



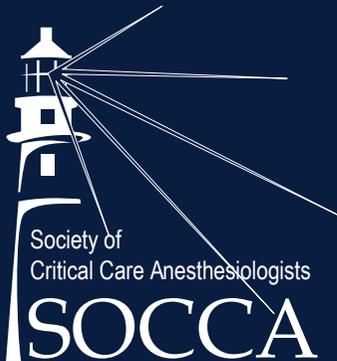
Syllabus

SOCCA

Presented prior to the IARS 2014 Annual Meeting and International Science Symposium

27th Annual Meeting and Critical Care Update

May 16, 2014 | Fairmont The Queen Elizabeth Hotel | Montréal, Canada





The Society of Critical Care Anesthesiologists
would like to thank the following exhibitors
of the SOCCA 27th Annual Meeting:



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Continuing Medical Education (CME) Activity Information

Activity Overview

The Society of Critical Care Anesthesiologists 27th Annual Meeting seeks to optimize outcomes for critically ill patients and their families by providing updates and expert discussion on topics of interest to anesthesiologists practicing critical care and perioperative medicine through advancing knowledge, improving competence and enhancing performance of intensive care teams.

Target Audience

The SOCCA 27th Annual Meeting and Critical Care Update is designed for anesthesiologists in the clinical and laboratory setting who desire to improve development of anesthesiology teaching methods by engaging in an interchange of ideas as represented in this meeting.

Accreditation Statement

This activity has been planned and implemented in accordance with the accreditation requirements and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of the International Anesthesia Research Society (IARS) and the Society of Critical Care Anesthesiologists (SOCCA). The IARS is accredited by the ACCME to provide continuing medical education for physicians.

American Medical Association (AMA)

Credit Designation Statement

The International Anesthesia Research Society designates this live activity for a maximum of 7 *AMA PRA Category 1 Credits*[™]. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Educational Objectives

As a result of participation in this CME activity, learners should be able to:

- Recognize the current state of emerging knowledge and practice patterns and assess the relevance for their professional practice;
- Incorporate new knowledge from advances in anesthesiology practice into their professional practice areas; and
- Recognize gaps in their knowledge, behavior, and patient outcomes that may result in a need for additional education and training.

Disclosure

The IARS complies with ACCME Essential Areas, Standards and Policies regarding industry support of CME Activities. The IARS has implemented policies and practices with respect to the planning, implementation and presentation of this activity to identify and resolve potential conflicts of interest for all persons in a position to control content.

Session Learner Objectives

What's New in Management of Acute Pulmonary Hypertension in the ICU

- Recognize the different mechanism of acute pulmonary hypertension and its consequence on RV function.
- Develop an approach in the treatment of acute pulmonary hypertension in the ICU.

Role of Right Mechanical Heart Assist

- Recognize the risk factors for RV failure following LVAD implant.
- Review the management of RV failure.
- Evaluate the role of mechanical RV support.

Bedside Point of Care Ultrasound in Assessment of RV Function

- Recognize the pathophysiology of right ventricular failure.
- Recognize the common transthoracic views of the Right Ventricle.
- Integrate a comprehensive bedside Point of care US assessment.

Important Publications You Might Have Missed

- Identify key articles that influence medical care in critically ill patients.
- Describe the population, intervention, control group, and outcome for each study discussed.

Innovative Informatics Approaches to Improve Healthcare Delivery in the ICU

- Identify opportunities for improvement in current clinical workflows and their outcomes.
- Illustrate how to improve situational awareness and decision support with innovative informatics to impact ICU patient outcomes.
- Elucidate barriers to the use of technology to drive patient care.
- Address security concerns in the ICU with medical technology.

Tele-ICU: Optimizing Care Delivery in Under-Resourced ICUs

- Recognize the need for Tele-ICU services in the US.
- Discuss the outcome data related to Tele-ICU utilization.
- Identify what the future of Tele-ICU might look like.

The Future of Clinical Informatics in the ICU

- Recognize the existing and future electronic medical record infrastructure in critical care environments.
- Discuss lost data and the value that may exist in this data.
- Use these data systems to improve quality and conduct research.

Interactive Case Management

- List considerations for induction of anesthesia in unstable patients.
- Discuss current controversies in perioperative fluid management.
- Characterize risks in emergent blood product transfusion.

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The following program committee members/faculty have no relevant financial relationships to disclose:

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Poster Presenter

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Stephen Heitner, M.D.

Matthias Merkel, M.D., Ph.D.
Patrick L. Purdon, Ph.D.
Peter Schulman, M.D.
P. Andrew Stephens, M.D.

Disclosure

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Onyx Pharmaceuticals
4 - Actelion Pharmaceuticals. Inc.
3, 9 - Masimo
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The following program poster presenters have no relevant financial relationships to disclose:

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Peggy White, M.D.
Christopher E. White, M.D.
Edward C. Yang, M.D.
Andrew Young, M.D.



Awards

Lifetime Achievement Award

Attendees of the SOCCA 27th Annual Meeting will honor **Clifford S. Deutschman, M.S., M.D., MCCM** as this year's Lifetime Achievement Award recipient. This award recognizes Dr. Deutschman's distinguished service and outstanding contributions to critical care medicine. Dr. Deutschman's presentation is entitled "Laws and Axioms to Live By – or – Some Stuff I've Learned Along The Way".

Young Investigator Award

This award is presented annually to the individual whose research exemplifies the Society's mission to educate anesthesiologists in the care of critically ill patients and to foster the knowledge and practice of critical care medicine by anesthesiologists. The recipient of the Young Investigator Award will make an oral presentation of their work at the SOCCA 27th Annual Meeting. SOCCA is proud to announce the 2014 Young Investigator Award recipient as **Andrew Young, M.D.**, Oregon Health & Science University for his paper entitled "Focused Echocardiography During Glucagon Administration To Diagnose Beta Blocker-induced Cardiomyopathy".

Burchardi Award

Attendees of the SOCCA 27th Annual Meeting will honor **Michael F. Heine, M.D.** as the recipient of this year's Burchardi Award. This award recognizes Dr. Heine's considerable contributions to the specialty.



Program Schedule

Friday, May 16, 2014

6:30 a.m. – 5:00 p.m. **Registration**

7:30 – 8:00 a.m. **Continental Breakfast – Exhibits Open**

8:00 – 8:05 a.m. **Welcome and Introduction**
Carlee Clark, M.D.; Daryl Kor, M.D.

8:05 – 8:15 a.m. **IARS Leadership Opening Remarks**
Denise J. Wedel, M.D. – Board Chair

Session I - RV: The Forgotten Ventricle in the ICU

8:20 – 8:50 a.m. **What's New in Management of Acute Pulmonary Hypertension in the ICU**
Andre A. Denault, M.D.

8:55 – 9:25 a.m. **Role of Right Mechanical Heart Assist**
Vivek Rao, M.D., Ph.D., FRCPS

9:30 – 10:00 a.m. **Bedside Point of Care Ultrasound in Assessment of RV Function**
Massimiliano Meineri, M.D.

10:00 – 10:15 a.m. **Break and Visit with Vendors**

Session II - Celebrating Science

10:20 – 11:10 a.m. **Important Publications You Might Have Missed**
Vivek Moitra, M.D. – Moderator

Panelists:

Steven B. Greenberg, M.D.
Erin K. Hennessey, M.D.
Brian Wessman, M.D.

11:15 – 11:30 a.m. **Young Investigator Award and Poster Presentation**
“Focused Echocardiography During Glucagon Administration To Diagnose Beta Blocker-induced Cardiomyopathy”
Andrew Young, M.D.

11:35 – 11:40 a.m. **Introduction of ASA Representative**
Aryeh Shander, M.D., FCCM

11:45 – 11:55 a.m. **ASA Address**
John P. Abenstein, M.D. – ASA President-Elect

12:00 – 1:00 p.m. **Lunch and Burchardi Award**
Burchardi Award Recipient
Michael F. Heine, M.D.

Session III – Biomedical and Health Informatics in the ICU

1:00 – 1:20 p.m. **Innovative Informatics Approaches to Improve Healthcare Delivery in the ICU**
Lisa Weavind, M.D.

1:25 – 1:45 p.m. **Tele-ICU: Optimizing Care Delivery in Under-Resourced ICUs**
Ruben Azocar, M.D.

1:50 – 2:10 p.m. **The Future of Clinical Informatics in the ICU**
James Blum, M.D.

2:15 – 2:50 p.m. **Moderated Poster Session**

2:15 – 2:50 p.m. **Break and Vendor Visits**

2:55 – 3:25 p.m. **Lifetime Achievement Award Presentation**
“Laws and Axioms to Live By – or – Some Stuff I've Learned Along The Way”
Clifford S. Deutschman, M.S., M.D., MCCM

Session IV – Interactive Case Management

3:30 – 3:35 p.m. **Introduction**
Daniel R. Brown, M.D., Ph.D., FCCM - Moderator

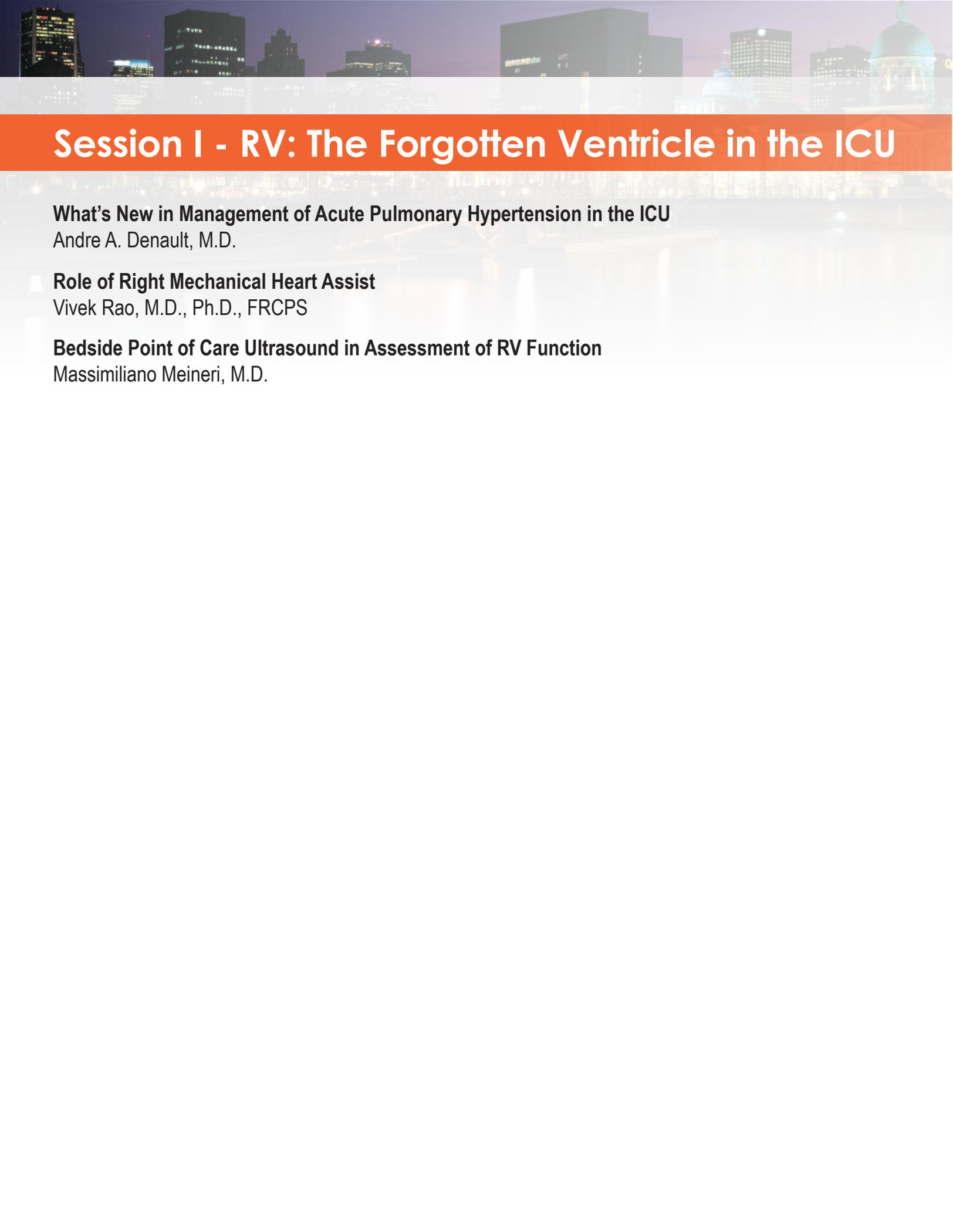
3:35 – 4:55 p.m. **Panelists:**
Brenda G. Fahy, M.D., MCCM
Michael F. O'Connor, M.D., FCCM
Avery Tung, M.D., FCCM

4:55 – 5:00 p.m. **Closing Remarks**

5:00 – 5:45 p.m. **SOCCA Annual Business Meeting**

5:00 – 6:00 p.m. **Resident/Fellow Program**

5:45 – 7:00 p.m. **Welcome Reception**

A background image of a city skyline at night, featuring various skyscrapers and a prominent domed building on the right. The scene is reflected in water in the foreground.

Session I - RV: The Forgotten Ventricle in the ICU

What's New in Management of Acute Pulmonary Hypertension in the ICU

Andre A. Denault, M.D.

Role of Right Mechanical Heart Assist

Vivek Rao, M.D., Ph.D., FRCPS

Bedside Point of Care Ultrasound in Assessment of RV Function

Massimiliano Meineri, M.D.



RV: The Forgotten Ventricle in the ICU
What's New in Management of Acute Pulmonary Hypertension in the ICU

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SOCCA
 May 16 2014
 Montreal, Canada

ICM  Université de Montréal

No disclosure



**FONDATION
 INSTITUT DE
 CARDIOLOGIE
 DE MONTREAL**

Learning objectives

- Appreciate the different mechanism of acute pulmonary hypertension and its consequence on RV function
- Develop an approach in the treatment of acute pulmonary hypertension in the ICU

Current Cardiology Reviews, 2010, 6, 1-14

Pulmonary Hypertension in Cardiac Surgery

André Denault*, Alain Deschamps, Jean-Claude Tardif, Jean Lambert and Louis Perrault

Montreal Heart Institute and Université de Montréal, Montreal, Quebec, Canada

 **Perioperative right ventricular dysfunction**

André Yvan Denault^{MD}, François Haddad^{MD}, Eric Jacobsohn^{MD}, and Alain Deschamps^{MD}

67 yo ♂ ans: altered mental state and hemodynamic instability: emergency room



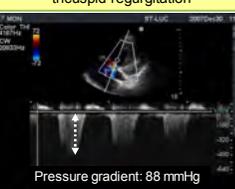

67 yo ♂ ans: altered mental state and hemodynamic instability: emergency room

Right atrial diameter measurement



7.3 cm

CW Doppler on tricuspid régurgitation



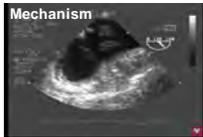
Pressure gradient: 88 mmHg

RA normal: 2.9-4.5 cm

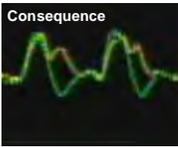
Definition



Mechanism



Consequence



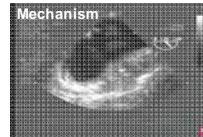
Treatment



Definition



Mechanism



Consequence



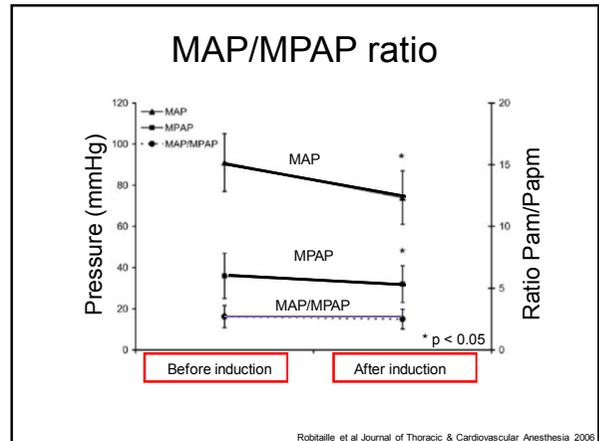
Treatment



Definition of pulmonary hypertension

- Absolute values**
 - Systolic Pap: >30 or ≥ 40 mmHg
 - Systolic Pvd: > 35 mmHg
 - Mean Pap: > 25 mmHg
 - Pulmonary vascular resistance: $>125-200$ dynes/s/cm⁵
- Relative values**
 - Systemic to pulmonary vascular resistance ratio: $> 10\%$
 - Pulmonary to systemic mean pressures: $>33-50\%$
 - Systemic to pulmonary mean pressures: (ratio MAP/MPAP: normal ≥ 4 and abnormal < 4)

Denault et al Cardiologie Contemporaine 2006



Importance of Relative Pulmonary Hypertension in Cardiac Surgery: The Mean Systemic to Pulmonary Artery Pressure Ratio

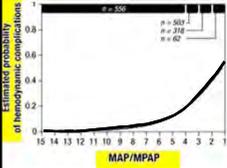
Arnaud Robitaille, MD,* André Y. Denault, MD, FRCP,* Pierre Couture, MD, FRCP,* Sylvain Bétisle, MD, FRCP,* Amrik Fortin, MSc,† Marie-Claude Guertin, PhD,† Michel Carrier, MD, FRCS,† and Raymond Martineau, MD, FRCPC*

1439 patients undergoing cardiac surgery in 1999

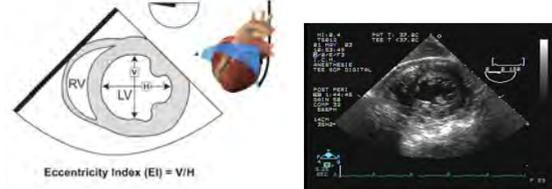
Primary end-point: composite index of death, cardiac arrest, vasoactive support >24 hours, post-operative IABP

Results: 302 patients (21%)

- MAP/MPAP ratio only hemodynamic predictor odds ratio **1.3 (95% CI: 1.1-1.5)**
- DSB: odds ratio 3.5 (95% CI: 2.5-5.1)



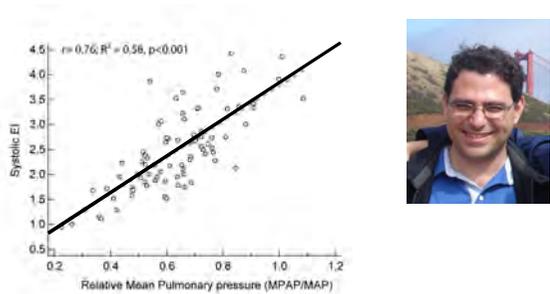
JTCVA June 2006



Eccentricity Index (EI) = V/H

Normal = 1

Independent Determinants of Septal curvature in patients with Pulmonary Arterial Hypertension: the importance of the Interventricular Pressure Gradient, relative Right Ventricular Size and Interventricular Mechanical Delay



Echocardiography 2013

46 yo ♀ aortic stenosis: PHT?

Hemodynamic profile

- Pa = 96/47 (65) mmHg
- Ppa = 34/16 (23) mmHg
- Pra = 14 mmHg
- PCWP = 16 mmHg
- CI = 2.2 L/min/m²
- SVRI = 1824 dynes • sec • cm⁵/m²
- PVRI = 286 dynes • sec • cm⁵/m²

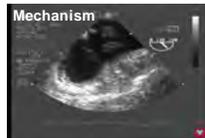
MAP/MPAP ratio
65/23 = 2.8 (Normal ≥4)

Journal of Thoracic & Cardiovascular Anesthesia 2006

Definition



Mechanism



Consequence



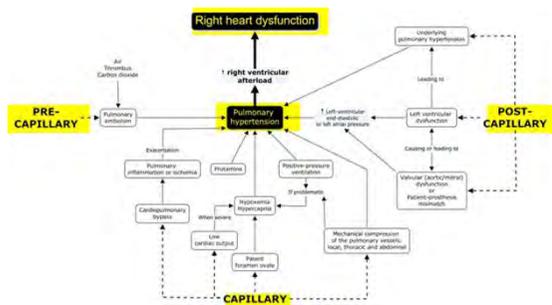
Treatment



Classification of Pulmonary Hypertension

1. Pulmonary arterial hypertension
2. Pulmonary hypertension with left heart disease
 - 2.1. Left-sided atrial or ventricular heart disease
 - 2.2. Left-sided valvular heart disease
3. Pulmonary hypertension associated with lung diseases and/or hypoxemia
 - 3.1. Chronic obstructive pulmonary disease
 - 3.2. Interstitial lung disease
 - 3.3. Sleep-disordered breathing
 - 3.4. Alveolar hypoventilation disorders
4. Pulmonary hypertension due to chronic thrombotic and/or embolic disease
5. Miscellaneous

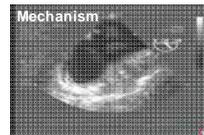
Simoneau G JACC 2004;43:5S-12S



Definition



Mechanism



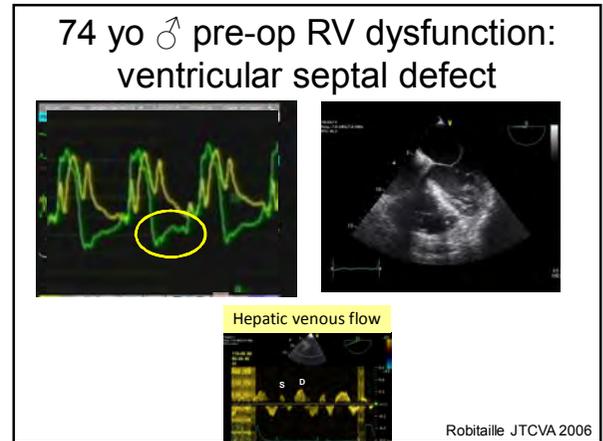
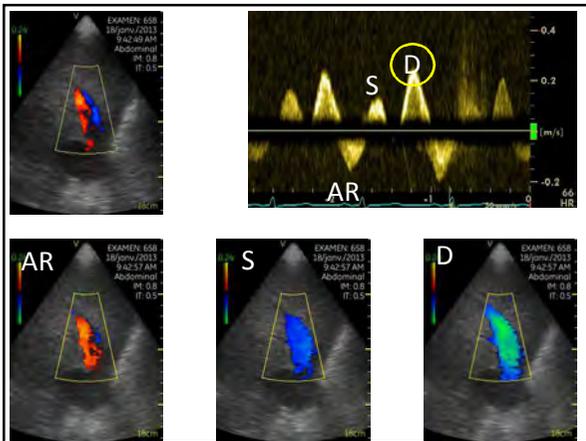
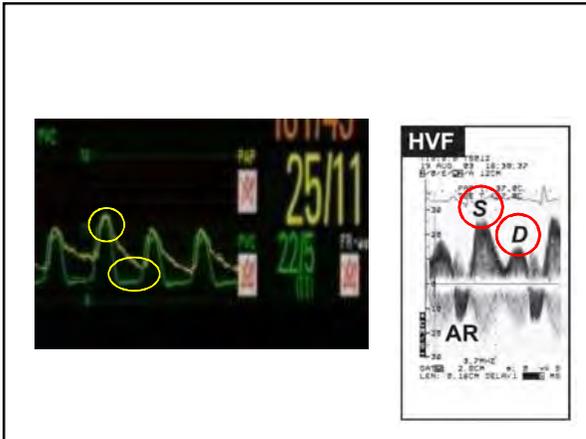
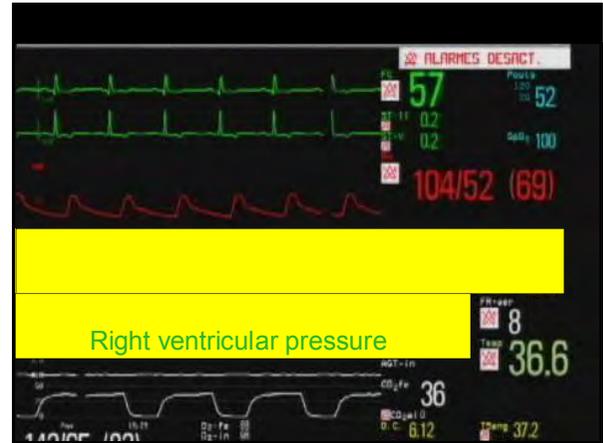
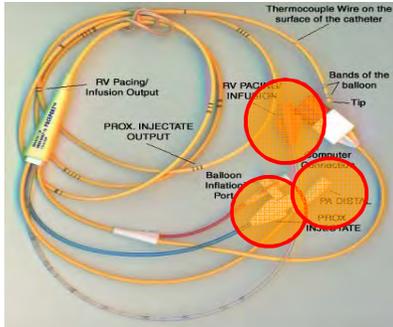
Consequences



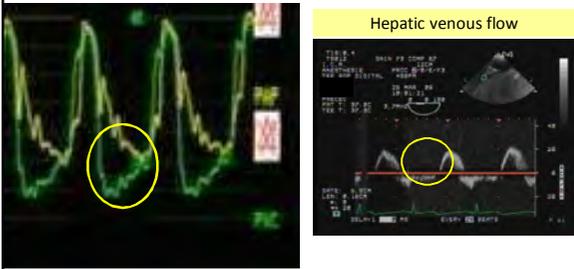
Treatment



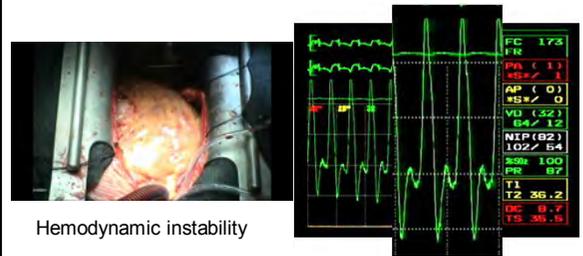
Is the standard Swan-Ganz useful in diagnosing RV dysfunction?



