AUCKLAND CITY
SYMPOSIUM

AUCKLAND CITY SYMPOSIUM

Fluids, fluids everywhere, but which one should we use?

New Directions in Perioperative Fluid Management

Programme and Abstracts

Saturday 14th March 2015

School of Medicine, The University of Auckland, New Zealand

www.acs.ac.nz

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Welcome to Auckland City Symposium 2015

Dear Colleagues,

Welcome to Auckland City Symposium 2015. This year we are focusing on perioperative fluid management with the theme: Fluids, fluids everywhere, but which one should we use?

In addition, we will review tools for haemodynamic monitoring, ERAS for colorectal surgery and goal directed fluid therapy.

Our keynote speakers are Professor Andrew Shaw and Professor Dileep Lobo.

Prof. Shaw is Chief of Cardiothoracic Anesthesiology at Vanderbilt University Medical Center in Nashville, Tennessee. His clinical practice is cardiothoracic anesthesia and surgical critical care medicine. His clinical research interests include observational and interventional trials of candidate interventions to prevent and treat adverse outcomes following surgery.

Prof. Lobo is Professor of Gastrointestinal Surgery and Consultant Hepatopancreaticobiliary Surgeon at Queen's Medical Centre, Nottingham, UK. His clinical interests focus on surgery of the pancreas and biliary tree and laparoscopic surgery. His research interests include surgical nutrition and metabolism, fluid and electrolyte balance and pancreatic cancer.

Our international speakers will be joined by our excellent local faculty and we would like to acknowledge their hard work and commitment to our meeting.

Thanks to all our sponsors for ACS 2015 with a special acknowledgment of the generous support of our platinum sponsor, Baxter. Please visit their booths during the breaks.

This year we are transitioning over to a new website and registration process. There have been some technical issues for some delegates; thank you for your patience and we hope the process will be smoother next time. Karen Patching has continued to help with all the details required to host a successful meeting and we would like to thank her for her effort.

To our delegates, thank you for your support and we trust you will enjoy the day.

Kind regards,

Dr Kerry Gunn
Co-convenor

Dr Neil MacLennan
Co-convenor

PROGRAMME

Saturday, 14th March 2015

0730	**Registration Desk Open** –	Exhibitor Area, Atrium, School of Medicine
0800 - 0810	Welcome and introduction	***Kerry Gunn***
SESSION 1	**The trouble with fluid(s)**	
0810 - 0840	Finding the right balance in crystalloid composition	***Andrew Shaw***
0840 - 0910	Perioperative renal function and fluid therapy	***Jevon Puckett***
0910 - 0940	A brief history of colloids in fluid therapy	***Shay McGuiness***
0940 - 1010	**Morning Break –**	Exhibitor Area, Atrium, School of Medicine
SESSION 2	**New approaches to fluid management**	
1010 - 1040	Fluid management guidelines (NICE/GIFTASUP)	***Dileep Lobo***
1040 - 1110	How I give fluid intraoperatively...	***Andrew Shaw***
1110 - 1140	Tools for intraoperative haemodynamic monitoring	***Martin Misur***
1140 - 1240	**Lunch Break –**	Exhibitor Area, Atrium, School of Medicine
SESSION 3	**All about ERAS**	
1240 - 1310	ERAS for colorectal surgery; an integrated approach	***Dileep Lobo***
1310 - 1340	GDT: a critical appraisal of the literature	***Matt Taylor***
1340 - 1410	What are the essential components of a colorectal ERAS programme? (Panel Discussion)	
1410 - 1440	**Afternoon Break –**	Exhibitor Area, Atrium, School of Medicine
SESSION 4	**Putting it all together**	
1440 - 1510	The dilemma of the transfusion threshold	***Chang Kim***
1510 - 1540	What makes fluids act like something of substance?	***Kerry Gunn***
	Case presentations and case vignettes (Panel Discussion)	
	Future Meetings	
1700	Meeting concludes	
1700 - 1800	**Drinks and Nibbles –**	Exhibitor Area, Atrium, School of Medicine

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UPDATE IN ANAESTHESIA
Annual Queenstown Update in Anaesthesia
August 20-22, 2015
KEYNOTE SPEAKER:
Prof Kate Leslie
Head of Research,
Royal Melbourne Hospital
Dept of Anaesthesia and
Pain Management
VENUE:
Heritage Hotel, Queenstown,
New Zealand
www.aqua.ac.nz
NZSA
SALUS PER SCIENTIAM


BATS on Ice
BETTER ANAESTHESIA
THROUGH SONOGRAPHY
A one-day ultrasound regional anaesthesia workshop
Thursday, 20 August 2015
Heritage Hotel, Queenstown, New Zealand
www.bats.ac.nz

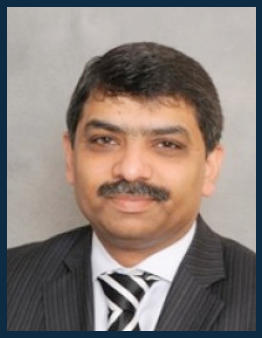
SPEAKERS

We are proud to announce the following Keynote speakers for Auckland City Symposium 2015.

Prof Andrew Shaw

Vanderbilt University, Tennessee, USA

Dr Shaw is Professor and Chief, Cardiothoracic Anesthesiology at Vanderbilt University Medical Center in Nashville, Tennessee. His clinical practice is cardiothoracic anesthesia and surgical critical care medicine. His translational research interests include the use of metabolomics, proteomics and genomics for biomarker discovery in acute illness and injury; his clinical research interests include observational and interventional trials of candidate interventions to prevent and treat adverse outcomes following surgery.



Prof Dileep Lobo

University of Nottingham, UK

Dileep N Lobo, MB BS, MS, DM, FRCS, FACS, FRCPE is Professor of Gastrointestinal Surgery and Consultant Hepatopancreaticobiliary Surgeon at Queen's Medical Centre, Nottingham, UK. His clinical interests focus on surgery of the pancreas and biliary tree and laparoscopic surgery. His research interests include surgical nutrition and metabolism, fluid and electrolyte balance and pancreatic cancer. Dileep is the Surgical Lead for the Nottingham Digestive Diseases Centre NIHR Biomedical Research Unit.

LOCAL FACULTY

Jevon Puckett — *Fellow, Department of Surgery, University of Auckland*

Shay McGuinness — *Specialist Anaesthetist, Auckland City Hospital*

Martin Misur — *Specialist Anaesthetist, Auckland City Hospital*

Matt Taylor — *Specialist Anaesthetist, Middlemore Hospital*

Chang Kim — *Fellow, Department of Anaesthesia, Auckland City Hospital*

Kerry Gunn — *Specialist Anaesthetist, Auckland City Hospital*

Crystalloids, colloids and the appeal for balanced solutions. What's the evidence and does it really matter?

Prof Andrew Shaw
Division of Cardiotoracic Anesthesiology, Tennessee, USA

Crystalloids and the appeal for balanced solutions. What's the evidence and does it really matter?

Andrew Shaw

MB FRCA FCCM FFICM

Professor and Chief

Cardiothoracic Anesthesiology

Vanderbilt University Medical Center

VANDERBILT UNIVERSITY MEDICAL CENTER

Vanderbilt University

Dr Robert Lewins

DR. LEWINS ON INJECTION OF SALINE SALTS INTO THE VEINS. 257

INJECTION
OF
SALINE SOLUTIONS INTO THE VEINS,
Adopted with success in Malignant Cholera.

[We have been favoured with the following curious and interesting document, addressed to the Central Board of Health.]

Sir,—I conceive it to be my duty to let you know, for the information of the Central Board of Health, that the great desideratum of restoring the natural current to the veins and arteries, of improving the colour of the blood, and recovering the function of the lungs in cholera asphyxia, may be accomplished by injecting a weak saline solution into the veins of the patient. To Dr. Thomas Latta, of this place, is due the merit of first having recourse to this practice. He has tried it in six cases; three of which I have seen, and assisted to treat. The most wonderful and satisfactory effect is the immediate consequence of the injection. To produce the effect referred to, a large quantity must be injected, from *five to ten pounds* in an adult, and repeated at longer or shorter intervals, as the state of the pulse, and other symptoms, may indicate. Whenever the pulse fails, more fluid ought to be thrown in to produce an effect upon it, without regard to quantity. In one of the cases I have referred to, 120 *ounces* were injected at once, and repeated to the amount of 330 ounces in 12 hours. In another, 376 ounces were thrown into the veins between Sunday, at 11 o'clock A.M. and this day, (Tuesday) at 4 P.M.; that is, in the course of 53 hours, upwards of 31 pounds!

The solution that was used consisted of two drachms of muriate, and two scruples of carbonate of soda, to sixty ounces of water. It was at the temperature of 108 or 110°.

The apparatus employed for injecting was merely one of Read's common syringes, (the fluid being put into a vessel rather deep and narrow) with a small pipe fitted, that it might easily be introduced into an incision in the veins of the usual size that is made in bleeding. It may, however, be well to keep in mind that in the event of the operation being frequently repeated, it may be advisable to inject by different veins.

I forbear at present to enter further into the particulars, nor have we had sufficient experience to speak decisively on the subject. I may, however, mention that the idea of having recourse to this remedy in cholera occurred to Dr. Latta, from being convinced (which I am also) that the evacuations upwards and downwards are in reality the serum of the blood; that it is the duty of the physician to replace it as speedily as possible by injecting a fluid, as similar to the serum as can be formed artificially, directly into the veins, which has been done here with wonderful, and so far as we can yet judge, excellent effect. An immediate return of the pulse, an improvement in the respiration and in the voice, an evolution of heat, an improvement in the appearance of the patient, with a feeling of comfort, are the immediate effects. The quantity necessary to be injected will probably be found to depend upon the quantity of serum lost, the object of the practice being to place the patient in nearly his ordinary state as to the quantity of blood circulating in the vessels.

I have, &c.
(Signed) ROBERT LEWINS, M.D.
Fellow of the Royal College of Physicians, and Member of the Leith Board of Health.

Leith, 6, Quality-Street, May 15, 1832.

To W. Maclean, Esq.
Secretary to the Central Board of Health.

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" Whenever the pulse fails, more fluid ought to be thrown in, without regard to quantity"

"The most wonderful and satisfactory effect is the immediate consequence of the injection."

"The quantity necessary to be injected will probably be found to depend upon the quantity of serum lost."

"The solution that was used consisted of two drachms of muriate, and two scruples of carbonate of soda to sixty ounces of water."

Lewins: London Medical Gazette 1832

Lancet editorial 1832

384 THE CASES OF CHOLERA SUCCESSFULLY TREATED

THE LANCET.

London, Saturday, June 2nd, 1832.

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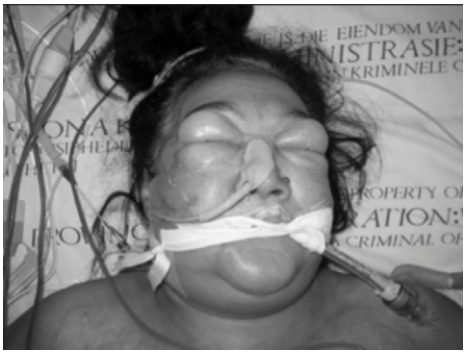
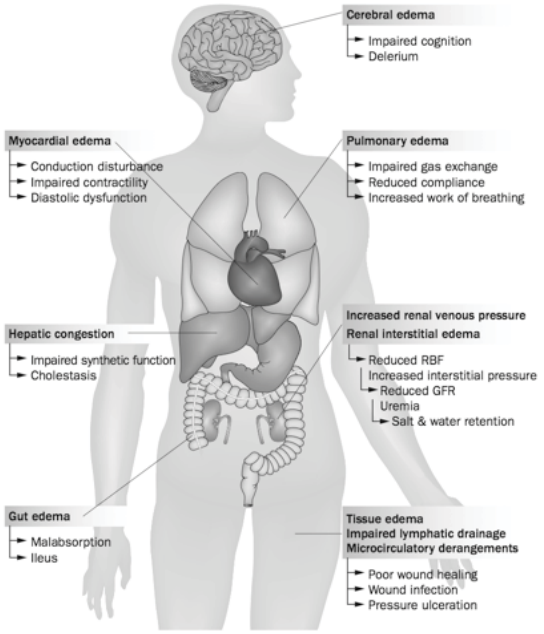
"A suitable clinical investigation is required… the mass of the profession is unable to decide; and thus, instead of any uniform mode of treatment, every town and village has its different system or systems, while the daily lists of mortality proclaim the general inefficiency of the whole."

Lancet 1832

Is fluid *amount* important ?



Organ function effects of volume overload



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Outcome benefits of fluid restriction

Reference	Study type	Population	*n*	Average fluid balance in less-positive group	Average fluid balance in more-positive group	Renal function measure	Renal outcome with more-restrictive fluid balance strategy	Principal outcome with more-restrictive fluid balance strategy
ARDS Clinical Trials Network (2006)[88]	Multicenter RCT	ARDS	1,000	−136 ml on day 7	+6,992 ml on day 7	Need for RRT; change in creatinine	No difference	Shorter duration of ventilation and ICU stay
Martin *et al.* (2005)[86]	Single-center RCT	Mixed ALI	40	−5,480 ml on day 5	−1,490 ml on day 5	Change in creatinine	No difference	Improved oxygenation
Martin *et al.* (2002)[85]	Single-center RCT	ALI after trauma	37	−3,300 ml on day 5	+500 ml on day 5	Change in creatinine	No difference	Improved oxygenation
Mitchell *et al.* (1992)[127]	Single-center RCT	Mixed ICU needing PAC	102	+142 ml	+2,239 ml	Change in creatinine	Small rise in creatinine	Shorter duration of ventilation and ICU stay
Bouchard *et al.* (2009)[25]	Retrospective observational	Mixed ICU with AKI	542	<10% rise	>10% rise	Dialysis independence	Improved	Decrease in mortality
Payen *et al.* (2008)[6]	Retrospective observational	Mixed ICU with or without AKI	3,147	−1,000 ml	+3,000 ml	Renal SOFA score	Improved	Decrease in mortality in patients with AKI
Vidal *et al.* (2008)[72]	Prospective observational	Mixed ICU with elevated or normal IAP	83	+5,000 ml	+9,000 ml	Renal SOFA score	Improved	Normal IAP associated with less organ failure and shorter ICU stay
Adesanya *et al.* (2008)[128]	Retrospective observational	Surgical ICU	41	+5 kg	+8.3 kg	Change in creatinine	No difference	Shorter duration of ventilation and ICU stay
McArdle *et al.* (2007)[87]	Retrospective observational	Surgical ICU	100	+7,500 ml	+10,000 ml	Change in creatinine	No difference	Decrease in postoperative complications
Arlati *et al.* (2007)[99]	Prospective observational	Burns ICU	24	+7,500 ml	+12,000 ml	Urine output	No difference	Decrease in organ dysfunction score

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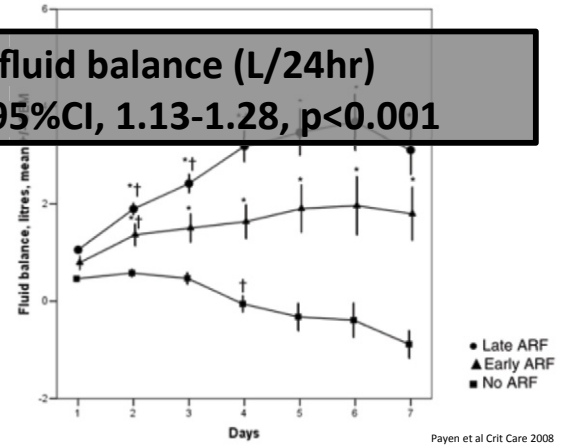
Prowle, J. R. *et al. Nat. Rev. Nephrol.* **6**, 107–115 (2010)

Research **Open Access**

A positive fluid balance is associated with a worse outcome in patients with acute renal failure

Didier Payen[1], Anne Cornélie de Pont[2], Yasser Sakr[3], Claudia Spies[4], Konrad Reinhart[3], Jean Louis Vincent[5] for the Sepsis Occurrence in Acutely Ill Patients (SOAP) Investigators

Mean fluid balance (L/24hr)
HR 1.21, 95%CI, 1.13-1.28, p<0.001



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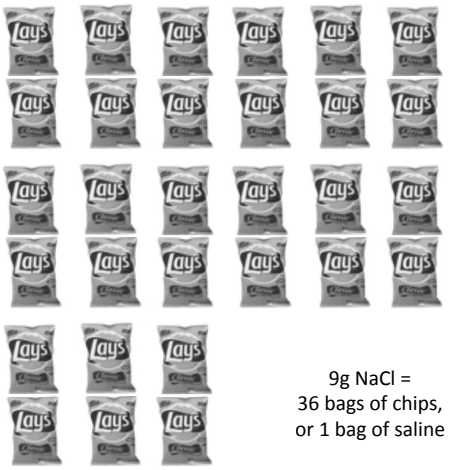
Payen et al Crit Care 2008

Is fluid *amount* important ?

