity, pre-existing health status, medication profile, fluid therapy and post-operative pain. While surgical proficiency is generally of high standard, perioperative morbidity is an increasing problem due to an aging population, an increasing number of redo surgeries, failed angioplasties and complex corrective pediatric procedures. Currently, in adults, ~10% of CABG patients will experience reduced left ventricular function lasting days to weeks, ~25% will have post-operative atrial fibrillation, 3-16% will experience respiratory complications, up to 40% will have some form of acute renal injury, 30 to 40% will have transient cognitive dysfunction and delirium, and 2 to 3% of patients will suffer a stroke. Here we discuss the possible role of hyperkalemic cardioplegia to exacerbate the stress response, and review the new developments of a normokalemic cardioplegia comprising adenosine, lidocaine and Mg2+ (ALM), along with an intravenous ALM 'drip' that is currently being developed with the US military for catastrophic far-forward trauma. The challenge for the future in cardiac surgery is to think beyond hyperkalemic cardioplegia, and include ways to better protect the heart and whole body against the trauma of surgery.

# SHOULD ECMO BE USED IN POST CARDIAC SURGERY ELDERLY PATIENTS?

# Pankaj Saxena, FRACS, PhD, Associate Professor, James Cook University; Cardiothoracic Surgeon, The Townsville Hospital, Townsville, Australia and James Neal, CCP, Department of Perfusion, Mayo clinic, Rochester, MN, USA

**Introduction:** Complex cardiac surgery is performed more often in elderly patients in the present era as the population continues to grow older. We reviewed our experience with the use of extracorporeal membrane oxygenation (ECMO) support in patients 70 years or older following cardiac surgery.

Materials and methods: Forty five patients, 70 years or older required ECMO support following cardiac surgery over a period of 10 years (2003-2013) at the Mayo Clinic, Rochester, MN.

**Results:** There were 31 males (68.9%). The mean age was 76.8 years. Forty four patients were in cardiogenic shock preoperatively. Mean duration of ECMO support was  $103.8 \pm 74.3$  h. Twenty four patients were weaned off ECMO initially and 11 patients were discharged from the hospital. In hospital mortality was 75.6%. Post-operative complications included acute kidney injury in 30 patients (44.4%), pneumonia in 12 (26.7%) and sepsis in 11 (24.4%). Preoperative atrial fibrillation, chronic kidney injury, lactic acidosis on ECMO and persistent coagulopathy were associated with higher mortality.

**Conclusions:** Post cardiac surgery ECMO support is associated with higher morbidity and mortality in elderly patients. Further studies are required to define protocols for use of ECMO support in elderly patients.

# ADVANTAGES OF USING PULSATILE EXTRACORPOREAL MEMBRANE OXYGENATION IN EMERGENCY MEDICINE

# Hideshi ITOH, Ph.D., Associate Professor, Junshin Gakuen University, Fukuoka, JAPAN

Extracorporeal membrane oxygenation (ECMO) is well known as an effective medical support tool in emergency medicine for patients with circulatory and respiratory failure. The ELSO (Extracorporeal Life Support Organization) Registry reported in 2015, that the rate of survival among patients using ECMO is 71%, and the discharge rate for patients using ECMO is 59%. However, the rate of survival when ECMO is used for emergency cardio-pulmonary resuscitation is only 40% in neonates, 41% in pediatric patients, and 28% in adults. Outcomes of patients on ECMO have been improving; however, there is scope for further improvement in this area.

How can we improve the outcomes of patients on ECMO? The use of ECMO is associated with specific disadvantages; it leads to mismatches in preload and afterload. The management of these mismatches affects the outcomes of patients on ECMO. Furthermore, anticoagulation, durability, and biocompatibility of ECMO devices may affect the outcomes of patients on ECMO. We have studied the use of ECMO with the aim of improving outcomes of patients who use this device. We believe that an aggressive ECMO approach, improvement in the physiological hemodynamics of ECMO, development of more effective ECMO devices, and increasing hemodynamic energy may improve the outcomes of patients on ECMO.