46 yo ♂ with RV dysfunction

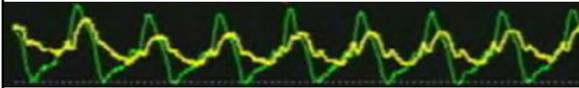


Severe RV dysfunction



Severe RV dysfunction

Diastolic equalisation



RV Pulsus tardus and ↓ pulse pressure

Before CPB

After CPB

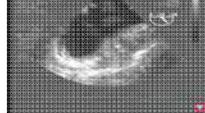


Returning on CBP

Definition



Mechanism



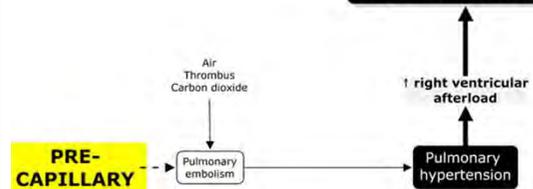
Consequence



Treatment



Right heart dysfunction



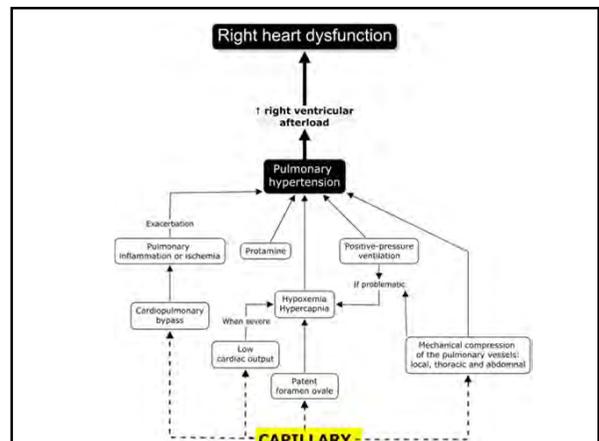
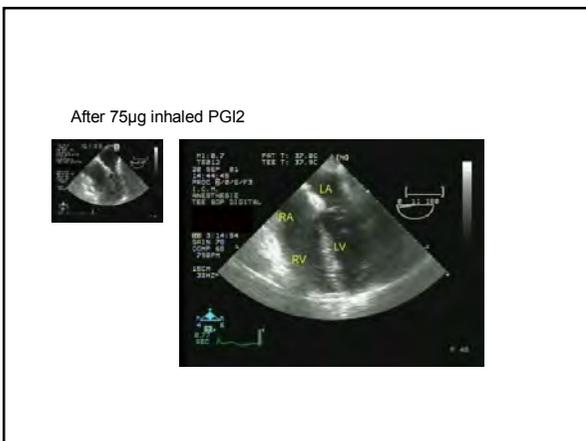
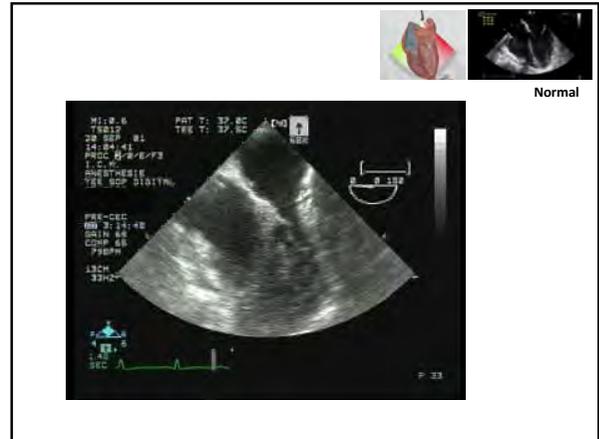
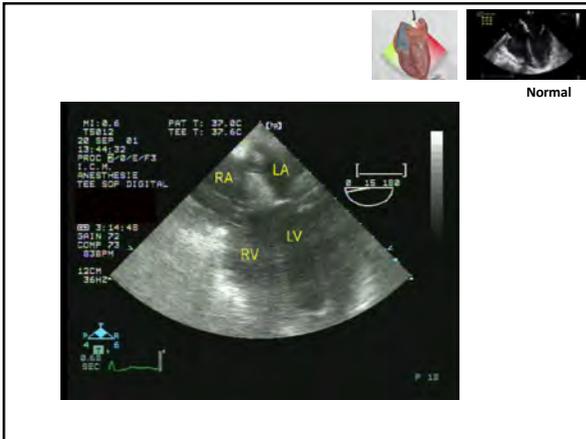
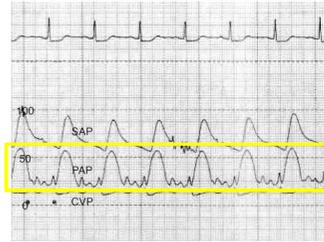
64 yo ♀ saphenoscopic surgery
Sudden increase in end-tidal CO₂

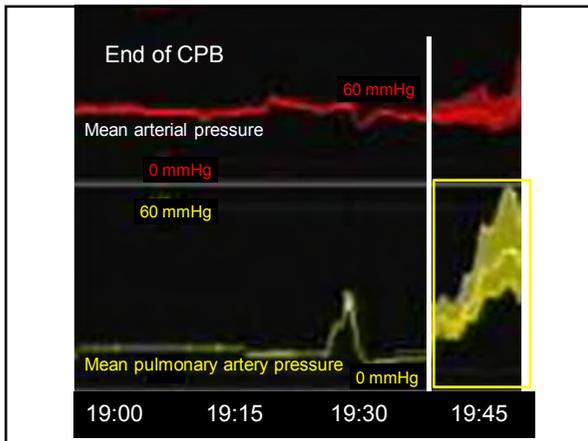


Martineau A&A 2003

64 yo ♀ saphenoscopic surgery
Sudden increase in end-tidal CO₂

Hemodynamic instability: severe pulmonary hypertension





Acute PHT Hypertension during CPB weaning

Vasopressin 1 unit

Inhaled milrinone 5 mg (70 μ /kg)

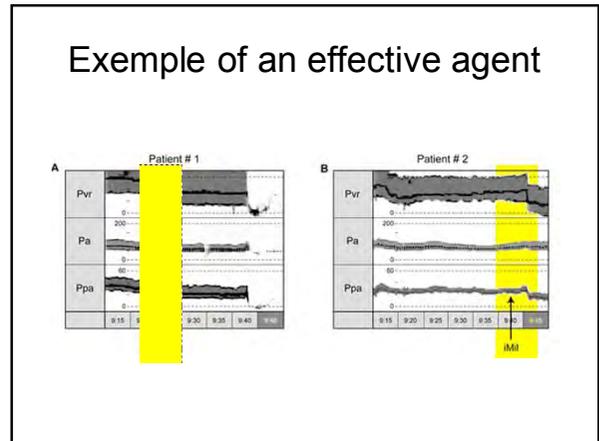
Noradrenalin reduced from 27 à 1.3 ug/min

Lamarcho et al. Cardiopulmonary Support and Physiology

Inhaled but not intravenous milrinone prevents pulmonary endothelial dysfunction after cardiopulmonary bypass

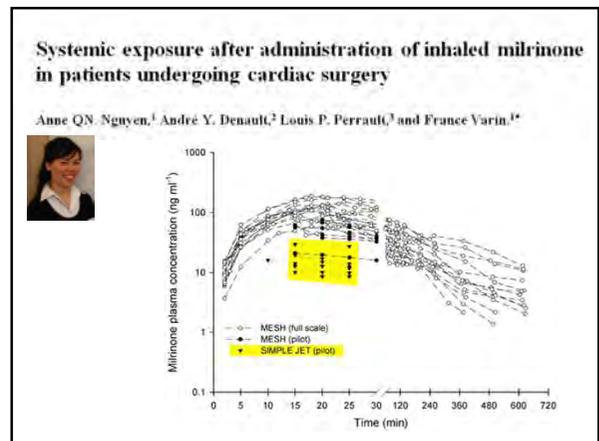
Y. Lamarcho, MD,^{1,3} O. Malo,¹ and L. P. Perrault, MD, PhD^{1,3}

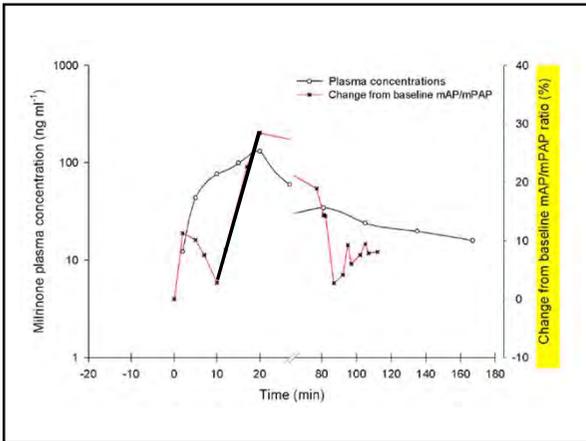
The Journal of Thoracic and Cardiovascular Surgery • July 2005



Post-CPB Unstable

Inhaled milrinone



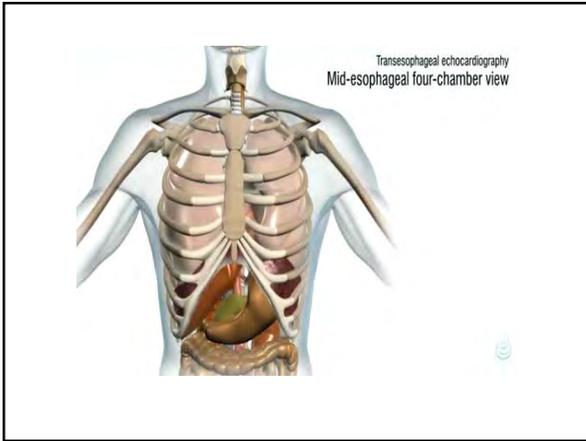
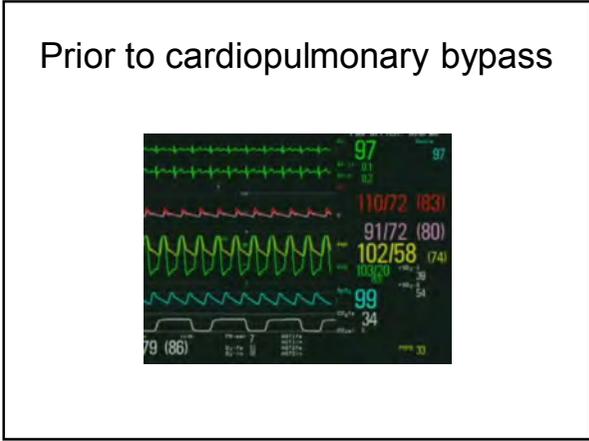


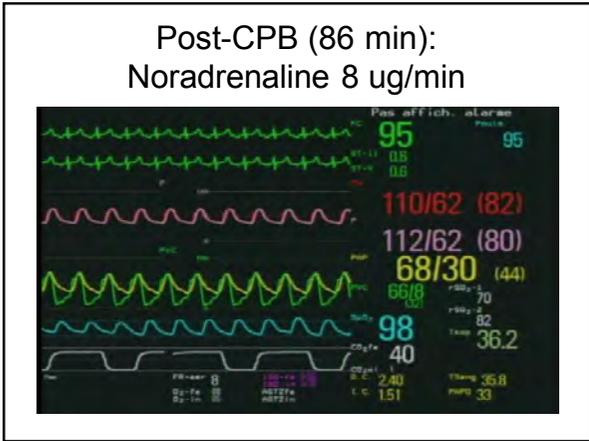
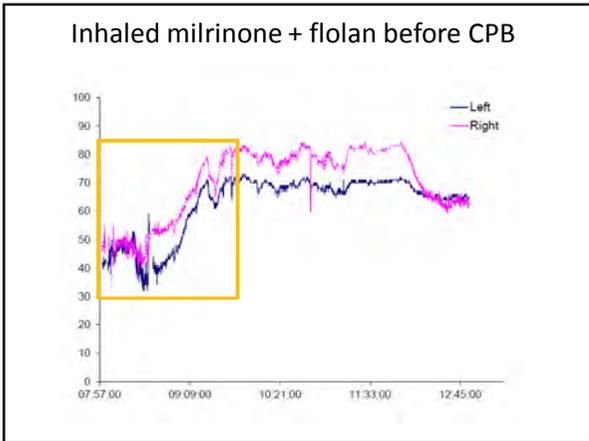
Inhaled Milrinone and Epoprostenol in a Patient With Severe Pulmonary Hypertension, Right Ventricular Failure, and Reduced Baseline Brain Saturation Value From a Left Atrial Myxoma

Patrick St-Pierre, MD,^{1*} Alain Deschamps, MD, PhD,^{2*} Raymond Cartier, MD,¹ Arsène J. Basmadjian, MD,¹ and André Y. Denault, MD, PhD³

- 23 yo ♀ with leg edema and tiredness
- No past medical history
- INR = 1.7
- Transthoracic examination: left atrial myxoma (5.5 X 8 cm)
- Brought to the MHI for surgery

JTCVA 2013

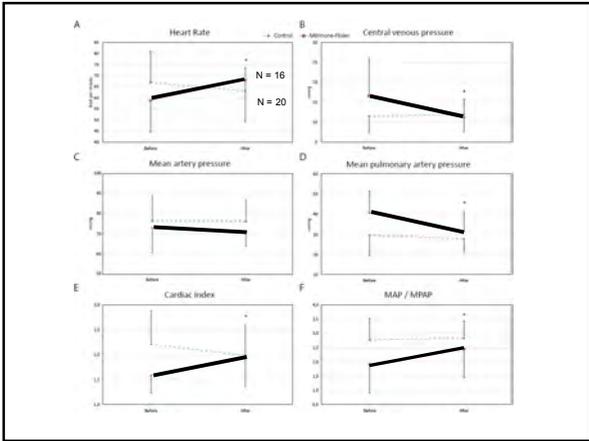
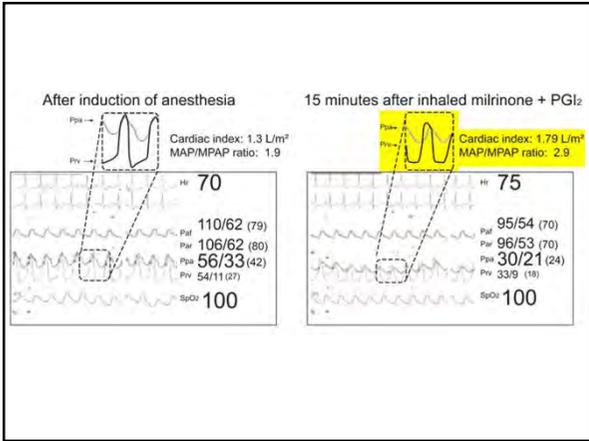




Preliminary Experience with Combined Inhaled Milrinone and Prostacyclin in Cardiac Surgery in Patients with Pre-operative Pulmonary Hypertension or Right Ventricular Dysfunction

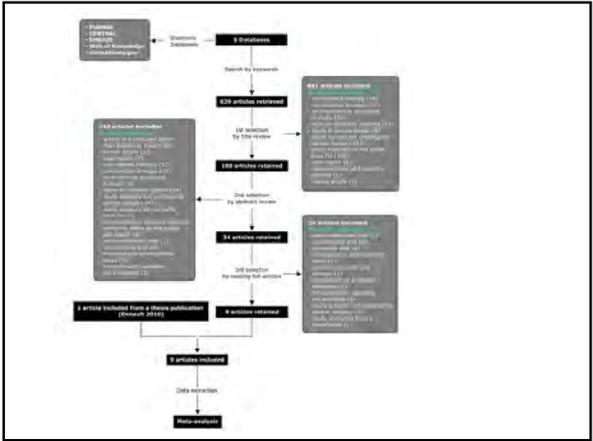
Maxime Laflamme, MD,* Louis P. Perrault, MD, PhD,* Michel Carrier, MD, MBA,+
Malissa Elm-Sarabi,* Annik Fortier,* André Y. Denault, MD, PhD,†

(n = 61) (Under revision JTCVA 2014)



Inhaled Therapy For Pulmonary Hypertension In Cardiac Surgical Patients: A Systematic Review and Meta-Analysis

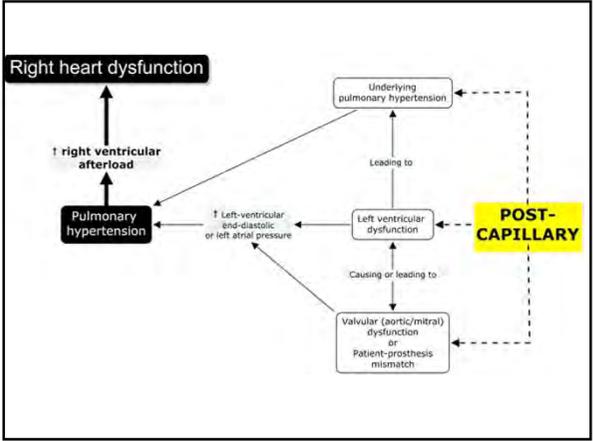
Mahsa Elmi-Sarabi¹, Alan Deschamps¹, Stéphane Delisle², Yoan Lamarque³, Hosham Aoud¹, Louis P. Perrault⁴, Jean Lambert⁵, André Y. Denault¹

Results

Meta-analysis

- 9/ articles
- Total of 323 patients
- Interventions: iNO, inhaled milrinone, iPG₂, inhaled iloprost
- Comparators: IV milrinone, IV nitroglycerin, IV sodium nitroprusside, placebo

“Patient-prosthesis mismatch” (PPM)

Indexed effective valvular orifice area (EOA) of the prosthesis

- Aortic PPM :**
 - Moderate: $iEOA = [0.65-0.85 \text{ cm}^2/\text{m}^2]$
 - Severe: $iEOA \leq 0.65 \text{ cm}^2/\text{m}^2$
- Mitral PPM**
 - Moderate: $iEOA = [0.9-1.2 \text{ cm}^2/\text{m}^2]$
 - Severe: $iEOA \leq 0.9 \text{ cm}^2/\text{m}^2$

71 yo ♂ (1.89 m²)
Redo AVR symptomatic
Pap 60/15
AVR 4 years ago
Type CM #19 (AVE = 1.06 cm²)
 $iEOA \text{ of } 1.06/1.89 = 0.56 \text{ cm}^2/\text{m}^2$



Denault et al TEE Multimedia Manual 2005

Perioperative right ventricular dysfunction

André Yvan Denault^{a,b}, Francois Haddad^c, Eric Jacobsohn^d, and Alain Deschamps^a

Curr Opin Anesthesiol 2013, 26

In summary

- Diagnosis and severity of PH can be easily assessed using the MAP/MPAP ratio
- The treatment has to be tailored to the underlying etiology
- Inhaled agents can be used to reduce the severity of PH
- Their impact on outcome remain to be determined

Pulmonary Hypertension in Cardiac Surgery

André Denault*, Alain Deschamps, Jean-Claude Tardif, Jean Lambert and Louis Perrault

Montreal Heart Institute and Université de Montréal, Montreal, Quebec, Canada

Abstract: Pulmonary hypertension is an important prognostic factor in cardiac surgery associated with increased morbidity and mortality. With the aging population and the associated increase severity of illness, the prevalence of pulmonary hypertension in cardiac surgical patients will increase. In this review, the definition of pulmonary hypertension, the mechanisms and its relationship to right ventricular dysfunction will be presented. Finally, pharmacological and non-pharmacological therapeutic and preventive approaches will be presented.

Keywords: Cardiac surgery, pulmonary hypertension, pharmacological therapy.

1. DEFINITION OF PULMONARY HYPERTENSION

There are several hemodynamic parameters that are used in defining pulmonary hypertension (PHT) (Table 1) [1]. Several of these definitions have been used in various studies. In cardiac surgery, we obtain information on PHT before the procedure and this is usually from an awake patient. This preoperative information is either acquired through preoperative catheterization or, more frequently, estimated *via* transthoracic echocardiography by using the Bernoulli's equation. In the presence of tricuspid regurgitation, as shown in Fig. (1), [2] the simplified Bernoulli's equation will give an estimation of the pressure gradient across the tricuspid valve. This pressure gradient is equal to the difference between the systolic pressure of the right ventricle (RV) and the right atrium (RA). Therefore, knowledge (or estimation) of the right atrial pressure allows the estimation of the right ventricular systolic pressure. In the absence of right ventricular outflow tract obstruction (RVOTO) or pulmonic valve stenosis, this value will be an estimation of the systolic pulmonary artery pressure (SPAP).

Following the induction of general anesthesia, a reduction of both the systemic and the pulmonary artery pressures will be observed. Consequently, absolute values of SPAP used in defining PHT will tend to underestimate its severity. In 2006, we addressed this issue and published a study involving 1557 patients who underwent cardiac surgery [3]. We first demonstrated that the induction of general anesthesia in 32 patients was associated with a significant reduction in mean arterial pressure (MAP), mean pulmonary artery pressure (MPAP) but the MAP/MPAP ratio did not change (Fig. 2). Therefore, this ratio (normal value > 4) seems to be a very robust estimator of the severity of PHT. To demonstrate the utility of the MAP/MPAP ratio, we compared it to the other hemodynamic parameters listed in Table 1 in 1439 patients undergoing cardiac surgery after the induction of general anesthesia but before cardio-

pulmonary bypass (CPB). We observed that the MAP/MPAP ratio behaved similarly to the other hemodynamic parameters (Fig. 3), and had the highest receiver operating curve value to predict hemodynamic complications after cardiac surgery. The hemodynamic complications were defined as postoperative death or requirement for an intra-aortic balloon pump, cardiac arrest and vasoactive support for more than 24 hours. Finally, using transesophageal echocardiography (TEE), we can confirm that the presence of an abnormal MAP/MPAP ratio is almost invariably associated with abnormal systolic or diastolic cardiac function (Fig. 4) [3]. This concept of using the relative instead of absolute value of PHT indices is currently used in congenital cardiology [4, 5].

Finally, PHT is typically classified as capillary, pre-capillary or post-capillary, depending on the site where the cause of PHT is present. The 2003 World Symposium on PHT proposed a classification based on 5 groups: 1-Pulmonary arterial hypertension, 2-PHT secondary to left heart disease, 3-PHT secondary to lung disease and/or hypoxia, 4-PHT secondary to thrombotic and/or embolic disease and 5-A miscellaneous category [6]. In cardiac surgery, it is typically post-capillary or group 2 because the cause of PHT is of cardiac origin and consequently localized after the pulmonary capillary. This is confirmed using pulmonary artery catheterization during which the diastolic pulmonary artery pressure is equal to the pulmonary artery occlusion pressure (PAOP). In a situation where the diastolic pulmonary artery pressure (DPAP) is significantly higher than the PAOP in the absence of tachycardia, a capillary or pre-capillary cause could be sought [1].

In summary, PHT in cardiac surgery should be carefully defined. It is generally a post-capillary PHT. In awake patients, the absolute values have been used and correlated with outcome. However, in patients under general anesthesia, the relative value seems to be more appropriate.

2. PULMONARY HYPERTENSION IN CARDIAC SURGERY: MECHANISM AND ETIOLOGY

The mechanism of PHT in cardiac surgery is complex and can result from several mechanisms acting alone or in

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Table 1. Definitions of Pulmonary Hypertension Used in Clinical Research

Hemodynamic Parameter [1]	Normal Value	Abnormal Value
Systolic pulmonary artery pressure (SPAP)	15-30 mmHg	> 30 or ≥ 40 mmHg
Mean pulmonary artery pressure (MPAP)	9-16 mmHg	Moderate: > 18 mmHg Significant: > 25 mmHg Exercise-induced: > 30 mmHg
Pulmonary vascular resistance (PVR) = (MPAP – PAOP) X 80/CO	60-120 dyn·s·cm ⁻⁵ 1.1-1.4 Wood unit	Mild: > 125 dyn s cm ⁻⁵ Moderate: > 200-300 dyn s cm ⁻⁵ Severe: > 600dyn s cm ⁻⁵
Indexed pulmonary vascular resistance (PVRI) = (MPAP – PAOP) X 80/CI	250-340 dyn·s·cm ⁻⁵ ·m ⁻²	
Pulmonary to systemic vascular resistance index (PVRI/SVRI) X 100%	< 10%	
Trans-pulmonary gradient (MPAP – PAOP)	< 14 mmHg	
Mean pulmonary to systemic pressure ratio (MPAP/MAP) X 100%	< 25%	Moderate: 33-50% Severe: > 50%
Mean systemic to pulmonary pressure ratio (MAP/MPAP) X 100%	> 4	< 4 [3]

CO: cardiac output, CI: cardiac index, PAOP: pulmonary artery occlusion pressure.

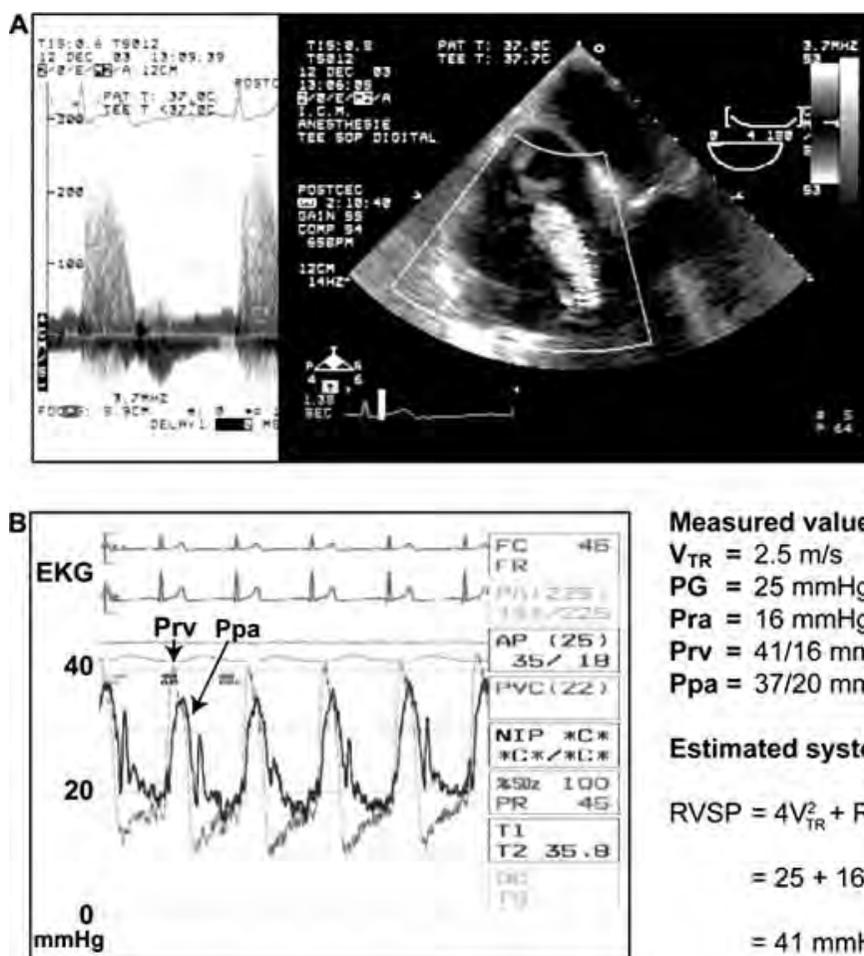


Fig. (1). (A) Estimation of right ventricular systolic pressure (systolic Prv or RVSP) using the pressure gradient (PG) obtained from tricuspid regurgitation (TR) and right atrial pressure (Pra or RAP). (B) Note that the RVSP is higher than the systolic pulmonary artery pressure (Ppa) due to a small gradient across the pulmonic valve (EKG: electrocardiogram, V: velocity). With permission from Denault *et al.* [2] (Chapter 5, p.111).