- **Excess fluid leads to adverse outcomes**
- **When fluid given is blinded – the ratio of crystalloid : colloid is generally 1.3 : 1**
- **NOT 3:1 as is widely believed**
- **Why are fluids not afforded the same respect as other intravenous drugs?**

Is *crystalloid type* important ?

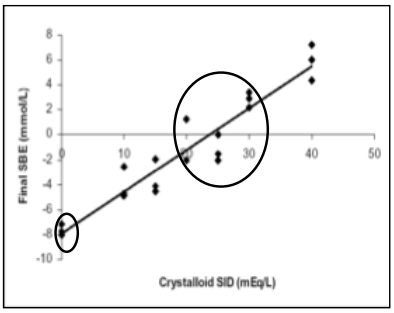


9g NaCl =
36 bags of chips,
or 1 bag of saline

Fluid Therapy Basics

Not all IV Fluids are created equal…

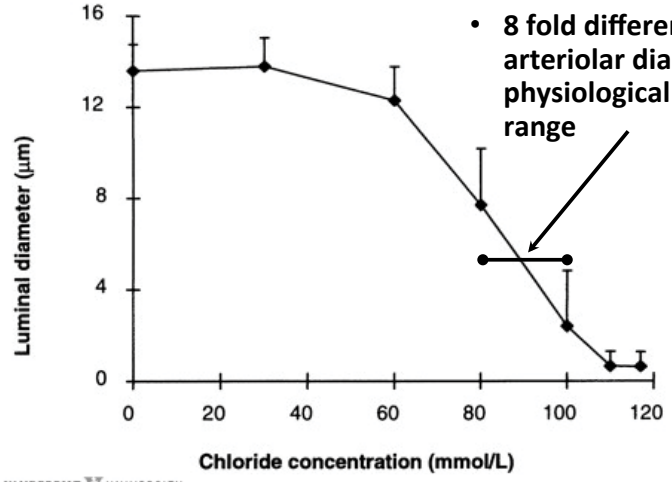
- **A "balanced" fluid has the physiological electrolyte composition of plasma**
- **Balanced fluids do not cause the hyperchloremic acidosis associated with 0.9% saline**



•Base excess after infusion is determined by the strong ion difference (SID) of the fluid infused.

•The red circle represents 0.9% NaCl, the blue circle represents balanced crystalloid

The importance of chloride: arteriolar vasoconstriction



- 8 fold difference in arteriolar diameter within physiological chloride range

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Hansen et al, Hypertension 1998; 32:1066–1070

Abnormal Saline

- 0.9% saline contains Na and Cl in equal amounts (154 meq/l)
- Unlike plasma
- Adding NaCl to plasma increases the relative Cl concentration more than that of Na
- 0.9% saline reduces plasma SID and leads to hyperchloremic metabolic acidosis

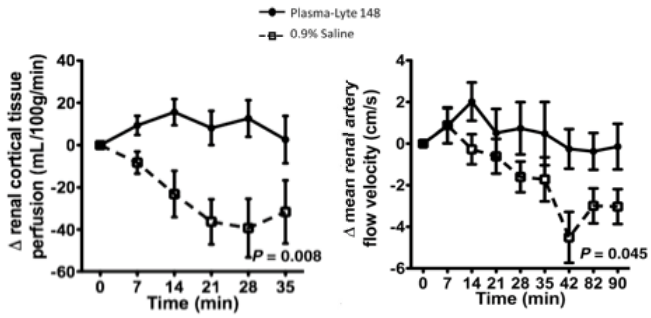
The Abuse of Normal Salt Solution

George H. Evans, JAMA 1911

"One cannot fail to be impressed with the danger…(of) the utter recklessness with which salt solution is frequently prescribed, particularly in the postoperative period…"

"…the disastrous role played by the salt solution is often lost in light of the serious conditions that call forth its use."

2L of Saline versus Balanced Crystalloid in Healthy Volunteers



Chowdhury et al (2012) Ann Surg

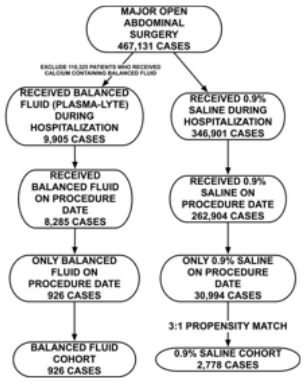
FEATURE

Major Complications, Mortality, and Resource Utilization After Open Abdominal Surgery

0.9% Saline Compared to Plasma-Lyte

Andrew D. Shaw, MB, FRCA, FCCM, Sean M. Bagshaw, MD,† Stuart L. Goldstein, MD,‡ Lynette A. Scherer, MD,§ Michael Duan, MS,‖ Carol R. Schermer, MD,¶ and John A. Kellum, MD#*

- **Retrospective analysis of a prospectively collected data asset (Premier database)**
- **Major (non cardiac) surgery**
- **>30,000 patients who received 0.9% saline or balanced crystalloid alone on day of surgery**



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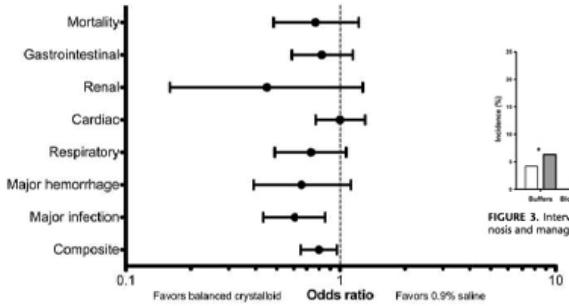
(*Ann Surg* 2012;00:1–9)

Risk adjusted major complications and resource use - All patients



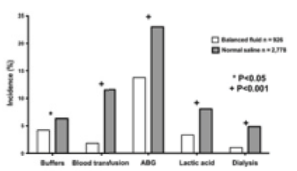


FIGURE 3. Interventions related to metaboloic acidosis diagnosis and management.

Shaw et al (2012) Ann Surg

Risk adjusted outcomes non-elective surgery

(NNT 29)

Outcome	Balanced	Saline	P value
n	296	20,047	-
All cause mortality (%)	3.7	7.2	0.019
Major morbidity (%)	36	41	0.01
Minor morbidity (%)	35	42	0.01
Post op ventilation (%)	13.2	18.7	0.02
Discharged home (%)	86	78	7×10^{-6}
30 day readmit rate (%)	30	31	NS

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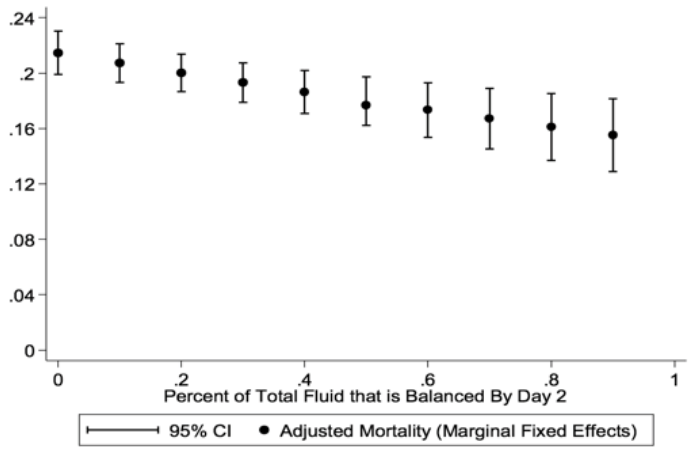
Shaw et al (2012) Ann Surg

Association Between the Choice of IV Crystalloid and In-Hospital Mortality Among Critically Ill Adults With Sepsis

Karthik Raghunathan, MD, MPH[1,2]; Andrew Shaw, MB, FRCA, FFICM, FCCM[1]; Brian Nathanson, PhD[3]; Til Stürmer, MD, PhD[4]; Alan Brookhart, PhD[4]; Mihaela S. Stefan, MD[5]; Soko Setoguchi, MD, DrPH[6]; Chris Beadles, MD, PhD[2]; Peter K. Lindenauer, MD, MSc[7]

Mortality Dose Response



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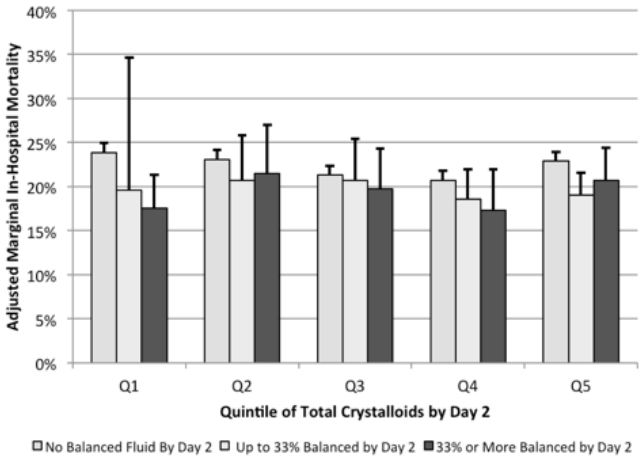
Raghunathan et al (2014) Crit Care Med

Total Volume Received Dose Response



Yunos et al 2012

PRELIMINARY COMMUNICATION

Association Between a Chloride-Liberal vs Chloride-Restrictive Intravenous Fluid Administration Strategy and Kidney Injury in Critically Ill Adults

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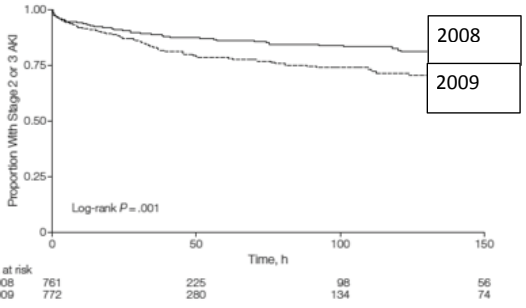
Yunos et al (2012) JAMA

KDIGO 2&3

Figure 1. Development of Stage 2 or 3 Acute Kidney Injury (AKI) While in the Intensive Care Unit (ICU)



Stage 2 or 3 defined according to the Kidney Disease: Improving Global Outcomes clinical practice guideline.

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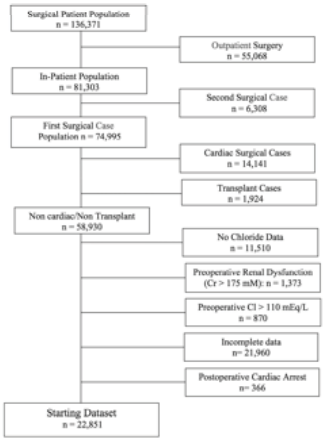
Yunos et al (2012) JAMA

Hyperchloremia After Noncardiac Surgery Is Independently Associated with Increased Morbidity and Mortality: A Propensity-Matched Cohort Study

Stuart A. McCluskey, PhD, MD,* Keyvan Karkouti, MSc, MD,*† Duminda Wijeysundera, PhD, MD,* Leonid Minkovich, PhD, MD,* Gordon Tait, PhD,* and W. Scott Beattie, PhD, MD*

- **Observational cohort study**
- **Major (non cardiac) surgery**
- **23000 patients**
- **4266 of 4955 who developed high serum chloride propensity matched to patients who did not**



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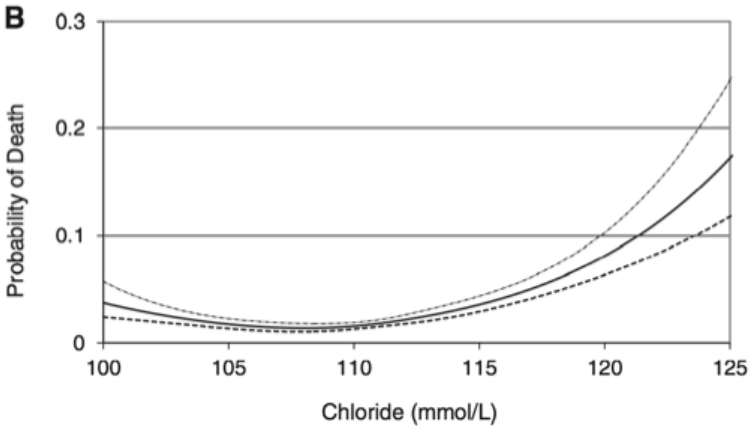
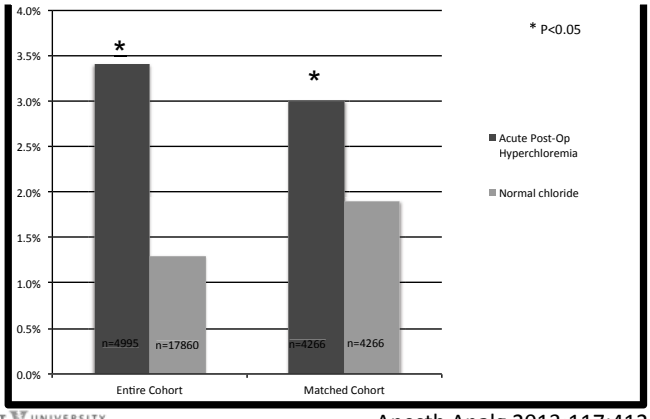
Chloride and mortality



CME

Hyperchloremia After Noncardiac Surgery Is Independently Associated with Increased Morbidity and Mortality: A Propensity-Matched Cohort Study

Stuart A. McCluskey, PhD, MD,* Keyvan Karkouti, MSc, MD,*† Duminda Wijeysundera, PhD, MD,* Leonid Minkovich, PhD, MD,* Gordon Tait, PhD,* and W. Scott Beattie, PhD, MD*



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Anesth Analg 2013;117:412–21

Intensive Care Med
DOI 10.1007/s00134-014-3505-3

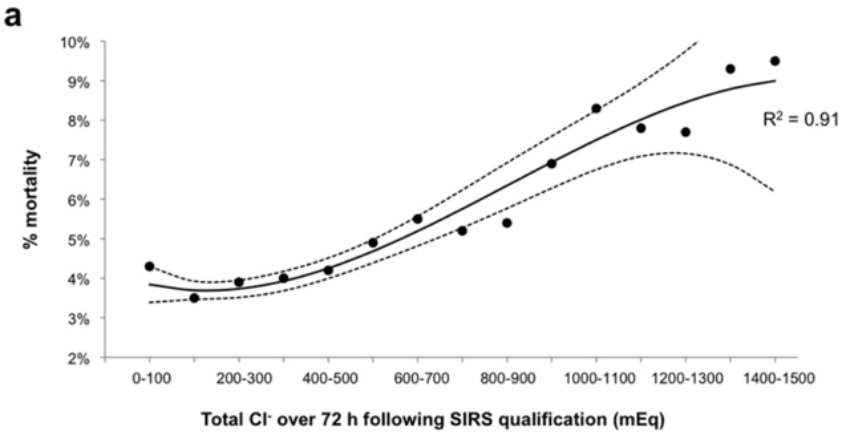
ORIGINAL

Andrew D. Shaw
Karthik Raghunathan
Fred W. Peyerl
Sibyl H. Munson
Scott M. Paluszkiewicz
Carol R. Schermer

Association between intravenous chloride load during resuscitation and in-hospital mortality among patients with SIRS

- **109,836 adult patients with SIRS from Cerner health facts database**
- **Baseline risk adjustment as well as APS included in outcomes model**
- **Effect of volume adjusted chloride load on mortality**

Administered chloride



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Shaw et al (2014) Under review

Systematic review

Meta-analysis of high- *versus* low-chloride content in perioperative and critical care fluid resuscitation

M. L. Krajewski[1], K. Raghunathan[1,2], S. M. Paluszkiewicz[3], C. R. Schermer[4] and A. D. Shaw[5]

[1]Department of Anesthesiology, Duke University Medical Center, and [2]Anesthesiology Service, Durham VA Medical Center, Durham, North Carolina, [3]Boston Strategic Partners, Boston, Massachusetts, [4]Baxter Healthcare Corporation, Deerfield, Illinois, and [5]Department of Anesthesiology, Vanderbilt University Medical Center, Nashville, Tennessee, USA

Correspondence to: Professor A. D. Shaw, Division of Cardiothoracic Anesthesiology, Vanderbilt University Medical Center, Nashville, Tennessee 37232–8274, USA (e-mail: andrew.shaw@vanderbilt.edu)