Here, we studied the use of pulsatile ECMO, which produces more physiological hemodynamics and hemodynamic energy than non-pulsatile ECMO. Moreover, pulsatile ECMO improves microcirculation in patients. Increase in hemodynamic energy by using pulsatile ECMO leads to an increase in mechanical shear stress and stimulates endothelial cells. This reduces peripheral vascular resistance, improves microcirculation as a result of nitric oxide secretion from the endothelium, and prevents platelets aggregation. For these reasons, pulsatile ECMO may improve outcomes of patients on ECMO in emergency medicine.

# MANAGING BLEEDING ON ECMO

# Phillip Scott, The MAYO Clinic, Rochester, USA

Without doubt, the most challenging struggle for medical professionals caring for ECMO patients is coagulation management. Too much anticoagulation results in bleeding which further attenuates the rise of plasma antibodies via blood product administration. When the panel reactive antibody (PRA) number becomes too large, an ECMO patient may no longer be eligible for transplant. Yet, insufficient suppression of coagulation results in fibrin deposition of the circuit and product consumption by the patient (which also increases blood product administration). Balancing the scales between these two extremes often proves difficult.

This lecture will offer insight stemming from recent alterations of coagulation management in our mechanical assist device program at Mayo Clinic Rochester. The realization that heparin by itself can be detrimental to an ECMO patient has given cause for altering our strategies of anticoagulation management. Platelet suppression is becoming a focus and the awareness that insufficient anticoagulation oftentimes causes bleeding is real. Great benefit has stemmed from categorizing and labelling patients of differing etiologies and pairing them with vastly different coagulation management strategies.

The systematic gain from better anticoagulation management of these patients is exponential. This lecture will discuss topics such as why extrinsic cascade monitoring cannot be ignored on ECMO, why sepsis patients on ECMO must be aggressively anticoagulated, and why liver failure patients are so different from those with a healthy liver metabolism. Long term device patients are here to stay, and ten-month VA ECMO runs are as real as patients who present for a heart transplant having lived on ventricular assist for six years. The management of bleeding during the early days of ECMO proves critical for minimizing blood product administration and maintaining lower PRA's for patients awaiting transplant.



# 2017 AmSECT quality&outcomes October 18-21, 2017 | Portland, OR



# *NO? YES! NITRIC OXIDE AND* CARDIOPULMONARY BYPASS.

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

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 presentation will review where NO comes from, and the mechanisms involved in these therapeutic effects. I will report 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 Circulation this year.

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, 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.

# AVOIDING SUDDEN ONSET CATASTROPHIC THROMBOSIS THROUGH PROTAMINE AND PLATELET ADMINISTRATION

# Phillip Scott, The MAYO Clinic, Rochester, USA

This lecture will discuss a low-frequency high-risk event which many perfusionists will recall having occurred at least once in their career. Sudden catastrophic thrombosis occurs within minutes, is irreversible and results in death. Yet these tragedies are completely avoidable.

This lecture uses empirical case studies involving the care of more than twenty patients experiencing fatal catastrophic sudden thrombosis. Though this phenomenon may also occur to a lesser degree, the results still prove devastating. It is the desire of this author to share accumulated experiences from multiple centers that have lost patients because of the administration of foreign platelets shortly after protamine administration. Given the proper ingredients for a perfect storm, activated native platelets appear to aggressively interact with the foreign platelets causing an instantaneous and complete consumptive thrombotic event.

This lecture will share laboratory values and demonstrate the complete consumption of clotting agents which occurs within minutes. The thrombus formed through this process is systemic and irreversible. It is the goal of this lecture to raise awareness of this catastrophic syndrome and offer tips to avoid its occurrence.