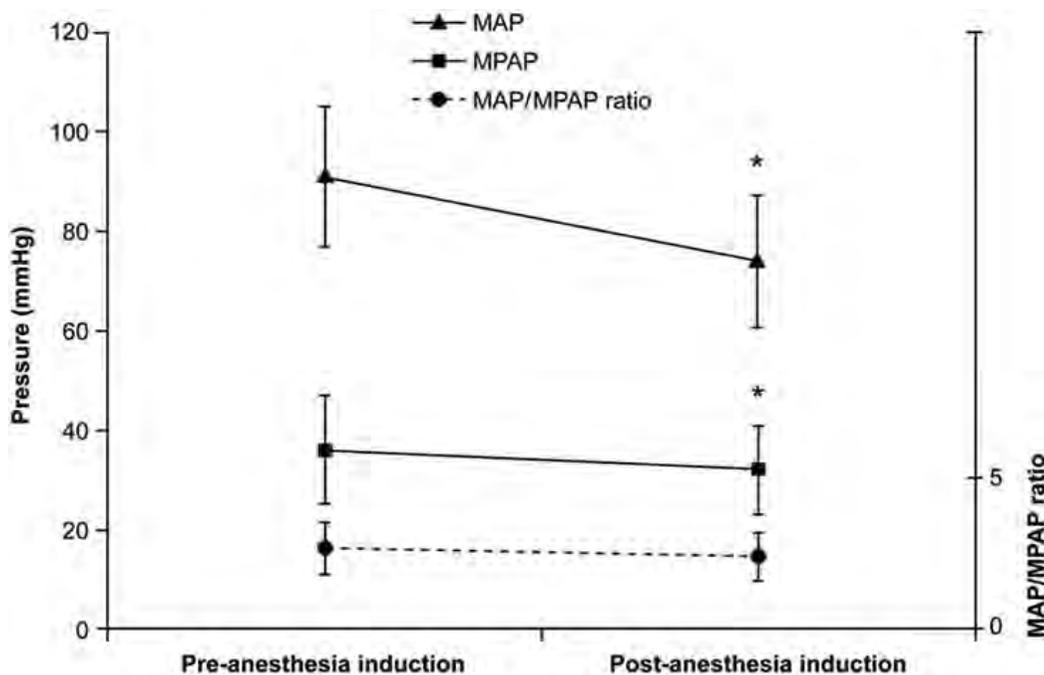


Fig. (2). Change in MAP, MPAP, and the MAP/MPAP ratio after the induction of anesthesia in 32 patients with preoperative pulmonary hypertension. No significant change in the MAP/MPAP ratio was observed ($*p < 0.05$). (MAP: mean arterial pressure, MPAP: mean pulmonary artery pressure) [3].

combination. These mechanisms can be present before the operation, secondary for instance from valvular heart disease. The cause of PHT can appear after CPB from mechanical failure or from the pulmonary reperfusion syndrome. Finally, PHT can be present or persist postoperatively secondary for instance from/to a mitral or aortic patient-prosthesis-mismatch (PPM). Fig. (5) focuses on the role of PHT in cardiac surgery [7] based on current literature, our research findings and our experience of this population.

2.1. Review of the Factors Involved in Pulmonary Hypertension in Cardiac Surgery

The 6 most important causes of PHT in cardiac surgery factors are illustrated in Fig. (5).

- 1) Left ventricular systolic or diastolic dysfunction and mitral or valvular disease either pre- or postoperative are the most common causes of PHT in cardiac surgery. Aortic PPM through a reduction in coronary reserve could also contribute to postoperative PHT [8].
- 2) During cardiac surgery, the extent of the systemic inflammatory response, the pulmonary reperfusion syndrome and the need for blood transfusions may exacerbate PHT (Fig. 6) [9, 10]. The mechanism of pulmonary damage during extracorporeal circulation is thought to be mainly triggered by 1) release of cytokines [11] through endotoxin production, 2) complement activation and 3) ischemia reperfusion injury [12, 13]. This leads to the production of free radicals, endothelin and prostacyclin derivatives with nitric oxide inhibition [12].
- 3) The administration of protamine can induce catastrophic pulmonary vasoconstriction in up to 1.8% of patients [14]. Protamine can also activate complement and, when

given at the end of CPB, can induce PHT associated with adverse hemodynamic responses ranging from minor perturbations to cardiovascular collapse. Three types have been described: systemic hypotension, anaphylactoid reaction and catastrophic PHT [15]. The mechanism of PHT with protamine is thought to occur through an imbalance of vasoconstrictors and vasodilators which leads a reduction in the release of nitric oxide (NO) from the pulmonary vasculature [15].

- 4) Mitral PPM is another recently described cause of residual postoperative PHT. Magne *et al.*[16] studied 929 patients who underwent mitral valve replacement (MVR) and followed them up for 15 years. Mitral valve PPM was defined as not clinically significant if $> 1.2 \text{ cm}^2/\text{m}^2$, as moderate if > 0.9 and $\leq 1.2 \text{ cm}^2/\text{m}^2$, and as severe if $\leq 0.9 \text{ cm}^2/\text{m}^2$. The prevalence of moderate PPM was 69% and that of severe PPM was 9%. Severe PPM was found to be associated with residual PHT and a 3-fold increase in postoperative mortality after adjustment for other risk factors. This new and relevant information is currently absent from the majority of the studies dealing with predictors of survival in mitral valvular surgery.
- 5) Hypoxia, hypercarbia and pulmonary embolism are other causes of PHT. They can appear before, during or after CPB. For instance, PHT can cause RV dysfunction, which will lead to an increase in right atrial pressure. This can lead to the opening of a patent foramen ovale (PFO), which is present in 20-30% of the general population [17]. The consequence of the opening of a PFO would be a right-to-left shunt. This would increase the severity of hypoxia and lead to an exacerbation of PHT. Pulmonary vessels constrict with hypoxia (Euler-Liljestrand reflex) and relax in the presence of hyperoxia

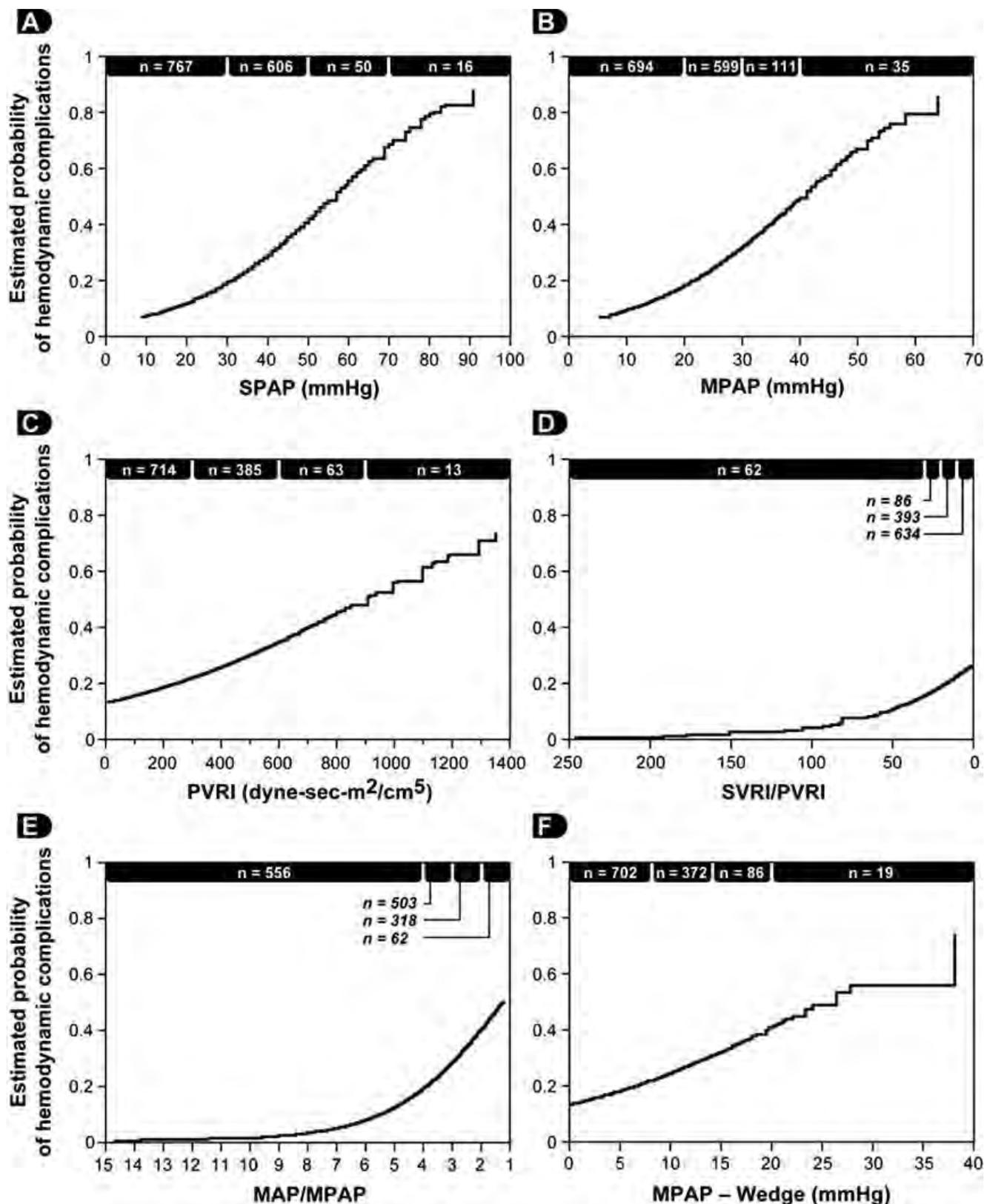


Fig. (3). Relationship between the estimated probability of hemodynamic complications and variables used in the evaluation of pulmonary hypertension: (A) systolic pulmonary artery pressure (SPAP), (B) MPAP, (C) PVRI, (D) the ratio of SVRI to PVRI, (E) the MAP/MPAP ratio, and (F) the transpulmonary gradient defined as MPAP minus pulmonary artery occlusion pressure (PAOP). For easier comparison, the scale of the x axis of the SVRI/PVRI and the MAP/MPAP are inverted. (*n* = number of patients). (MAP: mean arterial pressure, MPAP: mean pulmonary artery pressure, PVRI: indexed pulmonary vascular resistance, SVRI: indexed systemic vascular resistance) [3].

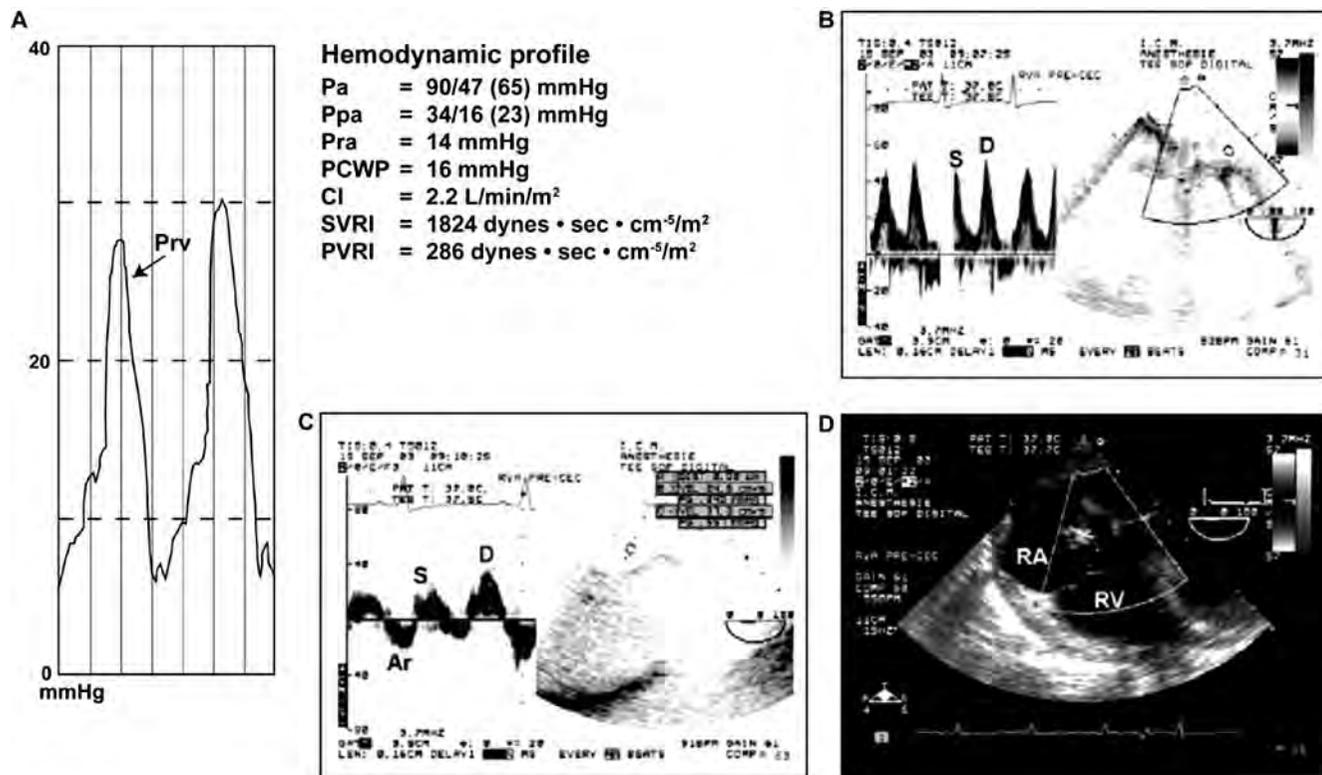


Fig. (4). Hemodynamic and transthoracic echocardiographic evaluation of a 46-year-old woman scheduled for aortic valve surgery. Despite a normal pulmonary artery pressure of 34/16 mmHg and PVRI at 286 dyn-s-cm-5-m-2, this patient had an abnormal right ventricular diastolic filling pressure waveform characterized by a rapid upstroke (A) and reduced systolic (S) to diastolic (D) pulmonary (B) and hepatic (C) venous flow consistent with left and right ventricular diastolic dysfunction. In addition, a dilated right atrium and ventricle were present without significant tricuspid regurgitation in a mid-esophageal right ventricular view (D). The MAP/MPAP ratio was 65/23 or 2.8. (CI: cardiac index, Pa: arterial pressure, PCWP: pulmonary capillary wedge pressure, Ppa: pulmonary arterial pressure, Pra: right atrial pressure, Prv: right ventricular pressure, PVRI: pulmonary vascular resistance index, RA: right atrium, RV: right ventricle, SVRI: systemic vascular resistance index) [3].

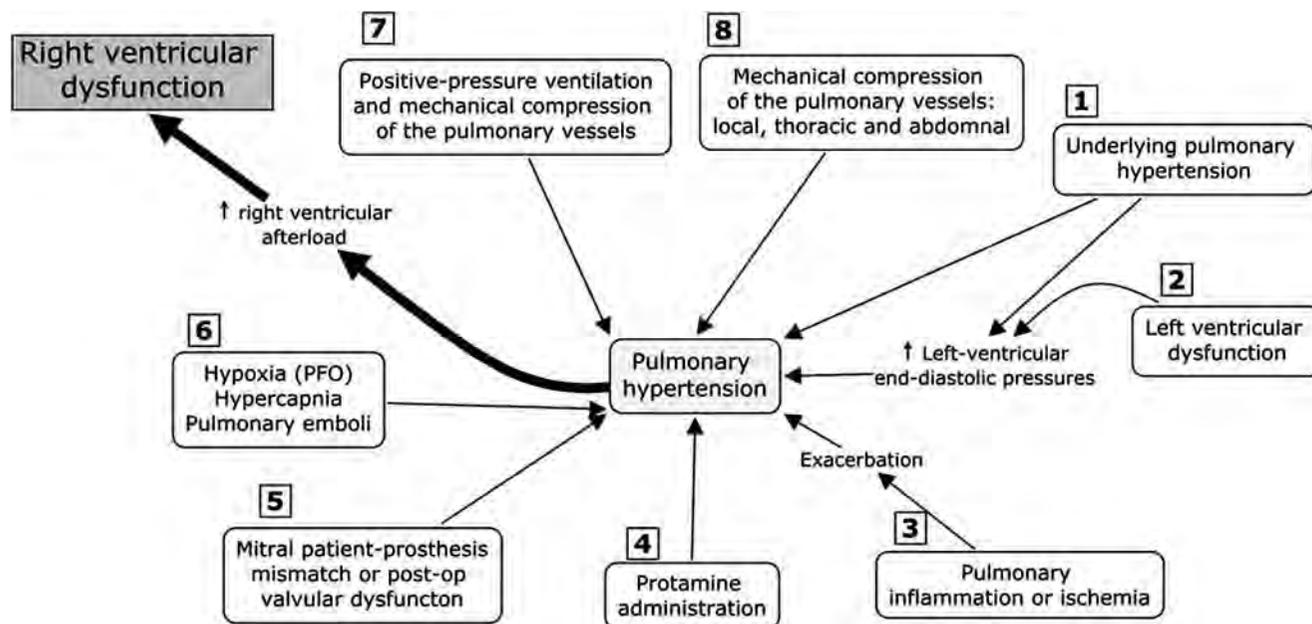


Fig. (5). The most common mechanisms that could induce pulmonary hypertension in cardiac surgery. (See Section 2.1 for details) (PFO: patent foramen ovale).

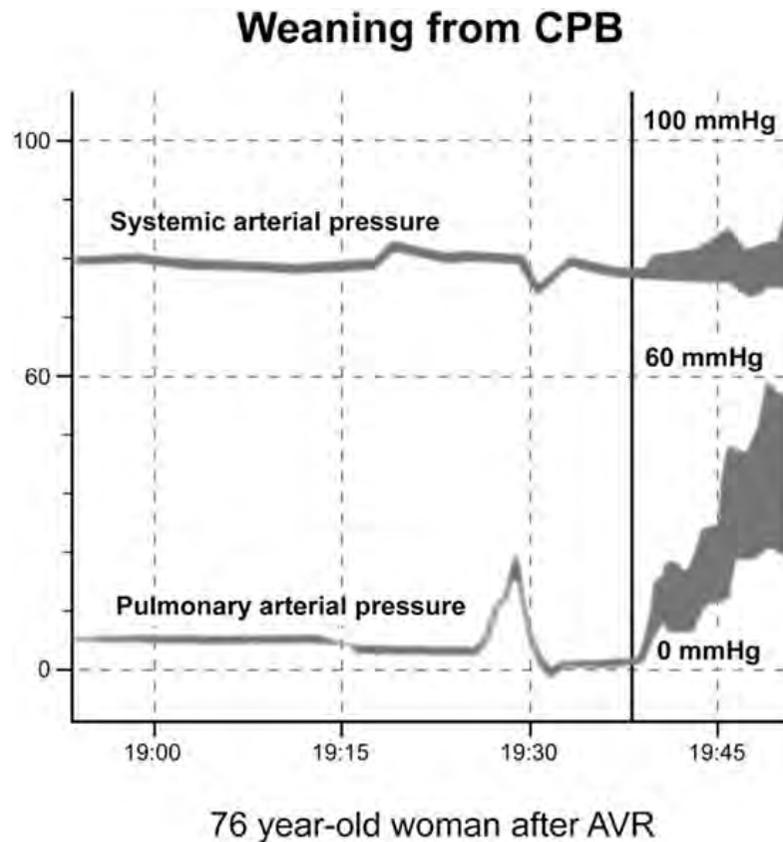


Fig. (6). Unexpected pulmonary hypertension upon weaning from cardiopulmonary bypass (CPB) in a 76-year-old woman after aortic valve replacement (AVR). The CPB duration was 71 minutes. A significant increase in pulmonary arterial pressure in relation to the systemic arterial pressure was observed as the patient was weaned from CPB. No mechanical causes were found.

[18]. Hypercarbia can occur particularly if acute lung injury occurs during or after the procedure. The increase in PCO_2 will increase PHT through vasoconstriction. Finally, although pulmonary embolism is rare in the immediate postoperative period, they can occur particularly in patients with predisposing factors (Fig. 7).

- 6) Lung volumes have a differential effect on intra- and extra-alveolar vessels, which accounts for the unique U-shaped relationship between lung volume and pulmonary vascular resistance (PVR). PVR is minimal at functional residual capacity and increased at large and small lung volumes (Fig. 8). Clinically, this may be observed when hyperinflation of the lungs greatly increases PVR [18]. Application of high levels of positive end-expiratory pressure (PEEP) may narrow the capillaries in the well ventilated lung areas and divert flow to less well ventilated or non-ventilated areas, potentially leading to hypoxia. An increase in cardiac output distends open vessels and may recruit previously closed vessels, decreasing PVR. Regional blood flow to lung is also influenced by gravity; pulmonary blood flow is greater in the dependant areas of the lung. In addition, increase in intrathoracic pressure will be transmitted to the surrounding cardiac pressure and contribute to elevate pulmonary artery pressure. Mechanical compression of pulmonary vessels can be caused by hemothoraces or tension pneumothoraces.

Finally, multiple molecular pathways are important for the regulation of PVR. These include the nitric oxide, prostacyclin, endothelin-1 and serotonin pathways [19]. Nitric oxide and prostacyclin are endogenous vasodilators produced in the pulmonary vascular endothelium. Endothelin-1 is an endogenous vasoconstrictor peptide secreted by the vascular endothelium and plays a role in pulmonary vasoconstriction and vascular smooth muscle proliferation [20]. The neurotransmitter serotonin and the serotonin receptor transporter have also been implicated in the regulation of pulmonary vascular tone. An imbalance in these pathways may lead to vasoconstriction and vascular remodelling, potentially leading to progressive pulmonary vascular disease.

The most dreadful consequence of PHT is the increase in RV afterload and RV dysfunction; this issue will be addressed here.

2.2 Right Ventricular Dysfunction

There is growing evidence that morbidity and mortality associated with PHT are dependent on RV adaptation to disease rather than on the absolute value of pulmonary arterial pressure [21-25]. In studies addressing hemodynamic variables and survival in idiopathic pulmonary arterial hypertension, high mean right atrial pressures and low cardiac output (CO) were consistently associated with poorer survival when contrasted with pulmonary arterial pressure

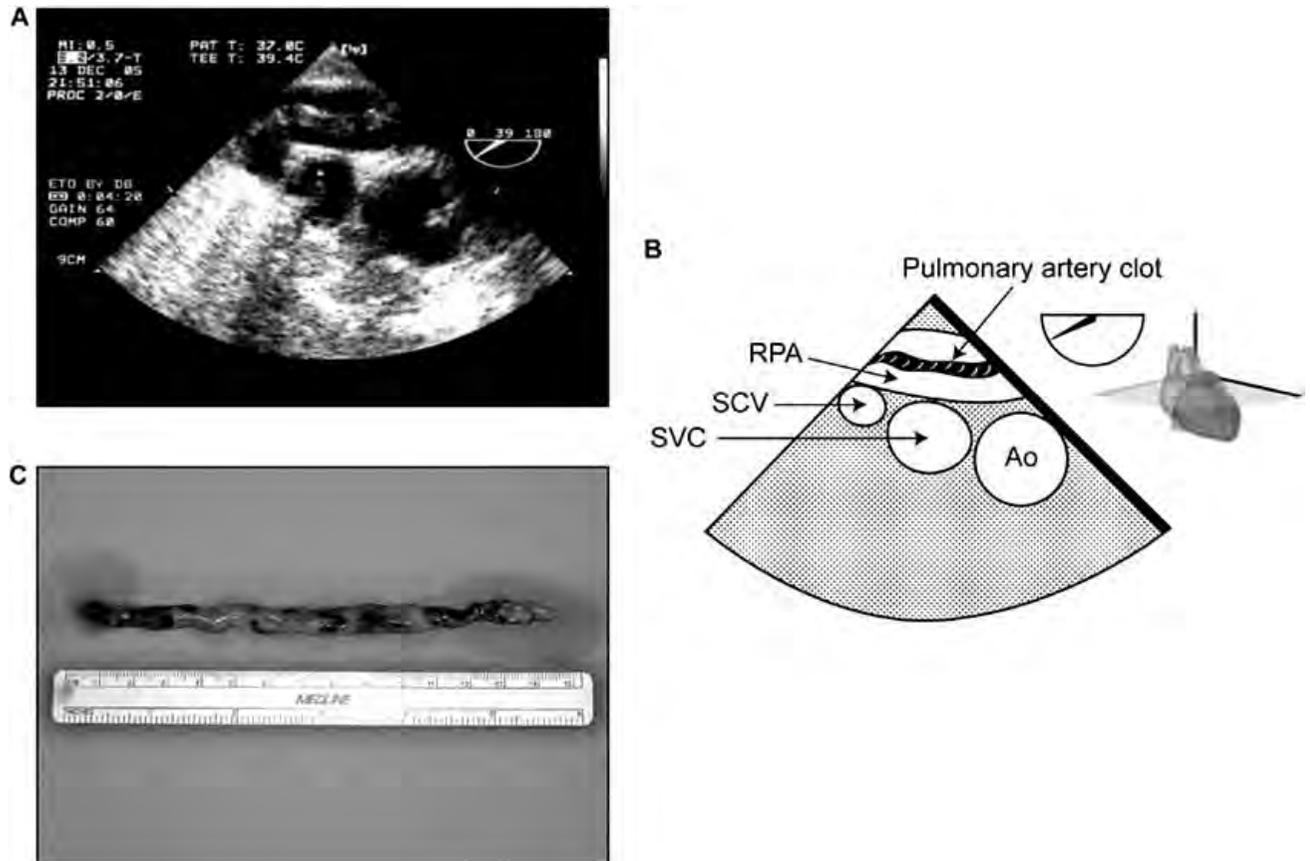


Fig. (7). Pulmonary embolism immediately after coronary revascularization. This patient was hospitalized and waiting for more than a week before the procedure could take place. At the end of the procedure while she was transferred in her bed, she became hemodynamically unstable. Immediate transesophageal echocardiographic exam was performed and showed the appearance of a clot in the right pulmonary artery (A-B). She was brought back to the operating room for urgent embolectomy and a clot was removed (C). She was discharged from the hospital in good condition. (Ao: aorta, RPA: right pulmonary artery, SCV: subclavian vein, SVC: superior vena cava) (Courtesy of Dr. David Braco and Dr. Nicolas Noiseux).