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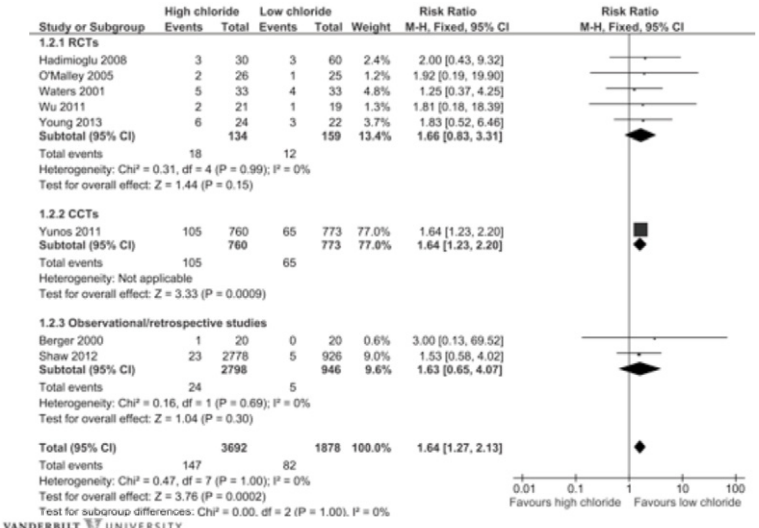
Krajewski et al (2014) Brit J Sur

AKI



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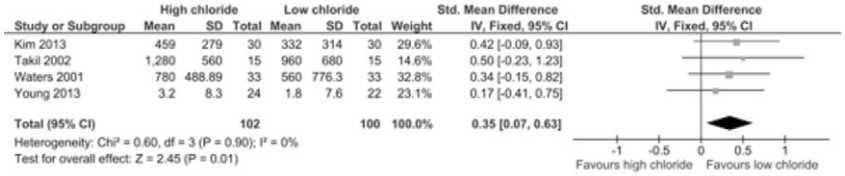
Blood transfusion volume



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ORIGINAL ARTICLE

Saline Versus Plasma-Lyte A in Initial Resuscitation of Trauma Patients

A Randomized Trial

Jason B. Young, MD, PharmD, Garth H. Utter, MSc, MD, Carol R. Schermer, MD, MPH, Joseph M. Galante, MD, Ho H. Phan, MD, Yifan Yang, MD, Brock A. Anderson, MD, and Lynette A. Scherer, MD

- **DBRCT of 65 trauma patients**
- **Plasma Lyte vs 0.9% saline**
- **Primary EP change in Base Excess first 24 hrs**
- **Deferred consent to allow immediate inclusion**

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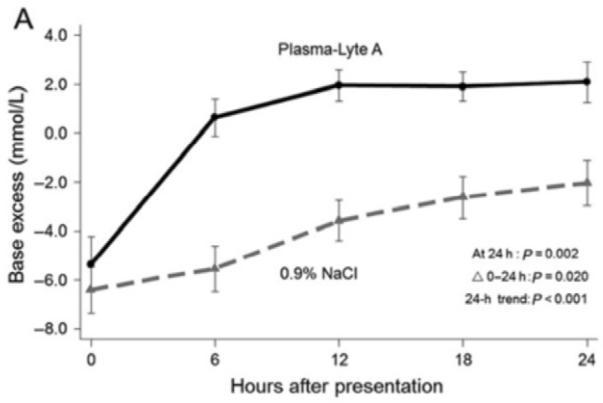
(*Ann Surg* 2013;00:1–8)

Base Excess



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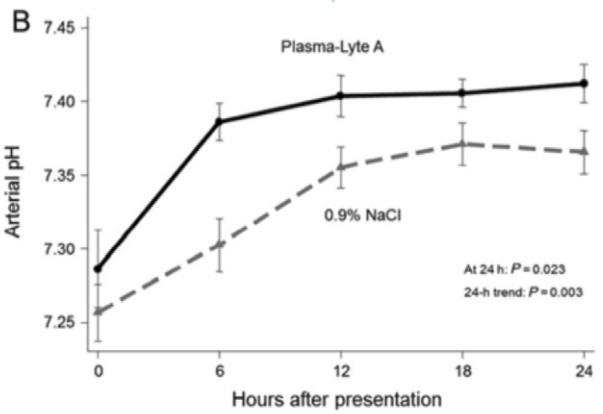
pH



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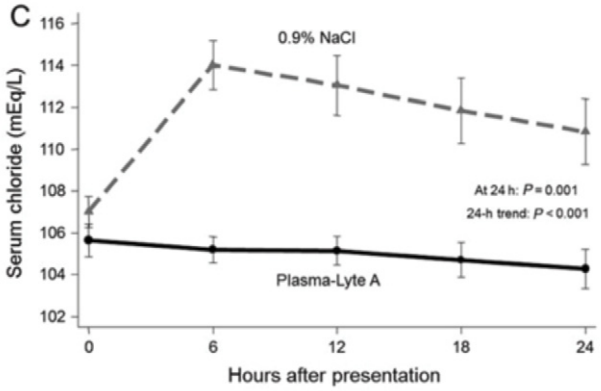
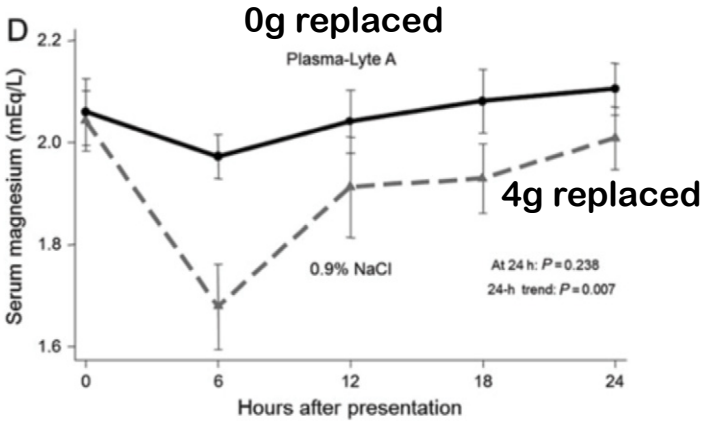
Serum chloride



Serum Magnesium



Mg supplementation

A thorough cost analysis is beyond the scope of this article, but based on information from our center, a 1-L bag of 0.9% NaCl costs $0.86/L and Plasma-Lyte A costs $1.62/L, a cost difference of 76 cents. The cost of standard IV magnesium replacement is $5.19 per 2 g and does not include the cost for nursing care to administer the infusion.

Cost-minimization analysis of two fluid products for resuscitation of critically injured trauma patients

CAITLIN A. SMITH, JEREMIAH J. DUBY, GARTH H. UTTER, JOSEPH M. GALANTE, LYNETTE A. SCHERER, AND CAROL R. SCHERMER

Cost-minimization analysis of two fluid products for resuscitation of critically injured trauma patients

Table 2.
Electrolyte Levels and Replacement Amounts, by Treatment Group[a]

Variable	0.9% Sodium Chloride Injection (*n* = 24)	Plasma-Lyte A (*n* = 22)	*p*[b]
Mean ± S.D. concentration			
Magnesium, mg/dL			
6 hr	1.7 ± 0.40	2.0 ± 0.20	0.004
24 hr	2.0 ± 0.30	2.1 ± 0.24	0.24
Potassium, meq/L			
6 hr	4.0 ± 0.62	3.8 ± 0.49	0.22
24 hr	4.1 ± 0.52	4.2 ± 0.63	0.34
Calcium, mg/dL			
6 hr	7.9 ± 0.95	7.9 ± 1.28	0.82
24 hr	8.1 ± 0.54	8.3 ± 0.60	0.28
Phosphate, mg/dL			
6 hr	3.7 ± 1.01	3.0 ± 0.88	0.03
24 hr	3.2 ± 0.92	3.4 ± 0.96	0.59
Amount replaced within 24 hr, median (IQR)			
Magnesium, g	4.0 (2.5–4.0)	0 (0–2.0)	<0.001
Potassium, meq	0 (0–20)	5 (0–20)	0.49
Calcium, g	0.5 (0–3.0)	1.50 (0–5.25)	0.46
Phosphate, mmol	0	0	0.52
Patients receiving electrolyte replacement within 24 hr, no. (%)			
Magnesium	21 (87.5)	6 (27.3)	<0.001
Potassium	9 (37.5)	12 (54.5)	0.25
Calcium	12 (50)	12 (54.5)	0.76
Phosphate	1 (4.2)	2 (9.1)	0.60

[a]IQR = interquartile range.
[b]Calculated via chi-square test, Student's *t* test, or Wilcoxon rank sum test.

Cost-minimization analysis of two fluid products for resuscitation of critically injured trauma patients

Table 3.
Average Daily Cost of Magnesium Replacement at Study Site, by Treatment Group

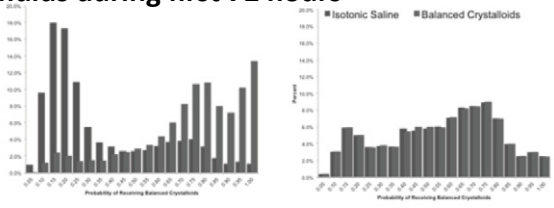
Expense Item	0.9% Sodium Chloride Injection (n = 24)			Plasma-Lyte A (n = 22)
	Cost ($) Including Labor		Cost ($) Excluding Labor	
	Administration of 2 g Magnesium	Administration of 4 g Magnesium	Administration of 4 g Magnesium	
Mean ± S.D. resuscitation fluid cost (per 24 hr)[a]	7.65 ± 4.92	7.65 ± 4.92	7.65 ± 4.92	20.46 ± 12.94[b]
Safety syringe with attached needle (1 unit)	0.72[b]	0.72[b]	0.72[b]	0.00
0.9% sodium chloride i.v. flush, 10 mL	1.34[b]	1.34[b]	1.34[b]	0.00
Surgical gloves (pair)	0.17[b]	0.17[b]	0.17[b]	0.00
Tubing (single tubing)	1.82[b]	1.82[b]	1.82[b]	0.00
Alcohol wipes (per swab)	0.01[b]	0.01[b]	0.01[b]	0.00
Drug acquisition cost	6.77[b]	13.54	13.54[b]	0.00
Labor cost (for average)[d]	7.56[b,c]	7.56[b,c]	…[d]	…
Total (per 24 hr)	26.04	32.81	25.25	20.46

[a]Based on obfuscated threshold cost of $0.85/L for 0.9% sodium chloride injection and $2/L for Plasma-Lyte A.
[b]Cost reflects administration of two 2-g doses of magnesium sulfate, which is the common practice for magnesium replacement at the study site.
[c]Calculated using U.S. Bureau of Labor Statistics data on mean nurse compensation in California; assumes mean nursing time of 9.2 minutes (4.6 minutes per infusion).
[d]Not applicable.

New cardiac surgery data (1)

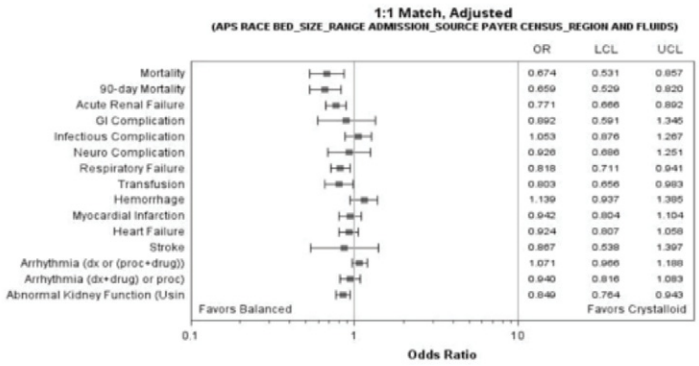
Choice of Intravenous Crystalloid Therapy and Major In-hospital Outcomes among Adult Patients Undergoing Cardiac Surgery

- **Cerner Healthfacts Database**
- **5641 on-pump patients receiving 0.9% saline PS matched with 5641 receiving balanced fluids during first 72 hours**



New cardiac surgery data (1)

Choice of Intravenous Crystalloid Therapy and Major In-hospital Outcomes among Adult Patients Undergoing Cardiac Surgery



New cardiac surgery data (2)

TITLE: THE ASSOCIATION BETWEEN CHOICE OF BALANCED INTRAVENOUS CRYSTALLOID AND SUBSEQUENT MAJOR IN-HOSPITAL OUTCOMES AMONG ADULT PATIENTS UNDERGOING CARDIAC SURGERY

Raghunathan K [1]; Khangulov VS [2]; Peyerl FW [2]; Shaw AD [3]

[1] Department of Anesthesiology, Division of Veterans Affairs. Duke University, Durham, NC, USA; [2] Boston Strategic Partners, Inc., Boston, MA, USA; [3] Department of Anesthesiology, Cardiac Division, Vanderbilt University Medical Center, Nashville, TN, USA

- **299 patients receiving Plasmalyte or Normosol matched with 299 who received LR**
- **OR for 90 day death 0.96 (0.94-0.97)**

New SIRS data

IV fluid in SIRS

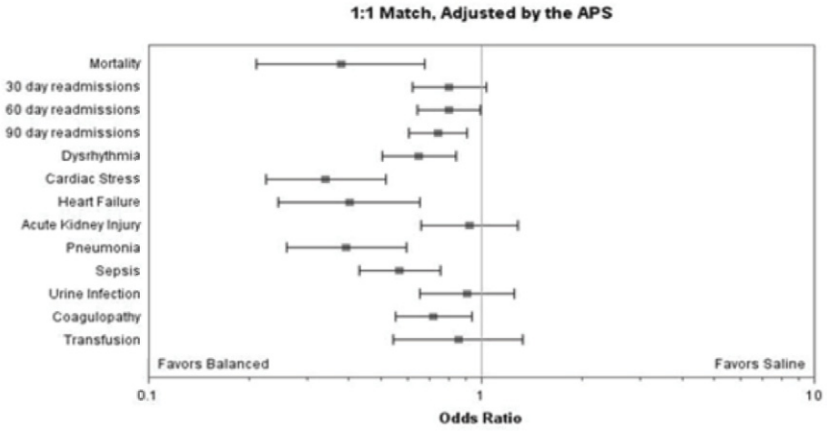
Impact of Intravenous Fluid Composition on Outcomes in Patients with the Systemic Inflammatory Response Syndrome

Andrew D. Shaw, MBBS[1]; Carol R. Schermer, MD MPH[2]; Dileep N. Lobo, MBBS, DM[3]; Sibyl H. Munson, PhD[4]; Victor Khangulov, PhD[4]; David Hayashida, BA[4]; and John A. Kellum, MD[5]

Bal v Sal in SIRS

- **3116 patients from Cerner with SIRS**
- **2 SIRS criteria and received at least 500 ml**
- **1558 Bal patients PS matched with 1558 Sal**
- **Outcomes: mortality, morbidity, resource use**
- **Adjusted for baseline comorbid disease, and APS score (for severity of illness)**

Results



When should we give abnormal saline?

- **Rarely**
- **Traumatic brain injury**
- **HCl loss (severe vomiting)**

Conclusions

- **The circumstantial evidence that high chloride solutions are harmful continues to mount**
- **There are no data suggesting 0.9% saline is beneficial**
- **New multicenter cardiac surgical data suggest balanced crystalloids are the fluids of choice for both cardiac surgery and in SIRS patients.**

Perioperative renal function and fluid therapy

Jevon Puckett
Fellow, Department of Surgery, University of Auckland

Introduction

Major surgery is a cornerstone of modern medicine. With an estimated 45% of people living in the industrialised world undergoing some form of major abdominal surgery within their lifetime, the reduction of perioperative mortality and morbidity is paramount.

Over recent decades, perioperative surgical care has undergone considerable advancement. With the advent of new surgical techniques(1) and the optimisation of perioperative care pathways(2), clinical outcomes after surgery have improved(3). Examples include Enhanced Recovery After Surgery (ERAS) protocols that have been developed with the specific aim of improving patient journeys through the entire surgical process.

Since the early 2000s, there have been a multitude of studies supporting clinical observations that fluid overloading in the perioperative period lends itself to an increase of all complications(4) (the first major randomised controlled trial being conducted by Bridgette Brandstrup in 2003(5)). Despite this overwhelming evidence and the instigation of Enhanced Recovery After Surgery (ERAS) protocols, anecdotal observations and published literature has found that maintaining a euvolaemic perioperative fluid balance is difficult in the hospital setting(6). One such hypothesis is the clinical treatment of low urine output states, historically defined as <0.5ml/kg/h(7), with fluid administration to avoid presumed acute kidney injury (AKI).