# LEVELS OF CIRCULATING AND URINARΥ MICRORNAS ARE INCREASED DURING CARDIAC SURGERY WITH CARDIOPULMONARY BYPASS

# Mazzone, A.L., Baker, R.A., McNicholas, K., Michael, M.Z. and Gleadle, J.M.

**Purpose:** To investigate if levels of hypoxically regulated microRNAs miR-210 increase in blood and urine of patients undergoing cardiac surgery. This pilot study is the first clinical evaluation of circulating levels of miRNAs in patients during and after cardiac surgery with cardiopulmonary bypass.

**Methods:** Serial blood and urine samples were taken from patients undergoing cardiac surgery with cardiopulmonary bypass (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 reverse transcription polymerase chain reaction. Levels of plasma free haemoglobin, troponin T, creatine kinase and creatinine were measured.

**Results:** Serum levels of miR-210 and miR-16 were significantly elevated compared to preoperative levels in patients undergoing cardiac surgery with cardiopulmonary bypass during the perioperative (pre vs CPB p< 0.05, pre vs rewarming p<0.05) and postoperative periods (p=0.09). There was an increase of greater than 200% in miR-210 levels and 3000% in miR-16 levels in urine when normalised to urinary creatinine concentration. Serum levels of miR-16 were relatively constant during off pump surgery, though miR-210 levels significantly increased in off pump patients perioperatively (P<0.05). A significant correlation was seen between levels of miR-16 and miR-210 and plasma free haemoglobin (plasma free haemoglobin vs miR-16 (r=-0.501, p <0.0001), or vs miR-210 (r=-0.513, p<0.0001).

**Conclusions:** Serum and urine concentrations of miR-210 and miR-16 increase in cardiac surgery utilising cardiopulmonary bypass when compared to off pump surgery. The correlation with plasma free haemoglobin is consistent with microRNA release due to haemolysis and appears to partially account for the release profiles, however release of miR-210 from other hypoxic tissues seems likely. These molecules may have utility in indicating severity of cardiac, red cell and renal injury during cardiac surgery.

# WHAT IS THE EFFECT OF REMOVING FRUSEMIDE FROM CPB PRIME?

# Keith Adkins, St Vincent's Hospital, Sydney

The aim of this study is to investigate the role of routine use of frusemide on cardiopulmonary bypass, especially focusing on the perioperative period and the immediate post-operative period.

The end points of this study are fluid balance, Rifle-R rate, transfusion rate, and ventilation and ICU time.

There will be two groups; a frusemide and a non-frusemide group. There will be 50 patients in each group. The patients will be undergoing CABG surgery only. Only 1st time sternotomy patients will be assessed.

# *IS PLASMA LACTATE A PREDICTOR OF* OUTCOME FOR PAEDIATRIC VA ECMO?

# Clarke Thuys, The Royal Children's Hospital, 50 Flemington Road, Parkville, Victoria 3052 Australia

This retrospective study sought to determine whether the peak lactate level or the lactate level at 12 hours, 24 hours or 48 hours after initiation of VA ECMO was a predictor of outcome for any paediatric patient. Outcomes were survival or non-survival to hospital discharge. Lactate values were stratified as: Normal lactate  $\leq 2 \text{ mmol/L}$ , intermediate lactate  $\geq 2 < 4 \text{ mmol/L}$  and high lactate  $\geq 4 \text{ mmol/L}$ .

Analysis of data from 267 patients supported with VA ECMO between 2005 and 2012 showed that 70.0% had a high level peak lactate, 17.3% had a peak lactate at the intermediate level while only 12.7% had a peak lactate that was considered normal.

There were no significant differences in outcome across the lactate groups for peak levels and at the 48 hour time point. At 12 hours and 24 hours the normal lactate group had a significantly better rate of survival (70%, 70%) to discharge than the intermediate (55%, 49%) and high lactate level groups (43%, 37%).