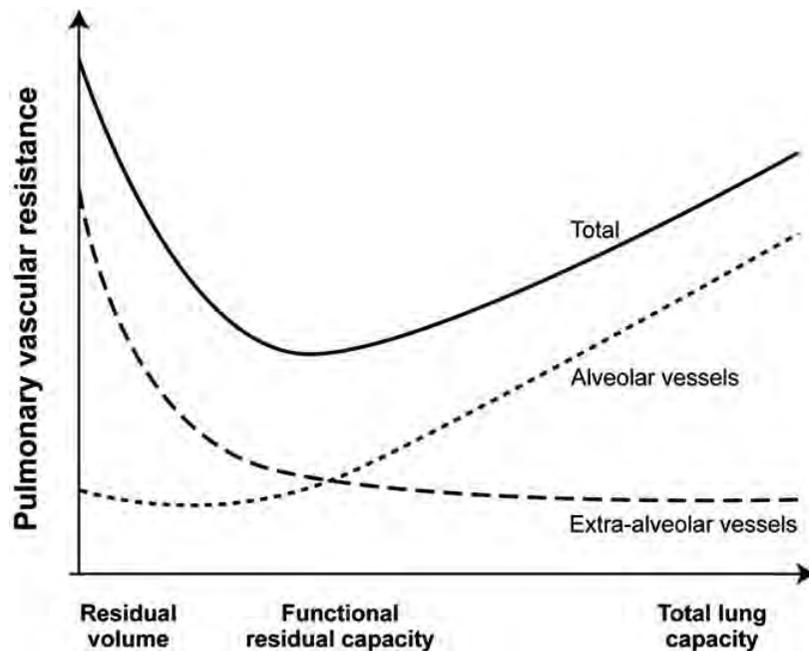


Fig. (8). Relationship between lung volume and pulmonary vascular resistance (PVR). PVR is minimal at functional residual capacity (FRC) and increased at large or total lung capacity (TLC) and small lung volumes residual volume (RV) decreases. The differential effect on intra- and extra-alveolar vessels accounts for the U-shaped relationship of PVR and lung volume. (Adapted from Fischer *et al.* [18]).

alone, which was only moderately related to outcome [21, 26].

The importance of RV function in cardiac surgery has been demonstrated in a variety of clinical settings such as high risk coronary or valvular heart disease, congenital heart disease, heart transplantation, in patients requiring mechanical assist devices and in the unstable postoperative patient (Table 2) [25]. However, most of the evidence that supports the importance of RV function is based on retrospective or small prospective studies. To date, parameters of RV function have not been included in large scale risk stratification models and therefore their incremental value to the Parsonnet Score or the EuroSCORE have not been well established [27-30]. A recent panel from the National Institute of Health (NIH) has stressed the importance of research in the understanding of RV failure [24].

2.2.1. Before the Procedure

In patients presenting with severe aortic stenosis, Boldt *et al.* have demonstrated that preoperative RV dysfunction was associated with a greater requirement of postoperative inotropic support [31]. In a retrospective study of patients undergoing mitral and mitral-aortic valvular surgery, Pinzani *et al.* demonstrated that preoperative RV failure was associated with perioperative mortality. In this same study, postoperative RV failure was the most important independent predictor of late survival [32]. In a small prospective study of 14 patients with severe non-ischemic mitral regurgitation and high risk descriptors (LV ejection (LVEF) $\leq 45\%$ or RV ejection fraction (RVEF) $\leq 20\%$), Wencker *et al.* found that preoperative RVEF $\leq 20\%$ predicted late postoperative deaths [33]. In patients under-going coronary artery surgery, Maslow *et al.* [34] showed that RV dysfunction defined by a RV fractional area change (RVFAC) less than 35% in the context of severe LV systolic dysfunction (LVEF $\leq 25\%$) and non-emergent coronary artery bypass surgery was associated with an increased risk of postoperative morbidity and mortality. In their retrospective study ($n=41$), patients with RV dysfunction had a higher prevalence of diabetes mellitus and renal disease as well as a higher incidence of postoperative inotropic or mechanical support, longer intensive care unit and hospital stay and a decreased short term and long term survival.

To further assess the value of RV function relative/compared to other validated risk factors in open valvular heart surgery, we recently published our experience related to 50 patients undergoing valvular surgery [35]. We confirmed that, in patients with a RV myocardial performance index (RVMPI) above 50% ($n=20$), the number of patients with DSB (difficult separation from bypass) (16/20 (80%) vs. 6/30 (20%), $p<0.0001$) and the end-point of mortality of postoperative heart failure (14/20 (74%) vs. 3/30 (10%), $p<0.0001$) were significantly higher. On a multivariate analysis, among all other demographic, hemodynamic and echocardiographic variables, the RVMPI was the only independent predictor of heart failure and mortality (OR: 25.20, 95%; CI 5.24-121.15, $p<0.0001$).

2.2.2. After the Procedure

The presence of RV failure after CPB is associated with a mortality rate ranging from 44% to 86% [36]. The incidence

of post-cardiotomy acute refractory RV failure ranges from 0.04 to 0.1%. Acute refractory RV failure has also been reported in 2-3% patients after a heart transplant and in almost 20-30% patients who receive a left ventricular assist device support with a reported initial salvage rate is only 25-30% [10].

3. IMPORTANCE AND IMPACT OF PULMONARY HYPERTENSION IN CARDIAC SURGERY

PHT present before any cardiac procedure is associated with increased morbidity and mortality [27,37-40]. However, the presence of PHT is not routinely reported to the surgeon. This can be explained by the use of preoperative risk stratification models in cardiac surgery, in which only 4/19 models used PHT as a risk factor [41]. Interestingly, the EuroSCORE model, which had the highest discriminatory, is one of the models in which PHT is included. In a study that included 4351 CABG patients operated in Sweden, the receiver operating characteristics (ROC) of EuroSCORE model was 0.86 and 0.75 for the 30-day and one year mortality, respectively.

Using our database in 1999, the mean preoperative systolic pulmonary artery pressure (SPAP) was 31 ± 10 mmHg. Elevated SPAP above 30 mmHg were present mostly in mitral valve replacement ($n=80$, 40 ± 14 mmHg), followed by combined CABG and valve ($n=126$, 36 ± 13 mmHg), multiple valves ($n=60$, 36 ± 16 mmHg) and heart transplantation ($n=6$, 36 ± 14 mmHg). A total of 605 patients (42%) presented with elevated SPAP defined as above 30 mmHg. If we select patients with more severe PHT using a MAP/MPAP ratio < 2 , there were only 16 patients, all experienced difficult separation from CPB, 3 died (18.7% mortality) and half required vasoactive support for more than 24 hours after the procedure.

For this reason, it is relatively clear that the presence of PHT before the operation or appearing during or after it will have an impact on survival and mostly through its effect on right ventricular function. The next question is: how can we prevent or treat PHT and its consequence, RV failure?

4. PHARMACOLOGIC AND NON-PHARMACOLOGIC APPROACHES IN THE TREATMENT AND PREVENTION OF PULMONARY HYPERTENSION IN CARDIAC SURGERY

The choice of the appropriate therapy should be based on evidence-based medicine. A MEDLINE search was performed using the key words 'randomized controlled trial' (RCT), 'humans', 'adults', 'English' and 'PHT'. Articles related to cardiac surgery were then selected and classified according to the levels of evidence proposed by Sackett [42] for evidence-based medical practice. Using this strategy, a total of 10 articles were retrieved. In addition, the Consort statement group has developed guidelines to assess the quality of randomized controlled clinical trials [43]. These studies are summarized in Table 3.

4.1. Treatment of Pulmonary Hypertension

4.1.1. Pharmacological

The agents studied were: inhaled prostacyclin (PGI₂), nitric oxide (NO) and intravenous vasodilators such as

Table 2. Prognostic Value of Right Ventricular Function in Cardiac Surgery (Selected Studies)

Study	Population	Study Design	RV Dysfunction	Results
Reitchert <i>et al.</i> [65]	Unstable post-operative patients	Prospective <i>n</i> =60	RVFAC < 35%	RV dysfunction associated with high mortality rates
Pinzani <i>et al.</i> [32]	Mitral and combined mitro-aortic surgery	Retrospective <i>n</i> =382	Clinical definition	Post-operative RV failure is the strongest predictor of postoperative mortality
Cullen <i>et al.</i> [66]	Tetralogy of Fallot	Prospective <i>n</i> =35	Restrictive RV physiology	Restrictive physiology predicts longer intensive care unit stay post repair and lower cardiac output
Gatzoulis <i>et al.</i> [67]	Tetralogy of Fallot	Prospective <i>n</i> =41	Restrictive RV physiology	Restrictive physiology predicts smaller RV and better exercise tolerance
Kromos <i>et al.</i> [68]	LVAD and RV failure	Retrospective <i>n</i> =31	Clinical mean RVEF = 11.8%	Preoperative clinical factors such as fever, pulmonary edema, and intraoperative blood transfusions were associated with RVAD need
Hosenpud <i>et al.</i> [69]	Heart Transplantation	Retrospective International Society for Heart & Lung transplantation <i>n</i> =69,205	RV failure associated with circulatory failure	RV failure accounts for up to 20% of early deaths
Oehiai <i>et al.</i> [70]	LVAD	Retrospective <i>n</i> =245	RV failure requiring RVAD	23 patients (9%) required RVAD. The need for circulatory support, female gender, and non-ischemic etiology were predictors of RVAD need.
Maslow <i>et al.</i> [34]	CAD undergoing coronary bypass surgery with LVEF < 25%	Retrospective <i>n</i> =41	RVFAC < 35%	RV dysfunction is associated with decreased long term survival
Therrien <i>et al.</i> [71]	Tetralogy of Fallot	Prospective <i>n</i> =17	RV remodeling	Severe RV dilatation (RVEDV ≥ >170 ml/m ² or RVESV >85 ml/m ²) associated with incomplete RV remodeling
Webb <i>et al.</i> [72, 73]	Atrial septal defect	Retrospective series	RV remodeling	Older age at repair and abnormal RV myocardial relaxation were associated with incomplete RV remodeling
Denault <i>et al.</i> [74]	Patients undergoing bypass surgery	Retrospective and prospective <i>n</i> =800	Dynamic obstruction of RVOT (Gd > 25 mmHg)	Incidence: 4%, dynamic obstruction of RVOT was associated with a higher incidence of difficult weaning from bypass
Haddad <i>et al.</i> [35]	High risk valvular surgery	Prospective <i>n</i> =50	RVFAC < 32% or RVMPI > 0.50	Preoperative RV dysfunction was associated with a higher incidence of post-operative circulatory failure

CAD: coronary artery disease, Gd: gradient, LV: left ventricular, LVAD: left ventricular assist device, RV: right ventricular, RVAD: right ventricular assist device, RVES: right ventricular end-systolic volume, RVED: right ventricular end-diastolic volume, RVEF: right ventricular ejection fraction, RVFAC: right ventricular fractional area change, RVMPI: right ventricular myocardial performance index, RVOT: right ventricular outflow tract obstruction. Based on [25].

prostacyclin E1 (PGE1), nitroglycerin (NTG), nitroprusside (NTP), milrinone, enoximone and dobutamine. One large RCT compared heparinise to protamine and explored as a secondary end-point the prevention of PHT from protamine administration [44]. Most of the studies reviewed included a small number of patients and their primary end-points were hemodynamic changes.

In the most recent trial, Fattouch *et al.* [45] studied patients with PHT (*n*=58) undergoing MVR for mitral stenosis. Inhaled PGI₂ (iPGI₂) and iNO were compared to conventional intravenous vasodilators. The inhaled drugs

were given just before the end of CPB. Significant reductions in PHT indices as well as increase in cardiac output (CO) and in RV ejection fraction were observed in both inhaled groups compared to conventional treatment. In addition, in both inhaled groups, separation from CPB was easier, the amount of vasoactive drugs administered was smaller and the duration of stay in the ICU and hospital was shorter. The same group also compared the same three strategies in the treatment of PHT after MVR upon arrival in the intensive care unit (ICU) [46]. Inhalation of PGI₂ was associated with a reduction in PVR and an increase in stroke volume. Inhaled NO reduced PVR but did not increase

Table 3. Randomized Controlled Trial in the Treatment of Pulmonary Hypertension in Adult Cardiac Surgery

	Author	Country	Date	Agents Used	Design	N	Inclusion criteria	Primary End-Point	Level of Evidence
1	Fattouch <i>et al.</i> [45]	Italy	2006	iPGI ₂ vs iNO vs intravenous vasodilators	RCT Unicenter	58	MVR + PHT before the end of CPB	Hemodynamic	A1b
2	Ocal <i>et al.</i> [14]	Turkey	2005	iPGI ₂ vs NTG	RCT Multicenter	68	CABG with protamine reaction after CPB	Hemodynamic	A1b
3	Stafford <i>et al.</i> [44]	USA NC	2005	Heparinase vs protamine	Non-inferiority clinical trial design Multicenter	167	CABG on + off pump after CPB	Bleeding	A1b
4	Fattouch <i>et al.</i> [46]	Italy	2005	iPGI ₂ vs iNO vs intravenous vasodilators	RCT Unicenter	58	MVR + PHT in the intensive care unit	Hemodynamic	A1b
5	Hache <i>et al.</i> [53]	Canada	2003	iPGI ₂ vs placebo	RCT Unicenter	20	PHT before CPB	Hemodynamic	A1b
6	Solina <i>et al.</i> [47]	USA	2001	iNO vs milrinone	RCT Unicenter	62	PHT after surgery	Hemodynamic	B
7	Feneck <i>et al.</i> [49]	UK	2001	Milrinone vs dobutamine	RCT Multicenter	120	CO < 2 L/min/m ² et PAOP > 10 mmHg after cardiac surgery	Hemodynamic	A1b
8	Solina <i>et al.</i> [48]	USA	2000	iNO vs milrinone	RCT Unicenter	45	PHT after surgery	Hemodynamic	A1b
9	Schmid <i>et al.</i> [50]	Switzerland	1999	iNO vs NTG vs PGE1	Crossover Unicenter	14	PHT after surgery	Hemodynamic	B
10	Hachenberg <i>et al.</i> [51]	Germany	1997	Enoximone vs dobutamine+N TG	RCT Unicenter	20	HTP in MVR before and after surgery	Hemodynamic	A1b

CABG: coronary artery bypass graft, CO: cardiac output, CPB: cardiopulmonary bypass, iNO: inhaled nitric oxide, iPGI₂: inhaled prostacyclin, MVR: mitral valve replacement, NO: nitric oxide, NTG: nitroglycerin, OR: operating room, PAOP: pulmonary artery occlusion pressure, PGE1: prostaglandin E1, PGI₂: prostacyclin, PHT: pulmonary hypertension, RCT: randomized controlled trial, UK: United Kingdom, USA: United States of America.

stroke volume, and NTP was associated with a reduction in systemic arterial pressure and systemic vascular resistance.

The administration of protamine can be associated with severe PHT followed by RV failure. This condition requires immediate treatment. In a study of CABG patients ($n=3800$), Ocal *et al.* [14] compared two therapeutic approaches in the treatment of the protamine reaction observed in 68 patients (1.8%). One group received iPGI₂ and the other intravenous NTG in addition to standard vasoactive agents. The iPGI₂ group showed improved hemodynamics and only 14 patients (39%) had to return on CPB compared to all 30 patients (100%) in the NTG group. A tendency for shorter length of stay in the ICU and reduced mortality was observed in the iPGI₂ group, but the numbers were too small to be statistically significant.

To avoid protamine reaction, heparinase I, a heparin degrading enzyme, was compared in a multicentered randomized controlled trial [44]. However, the results of the trial were negative and heparinase I was not associated with any reduction in the intervention to treat PHT or any reduction in bleeding.

Solina *et al.* explore the dose-responsiveness of iNO given on termination of CPB at 10, 20, 30 and 40 ppm compared to intravenous milrinone [47]. Nitric oxide was associated with a reduction in PVR with a maximum dose of 10 ppm. No significant difference in reduction of PVR or inotropic requirement was observed compared to milrinone. The same authors compared NO 20 ppm and 40 ppm to milrinone in patients with PVR above 125 after cardiac surgery [48]. The drugs were started after CPB and for 24

hours in the intensive care unit. Higher systemic arterial pressure was observed in the 20 ppm group and higher RVEF were obtained in the 40 ppm NO group. The milrinone group required significantly more phenylephrine and tended to have higher heart rate than either of the NO groups in the ICU.

In patients with CO below 2 L/min/m² and PAOP > 10 mmHg, Feneck compared milrinone to dobutamine in 120 patients [49]. In a subset of patients with PHT defined as (PVR >200 dyne sec cm⁵; MPAP > 25 mmHg), milrinone had a similar effect to dobutamine on the reduction of PVR and increase in cardiac index (CI). The PAOP and systemic vascular resistance (SVR) were more reduced by milrinone.

Schmid *et al.* [50] compared three approaches (iNO vs PGE1 vs NTG) in a crossover study; these were used to treat PHT after cardiac surgery in 14 patients. Only stable patients were included in the study, which limits the application of the results. Inhaled NO decreased PVR without reducing SVR, did not change coronary perfusion pressure of the right coronary pressure and increased oxygen transport.

Finally, Hachenberger *et al.* [51] explored the role of enoximone compared to NTG and dobutamine, given after induction of anesthesia and then restarted before the end of CPB. Only enoximone was associated with a decrease in MPAP and PVR.

In our practice at the Montreal Heart Institute, we regularly use iPGI₂ [52, 53] and inhaled milrinone [54, 55] in the presence of pulmonary hypertension and RV dysfunction before and after cardiac surgery. Inhaled NO and oral sildenafil are used in refractory cases in the ICU.

4.1.2. Non-Pharmacological Approach

The non-pharmacological approach to the treatment of PHT will be directed to the cause or the consequence of PHT, as illustrated in Fig. (5). In the presence of PHT secondary to LV failure, intra-aortic balloon counterpulsation will facilitate recovery of LV dysfunction. If prosthetic valve dysfunction is present after CPB, then return on CPB and correction of the problem will be the treatment of choice. The correction of hypoxia, hypercapnia and surgical thrombo-embolectomy (when surgically indicated) can help control PHT. In patients with elevated intrathoracic pressure from accumulated air or blood, chest drainage will be the solution. However, in some patients undergoing long procedures and long CPB duration, chest closure can be associated with hemodynamic instability. This is a sort of "thoracic compartment" syndrome. In these situations, the chest temporarily can be left open to reduce the surrounding pressures. Finally, pulmonary artery balloon pump, RV assist device (RVAD) or cavopulmonary diversion have been described as potential treatments for severe RV dysfunction [10].

4.2. Treatment of Right Ventricular Failure

We summarize our approach to the treatment of RV failure in Fig. (9). Right ventricular function is evaluated visually, using the RV pressure waveform and TEE. Once RVOTO is ruled out, the etiology of RV systolic dysfunction is divided in two categories. If ischemia is suspected to

contribute to RV failure, then both the medical and the surgical treatment will be oriented toward the promotion of RV perfusion. In a non-ischemic etiology is suspected, the medical and surgical treatment will be oriented toward an increase in contractility (inotropes) and a reduction in RV afterload (iNO, iPGI₂, inhaled milrinone).

5. PREVENTION OF PULMONARY HYPERTENSION

5.1. Pharmacological Approach

The prevention of PHT and its consequences could represent a promising strategy to prevent RV failure. However, very few studies have addressed this issue. One of the potential targets could be the prevention of the pulmonary reperfusion syndrome. In that regard, our group has demonstrated in an animal model that iPGI₂ [56] and inhaled milrinone [54] could prevent endothelial dysfunction induced by CPB. Hache *et al.* [53] conducted a pilot RCT in patients with preoperative PHT and demonstrated that iPGI₂ was superior to placebo in reducing PHT. Furthermore, in patients who received iPGI₂, the amount of vasoactive support was reduced.

We have completed a randomized controlled trial on the use of inhaled milrinone administered before CPB in 21 patients. There were 8 males and 13 females of a mean age of 70±6.3 years and a mean Parsonnet Score of 32±9. All procedures were valvular surgeries, 14 of which were complex surgeries and 5 reoperations. Mean systolic pulmonary artery pressures (SPAP) were reduced in the inhaled milrinone group from 66±20 mmHg (pre-CPB) to 46±20 mmHg (after CPB) ($p<0.001$). No changes in SPAP were observed in the control group and no differences in systemic arterial pressure between the groups were observed. We also published our preliminary experience in the use of inhaled milrinone involving 70 high risk patients (Parsonnet Score of 27±14) [27, 55]. Compared with a control group with similar baseline characteristics, we observed that the administration of inhaled milrinone prior to CPB ($n=30$) was associated with a lesser rate of re-initiation of CPB (9 vs 1; $p=0.021$) in this very high risk group. We also observed postoperatively lower pulmonary artery pressures in the pre-CPB group. Further studies will be required to determine the efficacy of this approach.

5.2. Non-Pharmacological Approach

The selection of the type and size of an aortic prosthetic valve could be a very important strategy, because it has been shown that, if the effective orifice area (EOA) of the aortic valve is too small in relation to body size, the so-called PPM, the intraoperative and long-term mortality, is increased [57-64]. Hence, anticipatory strategies aiming at the prevention of PPM, such as the implantation of a better performing prosthesis (i.e. stentless bioprosthesis, new generation bileaflet mechanical valve, new generation supra-annular stented bioprosthetic valve) or the enlargement of the aortic root to accommodate a larger prosthesis could contribute to reduce PHT after cardiac surgery and facilitate the separation from CPB. On the other hand, some of the alternative options that can be used to prevent PPM are complex and may increase the risk of DSB by prolonging the duration of

RIGHT HEART DYSFUNCTION FOLLOWING CARDIOPULMONARY BYPASS

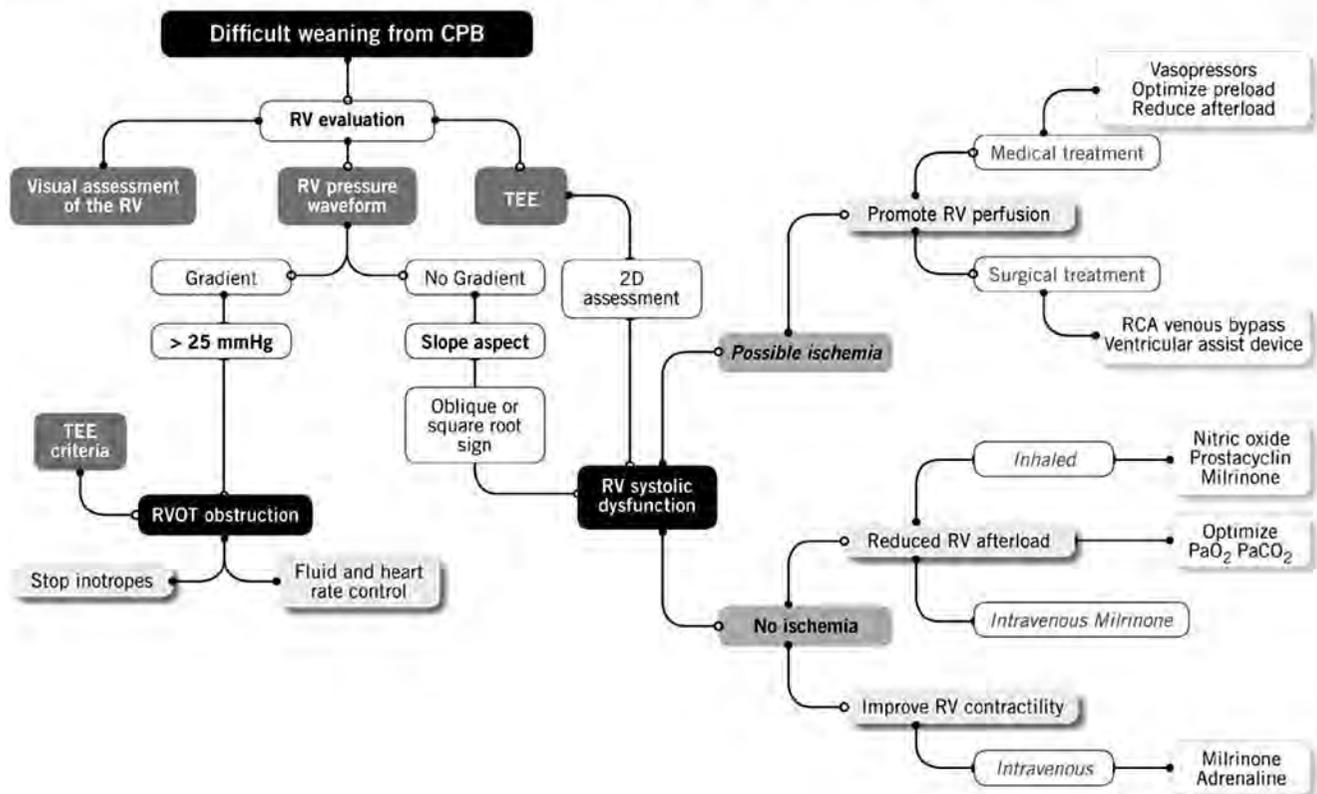


Fig. (9). Proposed approach in the treatment of right ventricular (RV) dysfunction. (RCA: right coronary artery, RVOT: right ventricular outflow tract, TEE: transesophageal echocardiography) (Presented by Dr. Y. Lamarche at the 2006 CCS meeting in Vancouver).

the surgical procedure and thus CPB time. As a consequence, in some cases, the drawbacks of using alternative procedures may overcome the benefits of avoiding PPM. It is therefore essential to establish accurate criteria to better assess the risk-benefit ratio with respect to the prevention of PPM. For mitral valve PPM, the best way would be to repair rather than replace the mitral valve. However, mitral valve repair cannot be provided to a significant number of patients and the options are more limited than for aortic valve replacement [16].

CONCLUSION

In summary, PHT in cardiac surgery is an important variable in cardiac surgery. It should be diagnosed before cardiac surgery and the impact of PHT on RV function determined. Future trials should address the role of pre-emptive reduction in the severity of PHT before cardiac surgery and their impact on post-operative outcome.

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What To Do When the Right Heart Fails? The Role of Right Mechanical Heart Assist

Vivek Rao, M.D., Ph.D., FRCPS

Insertion of a ventricular assist device (VAD) has become an established procedure for patients with both acute and chronic heart failure, with acceptable outcomes for either bridge to recovery (BTR) or bridge-to-transplant (BTT) indications. With technological advancement, non-pulsatile flow devices have produced superior outcomes compared to the earlier pulsatile devices [1-3].

Right ventricular (RV) dysfunction is a commonly observed complications after cardiac surgery. RV failure is variable in definition ranging from the need for mechanical RV support to prolonged inotropic support with or without inhalational therapies and even long term failure to thrive in the setting of isolated LVAD support. Although the prevalence of RV failure following LVAD implant appears to be lower in currently reported series, the wide range of definitions leads to a reported rate between 10% and 40% [1-5]. RV failure after LVAD implantation is associated with higher operative mortality and morbidity and longer stays in intensive care units and hospitals [6,7].

While most clinicians agree that early institution of RV support is preferable to “delayed” intervention, the key to avoiding RV failure is pre-emptive surgical optimization. Many authors have derived “risk-scores” for the development of RV failure. [4-8] Unfortunately, most of these risk scores were developed to predict the need for RVAD support after LVAD implant. However, regardless of the perceived risk of RV failure and independent of the planned surgical procedure, all patients should be medically optimized prior to surgical intervention. Our group believes that preoperative optimization is the single most important determinant of outcome following LVAD implant and any high risk surgical intervention. Preoperative medical therapy includes aggressive diuresis, pulmonary vasodilation and importantly nutritional support.