However, current surgical and anaesthetic teaching highlights the need for a perioperative urine output of ≥0.5ml/kg/h (8,9) to preempt AKI. Despite this teaching, few published studies exists in the literature clarifying this target. Furthermore what published studies are available have suggested no association between urine output and the subsequent development of AKI (10,11). These studies only represent low-grade evidence so neither support nor refute this target. Urinary catheters to measure hourly urine output are thus still routine following major abdominal surgery. Moreover, current practice within the renal physician community accepts a urine output target of <0.2ml/kg/h in the otherwise healthy population. With this in mind, should we be redefining our definition of perioperative oliguria?

Methods

Following a priori power calculation based on the biochemical detection of AKI using urinary neutrophil associated gelatinase lipocalin (uNGAL), 34 patients were required to participate in a non-inferiority study comparing a perioperative urine output target of <0.2ml/kg/h to the traditional 0.5ml/kg/h. To allow for drop-outs, 41 patients were ultimately randomised. Between 2012-2013, all adult patients undergoing elective colon or small bowel resection at the North Shore Hospital, Auckland, were eligible and screened at the outpatient clinic. Patients were excluded on a variety of criteria including severe chronic renal impairment (eGFR<30ml), age>85 years, ASA 4, ongoing need for nephrotoxic medication administration and Childs-Pugh B liver disease. At induction of anaesthesia, patients were randomised by off site computerized software to either attain a urine output target of 0.5ml/kg/h or 0.2ml/kg/h using boluses of Plasmalyte© (PL148). Fluid bolusing for isolated low urine output continued until 08:00 on postoperative day 2 (POD2).

The primary endpoint of the study was uNGAL at 08:00 on postoperative day 1 (POD1). Secondary endpoints included the measurement of current biochemical markers of renal health (creatinine and cystatin C), renal hormones (renin, aldosterone, angiotensin II) and effective renal plasma flow (para-aminohippurate) and glomerular filtration rate studies (sinistrin). PAH and sinistrin infusion studies were conducted at 08:00 approximately 1 week prior to surgery and at 08:00 on POD1 to test the hypothesis that epidural or spinal anesthetic led to reduced renal plasma flow and thus glomerular filtration rate.

All physiological data on blood pressure, heart rate and fluid balance were also recorded.

Results

41 patients were randomised to the high (n=18) vs low (n=23) groups. During the first 48 hours, the high group had statistically fewer episodes of oliguria as traditionally defined; 9.4(1.8) vs 21.3(1.6) ($p<0.0001$) and received more fluid volume; 8358 (580) vs 5435 (513) ml ($P<0.0005$). However, plasma creatinine concentrations on POD1 were similar; 69.3 (4.5) vs 74.3 (4.0) mMol/l ($P=0.41$). Importantly, the primary endpoint of uNGAL concentrations at 08:00 on POD1 were also non-significantly different; 17.1 (4.8) vs 20.2 (4.2) ng/ml ($P=0.63$).

There were no differences in length of stay ($P=0.34$) or 30-day incidences of minor ($P=0.52$) and major ($P=0.30$) complications (by Clavien-Dindo grade), although the trial was not powered to show a difference in clinical outcomes.

There was also no statistical difference between preoperative and postoperative effective renal plasma flow (504 vs 510ml/min $p=0.87$) and glomerular filtration rate (135 vs 139ml/min/1.73^2 $p=0.3$).

Conclusions

Despite resulting in significantly more episodes of oliguria as traditionally defined, accepting a lower urine output target of 0.2ml/kg/h in the elective colon or small bowel resection patient, was not inferior to maintaining the traditional target of 0.5ml/kg/h using a variety of biomarkers of renal health. Furthermore, the use of epidural or spinal anaesthesia did not result in reduced renal plasma flow and or a subsequent reduction in glomerular filtration rate.

Regarding the hypothesis, yes we can redefine the perioperative urine output target in this population.

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A brief history of colloids for fluid therapy

Shay McGuinness, CVICU
Auckland City Hospital

Fluid therapy with water and salts was probably first given in the 1830s for treatment of patients suffering from Blue Cholera.[1] Although case reports from the time describe a significant improvement in clinical symptoms outcomes were poor, attributed in part to the lack of a sustained effect. Over the next 70 years the use of IV crystalloid solutions became more widespread however the frequently observed short term effects lead clinicians to try and develop new solutions that would "remain in the circulation longer".[2] Gelatin, was the first fully artificial plasma substitute to be used extensively for shock treatment, in a large part due to the large number of casualties with hypovolaemic shock treated close to the battlefield during World War 1.[3] It is interesting to note that in his first case series Hogan commented on the relative lack of benefit of colloid fluid in non-haemorrhagic causes of shock.

By the end of world war II use of colloid for resuscitation was well established and dextrans as well as Gelatins used as a substitute to plasma for resuscitation. Hydroxyethyl starch (HES) solutions, derived from waxy-maize derivatives were introduced in the 1970s as a further attempt to more closely mimic the volume expanding effect and duration of action seen with albumin solutions. From the 1980s onwards there was rapid increase in the amount of synthetic colloids used, in particular HES, driven in part by the popular belief that colloids produced better resuscitation than crystalloids[4] and were safer and more cost-effective than donated blood derived products, especially albumin solutions.

By 1985 it was being suggested that the end of routine use of crystalloid solutions as volume expanders was approaching,[5] however over the following 13 years a number of systematic reviews and meta-analysis suggested that there may be adverse events and worse outcomes associated with the use of synthetic and natural colloids[6,7]. A landmark paper by the Cochrane collaboration in 1998 that implicated the use of human albumin in excess mortality sparked immediate publicity and the call for a ban on albumin use.[8]

In response to this controversy, the recently formed Australian and New Zealand Intensive Care Society Clinical Trials Group (ANZICS-CTG) proposed the SAFE study – a double blind evaluation of 4% albumin vs saline for volume resuscitation in intensive care. Published in 2004[9] the SAFE study represented the largest RCT performed in intensive care patients 9n=6999) and demonstrated that the use of albumin was safe (with the exception of patients with TBI), however no clinical benefits could be shown over saline.

Although the use of albumin decreased significantly following the Cochrane publication in 1998 the use of artificial colloids continued to increase, and this was demonstrated by the same group of investigators with an international observational study of iv fluid use in intensive care.[10] The increased use of synthetic colloids in general and of HES solutions in particular prompted the design of a trial comparing the effects of HES and Saline on clinical outcomes in the intensive care unit. The CHEST study,[11] published in 2012 demonstrated that the use of HES solutions was not associated with any improvement in outcomes and was actually associated with an increase in the use of renal replacement therapy and other adverse effects such as pruritus.

CHEST was published shortly after two other similar but smaller studies in patients with sepsis demonstrated higher mortality in patients who received HES solutions instead of crystalloid solutions.[12,13] Following the publication of these landmark papers there was an initial reaction from regulatory authorities around the world that ranged from a complete ban on HES use to less restrictive warnings and guidance. The CRISTAL study[14] suggested a possible benefit to colloids over crystalloids; however it has significant methodological limitations.

Recent consensus guidelines have attempted to distil the evidence from these large studies,[15,16] however the mobilisation of a significant industry promotional machine and the large amount of money involved in the IV fluid business worldwide suggest that the colloid story still has a few chapters left.

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Fluid management guidelines (NICE/GIFTASUP)

Prof Dileep Lobo
University of Nottingham, UK

To view this document in full, go to 2015 ACS Resources:

Nice Guidelines

How I give fluid intraoperatively...

Prof Andrew Shaw
Vanderbilt University, Tennessee, USA

How and why I give IV fluid

Andrew Shaw
MB FRCA FCCM FFICM

Professor and Chief
Cardiothoracic Anesthesiology
Vanderbilt University Medical Center

2015 Disclosures

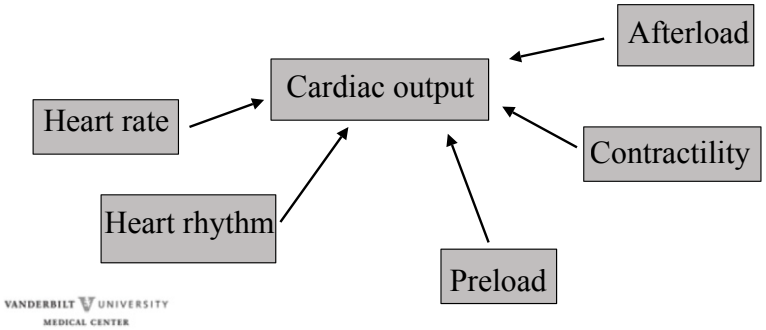
- **Consultant for Grifols – manufacturer of colloid (albumin) products**
- **Consultant for Baxter – manufacturer of crystalloid and colloid products**
- **No off label comments**

Fluids and public health

- 30% of ICU patients experience an episode of fluid resuscitation every day
- IV fluids are the commonest inpatient prescription in the world
- Fluid based GDT in the OR has been a cornerstone of ERAS

Basic physiology

- Cardiac output



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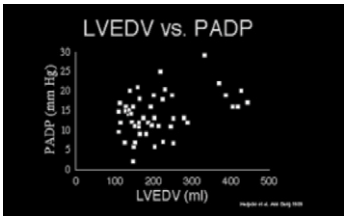
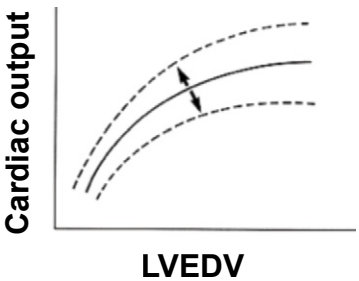
Preload

- Is a volume not a pressure!





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Correct Hemodynamics

- **Administer fluids, inotropes and vasoactive drugs to restore:-**
  - **An effective circulation**
  - **An effective mean arterial pressure**
  - **An effective oxygen carrying capacity**
- **Give fluids and drugs according to need and not just as a routine: make the patient earn their fluid (and blood and O_2)**
- **Deviate from guidelines with a clinical reason to do so**

Specific Endpoints

- **Blood Pressure: MAP is the main determinant of perfusion in a pulsatile circuit: at least 60 and sometimes 90**
- **Lactate: High levels correlate with poor outcome. Low levels do not rule out underperfusion**
- **SvO_2: Useful if low. Normal value does not rule out underperfusion**

Clinical Indices of Adequate Perfusion

- **Good urine output (1ml/kg/hr)**
- **No angina**
- **No reduction in conscious level**
- **Good capillary return**
- **Warm extremities**

Aims of Fluid Therapy

- **Convert hypodynamic situation to normal or hyperdynamic state**
- **Increase cardiac output until either effective circulation restored or plateau reached on Starling curve**
- **Blood: Always if Hb < 7 g/dl**
  Never if Hb > 10 g/dl
  For symptoms if 7-10 g/dl

Aims of Vasoactive Therapy

- **Restore MAP when optimum fluid therapy and appropriate inotropic therapy have not**
- **Vasopressor treatment may be needed emergently while fluid therapy is underway**
- **All who receive vasoactive therapy in the ICU should have an A-line in place**
- **A-lines: Radial – Brachial - Femoral – Axillary**

Fluids in Shock

- **50% of patients with hypotension will respond to fluid therapy alone**
- **Type not as important as how and how much**
- **Give by bolus and against an index of preload**
- **Encourage bedside generation of dynamic Starling curve**

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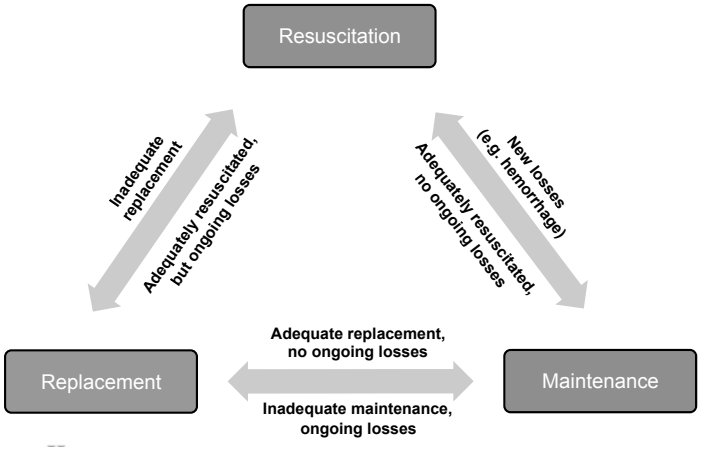
Thinking about fluids

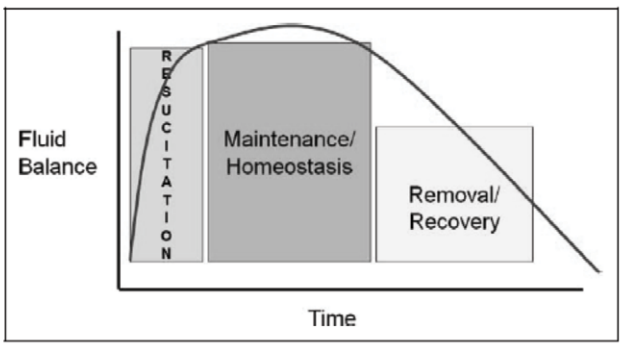
- Is amount of fluid given important?
- Is crystalloid type important?
- Is colloid type important?
- Which is better – crystalloid or colloid?

3Rs: Right amount of the Right fluid at the Right time

Reasons IV Fluids are Given



Fluid Balance During Hospital Stay



Challenges with IV Fluids

- Low awareness of the specific constituents of different fluids
- Little formal education and training exists on fluid management
- Wide variety in type of fluid charts used
- Fluid requirements are not re-assessed as patient status changes
- Insufficient attention to identify, treat and monitor fluid and electrolyte status

National Confidential Enquiry into Patient Outcome and Death (NCEPOD)

- Patients are dying as a result of infusion of too much or too little fluid by inexperienced staff

- Fluid prescription must be given the same status as drug prescription

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Extremes of Age: the 1999 report of the NCEPOD http://www.ncepod.org.uk/pdf/1999/99full.pdf

Basic Considerations

- Fluid therapy should be individualized
  - Understand the purpose and goals of giving IV fluid to your specific patient

- Prescribe IV Fluids like drugs
  - Specific dose
  - Specific indication

- Reassess routinely
  - Changes in patient status may require a change in fluid prescription

Indications and Goals

Indication	Goal
Resuscitation	• Restore / preserve intravascular fluid volume • Restore effective tissue perfusion • Re-establish and maintain a balance between tissue oxygen demand and supply
Fluid and Electrolyte Replacement	• Provide normal daily maintenance requirements plus compensate for abnormal losses • Aim to replace like with like: replace fluid lost with fluid of similar composition • Consider composition of balanced fluids vs plasma
Maintenance	Provide daily requirements of water and electrolytes • Water 25-35 ml/kg/day • Sodium 1 mmol/kg/day • Potassium 1 mmol/kg/day

Overall Goal for All Patients

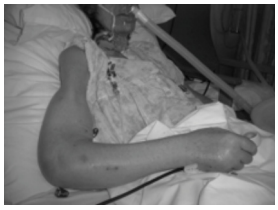
- **Right Amount**
- **Right Fluid**
- **Right Time**

Delivering the

RIGHT AMOUNT

Fluid Gain in the ICU

- **Patients with sepsis in the ICU may gain as much as 12.5 L of body water during the first 2 days of resuscitation**
- **Excretion of this excess load may take up to 3 weeks**
- **This is bad!**



How does this happen ?

- Patients receive lots of fluid, lots of sodium chloride
  - Kidneys can't excrete sodium load
  - Chloride causes renal vasoconstriction and exacerbates fluid retention and edema
  - Leaky capillaries in sick patients exacerbates edema

- Patients don't receive much potassium
  - Potassium depletion reduces ability to excrete sodium

Consequences of excess fluid

- Decreased renal blood flow and GFR
- Intra-mucosal acidosis
- Prolongation of gastric emptying time
- Ileus
- Hyperchloraemic acidosis
- Weight gain
- Low serum sodium due to ADH release
  - Can lead to administration of more sodium
- Cellular dysfunction

Sodium Chloride and Volume Overload: Clinical Effects

- Peripheral oedema
- Pulmonary oedema
- Renal oedema
- Gastro-intestinal oedema
- Impaired cardiac function
- CCF/arrhythmias
- Confusion
- Delayed mobilisation
- Pressure sores
- Increase in DVT

Sodium Chloride and Volume Depletion

- Reduced stroke volume – poor organ perfusion, hypotension
- Impaired renal perfusion - ARF
- Increased viscosity of mucus
- Reduced saliva
- Increased blood viscosity can lead to clots

Moderation

- The objective of care is restoration of normal physiology and normal function of organs, with a normal blood volume, functional body water, and electrolytes.