Stabilisation of lactate level at normal or intermediate, or movement from high to intermediate level, high to normal and intermediate to normal showed better rates of survival at the 12 and 24 hour time points than the high lactate groups.

Peak lactate and lactate at 48 hours do not appear to be predictive of outcome. Lactate levels at 12 and 24 hour points may give some insight into the expected outcome. Rate of lactate clearance at 12 and 24 time points may also provide predictive information about outcome.

Keywords: ECMO, Lactate, Outcome

# SHORT TERM FOLLOW UP OF LOW EJECTION FRACTION PATIENTS IN TOWNSVILLE

# Dr Anand Iyer, Dept Cardiac Surgery, The Townsville Hospital

**Purpose:** Patients with poor ejection fraction are often considered high risk candidates for cardiac surgery and require extensive pre-op multidisciplinary planning as they are often associated with increased morbidity and mortality.

**Methodology:** A retrospective audit was conducted on all patients with an ejection fraction (EF) of 30% or less undergoing cardiac surgery in Townsville between 2014 and 2015. Information was collected regarding: patient demographics; pre-op morbidities; EF; surgical details; use of Levosimendan and current patient status at most recent follow up (6 or 12 months post op).

**Results:** There were 25 patients, 84% male and 16% female, with a mean F of 26.3 (standard deviation (sd) of 8.0) and a mean age of 58.6 (SD 11.7). Hypertension (64%) and smoking (80%) were the most prevalent risk factors amongst the group. 64% presented urgently for surgery with 32% having had a STEMI. 92% had CABG or CABG with valve surgery. One patient had an AVR and one a Bentalls procedure. Pre-operative optimisation included the use of IABP (64%) and Levosimendan (36%). There was a 4% mortality rate at follow up and 72% had a post-operative echo, of which 76% showed an improvement of EF. The average improvement was 14% (SD 7.74%). 36% of these had IABPs, 36% had IABP and Levosimendan and 28% had neither. Of those, the patients who had both IABP and Levosimendan had the greatest increase in ejection fraction at follow up (17% as opposed to 13%) although this was not a statistically significant difference.

**Conclusion:** With more options available for stenting, an increasing number of low EF patients are presenting for surgery. Consideration of IABP or use of Levosimendan in conjunction with other pre-operative management may increase EF in short term follow up and could be improve morbidity for this group of high risk surgical patients.

# INTRAOPERATIVE DYSGLYCEMIA: PERFUSION REGISTRY DATA ON INCIDENCE, MANAGEMENT & POSTOPERATIVE OUTCOMES.

# Andrew Sanderson BSc CCP<sup>+</sup>, Richard Newland BSc CCP<sup>+</sup>, Rob Baker PhD CCP<sup>+</sup>.

<sup>†</sup>Ashford Hospital, <sup>\*</sup>Flinders Medical Centre and Flinders University, on behalf of the Australian and New Zealand Collaborative Perfusion Registry.

**Purpose:** In this report we aim to provide some insight into the prevalence of dysglycemia preoperatively and during cardiopulmonary bypass (CPB), examine potential mediators and the outcomes of clinical management.

Methods: Data collected in 24,495 cardiac surgical procedures using CPB from March 2007 – February 2016 from 9 centres in the Australian and New Zealand Collaborative Perfusion Registry (ANZCPR) was utilised for this report.

**Results:** Overall incidence of dysglycemia as defined by preoperative BGL >7.8 mmol/l was 17%, preoperative HbA1c >7 % was 21%, and diagnosis of diabetes was 27%. Incidence of CPB hypoglycaemia in the dataset is 0.9%. Quality improvement initiatives through benchmarking have improved maintaining CPB glucose between 4 - 10 mmol/l from 70% in 2010 to 81% in 2015. In patients with intraoperative hyperglycaemia, higher rates of mortality are seen in patients with diabetes or elevated preoperative glucose, but not elevated HbA1c.