Intraoperatively, there are several surgical details that help to preserve RV function. Firstly, the surgeon in conjunction with anaesthesia must limit volume administration, especially during induction when volume is typically administered to maintain hemodynamic stability. Other measures to preserve the RV include avoiding acidosis and hypercarbia, both associated with increased pulmonary vascular resistance. The RV receives coronary perfusion during both systolic and diastolic phases of the cardiac cycle; however, the RV is relatively more sensitive to perfusion pressure than the LV. Therefore, avoiding hypotension is critical especially early after weaning from cardiopulmonary bypass.

Some surgeons prefer to avoid cardiopulmonary bypass where possible to protect the RV and to reduce bleeding complications following high risk surgical intervention. However, we have a low threshold to employ cardiopulmonary bypass particularly if there is concomitant tricuspid insufficiency. Although severe tricuspid insufficiency is a common indication to repair the valve, we have recently corroborated the findings of previous reports suggesting that anatomic criteria (such as annular dilatation) is more important than the regurgitant volume when determining the need for TV repair. [9-10]

Finally, when all preoperative and intraoperative measures have failed and one is forced to consider mechanical RV support, one must also anticipate potential RVAD explant. The ability to facilitate RVAD explant by the use of pursestring sutures vs continuous anastomoses and pericardial closure will ensure that RV recovery persists after RVAD explant. Again, careful positioning of outflow cannulas will prevent RV compression rendering determination of RV recovery difficult.

In summary, RV failure is likely under recognized and suboptimally managed following cardiac surgery. Careful preoperative optimization, meticulous surgical technique and attention to perioperative fluid management can all minimize the risk of RV failure. When mechanical RV support is required, the same philosophy will ultimately lead to a favourable clinical outcome.

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Bedside Point of Care Ultrasound in Assessment of RV Function

Massimiliano Meineri, M.D.

Overview

Right Ventricular (RV) dysfunction is a common clinical scenario in the Critical Care setting and it is associated with increased morbidity and mortality^{1,2}.

Quantification of RV dysfunction is challenging and commonly based on pressure tracing and physical examination.

Echocardiography allow precise quantification of RV function and its measurements have been established as strong prognosticators³.

Qualitative RV assessment can be reliably accomplished with transthoracic Focused Cardiac Ultrasound (FCU) and integrated into the physical examination⁴.

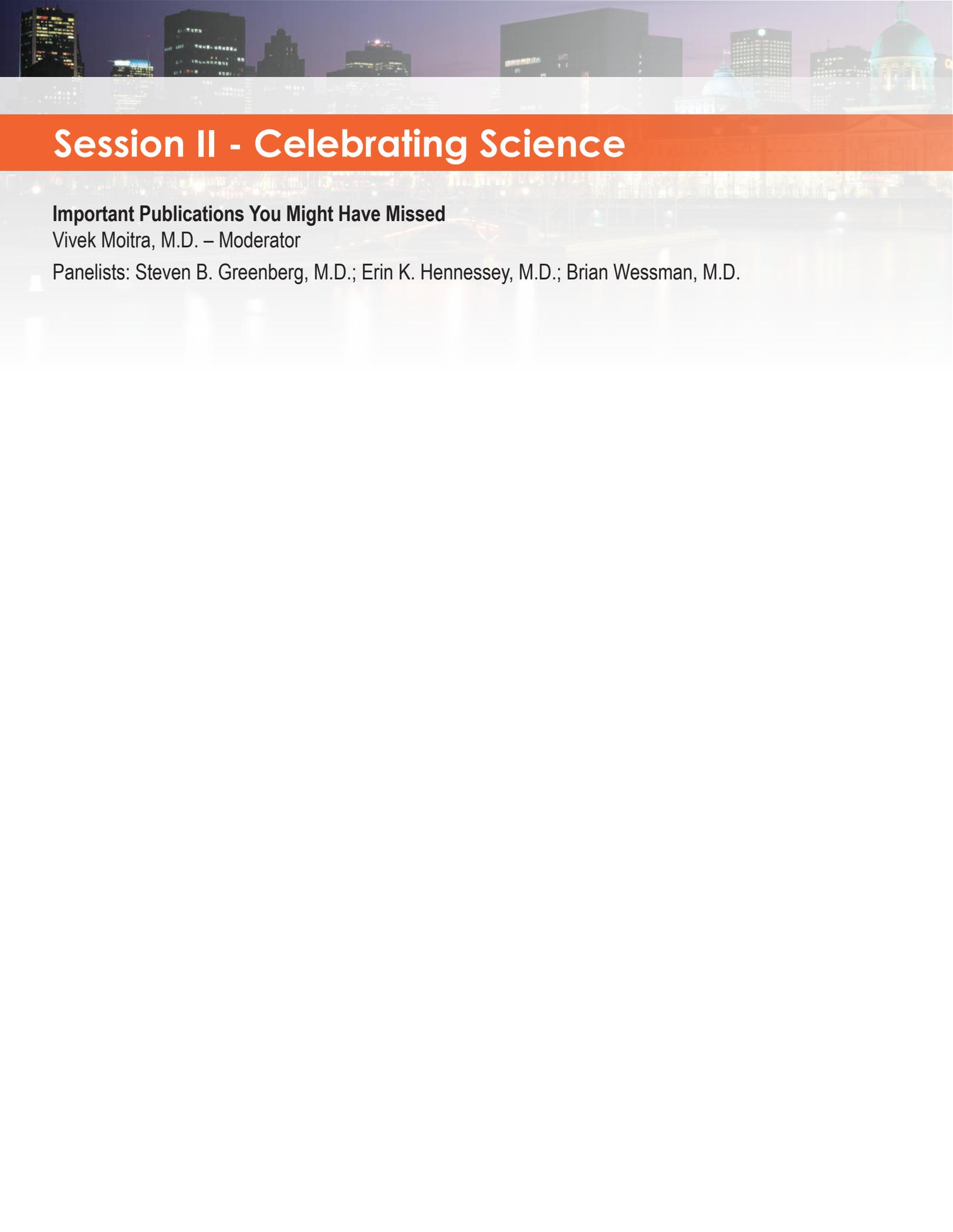
Quantitative RV assessment requires advanced transthoracic and trasesophageal echocardiographic skills and provides a deeper insight into RV pathophysiology⁵.

Learning Objectives

- Review RV physiology
- Discuss the indication and limitations of FCU in the assessment of RV function
- Discuss the prognostic value of Quantitative RV assessment
- Integrate echocardiography with standard bedside hemodynamic monitoring and standard imaging.

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A background image of a city skyline at night, featuring several illuminated skyscrapers and a prominent domed building on the right side. The image is partially obscured by a semi-transparent orange banner at the top.

Session II - Celebrating Science

Important Publications You Might Have Missed

Vivek Moitra, M.D. – Moderator

Panelists: Steven B. Greenberg, M.D.; Erin K. Hennessey, M.D.; Brian Wessman, M.D.

Important Publications You Might Have Missed

Vivek Moitra, M.D. – Moderator

Panelists: Steven B. Greenberg, M.D.; Erin K. Hennessey, M.D.; Brian Wessman, M.D.

The purpose of this panel is to provide you with an overview of carefully selected topics among the enormity of published literature over the past year. Our focus will be to highlight the applicability of the paper's conclusions to our patient populations.

These are the articles each of our curators have selected for brief discussion:

Steve Greenberg, M.D.:

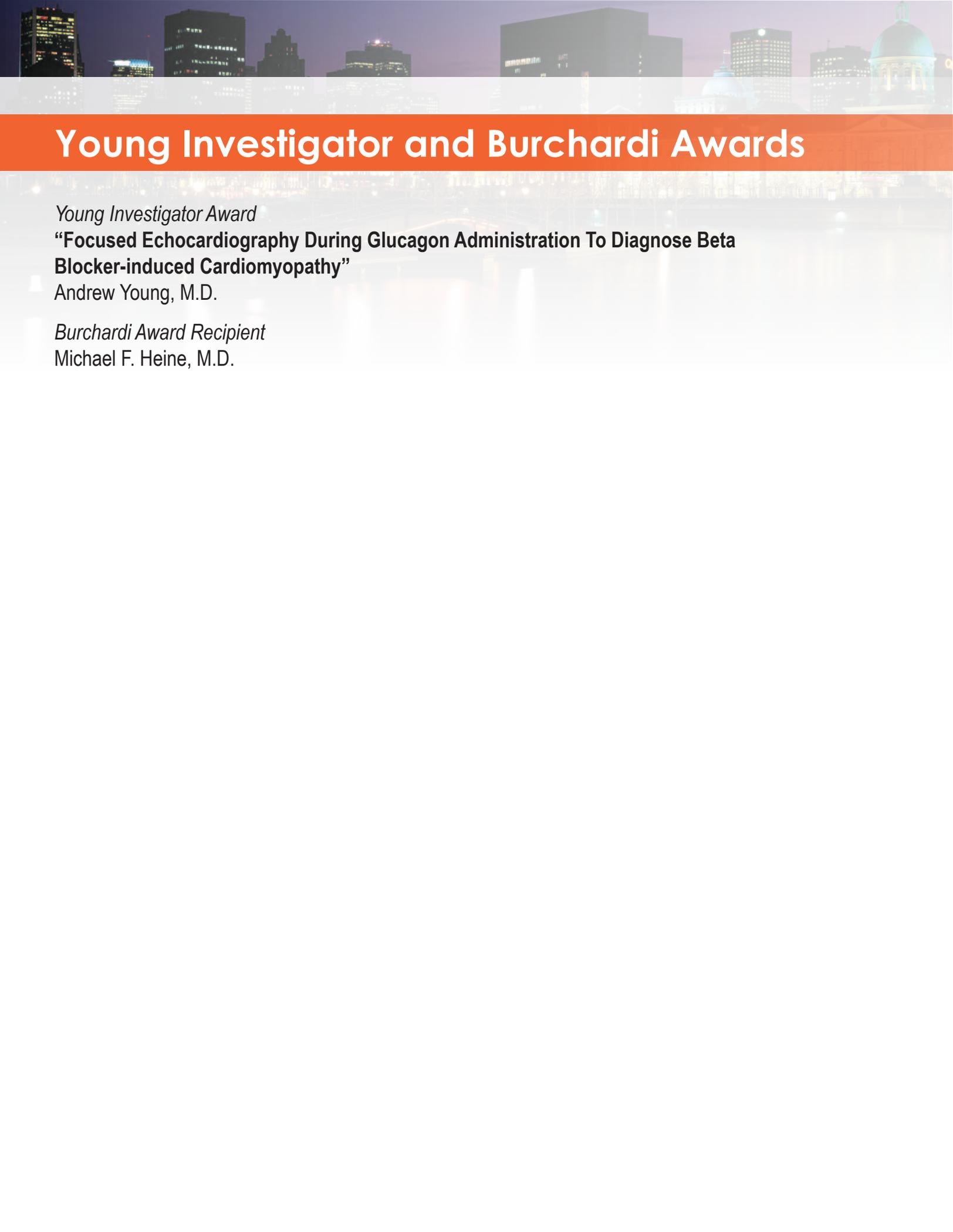
1. Targeted temperature management at 33°C versus 36°C after cardiac arrest.
Nielsen N, Wetterslev J, Cronberg T, Erlinge D, Gasche Y, Hassager C, Horn J, Hovdenes J, Kjaergaard J, Kuiper M, Pellis T, Stammet P, Wanscher M, Wise MP, Åneman A, Al-Subaie N, Boesgaard S, Bro-Jeppesen J, Brunetti I, Bugge JF, Hingston CD, Juffermans NP, Koopmans M, Køber L, Langørgen J, Lilja G, Møller JE, Rundgren M, Rylander C, Smid O, Werer C, Winkel P, Friberg H; TTM Trial Investigators.
N Engl J Med. 2013 Dec 5;369(23):2197-206.
2. Prone positioning in severe acute respiratory distress syndrome.
Guérin C, Reignier J, Richard JC, Beuret P, Gacouin A, Boulain T, Mercier E, Badet M, Mercat A, Baudin O, Clavel M, Chatellier D, Jaber S, Rosselli S, Mancebo J, Sirodot M, Hilbert G, Bengler C, Richecoeur J, Gannier M, Bayle F, Bourdin G, Leray V, Girard R, Baboi L, Ayzac L; PROSEVA Study Group.
N Engl J Med. 2013 Jun 6;368(23):2159-68.
3. A Randomized Trial of Protocol-Based Care for Early Septic Shock.
The ProCESS Investigators.
N Engl J Med. 2014 Mar 18 [Epub ahead of print].

Erin Hennessey, MD:

1. Effect of heart rate control with esmolol on hemodynamic and clinical outcomes in patients with septic shock: a randomized clinical trial.
Morelli A, Ertmer C, Westphal M, Rehberg S, Kampmeier T, Ligges S, Orecchioni A, D'Egidio A, D'Ippoliti F, Raffone C, Venditti M, Guarracino F, Girardis M, Tritapepe L, Pietropaoli P, Mebazaa A, Singer M.
JAMA. 2013 Oct 23;310(16):1683-91. doi: 10.1001/jama.2013.278477.
2. Effect of not monitoring residual gastric volume on risk of ventilator-associated pneumonia in adults receiving mechanical ventilation and early enteral feeding: a randomized controlled trial.
Reignier J, Mercier E, Le Gouge A, Boulain T, Desachy A, Bellec F, Clavel M, Frat JP, Plantefeve G, Quenot JP, Lascarrou JB; Clinical Research in Intensive Care and Sepsis (CRICS) Group.
JAMA. 2013 Jan 16;309(3):249-56.
3. Physician attire in the intensive care unit and patient family perceptions of physician professional characteristics.
Au S, Khandwala F, Stelfox HT.
JAMA Intern Med. 2013 Mar 25;173(6):465-7.

Brian Wessman, M.D.:

1. Effect of intravenous haloperidol on the duration of delirium and coma in critically ill patients (Hope-ICU): a randomised, double-blind, placebo-controlled trial.
Page VJ, Ely EW, Gates S, Zhao XB, Alce T, Shintani A, Jackson J, Perkins GD, McAuley DF.
Lancet Respir Med. 2013 Sep;1(7):515-23.
2. Transfusion strategies for acute upper gastrointestinal bleeding.
Villanueva C, Colomo A, Bosch A, Concepción M, Hernandez-Gea V, Aracil C, Graupera I, Poca M, Alvarez-Urturi C, Gordillo J, Guarner-Argente C, Santaló M, Muñoz E, Guarner C.
N Engl J Med. 2013 Jan 3;368(1):11-21.
3. A trial of intraoperative low-tidal-volume ventilation in abdominal surgery.
Futier E1, Constantin JM, Paugam-Burtz C, Pascal J, Eurin M, Neuschwander A, Marret E, Beaussier M, Gutton C, Lefrant JY, Allaouchiche B, Verzilli D, Leone M, De Jong A, Bazin JE, Pereira B, Jaber S; IMPROVE Study Group.
N Engl J Med. 2013 Aug 1;369(5):428-37.
4. Metastasis of E-mail at an Academic Medical Center.
Ian M. Paul, MD, MSc; Benjamin H. Levi, MD, PhD
JAMA Pediatr. 2014;168(3):290-291.

A background image of a city skyline at night, featuring various skyscrapers and a prominent domed building on the right. The image is partially obscured by a semi-transparent orange banner at the top.

Young Investigator and Burchardi Awards

Young Investigator Award

“Focused Echocardiography During Glucagon Administration To Diagnose Beta Blocker-induced Cardiomyopathy”

Andrew Young, M.D.

Burchardi Award Recipient

Michael F. Heine, M.D.

“Focused Echocardiography During Glucagon Administration To Diagnose Beta Blocker-induced Cardiomyopathy”

Andrew Young, M.D.

Andrew Young, M.D.; Matthias Merkel, M.D., Ph.D.; James Cooney, M.D.; Stephen Heitner, M.D.; Peter Schulman, M.D.
Oregon Health & Science University

Introduction: We report a case of acute cardiovascular collapse in a patient with a type B aortic dissection in which real time transthoracic echocardiography (TTE) was used during glucagon administration to diagnose beta-blocker (BB) overdose.

Case Report: A previously healthy 30 year-old African American man was admitted to the intensive care unit (ICU) for medical management of a type B aortic dissection. Adequate heart rate and blood pressure control required intravenous (IV) esmolol, diltiazem, nitroprusside, and oral labetalol, metoprolol and clonidine. 2.2 grams of labetalol and 1.3 grams of metoprolol were administered during the first 4 days.

On day 5 he developed acute hypotension unresponsive to fluid challenges, abdominal distention, and an elevated lactate (5.9 mmol/L) concerning for bowel ischemia. Norepinephrine and vasopressin infusions were initiated along with broad-spectrum antibiotics. During emergent surgery, 60 cm of ischemic small bowel was resected. Intraoperative transesophageal echocardiography revealed severely reduced LV function and epinephrine was started. Upon return to the ICU, Dobutamine was added for presumed septic cardiomyopathy, and the patient required a 10% dextrose infusion for persistent hypoglycemia.

Given his ongoing vasopressor requirement, hemodynamic instability and profound hypoglycemia, ultimately a BB-induced cardiomyopathy as a consequence of its altered enteral absorption was entertained. 5 mg IV glucagon was given, and then repeated 12 hours later, this time with the concurrent use of TTE to test the effect on cardiac function: within minutes the ejection fraction normalized. Hemodynamic support was

successfully weaned over the next 36 hours and he ultimately underwent endovascular aortic repair (figure).

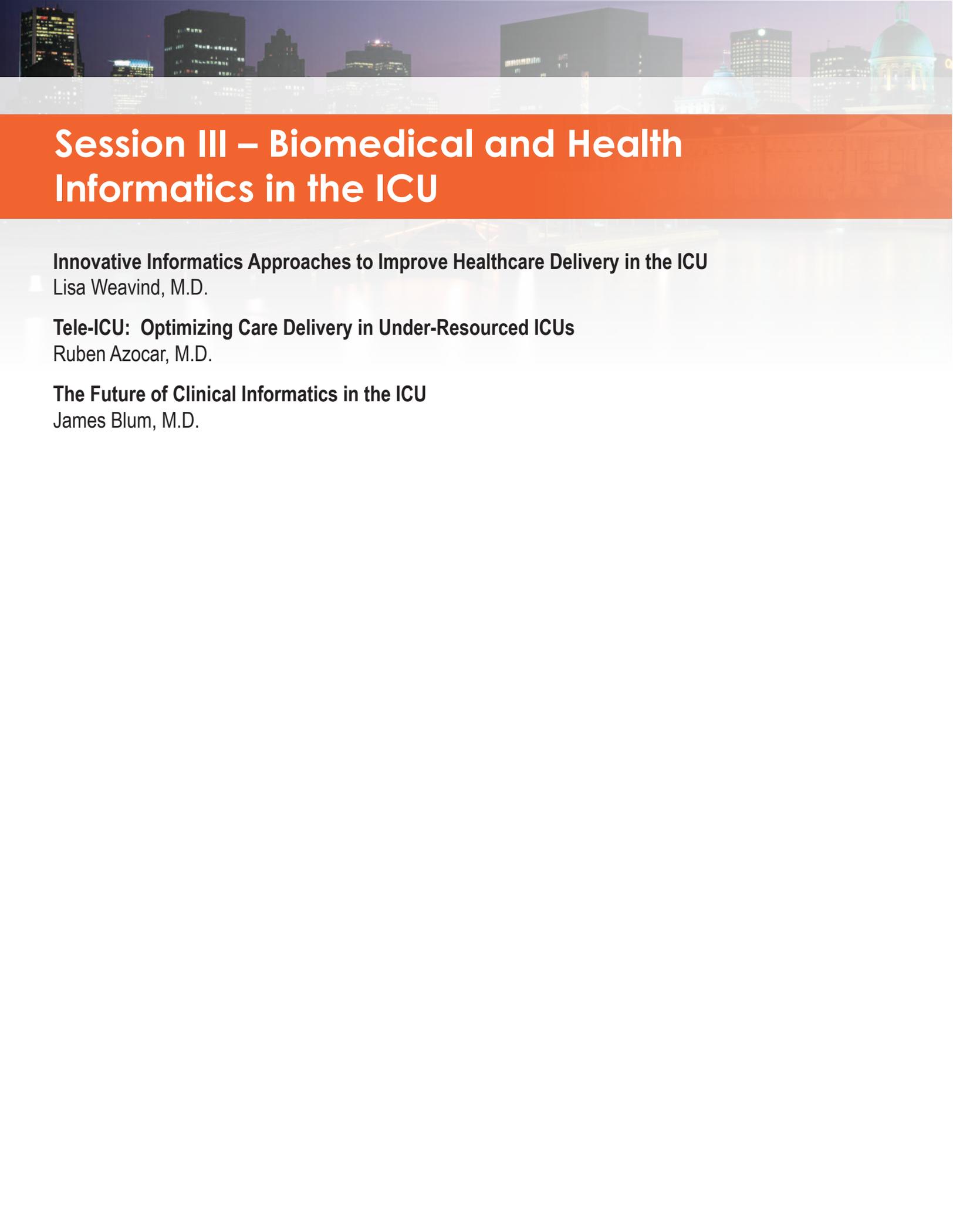
Conclusions: This case provides a new example of how real time TTE can be used to aid the diagnosis and management of a complex ICU patient with acute shock of unclear etiology. Although the benefits of focused bedside TTE in the ICU have been widely recognized¹, this is the first report describing its use during the administration of glucagon to guide diagnosis and treatment of acute BB toxicity.

Glucagon has positive chronotropic and inotropic effects through direct myocardial action via adenylyl cyclase that occur within minutes after administration². Glucagon reverses BB toxicity by providing the cAMP necessary for improved cardiac performance, stimulated via an alternative receptor. Its efficacy as an antidote for BB toxicity has been controversial in the literature; several case reports promote glucagon's efficacy³, though it has failed in other instances where the dose of BB ingestion was extremely high⁴.

In this case focused TTE allowed for an objective assessment of glucagon's immediate efficacy supporting the presumptive diagnosis of BB toxicity.

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A background image of a city skyline at night, featuring several illuminated skyscrapers and a prominent domed building on the right side. The image is partially obscured by a semi-transparent orange banner at the top.

Session III – Biomedical and Health Informatics in the ICU

Innovative Informatics Approaches to Improve Healthcare Delivery in the ICU

Lisa Weavind, M.D.

Tele-ICU: Optimizing Care Delivery in Under-Resourced ICUs

Ruben Azocar, M.D.

The Future of Clinical Informatics in the ICU

James Blum, M.D.

Innovative Informatics Approaches to Improve Healthcare Delivery in the ICU

Lisa Weavind, M.D.

My talk will be a high level review of some of the “Big Data” projects currently underway in a few ICU's and what that means for us in the future. I will then focus in on what we are doing at Vanderbilt University Hospital and how we are utilizing innovative informatics to assimilate and analyze multiple inputs of data from our ICU's to provide situational awareness to our clinicians. We will look at process and quality improvement initiatives, which should lead to improved healthcare delivery and patient safety outcomes.

Tele-ICU: Optimizing Care Delivery in Under-Resourced ICUs

Ruben Azocar, M.D.

Introduction

Intensive care units in the US care about six million for the sickest oldest patients yearly. They are responsible for rate highest mortality and cost in health care. An estimate 4.1% of the annual health care spending (nearly \$107 billion per year) is spent caring for the critically ill.¹ There is abundant data that demonstrates that critically ill patient have better outcomes and shorter length of stay when the care is provided by intensivist.²

As result groups such as the Leapfrog group, the leading organization of health care employers and purchasers, have established standards for intensivist care in the ICU. In the case of the Leapfrog group, the standard requires that only intensivist provide care in the ICU during daytime hours and that when they are no on-site or if telemedicine is not available, that intensivist should return pages within five minutes at least 95% of the time and that arrangements for a FCCS-certified physician or physician extender to reach the patients within five minutes exits.³

However, there are more than 5,000 hospitals in the United States and only about 6,000 intensivists. As only a minority of physicians train in critical care medicine, there is not solution for this gap in the near future. This is aggravated by the rapidly growing geriatric population, which will demand more critical care services as they live longer and undergo invasive procedures. It appears clear than Tele-ICU might be a sound alternative to meet standards and recommendations as the one stated above. The public has expressed their opinion on this matter. In Massachusetts, a study of the Massachusetts Technology Collaborative and the New England Healthcare Institute was designed to determine if Tele-ICU technology would meet criteria for their Fast Adoption of Significant Technologies (FAST) initiative.⁴ They determined that not only these technologies meet their criteria, but also they established a series of recommendations made public in December of 2010. Those include that ALL Massachusetts academic medical centers should implement Tele-ICU systems in their primary and affiliated hospitals by 2014 and that by 2015 ALL community hospitals having 10 or more beds with a 45% occupancy rate should do the same.

Tele-ICU in the US

Since the installation of the first Tele-ICU in the US only a relative small number of Hospitals have embraced this technology. The larger provider of Tele-ICU is eICU® from Phillips and their website on March 2014 reports only about 350 hospitals across the US using their product.⁵ It is very likely than no more than 20% of the hospitals in the US are using any Tele-ICU.

This is very surprising considering the Leapfrog standards and that there is solid data supporting the use of Tele-ICU's. Lilly and colleagues reported data before and after implementation of a Tele-ICU initiative in a single Academic Medical Center⁶. The hospital mortality rate was 13.6% during the pre-intervention period compared with 11.8% during the Tele-ICU intervention period. Shorter hospital length of stay was also noted (9.8 vs 13.3 days, respectively). They also reported better adherence to best practices such as prevention of deep vein thrombosis, prevention of stress ulcers, cardiovascular protection, ventilator-associated pneumonia and for catheter-related bloodstream infection. On a more recent review from 118,990 adult patients (11,558 control subjects, 107,432

intervention group patients) 56 ICU's and 32 hospitals in 19 different US health-care systems, similar results were reported.⁷ Mortality in the ICU telemedicine intervention group was significantly better than that of control subjects., adjusted hospital LOS was reduced, on average, by 0.5 days, and adjusted ICU LOS was reduced by 1.1 days. They suggest that interventions that were associated with lower mortality, reduced LOS, or both included (1) intensivist case review within 1 h of admission, (2) timely use of performance data, (3) adherence to ICU best practices, and (4) quicker alert response times.

If Tele-ICU allows critical care patients access to an intensivist, decreases mortality, length of stay and costs: Why is it not wide-spread? Why has it failed in other institutions?

The Barriers

There are multiple reasons for this phenomenon. Organization and physician resistance, technical incompatibilities, lack of a clear implementation plan, cross state license issues, lack of reimbursement for telemedicine services but probably most importantly the very high cost of implementation and subsequent expansion.