- This can never be accomplished by inundation.

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FD Moore & GT Shires, Ann Surg (1967)

Right Amount of Fluid Depends on Reason IV Fluid is Needed

- Resuscitation
  - Restore circulation and oxygen supply to vital organs with 250 – 500 mL of fluid immediately and monitor response (but what type?)
- Fluid and electrolyte Replacement
  - Amount should incorporate daily maintenance plus any abnormal losses
- Maintenance
  - Amount should be sufficient to maintain normal status in body fluid compartments, and allow kidney to excrete waste products

The Right Amount of Fluid Depends on the Type

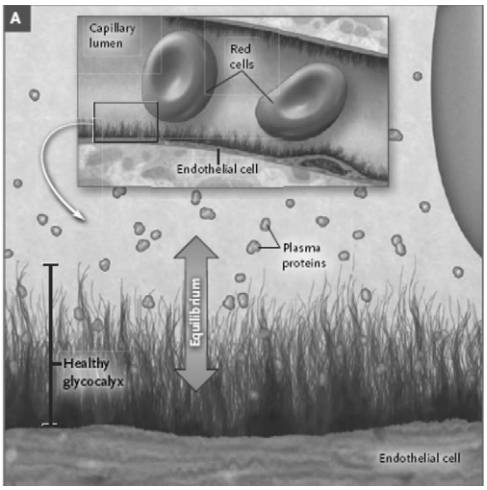
Volume effect of colloids:crystalloids was thought to be 1:3

Not True!

Recent data shows the ratio is more likely to be only 1:1.3

The Endothelial Glycocalyx

Healthy



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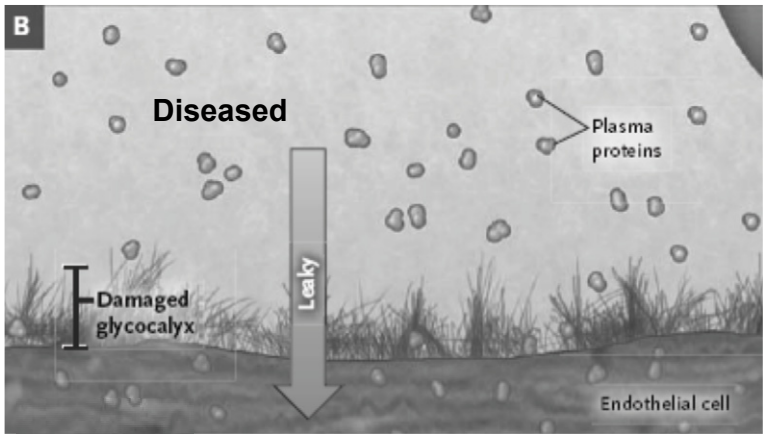
The Endothelial Glycocalyx



Restrictive or Liberal Strategy ?

- **Currently: trend towards restrictive fluid strategy**
- **Commonly accepted definitions of “restrictive” or “liberal” fluid strategies do not exist**
- **Definition, methodology and results not well-defined in the literature, precluding evidence-based guidelines for procedure-specific perioperative fixed-volume regimens**

Goal-directed Fluid Therapy

- **Meta-analyses have shown that cardiac output guided fluid management appears to reduce hospital stay and morbidity**
- **Goal-directed fluid therapy appears to reduce inflammation, morbidity, and mortality in patients who undergo major surgery**

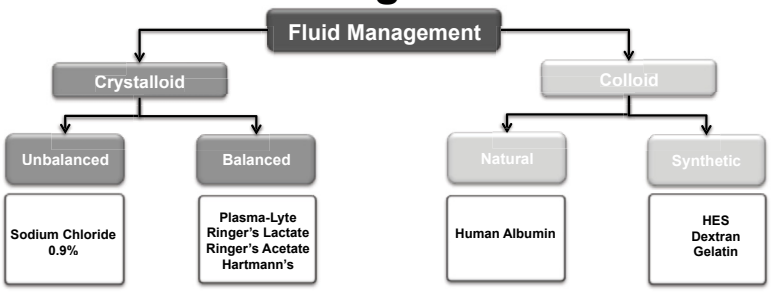
Summary: Right Amount of IV Fluid

- **Maximum effect with minimum sodium, chloride and water loading**
- **Before patients can recover, they must excrete the water, sodium and chloride given during resuscitation**
- **Reason IV fluid is needed must be considered when determining what to administer**
- **Fluids differ in electrolyte content; choice of fluid matters too**

Delivering the Right Amount of the

RIGHT FLUID

Treatment Choices for Fluid Management



Different fluids with different modes of action, and different side effects

Crystalloids and Colloids

Colloid solutions	Crystalloid solutions
• Contain large proteins or synthetic glucose polymers which are too large to pass through the walls of capillaries under normal conditions • Colloids are thought to have greater volume effect compared with crystalloids, but current research shows ratio to only be 1:1.3	• Contain electrolytes (e.g. sodium, potassium, calcium, chloride) • An isotonic crystalloid solution is distributed in the entire extracellular space (plasma plus interstitial space)

Delivering the Right Amount of the Right Fluid at the

RIGHT TIME

Criteria for IV Fluid Administration

- Fluids should be given to address a specific patient need, not because of routine practice
- Objective criteria should be used when:
  - Starting IV fluids
  - Increasing or decreasing IV fluids
  - Stopping IV fluids

How do we assess fluid balance?

- Physical exam
  - "Stethoscope findings" (rales, rhonchi), pulse, weight, skin perfusion/ temperature, urine output and electrolyte concentration, fluid balance charts
- Metabolic monitors
  - Lactate, SVO_2, ABG
- Static monitors
  - BP (MAP), CVP, PAOP
- Dynamic monitors
  - Pulse pressure variation
  - Cardiac output
  - Stroke volume variation
  - Passive leg raise
  - Continuous TEE

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Key Clinical Questions:
- Is the patient fluid deficient?
  If so…
- Is the patient responsive to fluids?

Volume Assessment Tools

Table 2 | Summary of volume assessment tools

Method	Invasive or noninvasive	Static or dynamic	Assess fluid responsiveness	Comments
Historical findings	Noninvasive	Static	No	Of limited value with poor correlation with invasive pressure measurements
Physical exam	Noninvasive	Static and dynamic	Yes	Of limited value but serial examinations may detect changes in organ perfusion
Chest radiograph	Noninvasive	Static	No	Requires use of standardized measures of vascular pedicle width and cardiothoracic ratio. Serial chest X-ray may be helpful in determining effects of fluid therapy
Central venous pressure	Invasive	Static	No	Poor correlation with fluid responsiveness
Pulmonary capillary wedge pressure	Invasive	Static	No	Poor correlation with fluid responsiveness
Echocardiogram	Noninvasive	Static	No	Single measures of cardiac chamber volume hard to assess. Serial measures may be helpful
Stroke volume or pulse pressure variation	Invasive (pulse oximeter method in noninvasive)	Dynamic	Yes	Requires sedated, mechanically ventilated patient
Esophageal doppler	Invasive	Dynamic	Yes	Not useful for continuous measurements
Vena cava diameter	Noninvasive	Dynamic	Yes	Body habitus dependent
Passive leg raising	Noninvasive (bioreactance, end-tidal CO_2) Invasive (FloTrac or PICCO or LiDOO)	Dynamic	Yes	Unreliable with intra-abdominal hypertension
End-expiratory occlusion	Passive leg raising	Dynamic	Yes	Requires 15-s end-expiratory occlusion
Bioimpedance	Noninvasive	Static	No	Not able to assess intravascular volume

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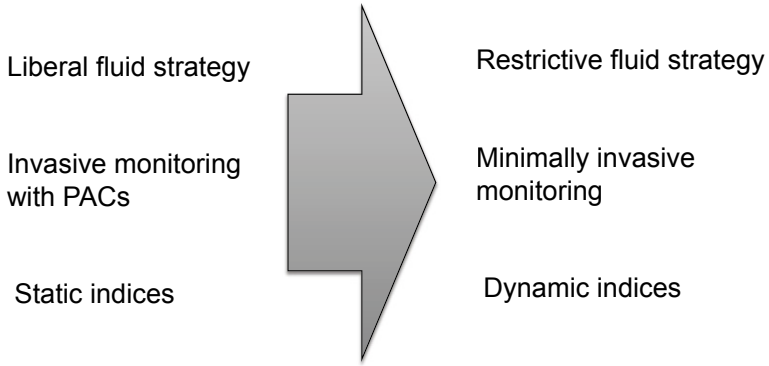
Kalantari et al. Kidney International (2013) 83,1017-1028.

Trends in Fluid Assessment



Signs That a Patient May be Hypovolemic

- **Systolic BP < 100 mmHg**
- **HR > 90 bpm**
- **Capillary refill > 2 seconds or extremities are cold to touch**
- **RR > 20 bpm**
- **Passive leg raising test is positive**
- **Blood pressure drop when sitting up**
- **Invisible/collapsing neck veins**
- **Thirst**
- **Low urine output**

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Functional Questions to Consider Prior to Starting Fluids

- Is tissue oxygenation adequate?
  - Surrogates:
    - Mixed venous oxygen saturation
    - Central venous oxygenation
    - Serum lactate
- Is the patient volume responsive?
- Is vasomotor tone increased or decreased?
- Is the heart able to sustain an adequate CO when arterial pressure is restored without going into failure?

Patient Assessment and Monitoring for Fluid Therapy

- Patient monitoring and reevaluation on a routine basis is crucial for safe fluid therapy
- Reason for IV fluids may change as patient status changes, so IV fluid orders should be re-evaluated frequently
- Goal is to stop IV fluid as soon as patient can meet needs enterally

Summary: The Right Amount of the Right Fluid at the Right Time

- Fluid therapy should be individualized
  - Understand the purpose and goals of giving IV fluid to your specific patient
- Prescribe IV Fluids like drugs
  - Specific dose and indication
- Choose a fluid based on composition and patient needs
  - Default fluid for critically ill should likely be a balanced crystalloid
- Reassess patient using objective measures and adjust fluid prescription accordingly

Haemodynamics – Understanding the Relationship

Martin Misur

Specialist Anaesthetist
Auckland City Hospital

I maintain that intravenous fluids are drugs. Like any drug they have indications, contraindications, appropriate doses and administration schedules. Amongst other things, haemodynamic monitoring aids us to determine the dose.

Anaesthetists appear as a group to embrace technology and to like numbers. As an inducement to register early for this meeting, the organisers were giving away a Masimo pulse oximeter for your smartphone. Why is pulse oximetry important? Well in 1972, Takuo Aoyagi (figure 1) was trying to develop a non-invasive method to determine cardiac output using cardiogreen dye and an ear oximeter. He found that the light transmission exhibited pulsatile artefacts that made it impossible to compute cardiac output using dye dilution.

He realised the implication and developed a two-wavelength ear pulse oximeter, which made use of heart pulsations to detect and measure arterial blood absorbance. The first commercially successful device was marketed in 1977 by the Biox Corporation (later purchased by Ohmeda).

Fig 1 – Takuo Aoyagi

Few of us would accept monitoring that did not include pulse oximetry and yet when first marketed it took a while for it to be accepted. Severinghaus and Honda[1] explain that "few foresaw its value in anesthesiology, intensive care, and other emergent situations." With concerns over accuracy, it was not until 1986 that the ASA recommended it as standard of care.

Just as with the early story of pulse oximetry, there appears to be a reluctance to monitor cardiac output.[2]

A pivotal goal of anaesthesia and intensive care is tissue oxygen delivery, and this can be related to outcome.[3,4] Haemodynamic monitors are tools that may assist us in this goal by providing information for us to act on. They are not treatments. In the words of Michael Pinsky,[5] "no monitoring tool, no matter how accurate, by itself has improved patient outcome."

Two key requirements for tissue oxygen delivery are perfusion pressure and flow (cardiac output). Advanced haemodynamic monitoring involves –

- Assessment of preload responsiveness (the customary role)
- Cardiac output (CO) monitoring
- Assessment of cardiac contractility
- Assessment of tissue perfusion

Advanced haemodynamic monitoring is an integrative model view of single parameters.

Some Basic Physiology

$$MAP = \mathbf{CO} \times SVR$$

And perfusion pressure is dependent on preload, afterload, rate, rhythm and contractility. If afterload is represented by SVR, then the other four parameters are the determinants of CO.

$$DO_2 = \mathbf{CO} \times [Hb] \times 1.31 \times SaO2$$

$$CO = HR \times \mathbf{SV}$$

Hence stroke volume, the volume of blood ejected by the ventricle per heartbeat, is pivotal in determining flow and oxygenation. It is affected by preload, afterload and contractility. Preload is the end-diastolic ventricular wall tension (/ pressure), or simply how "full" is my patient.

The Frank Starling law (figure 2) holds that the force of myocardial contraction is proportional to initial cardiac muscle fibre length. It describes the relationship of preload to cardiac output.

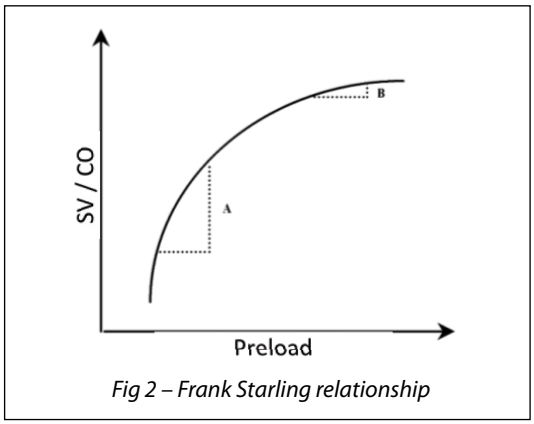


Fig 2 – Frank Starling relationship

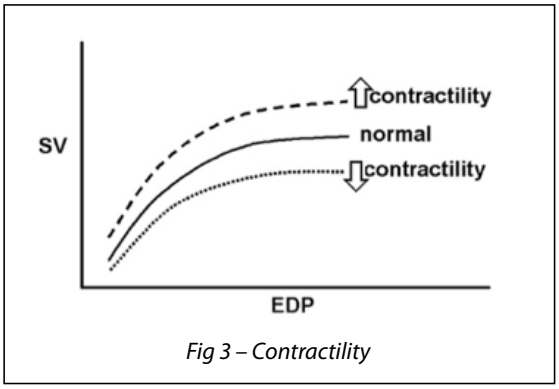


Fig 3 – Contractility

Contractility (figure 3) is the inherent ability of the cardiac muscle to contract regardless of preload or afterload status. It is estimated by analysis of the arterial waveform – maximum speed of the arterial pressure curve during ejection.

So DO_2 depends on CO. And CO depends on SV. And SV depends on preload. So the question for anaesthetists is will SV / CO improve with fluid resuscitation (aka fluid responsiveness)? To increase stroke volume is the *only* reason to give a patient a fluid challenge. However –

- Being fluid responsive does not mean the patient needs fluid
- Measurements of fluid responsiveness do not indicate what type of fluid is most suitable

Fluid therapy is a difficult balance –

- Too little fluid leads to –
  - Tissue hypoperfusion and organ dysfunction
  - Uncorrected hypovolaemia and inappropriate use of vasoconstrictors may worsen hypoperfusion and tissue ischaemia
  - We have evolved to cope with hypovolaemia[6]
- Too much fluid causes –
  - Tissue oedema, impaired wound healing
  - Bowel oedema, reduced motility, ileus, anastomotic leak, abdominal compartment syndrome
  - Lung oedema with increased respiratory complications
  - Impaired oxygen uptake and delivery
  - Worse outcomes, increased LOS
  - Volume overload is a recent phenomenon, usually iatrogenic, and humans lack compensatory mechanisms

Central Venous Pressure

Central venous pressure (CVP) is the pressure within the right atrium and great veins of the thorax. Whilst as a profession we were slow to adopt pulse oximetry and there is a reluctance to embrace advanced haemodynamic monitoring, CVP measurements are near universally used to make clinical decisions. Indeed a 2007 European survey of anaesthetists / intensivists[7] demonstrated that more than 90% used the CVP to guide fluid management.

Whether CVP is predictive of preload and fluid responsiveness has been questioned since 1971.[8] In 1984 Shipley et al[9] made over 1,500 simultaneous measurements of blood volume and CVP in 188 ICU patients. They were able to demonstrate no relationship between CVP and blood volume.

Receiver operating characteristic (ROC) plots compare different clinical tools with different diagnostic accuracies – figure 4. Plots located in the upper left-hand quadrant have better sensitivity and specificity.