**Conclusions:** Collection and reporting of variables related to dysglycaemia in the ANZCPR provides valuable insight into the incidence and intraoperative management. Comparing practice data provides a mechanism for process improvement. Further evaluation of the association between HbAlc and outcome is warranted, to identify improved management strategies.

# THE ROLE OF LACTATE MEASUREMENT IN PERFUSION THERAPY.

# Dr Adam Holyoak, Consultant Intensivist and Emergency Physician, The Townsville Hospital

Blood lactate levels during cardiopulmonary bypass have been used to determine adequacy of perfusion during cardiac surgical procedures. However, it has also been noted by some that cardiopulmonary bypass itself may alter lactate metabolism resulting in increased serum levels. Associations between increased serum lactate levels, bypass haematocrit and the amount of blood transfusion required intraoperatively have also been observed. Certainly, higher lactate levels post bypass have been associated with increased morbidity and mortality. The utility of the measured lactate level during cardiopulmonary bypass will be discussed. Given the multiplicity of parameters that may affect the lactate value, appropriate strategies to improve perfusion and patient management techniques intra-operatively need to be determined.

The next Annual Scientific Meeting will be held in Melbourne in 2017

# CALENDAR of EVENTS

### JANUARY 2017

8-11
14th Annual Winter Park Perfusion Conference
Vintage Hotel
Winter Park, Colorado, USA
http://www.hatravel.com/Page/WPPC2017MainPage

### 19-22

38th Annual Seminar of The American Academy of Cardiovascular Perfusion The Westin San Diego Hotel San Diego, California, USA http://www.theaacp.com/annual-meeting/

### FEBRUARY 2017

22-26

Cardiology 2017-Pediatric Cardiac Surgery Disney's Yacht and Beach Club Orlando, Florida, USA www.chop.edu/cardiology2017

### 23-MARCH 2017

33nd Annual Children's National Symposium: ECMO and the Advanced Therapies for Respiratory Failure Keystone Resort Keystone, Colorado, USA http://www.cvent.com/d/1fqxtc

### **APRIL 2017**

**5-8** Sanibel Symposium Sanibel Harbor Resort and Spa Fort Myers, Florida, USA http://www.perfusion.com/symposium/

### April 29-May 2

AmSECT 55th International Conference in Collaboration with American Association for Thoracic Surgery Boston Hynes Convention Center and Sheraton Hotel Boston, Massachusetts USA http://www.amsect.org/p/cm/ld/fid=1424

### **MAY 2017**

5-6 ANZCP 3rd Intraoperative Management of Blood Amora Hotel Sydney, NSW

### **JUNE 2017**

14-17 17th European Congress on Extracorporeal Circulation Technology Marseille, France http://fecect.org/

### 21-24

63rd International Conference for the American Society of Artificial Internal Organs Chicago, Illinois USA https://asaio.com/annual-conference/chi-2017-63rd-annual-conference/

### **JULY 2017**

16-21

7th World Congress of Pediatric Cardiology and Cardiac Surgery Centre Convencions Internacional de Barcelona Barcelona, Spain http://wcpccs2017.org/en/

### SEPTEMBER 2017

**7-9** AmSECT Pediatric and Congenitcal Perfusoin Committee Conference Epic Hotel Miami, Florida USA

### 7-9

Case Reports in the Sun XIII PGA National Golf Report and Spa Palm Beach Gardens, Florida, USA http://www.floridaperfusion.org/latest-news/case-reports-in-the-sun-x/

### Sept 28-October 1

37th Annual International Cardiothoracic Surgery Symposium Westin San Diego San Diego, California USA www.crefmeeting.com

### OCTOBER 2017

17-21 AmSECT Quality and Outcomes Portland, Oregon

### 21-23

The Canadian Society of Clinical Perfusion National Meeting Toronto, Ontario

# SAVE THE DATES

# 17-19TH AUGUST 2017

PERFUSION DOWNUNDER



Novotel - Barossa Valley