In the MTC/NEHI report a \$6 to 8 million in one time capital cost to set up the command center, acquire and install Tele-ICU systems and pay initial salaries for the Tele-ICU staff were quoted. Cost for a satellite hospital to acquire and install the Tele-ICU technology ranges form \$300,000- to \$500,000.⁴

In a recent publication, the authors reviewed and summarized the data of the existing literature on the cost of Tele-ICU's and collected data on the "live" implementation of a Tele-ICU in a network of Veteran; Administration Hospitals. Their review of the existing systems suggested a combined implementation and first year operation cost for Tele-ICU of \$50,000 to \$100,000 per monitored bed. Data form the VHA suggested cost of implementation and first year of operation of \$70,000 to \$87,000 per ICU bed depending on the method of depreciation.⁸ In an austere healthcare economy only a handful of hospitals can afford these costs

The Future

As communications become faster and more accessible, will telemedicine technology adapt and become more flexible, portable and affordable?

Summary

Tele-ICU has the potential to allow access of critically ill patients to intensivists and improve outcomes. However, multiple barriers for implementation and sustainability exist and are limiting the use of this technology. Cost remains as the most salient issue, but there are others that have a negative impact as well. As communication technology continues to advance, will there be a change in the way Tele-ICU is done?

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The Future of Clinical Informatics in the ICU

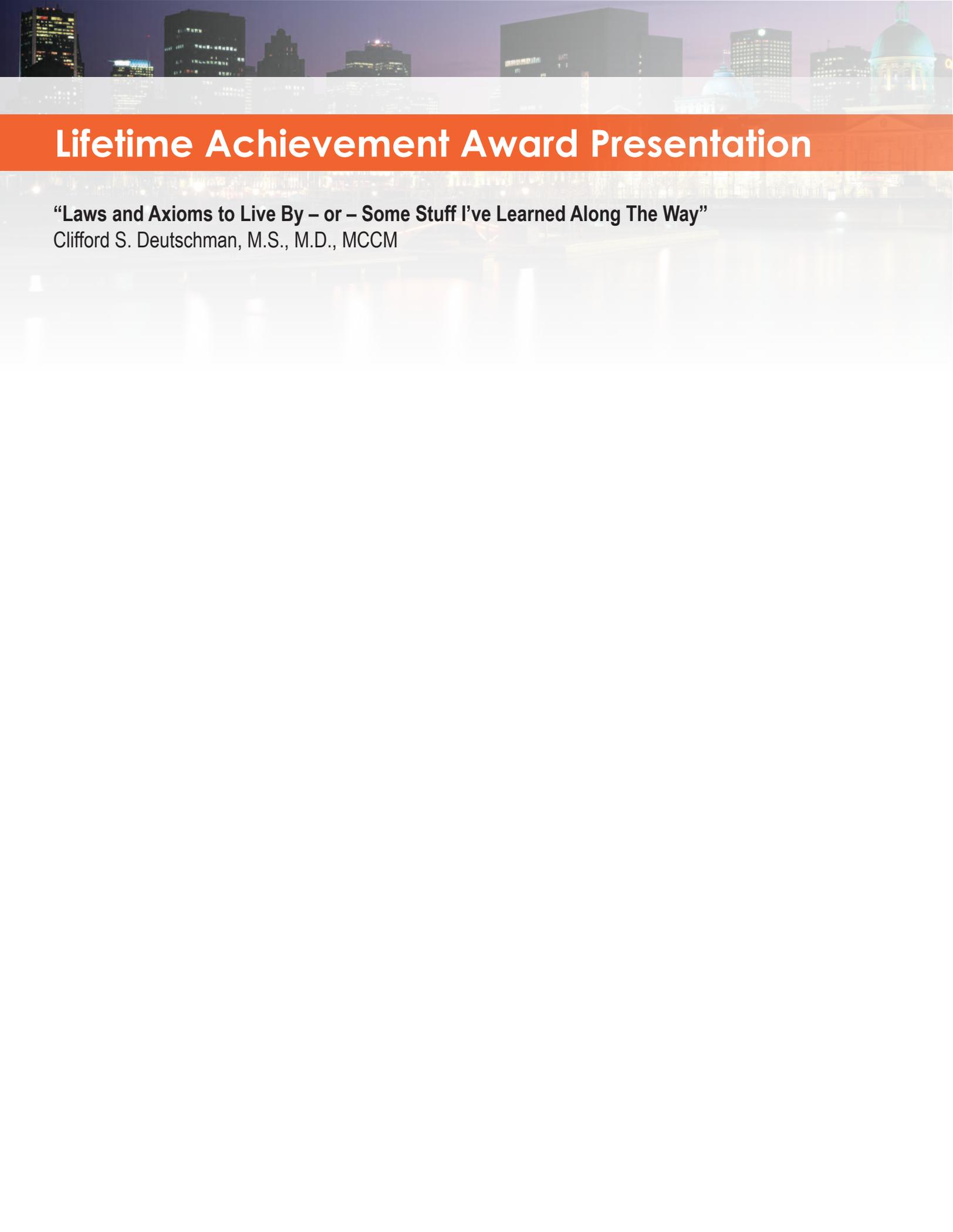
James Blum, M.D.

Over the past five years, there has been a profound increase in the use of electronic medical record systems in healthcare. Much of this has been driven through government incentives with the thought that such systems will increase efficiencies, reduce duplication of studies, and decrease the cost of care. Many institutions have now chosen to move from best in breed clinical information systems to unified single vendor solutions that provide solid integration.

With these implementations come an array of possibilities that will fundamentally change the way we provide care. By leveraging data from multiple data sources individual care and research will be greatly enhanced. In the future, there will be a drive towards increased clinical volume per clinician through the use of clinical informatics technologies and a drive toward increased utilization of quality metrics.

Examples of the way increased volume per clinician will be obtained are through the use of electronic ICU (eICU) coverage, improved clinical decision support, and advanced forms of monitoring that make use of new forms of information. The drive toward benchmarking and quality metrics will be facilitated by the aggregation of data similar to APACHE, which will provide quality data at both the facility and practitioner level.

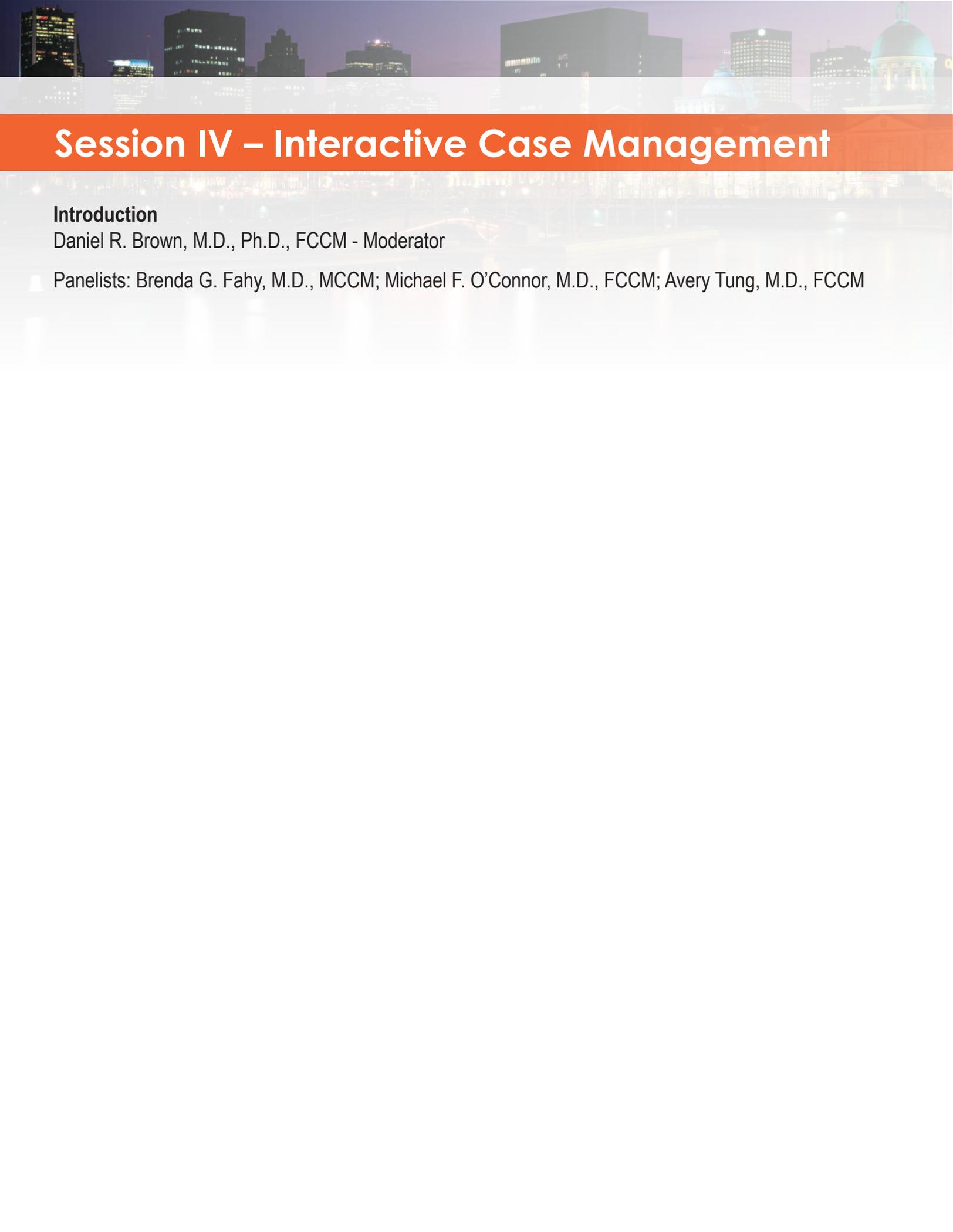
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Lifetime Achievement Award Presentation

“Laws and Axioms to Live By – or – Some Stuff I’ve Learned Along The Way”

Clifford S. Deutschman, M.S., M.D., MCCM

A background image of a city skyline at night, featuring several illuminated skyscrapers and a prominent domed building on the right side. The scene is reflected in water in the foreground.

Session IV – Interactive Case Management

Introduction

Daniel R. Brown, M.D., Ph.D., FCCM - Moderator

Panelists: Brenda G. Fahy, M.D., MCCM; Michael F. O'Connor, M.D., FCCM; Avery Tung, M.D., FCCM

Poster Presentations

- Poster # 1 **Methylene Blue Administration for Acute Septic Cardiomyopathy in a Severely Burned Patient**
Joseph Schlesinger, M.D.¹; Christina Burger, Pharm.D.²
Vanderbilt University Medical Center ¹; Saint Francis Hospital²
- Poster # 2 **Young Investigator Award**
Focused Echocardiography During Glucagon Administration To Diagnose Beta Blocker-Induced Cardiomyopathy
Andrew Young, M.D.; Matthias Merkel, M.D., Ph.D.; James Cooney, M.D.; Stephen Heitner, M.D.; Peter Schulman, M.D.
Oregon Health & Science University
- Poster # 3 **Pneumatosis Intestinalis and Portal Venous Gas: An Ominous Sign?**
Brendan Wanta, M.D.; Arun Subramanian, M.B.B.S.; Mark T. Keegan, M.D.
Mayo Clinic - Rochester
- Poster # 4 **A Case of Hyperacute Liver Failure After Volatile Anesthetic in a Patient with Quiescent Autoimmune Hepatitis**
David R. Wetzel, M.D.; Richard K. Patch, M.D.; Jeffrey B. Jensen, M.D.; Daniel R. Brown, M.D., Ph.D.
Mayo Clinic
- Poster # 5 **Serotonin Release Assay Negative in a Post-Whipple Patient with New Onset Thrombocytopenia and Thrombosis: Is It Heparin-Induced Thrombocytopenia?**
Edward C. Yang, M.D.¹; Nadia Haider, M.D.²
Advocate Illinois Masonic Medical Center¹; Edward J Hines, VA Medical Center ²
- Poster # 6 **Medically Challenging Case of TEN Associated with ITP Treated with Intravenous Immunoglobulin**
Michael D. Hermann, M.D.¹; Joe Schlesinger, M.D.¹; Christie Burger, Pharm.D.²
Vanderbilt University, Department of Anesthesiology, Division of Critical Care Medicine¹; Department of Clinical Pharmacy, Saint Francis Hospital, Memphis, TN²
- Poster # 7 **Utilization of a Web-Based Conference Series for State-Wide Learning and Collaboration: An e-Enhancement for Multi-institutional and Multidisciplinary Critical Care Fellowship Training**
Steven G. Venticinque, M.D.¹; Antonio Hernandez, M.D.²; Jason D. Aydelotte, M.D.³; Christopher E. White, M.D.⁴; John G. Myers, M.D.¹; Ronald M. Stewart, M.D.¹
University of Texas Health Science Center San Antonio, Department of Surgery, San Antonio, TX¹; Vanderbilt University Medical Center, Department of Anesthesiology, Nashville, TN²; Seton Healthcare, Austin, TX³; San Antonio Uniformed Services Health Education Consortium, Department of Surgery, Fort Sam Houston, TX⁴
- Poster # 8 **Mediastinal Compartment Syndrome After Type A Aortic Dissection Repair**
P. Andrew Stephens, M.D.; Heather McFarland, M.D.; Mitali Shah, M.D.
University Hospitals Case Medical Center
- Poster # 9 **Postpartum Headaches: A Multi-Disciplinary Concern**
Alexander N. Kahan, M.D.; John T. Denny, M.D.
Rutgers Robert Wood Johnson Medical School
- Poster # 10 **Efficient Vascular Access in a Hypovolemic Trauma Patient**
Kimberly I. McClelland, M.P.H.; Clairmont E. Griffith, M.D.; Besrat Mesfin, M.D.
Howard University College of Medicine
- Poster # 11 **Peri-Engraftment Respiratory Distress Syndrome: A Rare Cause of Acute Respiratory Failure**
Channing Twyner, M.D.; Arun Subramanian, M.B.B.S.
Mayo Clinic

Poster Presentations

- Poster # 12 **A Neurophysiological Approach to Electroencephalogram Monitoring During General Anesthesia and Sedation**
Patrick L. Purdon, Ph.D.; Emery N. Brown, M.D., Ph.D.
Massachusetts General Hospital
- Poster # 13 **Negative Pressure Pulmonary Edema with Superimposed Fat Embolism: The Value of the Differential Diagnosis**
David Graham, M.D.
Department of Anesthesia, University of Tennessee, Knoxville
- Poster # 14 **Making the Best of Tragedy: Brain Death in a Pregnant Mother Initially Presenting with a Nonviable Fetus**
David W. Miller, M.D.; Kenneth G. Smithson, M.D., Ph.D.; Andrew B. Barker, M.D.; Joseph R. Biggio, M.D.; Barton L. Guthrie, M.D.
University of Alabama at Birmingham
- Poster # 15 **Successful Use of Inhaled Nitric Oxide in Management of Critical Pulmonary Hypertension as Observed in Real Time with a Disposable ImaCor hTEE™ Probe**
Meghan Kirksey, M.D., Ph.D.¹; Robert Sladen, M.D.²
Hospital for Special Surgery¹; Columbia University²
- Poster # 16 **Gas Gangrene: Delayed Diagnosis Results in Mortality. A Case Report.**
Zyad J. Carr, M.D.
Dartmouth Hitchcock Medical Center
- Poster # 17 **Percutaneous Tracheotomy: A Modified Technique to Facilitate Placement and Improve Patient Safety**
Christopher P. Owen, M.D.; Philip G. Boysen, M.D., M.B.A.
Ochsner Clinic Foundation
- Poster # 18 **Malignant Cerebral Edema Secondary to Gliadel Wafers in the Early Postsurgical Period**
Ana Belen Fernandez, M.D.¹; Itahisa Cabrera, M.D.²; Liuva Pereira²; Eglis Lazo, M.D.²
Ntra Sra De Candelaria University Hospital ¹; Delaria Hospital²
- Poster # 19 **The Utility of a Fellowship Lecture Development Series: A Formal Process to Teach Fellows How to Create and Deliver Effective Presentations**
Steven G. Venticinque, M.D.; Stephen M. Cohn, M.D.
University of Texas Health Science Center San Antonio, Department of Surgery
- Poster # 20 **Goals and Pitfalls Involved in Selection of Agents for Hypertensive Augmentation for Vasospasm Treatment After Aneurysmal Subarachnoid Hemorrhage (aSAH) and Middle Cerebral Artery (MCA) Clipping with Superior Temporal (STA) to MCA Bypass**
Erin M. Etoll, M.D.; Peggy White, M.D.; Brenda Fahy, M.D.
University of Florida

Methylene Blue Administration for Acute Septic Cardiomyopathy in a Severely Burned Patient

Joseph Schlesinger, M.D.¹; Christina Burger, Pharm.D.²
Vanderbilt University Medical Center ¹; Saint Francis Hospital²

Introduction: A 60 year old male with no known past medical history was admitted to the Burn Intensive Care Unit with a 45% total body surface area burn secondary to a house fire.

Case Report: During hospitalization, the patient developed septic shock from a pneumonia that was poorly responsive to fluid therapy and vasoactive medications. Transthoracic echocardiography demonstrated a global hypokinesis with an ejection fraction of 10% (from a previous normal 55% ejection fraction). The patient was refractory to infusions of epinephrine, milrinone, norepinephrine, and vasopressin. A pulmonary artery catheter demonstrated a cardiac index of 0.9 L/min/m² and a systemic vascular resistance of 2100 mmHg. While methylene blue (MB) is typically used for vasoplegia and vasodilatory shock, there is literature that it can improve myocardial performance in the face of septic cardiomyopathy. Therefore we administered methylene blue to improve cardiac performance, weaned our vasoactive medications, and utilized a low-dose furosemide infusion for afterload reduction.

Conclusions: Nitric oxide (NO) is believed to contribute to the detrimental hemodynamic effects associated with septic shock. NO activates the second messenger guanylate cyclase, which converts cyclic guanosine triphosphate (cGTP) to cyclic guanosine monophosphate (cGMP), resulting in smooth muscle relaxation. In septic patients, bacterial endotoxins and inflammatory cytokines can activate production of an inducible nitric oxide synthase (iNOS), leading

to excessive production of NO and cGMP. iNOS is present in both the vasculature and the myocardium. The sustained production of NO from iNOS leads to profound vasodilation, hyporeactivity to catecholamines/vasopressors, and decreased inotropy. Methylene blue (MB) has been used successfully in the treatment of shock and acts through inhibition of eNOS and iNOS. Recent reports have shown that MB increases mean arterial pressure (with or without vasopressors) and reduces adrenergic support requirements in patients with sepsis. In addition, MB has been shown to increase cardiac contractility (increased SVI, LVSWI, RVSWI) through increased sensitivity to catecholamines (endogenous and exogenous) and/or by reducing the attenuating effect of NO on myocyte contraction.

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Young Investigator Award

Focused Echocardiography During Glucagon Administration To Diagnose Beta Blocker-Induced Cardiomyopathy

Andrew Young, M.D.; Matthias Merkel, M.D., Ph.D.; James Cooney, M.D.; Stephen Heitner, M.D.; Peter Schulman, M.D.
Oregon Health & Science University

Introduction: We report a case of acute cardiovascular collapse in a patient with a type B aortic dissection in which real time transthoracic echocardiography (TTE) was used during glucagon administration to diagnose beta-blocker (BB) overdose.

Case Report: A previously healthy 30 year-old African American man was admitted to the intensive care unit (ICU) for medical management of a type B aortic dissection. Adequate heart rate and blood pressure control required intravenous (IV) esmolol, diltiazem, nitroprusside, and oral labetalol, metoprolol and clonidine. 2.2 grams of labetalol and 1.3 grams of metoprolol were administered during the first 4 days.

On day 5 he developed acute hypotension unresponsive to fluid challenges, abdominal distention, and an elevated lactate (5.9 mmol/L) concerning for bowel ischemia. Norepinephrine and vasopressin infusions were initiated along with broad-spectrum antibiotics. During emergent surgery, 60 cm of ischemic small bowel was resected. Intraoperative transesophageal echocardiography revealed severely reduced LV function and epinephrine was started. Upon return to the ICU, Dobutamine was added for presumed septic cardiomyopathy, and the patient required a 10% dextrose infusion for persistent hypoglycemia.

Given his ongoing vasopressor requirement, hemodynamic instability and profound hypoglycemia, ultimately a BB-induced cardiomyopathy as a consequence of its altered enteral absorption was entertained. 5 mg IV glucagon was given, and then repeated 12 hours later, this time with the concurrent use of TTE to test the effect on cardiac function: within minutes the ejection fraction normalized. Hemodynamic support was

successfully weaned over the next 36 hours and he ultimately underwent endovascular aortic repair (figure).

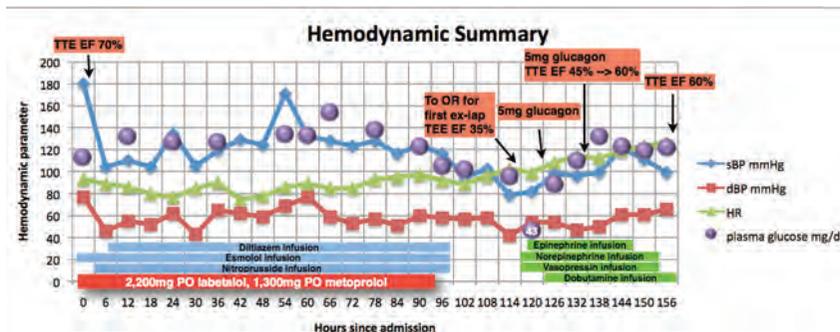
Conclusions: This case provides a new example of how real time TTE can be used to aid the diagnosis and management of a complex ICU patient with acute shock of unclear etiology. Although the benefits of focused bedside TTE in the ICU have been widely recognized¹, this is the first report describing its use during the administration of glucagon to guide diagnosis and treatment of acute BB toxicity.

Glucagon has positive chronotropic and inotropic effects through direct myocardial action via adenylyl cyclase that occur within minutes after administration². Glucagon reverses BB toxicity by providing the cAMP necessary for improved cardiac performance, stimulated via an alternative receptor. Its efficacy as an antidote for BB toxicity has been controversial in the literature; several case reports promote glucagon's efficacy³, though it has failed in other instances where the dose of BB ingestion was extremely high⁴.

In this case focused TTE allowed for an objective assessment of glucagon's immediate efficacy supporting the presumptive diagnosis of BB toxicity.

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Pneumatosis Intestinalis and Portal Venous Gas: An Ominous Sign?

Brendan Wanta, M.D.; Arun Subramanian, M.B.B.S.; Mark T. Keegan, M.D.
Mayo Clinic - Rochester

Introduction: Pneumatosis intestinalis (PI) is the radiographic finding of air in the intestinal wall. It is found in up to 0.37% of patients who undergo radiographic imaging and is becoming a more common finding with increased use of computed tomography (CT). [1] PI represents a spectrum of disease from benign to life-threatening, so it is important to determine disease severity to establish a treatment plan. When PI is accompanied by visualization of portal venous gas, the likelihood of the presence of bowel necrosis greatly increases.[2]

Review of Case: A 70 year old female with recurrent high-grade ovarian cancer (status-post debulking surgery and chemotherapy), intermittent small bowel obstruction, and atrial fibrillation was admitted to the hospital with nausea, vomiting and diarrhea of several weeks' duration. She was transferred to the intensive care unit (ICU) for the treatment of rapidly-developing respiratory failure and distributive shock. Laboratory analyses revealed a lactic acidosis and acidemia (lactate 5.9 mmol/L, pH 7.17, HCO₃- 12 mmol/L), neutropenia, coagulopathy, and acute kidney injury. An emergent abdominal CT scan (Figure 1), revealed PI and portal venous gas. A diagnosis of presumed acute bowel ischemia was made and the patient was appropriately resuscitated. Surgical

options were discussed, but deferred due to the patient's pre-admission impaired functional status, poor prognosis, and family wishes. Comfort cares were initiated and the patient expired shortly after. Autopsy revealed internal bowel herniation through thick adhesions and a large thrombus in the splenic vein.

Conclusions: Patients admitted to the ICU frequently undergo imaging studies, which may demonstrate PI, the significance of which needs to be determined. Intensivists need to distinguish conditions for which emergent surgical intervention is required from those in which the presence of PI is a benign finding. In this case, the additional finding of portal venous gas, coupled with the clinical manifestations of an acute abdomen, suggested the presence of catastrophic bowel ischemia.

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A Case of Hyperacute Liver Failure After Volatile Anesthetic in a Patient with Quiescent Autoimmune Hepatitis

David R. Wetzel, M.D.; Richard K. Patch, M.D.; Jeffrey B. Jensen, M.D.; Daniel R. Brown, M.D., Ph.D.
Mayo Clinic

Context: Volatile anesthetics are used extensively for safe, effective anesthesia. Volatile agents have been linked to adverse reactions. Rare case reports of sevoflurane causing severe liver failure exist (1-5). We present a case of hyperacute fulminant hepatic failure in a patient with a history of quiescent autoimmune hepatitis following uneventful primary hip arthroplasty with sevoflurane.

A 65 year old female underwent an elective total hip arthroplasty. Past history was notable for autoimmune hepatitis and steatosis successfully treated with a course prednisone and azathioprine two years prior. Preoperative labs were at baseline. Her history is also notable for prior volatile-based anesthetics without complications. The patient denied taking herbal remedies or over-the-counter medications.

Anesthesia utilizing sevoflurane was unremarkable. After an uneventful PACU recovery, the patient was admitted to the general care floor. Approximately six hours after PACU dismissal, the rapid response team was activated for evaluation and transferred to emergently to the ICU in shock. Hemodynamics stabilized on a norepinephrine infusion. An initial arterial blood gas (ABG) demonstrated a severe metabolic acidosis (6.98/22/288/5/-24) associated with a fever of 39.8 C and dark red-brown urine. Notable admission ICU lab data included: lactate 16 , INR 2.9, fibrinogen < 60, ALKPhos 173 , ALT 338 , total bilirubin of 3.5 and a creatinine increase to 1.6.

Cultures and broad spectrum antibiotics were initiated. STAT echocardiogram was unremarkable. Malignant hyperthermia was entertained resulting in empiric dantrolene administration. A repeat ABG revealed ongoing acidosis.

She was not considered a candidate for liver transplantation. Extensive evaluations for causes of acute hepatic failure were unrevealing. Multiorgan failure progressed and patient died on ICU day 2.

We present a case of hyperacute liver failure leading to multisystem collapse within hours of an uncomplicated total hip arthroplasty in a patient with history of quiescent autoimmune hepatitis. Evidence of liver injury, absent during her preoperative evaluation, was apparent upon ICU admission and ultimately resulted in the patient's demise. Volatile anesthetics have been implicated in immune mediated hepatic injury through metabolites bound to hepatic proteins (6). Sevoflurane hepatic metabolism results in a different metabolite which is ultimately excreted renally (6). Proposed mechanisms of injury relate to increased intracellular calcium ions causing cellular necrosis, free radical enzymes activation leading to sensitization and injury on re-exposure, or effects from compound A (5,7,8).