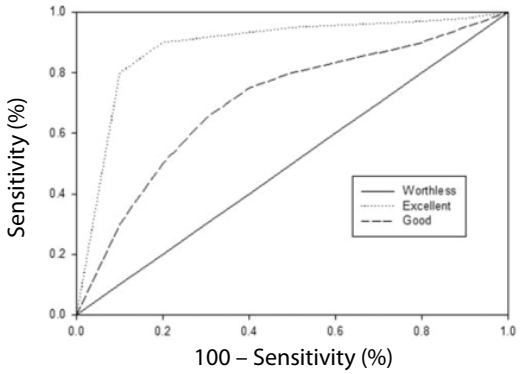


Fig 4 – Receiver operating characteristic (ROC) plot

In 2008 Marik et al published a review of 24 studies involving 803 patients.[8] They were able to show the correlation coefficient between CVP and measured blood volume was 0.16 (95% confidence interval [CI], 0.03-0.28). The area under the ROC curve was 0.56 (95% CI 0.51-0.61) – little better than a coin toss in determining fluid responsiveness. A patient has the same probability of being fluid responsive with a low or a high CVP. CVP is often used to follow 'trends.' In this paper, the correlation between ΔCVP and change in cardiac index was 0.11 (95% CI 0.015-0.21). The authors concluded that there is a very poor relationship between CVP and blood volume and that "CVP should not be used to make clinical decisions regarding fluid management."

Despite this the "Surviving Sepsis Campaign" publish internationally endorsed clinical guidelines[10] recommending a CVP target to guide fluid resuscitation. Miller's anaesthesia text[11] states that "the most important application of CVP monitoring is to provide an estimate of the adequacy of circulating blood volume," and that "trends in CVP during anesthesia and surgery are also useful in estimating fluid or blood loss and guiding replacement therapy."

There are now over 100 papers showing no relationship between CVP (or change in CVP) and fluid responsiveness.[12] Marik and Cavallazzi[13] updated their meta-analysis in 2013 to include 43 studies and compare ICU and operating room studies. Once again the area under the curve was 0.56 (95% CI 0.54-0.58) irrespective of whether the patient was in ICU or OR.

CVP may be useful in early detection of impaired cardiac function or high intra-thoracic pressure (not volume status) in –

- Heart transplant patients
- Right ventricular infarction
- Pulmonary hypertension
- Severe LV dysfunction
- Acute PE
- Tamponade
- Tension pneumothorax

Pathological CVP waveforms may assist in diagnosis.

Dynamic Parameters

Static parameters (CVP, PCWP) are insufficient to predict fluid responsiveness. More subtle changes in volume status need to be detected, and targeted endpoints need to be more sensitive and specific to allow for optimisation and possibly better outcomes.

Dynamic techniques rely on the change in preload resulting from mechanical ventilation and allow assessment of whether a patient is on the ascending portion of the Frank Starling Curve and has "recruitable" cardiac output – table 1.

The "swing in the trace" is better referred to as arterial pressure variation or inverse pulsus paradoxus. Intermittent positive-pressure ventilation induces cyclic changes in the loading conditions of the left and right ventricles –

Table 1 – Techniques for assessing fluid responsiveness. ROC, area under receiver operator characteristic curve; IVC, inferior vena cava; SVC, superior vena cava

Static pressure and volume parameters (ROC ~0.5–0.6) ▪ CVP ▪ PAOP ▪ IVC/SVC diameter ▪ Flow corrected time ▪ Right ventricular end-diastolic volume ▪ Left ventricular end-diastolic volume ▪ SVC/IVC variation during mechanical ventilation
Dynamic techniques based on heart–lung interactions during mechanical ventilation (ROC ~0.7–0.8) ▪ PPV ▪ SVV ▪ Pleth variability index ▪ Aortic blood flow (Doppler or echocardiography)
Techniques based on real or virtual fluid challenge (ROC ~0.9) ▪ PLR ▪ Rapid fluid challenge (100–250 cc)

- Mechanical insufflation →
  1. ↓ RV preload due to ↑ pleural pressure and ↓ venous return pressure gradient
  2. ↑ RV afterload due to ↑ transpulmonary pressure

- ↓ RV preload and ↑ RV afterload → ↓ RV stroke volume, which is at a minimum at the end of the inspiratory period

- ↓ RV ejection → ↓ LV filling (after a phase lag of two or three heart beats because of the long blood pulmonary transit time)

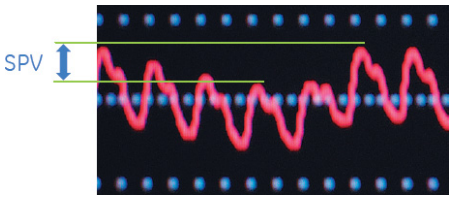
- ↓ LV preload → ↓ LV stroke volume, which is at its minimum during the expiratory period

The magnitude of these changes is dependent on the fluid status of the patient being of greater amplitude in hypovolemic patients where the ventricles operate on the steep portion of the Frank-Starling curve.

Measurement

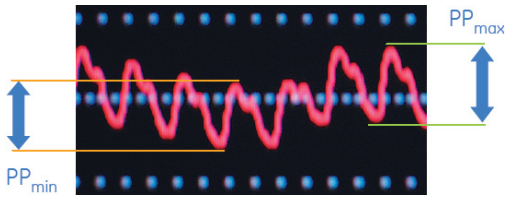
Systolic pressure variation (SPV, in mmHg) is a numerical quantification of the degree of swing in the arterial trace. Whether SPV is useful in spontaneously ventilating patients, whose respiratory physiology differs, is a matter of debate.

$$SPV = SBP_{max} - SBP_{min}$$



Pulse pressure variation (in %; aka dPP or ΔPP) is the ratio of the change in pulse pressure to the mean pulse pressure.

$$PPV = \frac{PP_{max} - PP_{min}}{(PP_{max} + PP_{min})/_2} \times 100$$



SPV and PPV have the attraction that they are effectively a free "by-product" of invasive arterial blood pressure monitoring. The major equipment manufacturers include software into their monitors which calculate these variables for us. However there are limitations –

- The patient must be mechanically ventilated (7 or 8ml/kg TV)
- They must make no spontaneous respiratory effort
- Cannot be used with an open chest
- High PEEP affects the result
- The patient must not have sustained cardiac arrhythmias as these result in variable cardiac filling time. More advanced standalone devices are able to filter out such arrhythmias to a degree
- Elevated intra-abdominal pressure / laparoscopic procedures
- Readings of invasive BP must be reliable (zeroed, no damping)
- RV failure
- There are questions around the effects of vasoconstrictors

A group based in Virginia[14] have investigated the ability of anaesthesia providers to eyeball SPV. They tasked 50 anaesthetists with estimating SPV as a percentage of SBP. Each was asked to look at 10 traces and determine whether the patient needed fluid. They found that visual estimates are within clinically reasonable limits 82% of the time and that erroneous management decisions were made in association with 4.4% of measurements.

When calculated manually PPV is termed PPV_{man} and is derived over a single mechanical breath. Continuous automated measurements (PPV_{auto}) are calculated over a longer period and hence the two values may differ. Cannesson et al[15]

investigated the ability of both measurements to predict fluid responsiveness during coronary artery bypass grafting. The agreement between PPV_{man} and PPV_{auto} was 0.7% ± 3.4%. In terms of predicting fluid responsiveness, the areas under the ROC curves (figure 5) were 0.923 ± 0.060 for PPV_{man} and 0.919 ± 0.058 for PPV_{auto}, showing that PPV_{auto} can be displayed continuously to predict fluid responsiveness. The authors caution though that a minute of haemodynamic stability is required before the value can be used clinically.

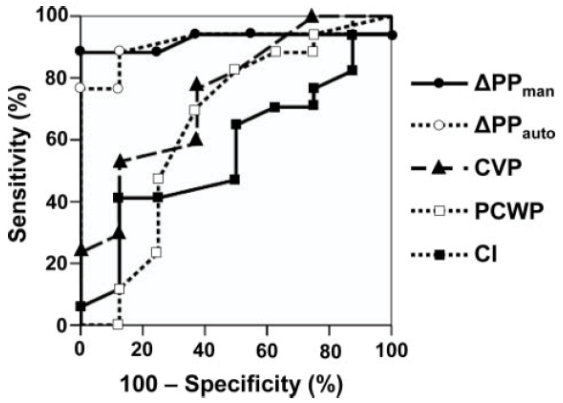


Fig 5 – Receiver operating characteristic curves comparing the ability of PPV_{man}, PPV_{auto}, CVP, PCWP, and CI at baseline to discriminate between responders and nonresponders to volume expansion

Cannesson et al have gone on to clarify the utility of PPV in a multicentre trial[16] and to clarify a "grey zone." This study again showed a strong predictive value with the area under the ROC curve being 0.89 (95% CI 0.86-0.92).

One problem with the ROC approach is that it leads to "black or white" decision making, seeking a value above which patients will be fluid responders, and this does not fit clinical practice. As they explain, a "grey zone technique proposes two cut offs that constitute the borders of the grey zone. The first cut off allows exclusion of the diagnosis (fluid responsiveness) with near certainty, whereas the second cut off is chosen to include the diagnosis with near certainty. Intermediate values included in the grey zone correspond to a prediction not precise enough for diagnostic decision making". They identified a grey zone of PPV values between 9 and 13%, between which fluid responsiveness cannot be reliably predicted.

Advanced Standalone Monitors

There exist a plethora of advanced haemodynamic monitors based on different technologies –

- Pulmonary artery catheters (PAC)
- Transoesophageal echo (TOE)
- Bioreactance
  - NICOM
- Continuous wave doppler
  - CardioQ (oesophageal doppler)
  - USCOM
- Pulse contour analysis
  - FloTrac
  - PiCCO
  - LiDCO, LiDCO-rapid
  - PRAM-MostCare
- Nonivasive pulse contour
  - Nexfin
- Partial carbon dioxide rebreathing
  - NICO

These offer an assessment of cardiac output as well as other haemodynamic parameters. Two of the most popular devices will be reviewed.

Pulse contour analysis – FloTrac

Advantages[17] –

- Continuous cardiac output
- Mini-invasive, "self-calibrating"

Disadvantages –

- Accuracy of output has been a concern
- Sensitive to changes in arterial resistance
- Requires a specific arterial pressure sensor

The FloTrac / Vigileo (now EV1000) system (Edwards Lifesciences) was introduced in 2005.[18] It utilises a pulse contour analysis technique that allegedly negates the need for external calibration and is therefore quick to use and less invasive. The proprietary waveform analysis calculates vascular impedance by combining the empirical estimation of large vessel elastance from patient demographic data with the quantitative analysis of the arterial waveform to determine dynamic resistance. These combined values generate a so-called auto-calibration scaling factor (Chi factor). The Chi factor, along

with a whole waveform assessment of pulse pressure, is then used to derive stroke volume. Further derivation provides other haemodynamic variables including stroke volume variability, a useful indicator of volume responsiveness, and cardiac output.

Stroke volume variation (SVV) is expressed as a percentage and is analogous to PPV. Values above 13% are considered to indicate fluid responsiveness. Hofer et al found that SVV reliably predicted fluid responsiveness.[19] The determination of SVV suffers from the same limitations as PPV described above. In determining fluid responsiveness using SVV, the area under the ROC curve has been calculated as 0.84 (95% CI 0.78-0.88).[20]

Slagt et al recently performed a systematic review of 65 CO validation studies with 2,234 patients and 44,592 data points.[18] They examined the performance of the FloTrac / Vigileo in three separate patient groups – general critical care including post-surgical patients and critical care patients with presumed normodynamics, a group of post-cardiac patients with presumed hypodynamics, and a group of patients with liver disease or sepsis with presumed hyperdynamics. They found that SVV predicted fluid responsiveness in 85% of studies examined and that "the accuracy and precision of the FloTrac / Vigileo system can be regarded as sufficient for rountine clinical use in hypo- or normodynamic conditions in the absence of large changes in vascular tone" with percentage errors at 30% or lower.

The relation between pulse pressure (PP) and SV is less fixed in hyperdynamic and vasodilated states such as liver disease, liver surgery, or septic shock. Moving down the arterial tree, PP normally increases, but in hyperdynamic conditions, the opposite occurs, leading to an underestimation of SV. Software releases 3 and 4 have sought to address this. The paper by Slagt et al reviewed software versions up to 3.02 (version 4 was released in 2014) and found performance in hyperdynamic states to be inadequate, that SVV may still be "useful" in predicting fluid responsiveness but that trending capacity "remains affected by changes in vascular tone."

Oesophageal Doppler – CardioQ

Advantages[17] –

- Less invasive than arterial-based systems

Disadvantages –

- Requires frequent manipulation for proper position
- Operator dependent[15]
- Validation data is old and little on CO trending[21]

Less invasive hemodynamic monitoring systems started in the 1990s.[17] One of the first systems to be described and developed was an oesophageal doppler system allowing for non-invasive monitoring of CO. The CardioQ-ODM (Oesophageal Doppler Monitor; Deltex Medical) utilises a probe placed in the oesophagus and aimed at the descending aorta. The waveform is very dependent on correct positioning and to optimise the signal requires frequent adjustments of depth, orientation, and gain. The CardioQ-ODM calculates the aortic cross-sectional area using a nomogram based on the patient's age, height and weight.[22] Calculation of cardiac output is dependent on five assumptions –

1. The distribution of blood caudally to the descending aorta and rosterally to the great vessels and coronary arteries maintains a constant ratio of 70% to 30%
2. That a flat velocity profile exists within the aorta
3. The estimated cross-sectional area is close to the mean systolic diameter
4. There is negligible diastolic blood flow
5. The velocity of blood flow in the aorta is measured accurately

The monitor offers a number of haemodynamic variables which can be used to guide treatment –

- Peak velocity – the peak velocity of blood in the aorta gives a good estimation of myocardial contractility
- Stroke volume (and thus CO) – stroke distance is the area under the velocity-time waveform; when multiplied by the aortic diameter this gives an estimate of the stroke volume. The stroke volume is usually averaged over a number of beats
- Corrected flow time (FTc) – the flow time is the duration of forward flow in the aorta. The flow time varies with heart rate and can be corrected to 60bpm. Anything that impedes filling or emptying of the left ventricle will cause a reduction in FTc. Most commonly this is seen in hypovolaemia

Unlike pulse contour analysis which require modelling of the circulation to produce an algorithm that converts pressure changes to blood flow, ultrasound measures blood flow directly. However the CardioQ does not measure true CO. Most of its validation data pre-date percentage error.[21] Dark and Singer's meta-analysis of 2004 used a statistic called "percentage of clinical agreement" (PCA) based on the number of data pairs that were within ±15% of mean bias.[23] Re-evaluation of data from this paper using mean CO and limits of agreement shows that the percentage error for many of these oesophageal Doppler studies was 40–50%.[21] However, the CardioQ-ODM does seem to track changes in CO, although there is little published data other than that found in a study by Valtier et al.[24]

Several studies (with small numbers) have shown a positive impact on postoperative complications in patients undergoing high-risk surgery.[17] Based on eight studies (six funded to some extent by the manufacturer) the UK National Health Service's National Institute for Heath and Clinical Excellence (NICE) has recommended the use of the CardioQ-ODM in high-risk surgical patients.25

Passive Leg Raise

A passive leg raise results in the transfer of blood from the legs and abdominal compartments and can be used to assess preload responsiveness. It is essentially a reversible autotransfusion with 45° leg elevation equivalent to a 500ml fluid bolus.12 The manoeuvre has been shown to be highly predictive with an area under the ROC curve of 0.95. It is useful in ED, ward and ICU settings, especially in patients with cardiac arrhythmias and spontaneous ventilation where dynamic parameters lose their predictive ability. It also avoids unnecessary fluids. Obviously utility in theatre is limited. Intra-abdominal hypertension (>16mmHg) impairs venous return and the ability to detect fluid responsiveness.

A Final Word

The use of the parameters and devices described above is often linked to a protocol for fluid and haemodynamic management – goal directed therapy. Despite all the advances in recent years it remains unclear which device to use in which setting.17 Crucially patient outcome data is missing. But can we expect a haemodynamic monitoring system by itself to affect outcome? After all, pulse oximetry has been evaluated in randomised trials including over 20,000 patients26 and has never been shown to improve patient outcome.

Competing Interests

I am a member of the medical advisory board of Edwards Lifesciences. I have previously been contracted for web development for the JAFA Trust, the organisers of this meeting.