Other cause assessments were unremarkable. We question if sevoflurane may have provoked acute hepatic failure in this patient.

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Serotonin Release Assay Negative in a Post-Whipple Patient with New Onset Thrombocytopenia and Thrombosis: Is It Heparin-Induced Thrombocytopenia?

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Advocate Illinois Masonic Medical Center¹; Edward J Hines, VA Medical Center²

Heparin-induced thrombocytopenia (HIT) is an immune-mediated complication of heparin therapy (1). Diagnosis can be challenging, requiring clinical suspicion coupled with laboratory data. A commonly used algorithm for scoring the likelihood of HIT is the 4T⁴ test (2), in conjunction with heparin-induced platelet antibody / platelet factor 4 and serotonin release assay (1). However, laboratory tests have a lag time, and false-positives and false-negatives remain a concern. We present a case of a post-Whipple patient suspected to have HIT with conflicting laboratory tests, but who clinically improved off heparin.

Case Description: A 78 year old male with history of coronary artery disease, peptic ulcer disease, and peripheral vascular disease presented for a Whipple. On post-operative day (POD) #8, he developed shock requiring emergent exploratory laparotomy, revealing gastric and left lobe of liver necrosis. On POD #11, patient's upper lip became necrotic, thought to be pressure necrosis from an endotracheal tube by otolaryngology. On POD #12, patient developed a cold lower extremity, but vascular surgery was unable to operate and recommended systemic heparin anticoagulation. Concomitantly, patient's platelets had trended down from pre-admission of 176,000 to a nadir of 67,000 by POD #12, and of note, prophylactic heparin had been started on POD#1. Given the timing and degree of thrombocytopenia and the suspicion of multiple sites of thrombosis, clinical suspicion for HIT was very high. Heparin was stopped and anticoagulation switched to bivalirudin; heparin induced platelet antibody / platelet factor 4 was positive at an optical density of 0.899, but serotonin release assay was negative. Clinical suspicion remained high and thus the patient was treated as HIT positive. Patient's

platelets improved immediately after heparin exposure was stopped and returned to greater than 150,000. Hematology-oncology consult recommended 6 months of anticoagulation.

Discussion: Heparin-induced thrombocytopenia is an antibody-mediated, adverse effect of heparin with life-threatening complications (1). Accurate and early diagnosis is critical; unfortunately, there is no "magic-bullet" laboratory test that definitely diagnoses HIT; even so-called confirmatory tests have a specificity and sensitivity that is not 100%. Diagnosis remains clinical, using scoring systems such as 4T⁴ (2) with supportive laboratory testing. Our case illustrates an example of a classic "false negative", where a supposed test (e.g. serotonin release assay) with greater than 98% sensitivity and specificity failed to predict a diagnosis. Clinical training relies heavily on "gold standard" laboratory testing, and although laboratory tests are useful for treatment, it is important to remember their limitations.

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Medically Challenging Case of TEN Associated with ITP Treated with Intravenous Immunoglobulin

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Introduction: This case report presents a unique circumstance in which a patient with a history of idiopathic thrombocytopenic purpura (ITP) was admitted with toxic epidermal necrolysis (TEN). Intravenous immunoglobulin (IVIG) was administered as a treatment modality for both diseases. This case report highlights the utility of IVIG for the treatment of multiple diseases as well as the need for monitoring for potential adverse effects.

Case Report: A 75 year old white female with a past medical history of ITP was a direct admission to a burn intensive care unit with 40% total body surface area (TBSA) TEN involvement (15% epidermal loss) without mucosal involvement. The patient previously acquired suspected *Pneumocystis jiroveci* pneumonia and trimethoprim-sulfamethoxazole (TMP-SMX) was prescribed. Within 48 hours of starting TMP-SMX, a rapidly progressing desquamating rash evolved on her back. On day 2 of her rash, she was admitted and started on IVIG and broad-spectrum antibiotics. TEN was confirmed by skin biopsy. Each IVIG infusion was preceded by administration of acetaminophen and diphenhydramine and the patient was monitored closely for adverse reactions by the ICU team. She experienced no immediate or late adverse events. After undergoing xenografting the patient was discharged on hospital day eleven.

Conclusions: ITP and TEN are examples of immune dysregulation for which IVIG is an adjunctive treatment¹⁻³. While IVIG has clinical utility, there are risks to the patient. Adverse effects are usually mild and immediate. Such reactions include tachycardia, headaches, nausea, wheezing, flushing, fevers, chills, and diarrhea⁴⁻⁵. Late reactions include acute renal failure, aseptic meningitis, hemolysis, and hyperviscosity related thromboembolic complications (cerebrovascular accident, myocardial infarction, deep venous thrombosis, and pulmonary embolism)⁴⁻⁵. Clinicians responsible for administering this blood product derivative should be familiar with these complications as well as proper management. This report highlights the use of IVIG to treat TEN in a critically ill patient with concomitant ITP and alerts clinicians to the potential for adverse events when using this agent.

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Utilization of a Web-Based Conference Series for State-Wide Learning and Collaboration: An e-Enhancement for Multi-institutional and Multidisciplinary Critical Care Fellowship Training

Steven G. Venticinque, M.D.¹; Antonio Hernandez, M.D.²; Jason D. Aydelotte, M.D.³; Christopher E. White, M.D.⁴; John G. Myers, M.D.¹; Ronald M. Stewart, M.D.¹

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Educational Objectives: Maintaining a high quality, independent conference series for critical care fellowship training programs can be difficult due to challenges such as few faculty, the breadth and complexity of critical care, the dispatch of fellows to other sites, and the need to maintain effective conferences. We describe our experience with a de-novo, internet-based conference series that serves as a foundational didactic element for our critical care fellowship-training consortium, and we describe the growth of our conference series within our state. The main objectives of the series are to enhance lecture access, trainee participation, lecture quality, and the exposure of trainees to a broader-base of expertise and opinion.

Program Description: In 2004, our fellowship training consortium began providing fellows, faculty, and other ICU team learners with weekly didactic conferences at our three main teaching sites utilizing a legacy video teleconference (VTC) platform. The VTC platform required dedicated hardware systems, which significantly limited the viewing and broadcast locations. Recently, we have converted to a web-based, software-only product called GoToWebinar®. This new technology allows conference viewing on any web-enabled device, and permits broadcast from any computer (figure 1).

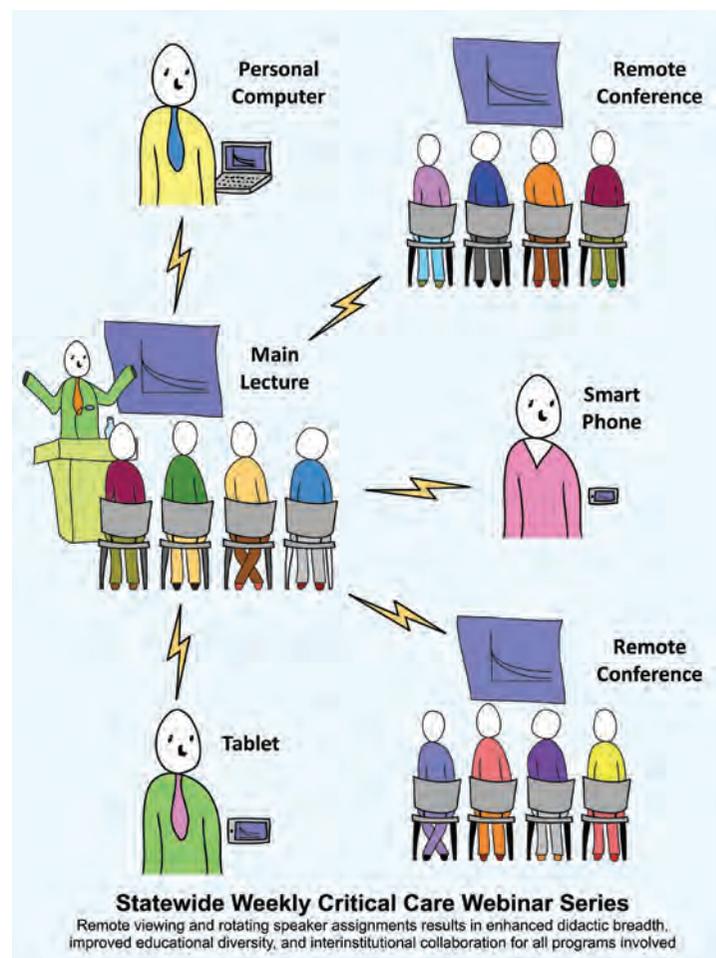
Outcomes: The use of GoToWebinar® has provided ease of access and improved video quality for our conference series, allowing faculty and trainees to view high-fidelity conferences from virtually any location. Further, other surgical and anesthesiology critical care fellowship programs within the state have now been able to join our conference series as viewers and presenters. In fact, six ACGME approved surgical and anesthesia critical care fellowship-training programs now participate in the webinar series. This has resulted in an unprecedented degree of collaboration and has broadened educational diversity. The webinar series also provides a new means of faculty development given its statewide footprint. Unanticipated challenges have been mainly technical in nature. Legacy webcast conference rooms are not configured for webinar viewing, and are ill equipped to optimally manage webinar audio and camera needs. Fortunately, relatively low-cost solutions are available

Future Plans: Web-based e-conferencing is an effective method of delivering medical education and maintaining a consistent didactic program within graduate medical education programs (1-3). The format lends itself to statewide multi-program and multi-institutional collaboration. We thus continue to seek other critical care programs, and individual clinicians, to join our webinar series, hoping to further

expand within the state and time zone. Future plans also include session archiving with on-demand viewing, CME offerings, and an objective assessment of the webinar's impact on fellow learning.

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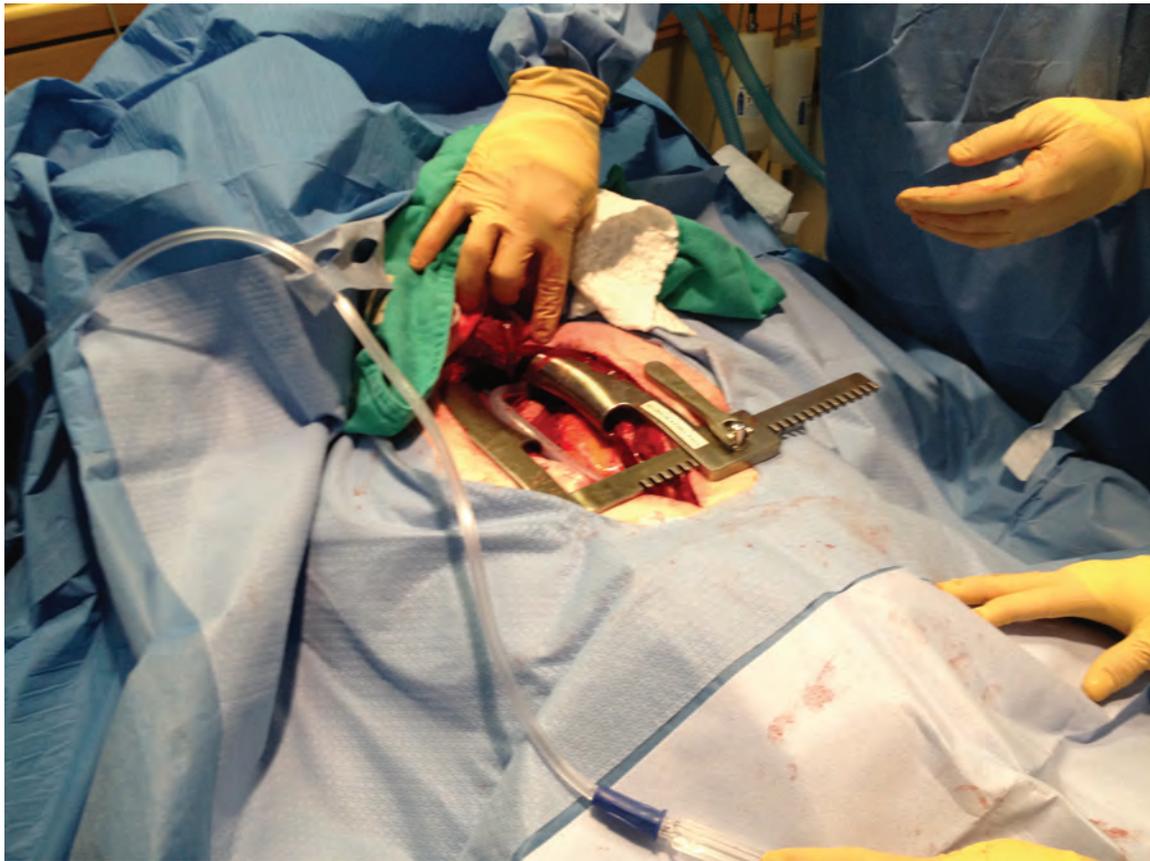
Mediastinal Compartment Syndrome After Type A Aortic Dissection Repair

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University Hospitals Case Medical Center

Compartment syndrome is defined as the dysfunction of organs/ tissue within the compartment due to limited blood supply caused by increased pressure within the compartment(1). Mediastinal compartment syndrome is rare, even in the busiest cardiac units, with less than 250 references in the literature since 1975 (1,2,3). We describe a 72 y.o. male that 18 hours post-op from a type A aortic dissection, had sudden increasing requirements for vasopressor and inotropic support, with decreasing Cardiac Index, ischemic rhythm, and dropping SvO₂. An emergent bedside opening of his mid-sternotomy and opening of the chest alleviated all symptoms within 2-3 minutes, in the absence of any clots or mediastinal blood. We will discuss the pathophysiology behind this infrequently seen complication and indication for the emergent procedure.

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Postpartum Headaches: A Multi-Disciplinary Concern

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Case Report: A 36-year-old female presented to the hospital with complaints of the "worst headache of her life". She was 7 days postpartum from a normal spontaneous vaginal delivery at an outside hospital, which records would later show, had an inadequately followed gestation due to poor patient compliance. The delivery was via an uneventful, Cervidil induction for oligohydramnios at 37 weeks. Pain control during that delivery was provided by an epidural catheter with no mention of any complications with regards to its placement or function. Her BP during the delivery ranged from 133-151/64-77.

On presentation to our institution, her headache was described as 10/10, fronto-temporal and unchanged with movement or position. It was associated with mild photophobia and phonophobia. She was afebrile, BP was 148/70, HR 55, 100% on RA. Her physical exam remained largely unremarkable except for 1+ pitting edema in her ankles b/l. Her liver function test and platelet levels were within normal limits, and her urine protein was normal after a 24 hour collection. She was admitted to the surgical ICU after a non-contrast CT of her head demonstrated the presence of bilateral subarachnoid hemorrhage near the convexities. An angiogram was negative for any aneurysm. Further imaging eventually ruled out venous thrombosis. Despite the absence of radiographic support, a full rheumatologic workup for vasculitis was done and was negative as well.

Discussion: In the setting of postpartum headaches, anesthesiologists are often consulted to address possible complications from oft-employed peripartum regional anesthetic techniques. However, it is important to realize that in the postpartum period, the differential diagnosis for headaches is very broad. Headaches in the postpartum period are

divided into 2 categories, primary and secondary. Primary headaches include migraine, tension, cervicogenic and cluster. Secondary headaches include postdural puncture headaches, intracranial pathology, and obstetric disease (e.g., preeclampsia) [1].

In a large prospective cohort study, primary headaches accounted for 75% of all postpartum headaches, whereas postdural puncture headaches accounted for only 4.7% of all postpartum headaches [2]. A history of migraines and preeclampsia have been found to be risk factors for the development of a postpartum headache [3]. In the case of postdural headaches, 90% present within 3 days of the procedure [4], and postural changes in the severity of the pain is essential to make the diagnosis [5].

The relative risk for developing a stroke in the peripartum period is 13 times that of the non-pregnant population, with the majority occurring in the postpartum period [6]. While many postpartum strokes occur with no identifiable cause, the majority occur secondary to hypertension, arteriovenous malformations, venous thrombosis or aneurysmal rupture [7].

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Efficient Vascular Access in a Hypovolemic Trauma Patient

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Introduction: The ability of the anesthesiologist to obtain vascular access in a trauma patient with hypovolemia is crucial—particularly when arterial blood gases are required for continuous monitoring of hemodynamic stability. This case serves as a discussion point for the implementation of various techniques for vascular access with respect to determining the most efficient method for fluid resuscitation and vasopressor administration.

Case Report: A 25-year-old man status-post multiple GSWs to the abdomen, scrotum and lower extremities was brought into the ED. Upon arrival, the patient was noted to be combative, agitated, diaphoretic, intoxicated and hypotensive. Bilateral dorsalis pedis pulse signals were not appreciated via Doppler ultrasound. Additionally, omental evisceration was observed, and the patient underwent an emergent exploratory celiotomy.

An arterial (A)-line was to be inserted in the radial artery prior to surgery; however, given the hypotensive state of the patient, coupled with previously failed attempts at arterial access that resulted in bilateral upper extremity hematomas, an ultrasound machine was brought into the OR to assist in artery location and cannulation.

A central line was placed via right internal jugular vein and the patient was anesthetized. Until the A-line was secured, real-time vital sign readings were continuously updated verbally, with infusion of colloids and administration of vasopressors.

Over the four-hour course of surgery, the patient received ten units of albumin, twelve units of PRBCs, six FFPs and one unit of platelets.

Technical setbacks included the patient's initial A-line becoming dislodged during the operation, necessitating the contralateral upper extremity to be cannulated, also using sonographic guidance. During his course in the SICU, Q-wave complexes were observed on the patient's EKG. He subsequently became unresponsive. After vigorous cardiopulmonary resuscitation and maximum vasopressor support, the patient expired.

Conclusion: There have been several techniques proposed and utilized by anesthesiologists in order to expedite the access of an artery or vein when time is of the essence (1). The presence of bilateral upper extremity hematomas in this trauma patient served to be a difficult obstacle to overcome, even with ultrasound-guided A-line placement. For an anesthesiologist, a venous cut-down may not have been possible in this case and may be considered outdated by some. Perhaps, in this patient, intraosseous infusion would have been another option for accessing the systemic venous system (2,3), since this technique is used in emergency situations when intravenous access is not an option. A comparison of intravenous, intramuscular, and intraosseous routes concluded that the intraosseous route is superior to intramuscular and comparable to intravenous administration (4).

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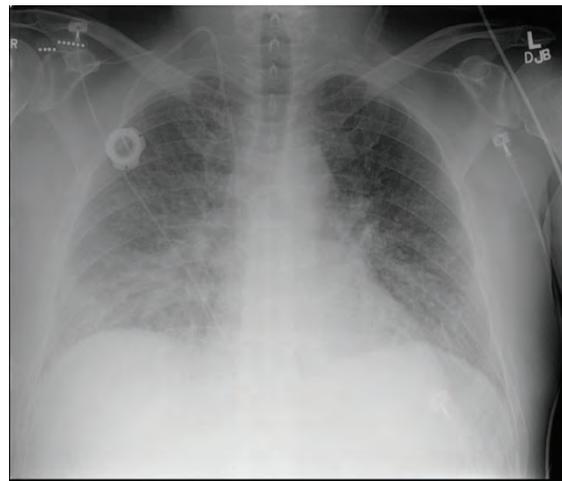
Peri-Engraftment Respiratory Distress Syndrome: A Rare Cause of Acute Respiratory Failure

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Mayo Clinic

Background: Following conditioning chemotherapy and autologous stem cell transplantation (ASCT), there is a period of time during which hematopoietic stem cells begin to form neutrophils, as evidenced by their appearance in the peripheral blood. This period of myeloid restitution is termed neutrophil engraftment, defined as an absolute neutrophil count greater than $0.5 \times 10^9/L$ on three or more consecutive days post ASCT. It is postulated that during this phase, a cytokine storm may drive a constellation of symptoms including non-infectious fever, skin rash, diarrhea, and increased capillary permeability (1). This is termed engraftment syndrome (ES). ES is associated with an increased risk of respiratory failure, with or without multi organ failure. If lung injury develops within 4 to 25 days post transplantation (median 11 days), in the absence of infectious and cardiac causes, the diagnosis of peri-engraftment respiratory distress syndrome (PERDS) is suggested (2). We report a case of PERDS that responded favorably to steroids.

Case: A 59-year-old man presented with neutropenic fever, fatigue, rash, and marked diarrhea 9 days after ASCT for multiple myeloma. At admission, he was profoundly neutropenic with a white cell count of $0.1 \times 10^9/L$. An infectious work up was instituted and broad spectrum antimicrobials with intravenous fluid repletion were commenced. Blood, sputum, and urine cultures, and chest films were negative. Over the next 4 days, even as his white cell count increased to $1.3 \times 10^9/L$, his clinical status deteriorated. He developed worsening dyspnea, hypoxia and oliguria, necessitating transfer to the intensive care unit (ICU). A portable chest film revealed bilateral interstitial infiltrates (figure 1). After careful consideration of other differential diagnoses, an empiric diagnosis of PERDS was made, and intravenous therapy with 1 gram of methylprednisolone daily was begun. Rapid clinical and radiographic improvement occurred over the next 2 days and he was discharged on an oral steroid taper.

Discussion: PERDS is an uncommon reason for admission to the ICU. Due to lack of uniform diagnostic criteria, the reported incidence in various series ranges from 7 to 59% (3). There are no specific blood tests and chest radiographs typically show bilateral interstitial infiltrates and pleural effusions. The similarity of clinical presentation and absence of diagnostic tests, can pose a challenge in distinguishing PERDS from the more frequently encountered sequelae in this patient population. It is imperative that infectious and non-infectious causes such as TRALI, TACO, cardiogenic pulmonary edema, diffuse alveolar hemorrhage and idiopathic pneumonia syndrome be excluded. Our case illustrates that PERDS can be easily overlooked unless astutely entertained in a patient with fever, rash, hypoxia, and capillary leak around the time of neutrophil engraftment. Timely steroid therapy can avoid the need for mechanical ventilation, multiple tests or prolonged ICU stay, and facilitate a dramatically favorable outcome, compared to other morbid complications of bone marrow transplantation.



A Neurophysiological Approach to Electroencephalogram Monitoring During General Anesthesia and Sedation

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Massachusetts General Hospital

General anesthetic and sedative drugs induce stereotyped changes in the electroencephalogram (EEG) that were first observed in the 1930's, providing the basis for present day anesthetic brain monitors. The most popular approach to EEG-based anesthesia monitoring has been to use empirically-derived indices that reduce the EEG to a single number between 0 and 100. Although these indices are constructed from the EEG, they do not relate directly to the neurophysiology underlying each anesthetic drug's effects on the brain and the EEG, and thus cannot provide a completely accurate picture of a patient's brain state or level of consciousness. Moreover, anesthetic and sedative drugs are known to act through different mechanisms, producing different states of altered arousal, and different EEG patterns or signatures, which are poorly characterized by a single number. These fundamental mechanistic differences are evident when these indices are used with ketamine or nitrous oxide, which can elevate index values, or with dexmedetomidine, which can produce index values mimicking general anesthesia in patients who are merely sedated and can be easily aroused.

These fundamental mechanistic differences suggest a powerful alternative approach: anesthesiologists could be trained to recognize the EEG signatures for different anesthetic drugs, allowing them to monitor the specific dose-dependent effects associated with each drug. To realize this approach, we developed a method to visualize the EEG signatures for different anesthetic/sedative drugs using the spectrogram, which characterizes the frequency content of signals over time. We recorded EEG during anesthetic brain monitoring in >700 patients receiving different anesthetics, and performed spectral analysis to

construct signatures for each anesthetic drug (Figure 1A). It is difficult to infer the anesthetic drug or dose from viewing traditional unprocessed EEG waveforms (Figure 1, left). However, using spectrograms (Figure 1, center), different drug- and dose-dependent effects are easily and precisely visualized. These spectral signatures can be viewed on existing EEG devices, and their structure can be understood in terms of each drug's underlying mechanisms. For instance, during propofol-induced unconsciousness, the predominant rhythms consist of frontal 8-12 Hz alpha oscillations, which likely reflect pathological thalamocortical oscillations [1], and < 1 Hz slow oscillations, indicative of a state of cortical fragmentation [2] (Figure 1, right). These oscillations imply a brain state incompatible with consciousness. Similar mechanistic associations can be made for ketamine, dexmedetomidine, inhaled anesthetics, and drug combinations.

These EEG signatures provide a parsimonious, physiologically-principled, and more informative alternative to index-based monitoring. The EEG signatures are relatively easy to learn, and are easier to read than traditional EEG waveforms. This paradigm is consistent with the pharmacological framework of anesthesiology, and fits seamlessly within existing models of clinical training and practice. We propose that this novel approach to monitoring could significantly enhance clinical management of anesthesia and sedation.

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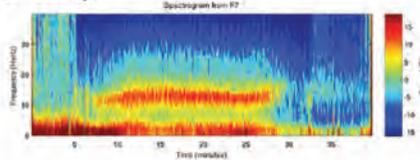
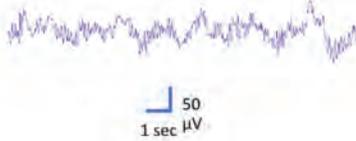
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Traditional Approach

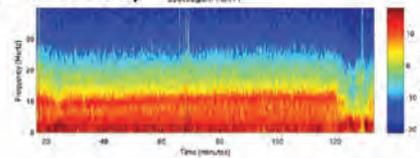
New "EEG Signatures" Approach

Links to Underlying Mechanisms

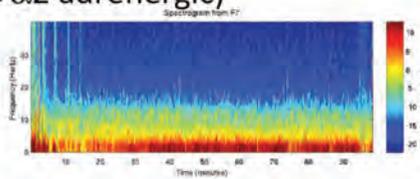
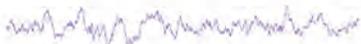
Propofol (+GABA)



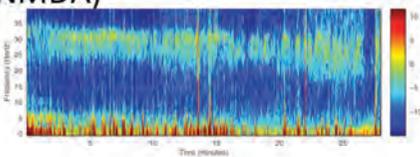
Sevoflurane (+/- various)



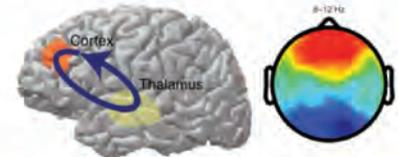
Dexmedetomidine (+ α 2 adrenergic)



Ketamine (-NMDA)

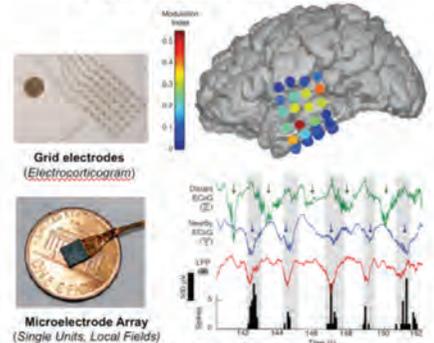


Alpha: Pathological Oscillation



Purdon et al. PNAS 2013; Cimenser et al. PNAS, 2011; Ching et al. PNAS, 2010

Slow: Cortical "Fragmentation"



Lewis et al. PNAS 2012

Figure 1. The EEG Signatures of General Anesthesia. Traditional EEG traces (left) are difficult to interpret, but the EEG frequency structure, displayed in the spectrogram (center), is much easier to read, and reveals specific frequency components tied to underlying neurophysiological (right) and molecular pharmacological mechanisms. The spectrogram (center) shows frequency on the y-axis, time on the x-axis (minutes), and signal energy in color. This approach provides a powerful new way of monitoring brain function during general anesthesia and sedation.

Negative Pressure Pulmonary Edema with Superimposed Fat Embolism: The Value of the Differential Diagnosis

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Syndrome: The Value of the Differential Diagnosis

Introduction: The cause of perioperative pulmonary edema can often be quickly elucidated by the patient's history and physical exam. A broader differential diagnosis must be considered based upon the patient's presentation, co-morbidities, and surgery.

Case: A 64-year-old male with a history of CABG and Obstructive sleep apnea (OSA) was admitted for an elective hip replacement. His surgery was uneventful with 600mL of blood loss and 300mL of urine. Fluid losses were replaced with 1L of albumin and 1.7L of ringers lactate. While in the recovery room, the patient appeared to be choking and was unable to breathe. He was re-intubated. A CXR revealed diffuse bilateral pulmonary opacities interpreted as pulmonary edema. He was given a diuretic, which produced 1.3L of urine. After a period of observation, he was transported to the ICU.

The patient's vital signs deteriorated during transport to the ICU. Upon arrival, his BP was 76/53 on 30 micrograms of phenylephrine and an ABG revealed a paO₂ of 50 on 100% FiO₂. Critical Care was consulted.

Physical exam revealed a morbidly obese male being aggressively bag-mask ventilated. He had an intermittent cough with pink frothy sputum. His trachea was midline and there was no JVD. He had a scar from a prior sternotomy, tachycardia, and bilateral diffuse rales. His skin was warm and dry in all four extremities without petechiae. He was sedated yet moved all four extremities.

Ventilatory support and vasopressors were optimized. A repeat CXR revealed persistent bilateral pulmonary edema suggestive of fluid overload. Empiric diuresis was attempted with little urine output. An emergent echocardiogram was ordered which showed an EF of 60% with a decreased left ventricular cavity size suggesting low intravascular volume. The patient was aggressively fluid resuscitated which improved urine output. He was empirically treated with heparin for a possible pulmonary embolism. The patient met the diagnostic criteria for ARDS and he quickly progressed to multi-organ failure.

Discussion: Although most commonly pulmonary edema in a patient with ischemic heart disease is usually volume overload, this patient's presentation was most consistent with Negative Pressure Pulmonary Edema (NPPE) with a superimposed Fat Embolism Syndrome. Other diagnoses considered included aspiration, pulmonary embolus, pneumothorax, myocardial ischemia, and Bone Cement implantation syndrome.

NPPE is a transudative edema produced by the exaggerated negative intrathoracic pressure generated by an inspiratory effort against a closed glottis. It is an uncommon post surgical complication of OSA. Death, ARDS, and multi-system organ failure secondary to NPPE have been reported. Early recognition of this syndrome is crucial to decrease morbidity in these patients. In addition, our patient met the Schonfeld Fat Embolism Syndrome Index criteria for Fat Embolism Syndrome. It is a well-known but uncommon cause of ARDS and multi-organ failure after hip surgery. Understanding the pathophysiology and simply considering these relatively uncommon perioperative diagnoses are crucial to their identification in Anesthesia Critical Care.

Making the Best of Tragedy: Brain Death in a Pregnant Mother Initially Presenting with a Nonviable Fetus

David W. Miller, M.D.; Kenneth G. Smithson, M.D., Ph.D.; Andrew B. Barker, M.D.; Joseph R. Biggio, M.D.; Barton L. Guthrie, M.D.
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Brain death is a heart-wrenching outcome in a pregnant woman. Recently, there has been increased attention to this subject.[1] The family and clinical services are presented with a terrible decision, especially when the mother presents with a nonviable fetus. Ultimately, the choice is between continuing hemodynamic and ventilatory support of the mother's body in an attempt to deliver a healthy infant versus stopping these resuscitative efforts, knowing the fetus will also succumb. There are multiple case reports in the literature of successfully supporting a mother's body after brain death but it does not always end with a viable delivery.[2-4] We present the case of a previously healthy 23-year-old female, pregnant for the first time, who presented to a local hospital at the end of her 22nd week of pregnancy with nausea, vomiting, and loss of consciousness. She required intubation and was diagnosed with a diffuse intraventricular hemorrhage by noncontrast computed tomography of the head. She was later found to have a right frontal arteriovenous malformation of the brain. She was urgently transported to our institution, but despite maximal surgical and medical interventions she progressed to brain death. The fetus had reached the age of viability a couple days prior to declaration of brain death. After discussion with the family, all involved clinical services, and the hospital Ethics service, the decision was made to proceed with support of the mother's body which was successful for the following 32 days. Due to maternal hemodynamic instability and nonreassuring fetal heart tones following a central line insertion, a low transverse Cesarean section was

performed with delivery of a viable female infant weighing 2 pounds, 4 ounces at 28 weeks of gestation. The infant was discharged home at 3 months of age with no significant complications or problems noted. This case is an example of how with multi-system support of the mother's body a fetus may reach an age of increased survival. However, this effort requires the support of the patient's family, which in our case was strengthened by favorable ultrasound imaging of the fetus.

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Successful Use of Inhaled Nitric Oxide in Management of Critical Pulmonary Hypertension as Observed in Real Time with a Disposable ImaCor hTEE™ Probe

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Introduction: Pulmonary hypertension (pHTN) has been observed in patients with hypertrophic obstructive cardiomyopathy, (HOCM) due to transmitted high left heart pressures. However, the pHTN seen in this setting is generally mild to moderate, with improvement observed after surgical correction of the left ventricular outflow tract (LVOT) obstruction.

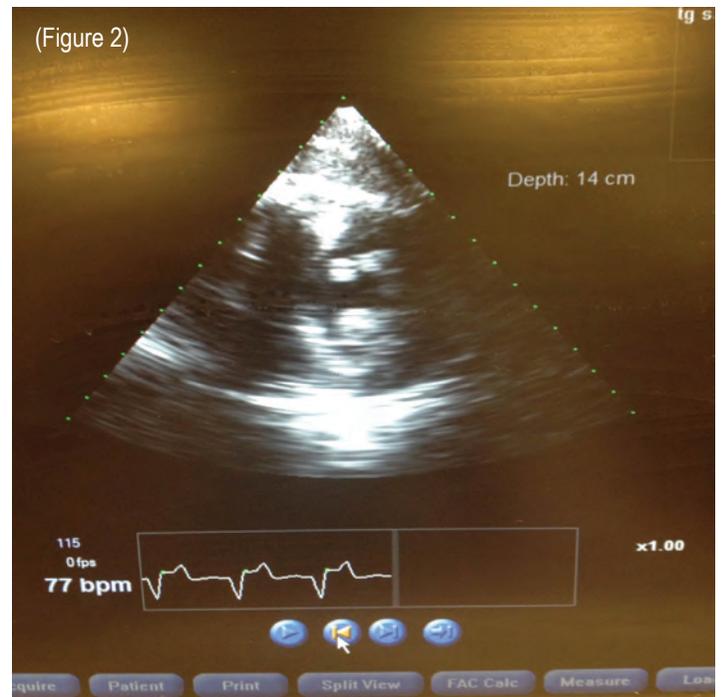
Case: A 34 year old woman with longstanding HOCM presented for septal myomectomy in the setting of severe and progressive dyspnea. She had experienced episodes of syncope during childhood, but these episodes had resolved with the addition of metoprolol eight years prior to this admission. 6 months prior to surgery, her LVOT gradient was found to be 103mmHg with severe mitral regurgitation (MR) and systolic anterior motion (SAM) of the mitral valve. On admission, her LVOT gradient was found to be 75, with asymmetric septal hypertrophy, SAM physiology, and moderate MR. On right heart catheterization she had near systemic pulmonary artery (PA) pressures (100/37).

After undergoing an uncomplicated septal myomectomy, she was admitted to the CTICU. Her post-operative TEE described resolution of the LVOT gradient (<10mmHg), resolution of SAM and mild residual MR. PA pressures on admission to the unit were 60% systemic. On the

night of post-operative day zero, her PA pressures increased gradually over the course of 10-15 minutes. She was unresponsive to a straight leg raise, and upon repositioning her flat, her systemic blood pressures dropped below her PA pressures. She became pulseless and chest compressions and 1mg of epinephrine were given.

After several minutes she regained her blood pressure and an ImaCor hTEE™ probe was emergently placed. On 4 chamber and short axis views, it was clear that she had no tamponade physiology, her right heart was full with bulging of the intra-atrial septum into the left atrium (Figure 1), and the LV was thick and underfilled. Inhaled nitric oxide was started in the middle of the exam with real time improvement in LV filling observed on hTEE coinciding with a drop in PA pressures on PA catheter tracing relative to systemic blood pressures on the arterial line tracing. (Figure 2).

Discussion: It is uncommon to encounter severe reactive pulmonary hypertension in the setting of surgically treated HOCM. Here we demonstrate the utility of inhaled nitric oxide and real-time hTEE in the management of highly dynamic cardiopulmonary physiology in the ICU setting.



Gas Gangrene: Delayed Diagnosis Results in Mortality. A Case Report.

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Gas gangrene is a relatively rare occurrence in the developed world, and is an infection with high mortality and morbidity requiring prompt treatment. We describe a longitudinal case of delayed diagnosis resulting in patient mortality.

Introduction: Gas gangrene is a relatively rare occurrence in the developed world, and is an infection with high mortality and morbidity requiring prompt treatment. Initial bacterial injury to host tissue impairs blood supply generating the conditions necessary for anaerobic clostridial species proliferation. The vast majority of clostridial wound infections are polymicrobial with the primary pathogen any one of the clostridial species including *C perfringens* (80%), *C novyi* (40%), *C septicum* (20%)¹. A variety of other bacterial isolates may be evident with organisms such as *Proteus*, *Bacillus*, *Escherichia*, *Bacteroides*, and *Staphylococcus* present in the tissues. Severity of infection is correlated to the density of clostridial load in the tissues with resultant exotoxin release (hemolysins, collagenases, proteases, and lipases). Endotoxemic damage results in liver necrosis, hemolytic anemia and renal failure. Signs and symptoms of systemic toxicity develop rapidly with most patients in multi-organ failure on presentation. We describe a case of delayed recognition of gas gangrene that rapidly progressed to multi-organ failure and mortality despite aggressive surgical intervention and maximal medical support.

Case Presentation*: A 56 year old male with a past medical history significant for alcoholic and hepatitis C associated cirrhosis, poorly controlled diabetes mellitus and peripheral neuropathy presented to an outside hospital with evidence of septic shock. On presentation he reported 4 days of severe left foot pain and swelling. Constitutional symptoms of fever and chills were also endorsed on review of systems. Physical examination was significant for an edematous forefoot with exquisite tenderness to palpation. A lateral radiograph of the foot demonstrated soft tissue swelling without evidence of gas formation (Figure 1). A chest X ray demonstrated a left lower lobe infiltrate, an X ray of the foot demonstrated chronic changes, and laboratory investigations revealed a leukocytosis (wbc $11.4 \times 10^3/\text{mcl}$), thrombocytopenia (plt 66,000/mcl), metabolic acidosis (lactate 5.8 mmol/L), moderate hypoxia (89% on room air) and acute renal failure (BUN 70, Cr 3.8 mg/dL). He was treated with azithromycin, ceftriaxone, and acetaminophen in the emergency department and admitted to the ward. His condition deteriorated over the course of several hours, with development of tachycardia (heart rate >100 bpm), hypotension (systolic blood pressure 80-90 mmHg) and deteriorating renal function. At this point, he was transferred to our tertiary care facility for multi-organ failure in the setting of presumed pneumonia.

Upon arrival to our facility, examination revealed a toxic appearing white male in respiratory distress (ABG pH 7.1/ pCo₂ 30mmHg/ pao₂ 172mmHg/ bicarbonate 17.3 mmol/L), altered mental status, and hypoxemia requiring the administration of 100% oxygen on noninvasive positive pressure ventilation. He was emergently intubated for airway protection. Examination demonstrated coarse breath sounds on

auscultation, normal heart sounds, a distended abdomen with signs and symptoms of ascites, and a dusky appearing foot with ecchymosis, and evidence of a dark fluid filled blister on the forefoot. Palpation elicited clear evidence of crepitus surrounding the blister (figure 2). He was persistently hypotensive with systolic blood pressures 75-90 mmHg requiring norepinephrine and aggressive fluid administration of three liters of normal saline. We administered empiric antibiotic therapy of piperacillin/tazobactam, clindamycin and vancomycin. Laboratory investigations demonstrated severe lactic acidosis (lactate 15.4mmol/L), anuric renal failure (BUN 62mg/dL, creatinine 3.22mg/dL), leukocytosis ($40.8 \times 10^3/\text{mcl}$) and anemia (9.3 to 10.2gm/dL). A chest X-ray demonstrated bilateral infiltrates. The presumptive diagnosis was gas gangrene with sepsis and ARDS. After confirmation of diagnosis with computed tomography (Figure 3), the patient proceeded urgently to the operating room for guillotine amputation of the left lower leg. Later that evening, despite source control, empiric antimicrobial therapy and continuous renal replacement therapy, the patient expired secondary to persistent lactic acidosis, and severe hypotension refractory to maximal medical therapy. Aerobic blood cultures demonstrated the presence of gram positive cocci that were identified as methicillin sensitive *Staphylococcus aureus*. Anaerobic cultures did not demonstrate microbial growth. Wound cultures grew abundant Group B streptococcus and methicillin sensitive *Staphylococcus aureus*.

*Dartmouth College Institutional Review Board approved for presentation.

Conclusion: The mainstay of treatment of gas gangrene remains early and aggressive surgical treatment with source control². Despite swift treatment, mortality remains high with many patients dying within 24 hours of hospital admission secondary to delayed diagnosis or challenging surgical debridement³. In this case, early radiography at the outside hospital did not demonstrate gas formation which led to delayed diagnosis, incorrect antimicrobial selection, delayed surgical intervention and mortality. This case serves to alert clinicians to remain vigilant for the clinical signs and symptoms of this rapidly progressing infection given its relative rarity in the developed world.

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Percutaneous Tracheotomy: A Modified Technique to Facilitate Placement and Improve Patient Safety

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Introduction: When a decision is made to perform tracheotomy for a critically ill patient in order to maintain long term ventilation, bedside percutaneous dilatational tracheotomy (PDT) has become the procedure of choice for most patients. As originally described by Ciaglia (1), the tracheotomy tube was introduced using a Seldinger, over the wire, technique following blind puncture of the trachea using anatomic landmarks. One challenge is to maintain mechanical ventilation after re-positioning of the endotracheal tube so it is out of the way for tracheal tube entry. We report a modified technique combining the video laryngoscope (VDL) and the flexible fiberoptic bronchoscope (FFB) to avoid "losing" the airway and to avoid complications of blind tracheal puncture.

Methods: After obtaining appropriate informed consent the patient was prepped and draped, a time out was performed, and infusions for analgesia/sedation/paralysis, were titrated to induce general anesthesia. The VDL was introduced to display the tube entering the larynx, and the tube was withdrawn to view the vocal cords and the proximal ring of the tube cuff. Adequacy of ventilation is confirmed. Using anatomic landmarks, a 2 cm vertical incision is made over the trachea; finger dissection identifies the tracheal rings. The trachea is punctured, air is aspirated, the Seldinger wire is passed, and the FFB is introduced through the endotracheal tube to view the wire in the trachea in desired position. Dilatation of the puncture site and introduction of the tracheal tube follows. The FFB is employed a second time to examine the trachea down to the carina, confirm position, and examine for blood,

trauma, etc. Adequacy of ventilation is again confirmed through the new airway access, and the endotracheal tube is removed.

Results: After IRB approval, we reviewed 79 patients in which this described procedure was performed. In no instance was there accidental extubation or loss of airway function; there were no instances of tracheal damage or a misplaced/dysfunctional tracheotomy. One patient had a cervical hematoma with no sequelae; one patient had a small right apical hemothorax which resolved. In no instance was the FFB damaged during the procedure.

Discussion: This modified technique to accomplish percutaneous tracheotomy, using technology widely available and part of the skill set of anesthesiologist/intensivists is safe, effective, easily accomplished, facilitates PDT, and improves patient safety vs. standard techniques. (2) The entry site is usually the 3rd tracheal ring, which is approximately 8.0 cm from the vocal cords with the neck slightly extended and padding under the shoulder. From the top of the balloon on an adult ET tube to the tip is 6.0 cm. Thus mechanical ventilation can be assured and uninterrupted with an inflated cuff with this method of repositioning, the field is clear for tracheotomy tube entry, and there is no damage to the FFB since it is not used for re-positioning the endotracheal tube.

References:

1. Chest (1985)87:715-9.
2. Critical Care (2006)10:202-11.

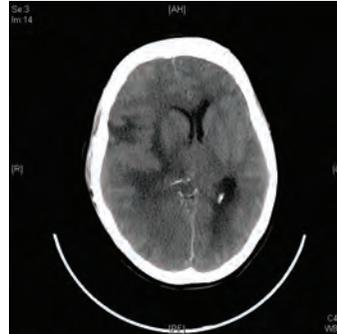
Malignant Cerebral Edema Secondary to Gliadel Wafers in the Early Postsurgical Period

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High grade gliomas are the commonest intrinsic brain tumours and account for more average years of life lost than all the common cancers. It has become the commonest cause of cancer death in men under the age of 45 and women under the age of 35. Although surgical resection can greatly reduce tumour bulk, complete excision is virtually impossible due to the infiltrative nature of these tumours. In an attempt to treat the infiltrating tumour cells, there has been much interest in using local therapies inserted at the time of surgery. The authors report a case of fatal cerebral edema unresponsive to aggressive medical and surgical assessment that finally evolved to premature death in the early postsurgical period, after the craniotomy and implantation of Gliadel wafers (Fig 1). They note that high doses of dexamethasone were insufficient to prevent cerebral edema and death. A search for corticosteroid use and dosing for patients treated with Gliadel wafers in the published literature revealed no recommendations on the doses of steroids to be administered. In our opinion this is a very important issue and maybe the key point for the treatment of this disease, and may need

to be addressed with treatment guidelines in the near future in order to ensure better results on patient's survival.

Prior to this case review there had been two similar report but a later presentation. So we think that this is the first case report of acute fulminant cerebral edema secondary to gliadel wafers in the early period.



The Utility of a Fellowship Lecture Development Series: A Formal Process to Teach Fellows How to Create and Deliver Effective Presentations

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Educational Objectives: Every physician is an educator (1). Effective information exchange is a physician core competency because doctors must be able to teach and communicate clearly with patients and families, learners, their peers, and other health professionals. In an effort to improve our fellow's communication skills, public speaking ability, and teaching skills we have historically tasked them to provide formal presentations within our critical care webinar series. After recognizing that public speaking and effective lecture delivery was not an instinctual behavior, we established a training process to help our fellows develop and deliver better presentations in order to become more effective clinician-educators.

Program Description: During the 2012-2013 academic year we developed a lecture development series based upon feedback from our fellowship annual review regarding fellow lecture quality, and from our fellow's perception of their readiness to provide formal presentations to a large and diverse audience. The program consists of three sessions where the trainees provide talks to a peer group of fellows and a few select faculty in a non-threatening setting. Constructive feedback is provided regarding time management, slide quality and content, speaking techniques, and the effectiveness of message delivery. The first session allots five slides in five minutes to cover a specific topic. For the second session, each fellow prepares 10 slides as part of a joint presentation focusing upon one topic. In the last session, a pro-con

format is utilized, allowing the fellows 10 minutes each to present 1-on-1 opposing points of view. The process and the usual outcomes are illustrated in figure 1.

Results: Using a small group, feedback driven, non-threatening, failure-to-success approach we have noted a distinct improvement in the quality of the fellow's formal lectures, as well as enhanced communication and presentation skills through each step of the lecture development series (2). The fellows have become more succinct and direct, their slides show greater clarity, and they exhibit greater confidence at the lectern.

Future Plans: Given the high yield of this simple program, we plan to maintain it as a permanent didactic element of our fellowship training consortium. Since it is a novel process for fellowship training, we would like to develop a means to objectively assess its effectiveness. We also plan to provide literature and material pertaining to lecture skill enhancement.

References:

1. Branch WT, Kroenke K, Levinson W. The clinician-educator - present and future roles. *J Gen Intern Med.* 1997;12 S2:1-4.
2. Pinsky LE, Irby DM. âœlf at first you don't succeed: Using failure to improve teaching. *Acad Med.* 1997;72:973-76.



Invited Speaker

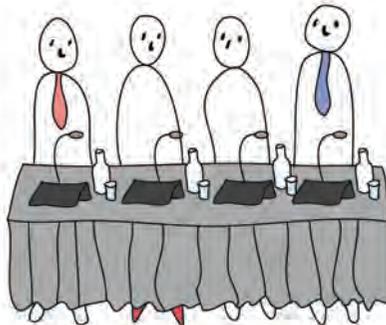
- Individual lectures
- 5 slides - 5 minutes

Session Focus

- Speaking skills
- Slide clarity
- Time management
- Succinctness

Usual Outcomes

- Cover only 3 of 5 slides
- Overly complex slides
- Imagery not complimentary
- Point not sold
- Need to improve self evident



Expert Panel

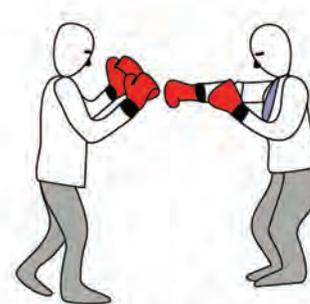
- Team topic
- 10 slides - 10 minutes

Session Focus

- Same as prior
- Team responsibility

Usual Outcomes

- Improved speaking skills
- Enhanced topic clarity
- Improved time management
- Work together as team
- Slides still very "busy"
- Recognize that additional growth is required



Pro-Con Debates

- 1-on-1 pro-cons
- 10 slides - 10 minutes

Session Focus

- Same as prior
- Brevity
- Competition as a driver for success

Usual Outcomes

- Speaking skills greatly improved
- Slides complimentary to goals
- Discussions are succinct and high yield
- Enhanced fellow confidence at the podium

Fellowship Consortium Lecture Skills Development Series

A Formal Process to Teach Fellows How to Create and Deliver Effective Presentations

Goals and Pitfalls Involved in Selection of Agents for Hypertensive Augmentation for Vasospasm Treatment After Aneurysmal Subarachnoid Hemorrhage (aSAH) and Middle Cerebral Artery (MCA) Clipping with Superior Temporal (STA) to MCA Bypass

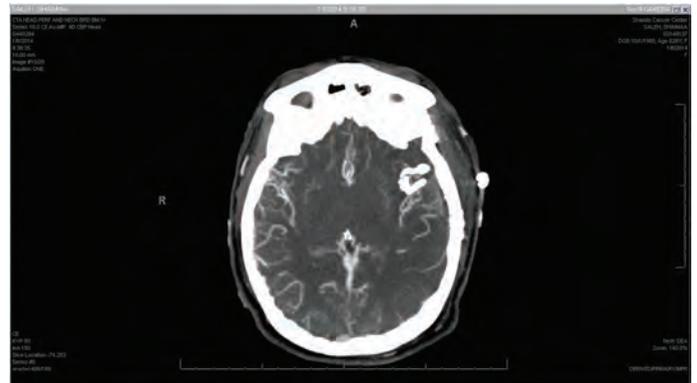
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University of Florida

Introduction: Treating the patient with vasospasm and delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage (aSAH) can be extremely challenging. Induced hypertension is utilized in an attempt to restore flow to ischemic brain. Agents of choice in this setting typically include phenylephrine and/or vasopressin. Vasopressor choice becomes more complicated when managing vasospasm in a patient with an extracranial by-pass. Dobutamine has the desired properties of increasing flow through beta one agonism. But also causes peripheral vasodilatation through beta 2 agonism, making it difficult to obtain adequate increase in systemic blood pressure. The use of any agent causing vasoconstriction may jeopardize the patency of the extracranial bypass.

Case Presentation: A 28 year old woman presented with aSAH and was found to have a left middle cerebral fusiform aneurysm that was unable to be coiled during angiography. The patient was put on nimodipine and a statin for vasospasm prophylaxis in anticipation for open clipping. Two days later the patient was brought to the operating room for definitive care. She underwent a left frontoparietal craniotomy for clipping which was technically difficult, requiring clip aneurysm trapping and a superficial temporal artery (STA) to middle cerebral artery (MCA) bypass. On postoperative day one, the patient developed mental status changes concerning for vasospasm and dci, confirmed with angiography. Hypertensive therapy was initiated with a dobutamine infusion and her neurologic status improved. Our goal was to provide blood pressure augmentation, but to avoid any alpha agonism that may cause vasoconstriction in the STA graft. The patient experienced occasional episodes of tachycardia to the 130's, and we were able to reach goal blood pressure. She also received intra-arterial verapamil. On postoperative day two she was no longer achieving the blood pressure goals and had a decline in her neurological status. After extensive discussion with the neurosurgeon it was agreed that a vasopressin

infusion would be an acceptable second agent. Although vasopressin causes vasoconstriction through action on the V1a receptor, almost all of this vasoconstriction occurs in the splanchnic and peripheral vascular beds. In addition, it wouldn't exacerbate the tachycardia issues already present from the dobutamine. Because of these properties, it was the least likely agent to damage the integrity of the STA to MCA bypass. The patient was treated with vasopressors and underwent intra-arterial verapamil injection almost every day over the next week. Her symptoms of vasospasm subsided after a week. At post-op day 11 the vasopressors were weaned off and she showed no signs of damage to the bypass.

Discussion: This case provided a challenge in balancing the need to induce hypertension and trying to avoid causing any stress to her STA MCA bypass from vasoconstriction. A combination of dobutamine and vasopressin was used successfully to induce hypertension in this patient. This along with multiple sessions of intra-arterial verapamil was used to improve cerebral perfusion during her period of severe vasospasm until it finally resolved.



Future Meetings



SOCCA 28th Annual Meeting and Critical Care Update

March 20, 2015
Hilton Hawaiian Village Resort
Honolulu, Hawaii



SOCCA 29th Annual Meeting and Critical Care Update

May 20, 2016
San Francisco Hilton, Union Square
San Francisco, California