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ERAS for colorectal surgery; an integrated approach

Prof Dileep Lobo
University of Nottingham, UK

To view this document in full, go to 2015 ACS Resources:

ERAS Guidelines Colon

ERAS Guidelines Rectum

All about ERAS

Goal Directed Therapy in ERAS: a critical appraisal of the literature

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Traditional liberal intraoperative fluid management for abdominal surgery was based on flawed historical concepts developed with flawed studies. With the advent of Enhanced Recovery After Surgery (ERAS) and avoidance of chemical and mechanical bowel preparation, fluid and electrolyte resuscitation following bowel preparation is unnecessary or at least attenuated significantly. There is also increasing realisation of the limitations of traditional approaches to fluid management. Thus:

- Overnight starvation does not result in a significant volume deficit
- "Third-space losses" as defined by Shires do not exist and actually represent an indicator artefact
- Bowel evaporative losses are actually minimal
- Evidence from the obstetric anaesthetic literature suggests that volume preloading of neuraxial blocks is ineffective at improving blood pressure or preventing the need for vasopressors.

Two concurrent streams of research evolved in the late 1990's to mid 2000's looking at intraoperative fluid management. One was from surgical research into nutritional sodium management and fluid restriction. This has matured into the "goal" of neutral fluid balance. The other was from anaesthetic/critical care work, refining earlier work by Shoemaker on superoptimisation. This focused on dynamic monitors for fluid status & optimal tissue perfusion using Oesophageal Doppler & Arterial pressure waveform analysers (Goal Directed Fluid Therapy-GDFT or GDT). Both strategies showed benefits over traditional "liberal" fluid management but meta-analysis reveals significant heterogeneity between studies that makes comparison and implementation difficult.

More recent meta-analyses (including these studies) have highlighted the lack of dominance of either strategy though overall a trend in favour of GDT may be implied. It is as yet unproven in a wider context and when compared to more modern fluid management concepts.

Because of uncertainty as to which particular strategy is more beneficial in elective colorectal procedures, recent studies (including two conducted in Australasia) have examined which of these regimes might be the better. These have shown that, within ERAS protocols utilising neutral fluid balance regimes, GDT conveys no added benefit in all comers and potentially harm in lower risk patients. Similarly, the primary outcomes and methods utilised in these studies are heterogeneous and difficult to compare. This is a valid criticism across the entire GDT literature with 118 differing goal/method combinations found in a recent systematic review of GDT.

In anaesthetic practice, the definition of what should be the "goal" is unclear within the context of a dynamically changing intraoperative state (e.g. laparoscopic surgery, changing pulmonary compliance, vasopressor adjustments & epidural sympatholysis). A recent observational study of healthy patients calls into question what exactly constitutes "optimal fluid balance". The original designers of GDT algorithms aimed for stroke volume maximisation to maximise DO2 to the GI tissues. Maximisation does not necessarily reflect optimisation of wider fluid balance. Given the evolution of our understanding of the microcirculation and the glycocaylx model, the "top of the starling curve" may in fact stray too close to the point at which atrial naturietic peptide (ANP) is released, allowing rapid increases in extravascular lung water and tissue oedema. These venous curves also vary with endothelial damage (e.g. sepsis). Whilst this would be expected to support the use of GDT in acute/unwell cases, there is very little evidence to support this indication (presumably due to difficulty in study design). It further questions where the "sweet spot"/"goal" should be of balancing cardiac output v tissue oedema and where optimal DO2 might lie.

More recent meta-analyses co-published with the Optimise study and including the studies in modern fluid administration context have highlighted the lack of dominance of either strategy though overall a trend in favour of GDT may be implied. However, this is as yet unproven in a wider context and when compared to these more modern fluid management concepts.

Multiple questions hence remains unresolved: What is "optimal fluid balance" and what goal do we aim for intraoperatively? Does the surrogate outcome of "fluid responsiveness" translate into "actually requiring fluid" and improved clinical

outcomes? What fluid management algorithms are most suitable now that starch-based colloids are falling from favour-given that GDT protocols have all been constructed and largely studied with starch or gelatin based colloid solutions? Which patient subgroups are likely to benefit from GDT?

It is the opinion of the presenter that the failure of GDT in recent studies to show significant improvements does not necessarily reflect a failure in the concept but rather a failure in implementation due to over simplification of the process and erroneous assumptions in physiology. It is also likely that wider implementation of a complex intervention such as this into wider clinical practice, coupled with the learning curves required, will reduce the expected clinical impact, especially during the early "learning phase".

GDT represented an improvement in fluid management compared with traditional liberal regimes but when compared with a neutral fluid balance protocol (a different sort of goal direction), such improvements are harder to demonstrate. It may be that GDT should be reserved for patient subgroups such as the physiologically unwell. Very little work has been conducted in acute colorectal surgery in either fixed fluid regimes or GDT. Extrapolating the results of elective surgical studies into patients with activated surgical stress responses and lack of preoperative optimisation may prove problematic.

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The dilemma of transfusion threshold

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Introduction

Until recently, the decision to transfuse red blood cell (RBC) was based on traditional "10/30 rule" – Hb should be kept above >10g/dL and Hct >30%.[1] However, concern regarding transfusion-related adverse events has resulted in a re-examination of this practice.[2] In the past two decades numerous trials have attempted to identify the optimal threshold for blood transfusion.[3-9] The common theme of these trials is that the benefits of blood transfusion should be considered along with the potential risks. This presentation will review the current literature on the blood transfusion threshold.

Why do we want to transfuse?

1. Delivery of oxygen

DO_2 = cardiac output x arterial oxygen content.

DO_2 is delivery of oxygen to tissues and arterial oxygen content is the multiple of [Hb] and oxygen saturation. In presence of anaemia, the arterial oxygen content would fall and the DO_2 will fall proportionately. And this may be the main reason a clinician would give transfusion.

There are other things that need to be considered:

1. At rest there is a large reserve in oxygen delivery (can be increased 4 fold).
2. In order to compensate for the fall in arterial oxygen content, there will be an increase in Cardiac Output (CO), an increase in tissue oxygen extraction ratio, and shifting of oxygen-dissociation curve to right to aid offloading of oxygen at tissue level.
3. Provided intravascular volume is maintained, oxygen delivery theoretically will be adequate until the haematocrit falls below 10%.
4. However this is largely theoretical and most sick patients will not be able to compensate by effectively increasing CO. In addition, delivery of oxygen may become more dependent on arterial oxygen content due to pathological reason altering the O_2 extraction ratio in presence of lactate.

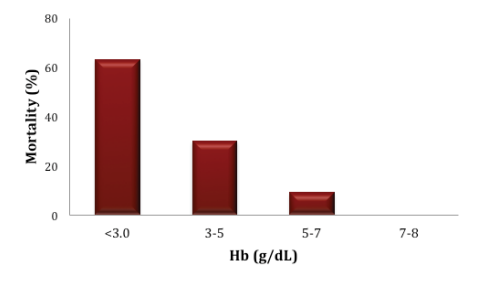
2. Anaemia is associated with increased mortality and morbidity

Pre-operative anaemia is associated with increased mortality, although there is no current evidence that correction of anaemia improves mortality.

There are many studies that demonstrate that even mild anaemia is unfavourable pre-operatively and the mortality rate could go as high as 42% in patients having hip fracture surgeries (FOCUS trial).[10] This was quite well proven from the study of 1958 Jehovah's Witness (JW) patients. Odds of death rose as the preoperative Hb fell and this was much higher in presence of underlying cardiovascular disease.[11]

Interestingly, post-op Hb had a different effect on mortality. In a subset of 300 patients with very low post-operative Hb, it was shown that post-operative Hb as low as 70-80 appeared to have no immediate adverse effect on mortality. Mortality proportionately increased below Hb of 7g/dL. **See figure 1.**

Figure 1. *Association between post-operative Hb and mortality*



Why do we want to avoid transfusion?

Risks and potential long-term complications of transfusion are very well known

- Infection (viruses, bacteria, parasites)
- Transfusion mediated immunosuppression
- Allergic and immune transfusion reaction
- Immunomodulation with recurrence in cancer
- Volume overload
- Hyperkalaemia
- Cost

What should the transfusion threshold be?

It would be extremely difficult to suggest a single number for all situations. The suggested threshold will be a recommendation based on available evidence up to date. It is vital not to forget we are clinicians and assessment of patients is vital component of the decision-making.

- Current clinical status
- Co-morbidities especially cardiovascular disease
- Presence of acute coronary syndrome
- Patient's view of transfusion

There are currently at least 8 recommendations available suggesting transfusion guidelines:

- American Society of Anesthesiology
- British Committee for Standards in Hematology
- Australian and New Zealand Society of Blood Transfusion
- Eastern Association for Surgery of Trauma (EAST) and the American College of Critical Care Medicine of the Society of Critical Care Medicine (SCCM)
- European Society of Cardiology (ESC)
- Society of Thoracic Surgeons and the Society of Cardiovascular Anesthesiologists
- AABB (formerly the American Association of Blood Banks)
- American College of Physicians

General recommendation:

Hb (g/dL)	
<6	Transfusion recommended except in exceptional circumstance
6-7	Transfusion generally indicated
7-8	Transfusion should be considered post-op including those with stable CVD after evaluating patient
8-10	Transfusion generally not indicated, but should be considered for some population (e.g. symptomatic anaemia, ongoing bleeding, ACS with ischaemia)
>10	Transfusion not indicated

These recommendations are based on a number of clinical studies and systematic reviews that have been published in the last 5 years.

Cochrane systematic review 2012[12]

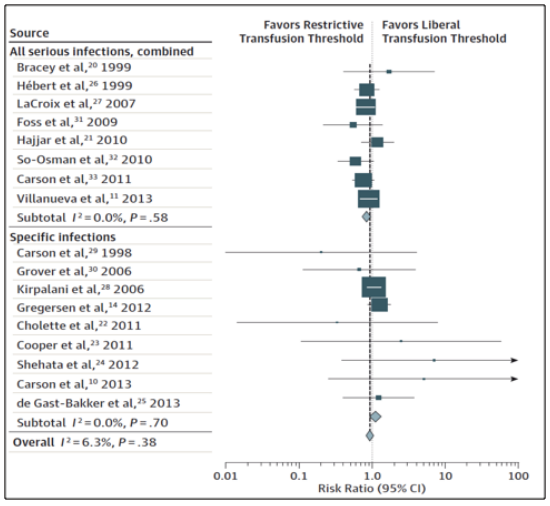
- 19 RCTs and 6264 patients
- Compared higher (10g/dL) versus lower (7-8 g/dL) transfusion thresholds
- Restrictive strategies resulted in:
  - 39% reduction in transfusion rate
  - 1.19 less RBC units/patient
  - Trend towards a lower 30-day mortality (RR 0.85; 95% 0.70-1.03)
  - Trend towards a lower overall infection rate (RR 0.81; 95% 0.66-1.00)
  - No difference in functional recovery
  - No difference in hospital/ICU length of stay
  - No increased risk of MI when all trials were included

Health care-associated infection after red blood cell transfusion: a systematic review and meta-analysis 2014[13]

- 17 trials and 7456 hospitalised patients
- Primary outcome - infection
- No difference in all infections rate (RR 0.92; 95% CI 0.82-1.04).
- Serious infections - Lower risk with restrictive strategy (RR 0.84; 95% CI 0.73-0.96)
- Subset analysis of 7 trial where all RBCs were *leukodepleted* - findings were consistent with infection happening less frequently with restrictive strategies (RR 0.83; 95% CI 0.69-0.99)

- See figure 2

Figure 2. **Meta-analysis: Transfusion strage and infection risks (13)**



Based on these results, it can be concluded that restrictive group is at least non-inferior and having Hb threshold of 7-8 g/dL is adequate for most haemodynamically stable medical and surgical patients.

Specific circumstances

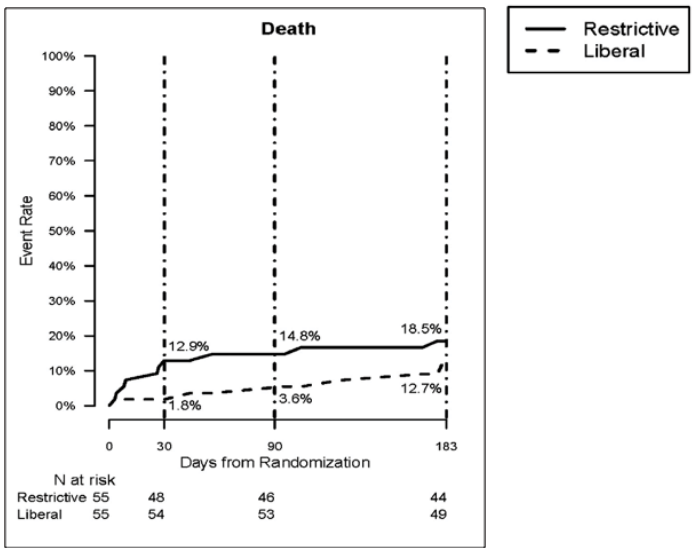
Acute Coronary Syndrome (ACS)

Transfusion threshold is largely unresolved.[14] Having a higher transfusion threshold did not confer improved mortality rates (Kansagara et al). In a more recent pilot trial of 110 patients with ACS[15], a threshold of 10g/dL provided better mortality rate @ 30 days compared to restrictive strategy (8 g/dL) (97% vs. 87%). **See figure 3**. But some experts in other papers prefer to have slightly lower threshold than this.

Based on limited evidence, the recommendation is:

Patients with Hb is <8 g/dL should have blood transfusion. Transfusion should be considered if Hb between 8-10. If the patient has ongoing ischaemia or symptoms, the recommendation is to maintain Hb >10 g/dL.

Figure 3. **Liberal vs. Restrictive strategy and mortality rate in patients with ACS**



Stable Ischaemic Heart Disease (IHD)

Functional Outcomes in Cardiovascular patient Undergoing Surgical Hip Fracture Repair, FOCUS trial10

- 2016 patients with pre-existing or risk factor of cardiovascular disease post-hip fracture surgeries
- All patients >50 years old (mean 82)
- Comparing liberal (10g/dL) vs. Restrictive (8g/dL)
- Primary outcome: Mortality or inability to walk 10 feet at the Day 60
- Secondary outcomes: Combined outcome of in-hospital MI, unstable angina, or death
- Similar mortality @ day 30 and 3 years.
- Similar rates of composite endpoint for ACS or death
- Separately,
    - Restrictive approach was associated with trend towards higher rate of MI
      - 3.8% vs 2.3% RR = 1.65; 95% CI 0.99-2.75
    - Liberal group associated with trend towards higher in-hospital mortality
      - 1.4% vs 2.0% OR 1.55; 99% CI 0.58-3.56

TRICC study (~800 ICU patients with anaemia and included CVD) which compared 10 g/dL vs 7 g/dL thresholds. Restrictive strategy was associated with lower mortality.

Based on this transfusion threshold of 8g/dL is acceptable

Symptomatic patients

Recommendation is that all symptomatic anaemia should be treated with transfusion if Hb <10 g/dL.[15,16] Symptoms may include:

- Symptoms of MI
- Orthostatic hypotension
- Tachycardia
- Unresponsive to fluid replacement

NB - generally symptoms of chronic anaemia (weakness, low exercise tolerance) are non-specific and not considered an indication for RBC transfusion.

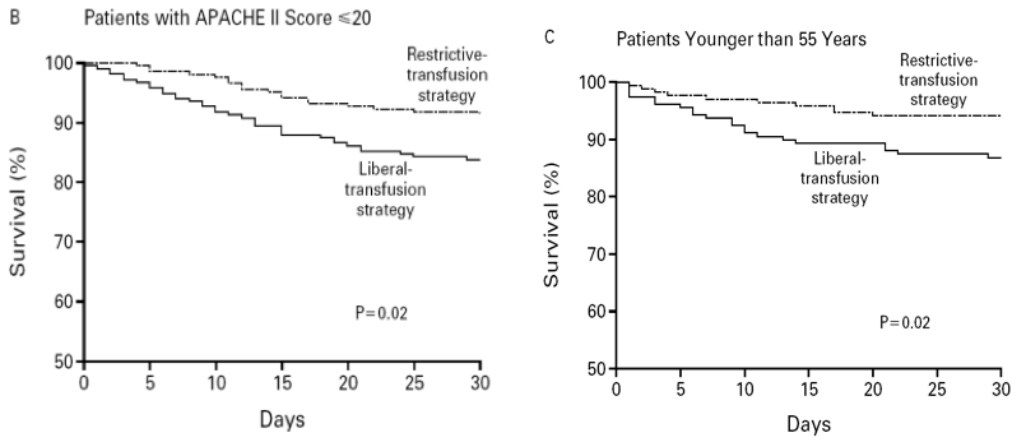
ICU/Septic shock patients

Restrictive strategy appears to be safe in medical patients in an ICU.

Transfusion requirements in critical care (TRICC trial)[17]

- Multicentre, Randomised controlled trial
- 838 patients
- Restrictive (7 g/dL) vs. Liberal (10 g/dL)
- Overall mortality similar
- Patients who were less acutely unwell and age <55 – Restrictive strategy was associated with better mortality outcome at 30 days. **(See figure 4)**.
- Morbidities were also lower in the restrictive transfusion strategy group
    - Myocardial Infarction (0.7% vs 2.9%)
    - Pulmonary Oedema (5.3% vs 10.7%)

Figure 4. **Comparing survival rates between lberal and restrictive strategies in TRICC trials**



Use of threshold of 7g/dL was also shown to be safe in patients with septic shock. The TRISS trial (transfusion requirements in septic shock) randomly assigned 998 patients with Hb <9 g/dL to restrictive (7 g/dL) and liberal (9 g/dL) strategy groups. Mortality and morbidities were similar.18

Summary

- Pre-operative anaemia is associated with poor outcome. It is unclear correcting this preoperatively with transfusion is associated with better outcome.

- Post-operative anaemia Hb as low as 7g/dL is generally not associated with increased mortality. Hb <7 g/dL is associated with increased mortality.

- Restrictive transfusion with threshold of 7-8 g/dL is:
  - Safe in most populations including ICU, septic patients and patients with stable ischaemic heart disease
  - At least non-inferior, may potentially be associated with less mortality and morbidity
  - Reduces the transfusion rate by 1/3 and amount of RBC consumption by half.

- Exceptions
  - Symptomatic patient should be transfused to at least 8g/dL and consider transfusing upto 10 g/dL.
  - Patient with ACS and ongoing ischaemia, liberal transfusion may provide better mortality outcome

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Fluids, blood and the microcirculation

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Most anaesthetists consider red cells an effective volume expander. Despite concerns about the lack of effective oxygen delivery from aged red cells, the product is often given when patients are hypotensive and tachycardic, and the general impression is that it supports the intravascular volume better than crystalloids and synthetic colloids. Studies have recently been undertaken to define what causes this improved effect, and how it may give insights into future colloid developments.

As haemodilution occurs, the characteristics of flow through the microcirculation changes. Flow through capillaries depends on the hydrostatic pressure to a certain degree, but more importantly on the tone of the pre-capillary arterioles, which dilate and constrict to preferentially allow flow to certain areas. Flexible red cells move through these so that red cells flow no more than 2 cell diameters away from cells, allowing oxygen diffusion to occur. In most circumstances oxygen delivery is not dependent of capillary flow, and this can be reduced significantly without hypoxia occurring. It can also increase in the presence of haemodilution, and oxygen delivery is maintained.

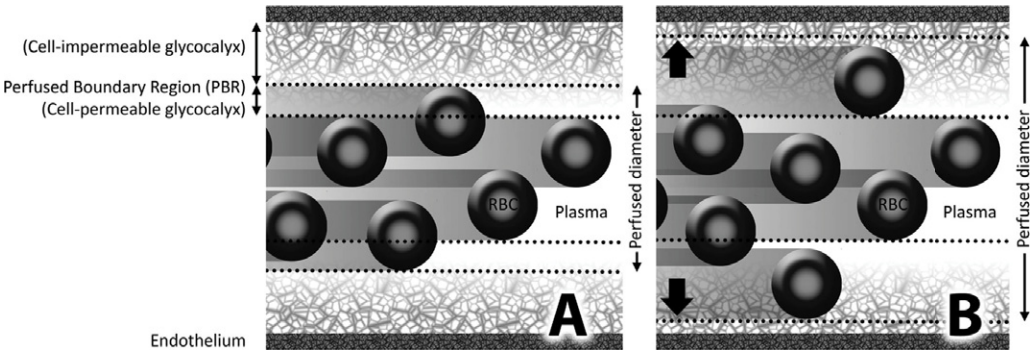
It is important to remember that capillary flow is not pulsatile, and arteriolar tone has a major effect on the rate of transcapillary transit. In addition, restoration of the microcirculation does not imply microcirculatory delivery of oxygen to tissues. Heterogeneity of blood flow appears to be a key characteristic of the disease state, even when adequate systemic oxygen carrying capacity is maintained.

Capillary diameter is also a function of the glycocalyx, and the endothelial cell diameter. In shock states these can both change, and impede capillary flow. This can have a profound effect on Functional Capillary Density. (FCD) This is the calculated capillary vessel numbers that are functioning with flow multiplied by the velocity.

Fluid viscosity is caused by the frictional (or viscous) resistance between the moving blood and the stationary vessel wall. Many suspended particulates in a fluid exhibit non-Newtonian behaviour, where the viscosity decreases when the velocity increases when travelling through a tube. Paint and blood are two examples.

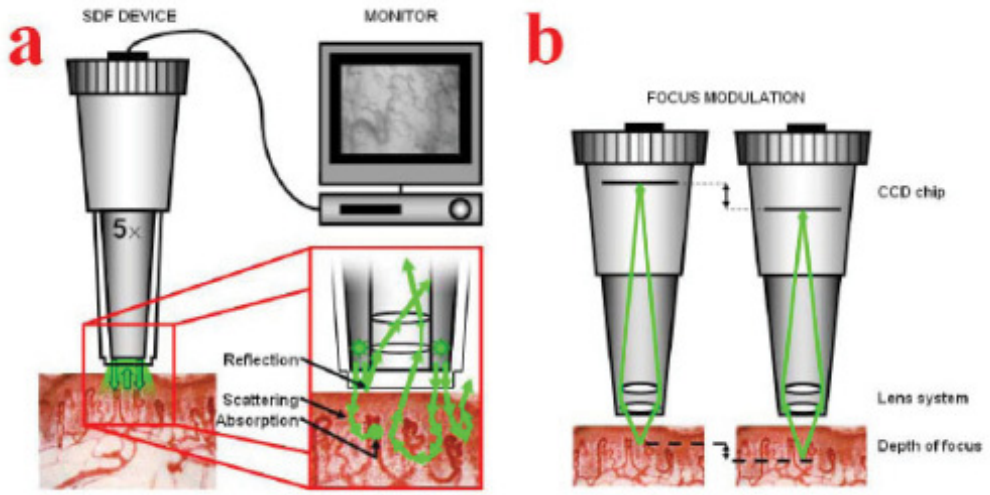
$$\tau = \frac{4\eta Q}{\pi R^3}$$

τ = fluid shear stress
η = blood viscosity
R = internal radius of vessel
Q = blood flow velocity

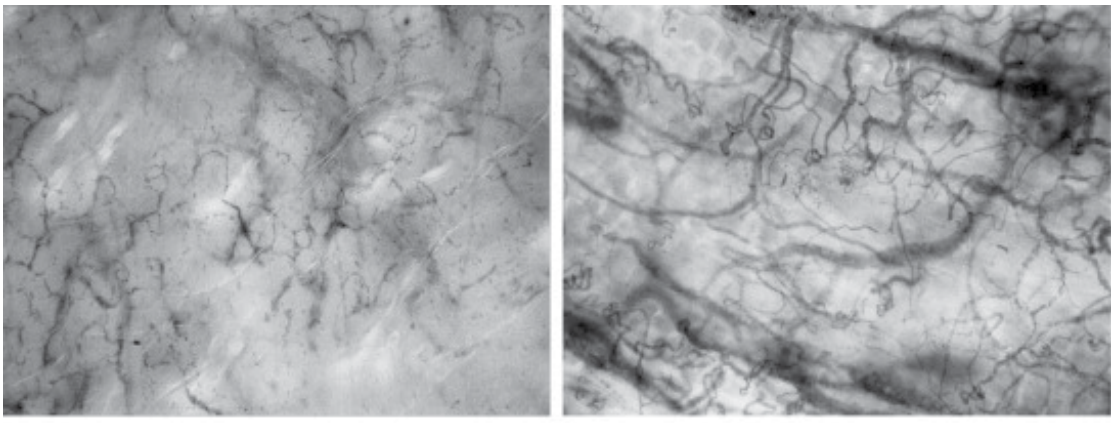


As blood acts as a Non Newtonian fluid; as its velocity increases, the spread of red cells becomes more aligned with the central portion of the flow. This obviously is more pronounced in anaemic patients. This reduces the shear forces on the lateral vessel wall. As the components of viscosity are from red cells and plasma (plasma proteins and fibrinogen), reductions in each of these will reduce the viscosity of the fluid. This reduces the outward fluid shear pressure on the arteriole, which reduces NO production and reduces arteriolar relaxation. This in turn reduces capillary flow, and induces homogenous hypoperfusion.

The Hamster chamber window model has been used as an experimental model to look at flow characteristics in hypoperfusion.



Functional capillary density (FCD) is one of the parameters obtained by microscopy using illumination of thin tissue layers. FCD, defined as the length of red cell-perfused capillaries per observation area (cm-1), has been used as an indicator of the quality of tissue perfusion in various animal models. Quantitative analysis of FCD in randomly selected regions of the tissue is performed by means of a computer-assisted video analysis system, which allows calculation of the length of RBC-perfused capillaries.



Low MFI, low capillary density

High MFI, high capillary density

Opportunities exist to use this information to create new and novel fluids that increase plasma viscosity and improve microcirculatory flow. While dextrans and starches have high viscosity, there may be other issues with these that reduce their clinical utility. Hypertonic saline, deoxygenated haemoglobin which has been pegylated, and PEG-Albumin are all under investigation to create alternate fluids that may better protect microvascular flow.

In addition photomicroscopy may offer a monitor for fluid management at the capillary level.

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References 1. Baxter Plasma-Lyte 148 Product Information. Date of most recent amendment, July 2014. **2**. Powell-Tuck J, Gosling P, Lobo DL *et al*. British Consensus Guidelines on Intravenous Fluid Therapy for Adult Surgical Patients (GIFTASUP). March 2011.

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Material reference number: ANZ/64/14-0004

Do physiological crystalloids deliver optimal clinical outcomes?

What's the problem with using an intravenous solution with a high chloride load?

Normal saline (0.9% Sodium Chloride) has a chloride level (154 mmol/L) well above normal human serum levels (98–106 mmol/L).[1] Studies have consistently shown that moderate-to-large-volume infusions of 0.9% Sodium Chloride are associated with hyperchloraemic acidosis.[2–10] A salt excess does not accompany infusions of 'balanced' (i.e. physiological) crystalloids, as these products (e.g. Hartmann's and Plasma-Lyte 148) have sodium and chloride levels similar to plasma.[11,12]

But is hyperchloraemic acidosis clinically relevant?

A recent study of 22,851 surgical patients with normal preoperative serum chloride concentration and renal function demonstrated a 22% incidence of acute postoperative hyperchloraemia.[13] Of the 4955 patients with hyperchloraemia after surgery, 4266 (85%) patients were propensity-matched with an equal number of patients who had normochloraemia postoperatively. Patients with hyperchloraemia were at increased risk of 30-day postoperative mortality (3.0 vs 1.9%; odds ratio 1.58 [95% CI 1.25–1.98]) and had a longer median hospital stay (7.0 days [interquartile range 4.1–12.3] vs 6.3 days [interquartile range 4.0–11.3], $p < 0.01$) than those with normal postoperative serum chloride concentrations.[13] Patients with postoperative hyperchloraemia were also more likely to have postoperative renal dysfunction as defined by a 425 decrease in GFR (12.9 vs 9.2%, $P < 0.01$).[13]

What's the evidence that balanced crystalloids lead to superior patient outcomes when compared to 0.9% Sodium Chloride?

A retrospective observational study evaluated adult patients undergoing major abdominal surgery who received either 0.9% Sodium Chloride (n = 30,994) or a calcium-free, balanced crystalloid solution [Plasma-Lyte]; n = 926) on the day of surgery and found that Plasma-Lyte was associated with less post operative morbidity.[14] Postoperative infection ($p = 0.006$), renal failure requiring dialysis ($p < 0.001$), blood transfusion ($p < 0.001$), electrolyte disturbance ($p = 0.046$), acidosis investigation ($p < 0.001$) and intervention ($p = 0.02$), were all more frequent in patients receiving 0.9% Sodium Chloride.[14] This study also showed the in-hospital mortality rate to be significantly lower in the Plasma-Lyte arm compared to the group receiving 0.9% Sodium Chloride (2.9 vs 5.6%; $p < 0.0001$).[14]

While there are no large randomised trials comparing 0.9% Sodium Chloride with balanced crystalloids, a strong signal is emerging from double-blinded trials[15–17] and large observational studies[13,14,18,19] that the high chloride content in 0.9% Sodium Chloride leads to numerous adverse pathophysiological effects (see Box 1) – and hence, worse patient outcomes.[20] These include an increased incidence of acute kidney injury (and need for renal replacement therapy) and pathological hyperchloraemia, which may increase postoperative mortality.[20] These same effects are not observed with balanced crystalloids.[20] A thorough review of the evidence cited here can be found in Lobo and Awad, 2014.[20]

Box 1. Adverse events related to intravenous therapy with 0.9% Sodium Chloride when compared with balanced crystalloids

Metabolic	• Hyperchloraemic acidosis • ↑ Need for buffers to correct acidosis
Body water	• Possible damage to endothelial glycocalyx • ↑ Intestinal fluid volume leading to oedema
Renal	• Renal oedema and capsular stretch leading to intrarenal tissue hypertension • Renal vasoconstriction, ↓ renal blood flow and renal tissue perfusion • ↓ Glomerular filtration rate, urine volume and sodium excretion
Gastrointestinal	• Gastrointestinal oedema, intestinal stretch • Illeus, impaired anastomotic healing
Haematological	• ↑ Intraoperative blood loss • ↑ Need for blood product transfusion
Clinical outcomes	• ↑ Post-operative complications • ↑ Mortality • ↑Incidence of acute kidney injury and need for renal replacement surgery

Evidence collected from animal studies, healthy volunteers, small-randomised clinical trials and large patient cohort studies, and thus cannot be regarded as Grade A. Adapted from Lobo and Awad, 2014 (see paper for full evidence review).

How do the physiological profiles of commonly used crystalloids differ?[1,11–12,22–23]

	mmol/L								Tonicity*
	Cations				Anions				
	Na^+	K^+	Ca^{++}	Mg^{++}	Cl^-	Acetate	Lactate	Gluconate	
0.9% SODIUM CHLORIDE	154	-	-	-	154	-	-	-	Isotonic
HARTMANN'S	131	5.0	2.0	-	111	-	29	-	Isotonic
LACTATED RINGERS	130	4.0	1.5	-	109	-	28	-	Isotonic
PLASMA-LYTE 148	140	5.0	0	1.5	98	27	-	23	Isotonic
PLASMA	136–145	3.5–5.0	2.2–2.6	0.8–1.2	98–106	Bicarbonate 21–30			Isotonic

No calcium
Compatible with blood

Physiological levels of sodium and chloride

Dual bicarbonate precursors: acetate and gluconate

Physiological osmolarity

0.9% Sodium Chloride, Hartmann's, Lactated Ringers and Plasma-Lyte 148 differ in both the type and amount of electrolytes they contain. Unlike 0.9% Sodium Chloride, Hartmann's, Lactated Ringers and Plasma-Lyte 148 are multiple electrolyte solutions with one or more bicarbonate precursors.[11,12] It can be seen from the figure to the left that Plasma-Lyte 148's physiological profile is closest to that of normal human plasma. It also does not contain calcium and, therefore, can be administered before, during or after blood administration.[4,12,21]

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US-1140471	K-3 Lancet 21G x 70mm
US-1140761	K-3 Lancet 21G x 90mm
US-1150371	K-3 Lancet 22G x 38mm
US-1150971	K-3 Lancet 22G x 70mm
US-1151261	K-3 Lancet 22G x 90mm
US-1160671	K-3 Lancet 23G x 70mm
US-1160961	K-3 Lancet 23G x 90mm
US-1182961	K-3 Lancet 25G x 90mm
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US-5331971	Pencil Point 24G x 90mm *w/ introducer* 20G x 38mm
US-5332071	Pencil Point 25G x 90mm *w/ introducer* 20G x 38mm
US-5332171	Pencil Point 25G x 120mm *w/ introducer* 20G x 38mm
US-5332271	Pencil Point 25G x 150mm *w/ introducer* 20G x 38mm
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US-5332471	Pencil Point 26G x 120mm *w/ introducer* 20G x 38mm
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Neostigmine or a neostigmine/ anticholinergic combination is contraindicated e.g. IHD, morbid obesity, COPD

Severe neuromuscular degenerative disease where neuromuscular block is required

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