1st Annual ResusCon Conference

September 13th 2018
The Grandview
176 Rinaldi Boulevard
Poughkeepsie, NY 12601
7:30am – 4:30pm
<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
</tr>
</thead>
<tbody>
<tr>
<td>7:30am – 7:55am</td>
<td>REGISTRATION</td>
</tr>
<tr>
<td>7:55am – 8:00am</td>
<td>Opening Remarks</td>
</tr>
<tr>
<td></td>
<td>- Faizan Arshad, MD, EMS Medical Director, Vassar Brothers Medical Center, Health-Quest Systems</td>
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<tr>
<td>8:00am – 8:40am</td>
<td>Sepsis: Forecasting &amp; Eliminating End Organ Dysfunction</td>
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<tr>
<td></td>
<td>- Timothy Buchman, PhD, MD, FACS, FCCP, MCCM, Professor of Surgery, Emory University School of Medicine; Professor of Anesthesiology, Emory University School of Medicine; Director, Emory Critical Care Center, Emory University School of Medicine Chief, Critical Care Service, Emory Healthcare</td>
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<tr>
<td>8:40am – 9:20am</td>
<td>Fluid Resuscitation in Trauma- Do No Harm</td>
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<tr>
<td></td>
<td>- Ronald Simon, MD, Chief, Division Acute Care Surgery Maimonides Medical Center, Brooklyn, New York</td>
</tr>
<tr>
<td>9:20am – 9:50am</td>
<td>ECMO in the Setting of Cardiac Arrest</td>
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<tr>
<td></td>
<td>- A. Reshad Garan, MD, Associate Director of the Cardiac Intensive Care Unit and Medical Director of the Acute Circulatory Support Program at the Columbia University Medical Center; Assistant Professor of Medicine at Columbia University Medical Center</td>
</tr>
<tr>
<td>9:50am – 10:10am</td>
<td>BREAK</td>
</tr>
<tr>
<td>10:10am – 10:40am</td>
<td>Return of Spontaneous Circulation- Care in the CCU</td>
</tr>
<tr>
<td></td>
<td>- Simon Gorwara, MD, FACC, President of the Heart Center, Chief of Cardiology, Vassar Brothers Medical Center</td>
</tr>
<tr>
<td>10:40am – 11:10am</td>
<td>Integrating the Links in the Chain of Survival - Out of Hospital Cardiac Arrest</td>
</tr>
<tr>
<td></td>
<td>- A. Reshad Garan, MD, Associate Director of the Cardiac Intensive Care Unit and Medical Director of the Acute Circulatory Support Program at the Columbia University Medical Center; Assistant Professor of Medicine at Columbia University Medical Center</td>
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<tr>
<td></td>
<td>- Timothy Collins, DO, FCCP, Director, Pulmonary/Critical Care/Sleep Medicine Adult Director, Cystic Fibrosis Program Vassar Brothers Medical Center, Health Quest Medical Practice</td>
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<tr>
<td>11:10pm – 11:40pm</td>
<td>tPA for Massive Pulmonary Embolus</td>
</tr>
<tr>
<td></td>
<td>- Timothy Collins, DO, FCCP, Director, Pulmonary/Critical Care/Sleep Medicine Adult Director, Cystic Fibrosis Program Vassar Brothers Medical Center, Health Quest Medical Practice</td>
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<tr>
<td>11:40pm – 1:00pm</td>
<td>LUNCH</td>
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<tr>
<td>1:00pm – 2:00pm</td>
<td>Advancing Care in New York State, How Developing Protocols Can Work for Our Patients</td>
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<tr>
<td></td>
<td>- Michael Dailey, MD, FACEP, Director Pre-Hospital Care &amp; Education, Albany Medical Center; Medical Director, Regional Emergency Medical Organization</td>
</tr>
<tr>
<td>2:00pm – 2:45pm</td>
<td>Training the Limbic System for High Performance During Resuscitation</td>
</tr>
<tr>
<td></td>
<td>- Faizan Arshad, MD, EMS Medical Director, Vassar Brothers Medical Center, Health-Quest Systems</td>
</tr>
<tr>
<td>2:45pm – 3:00pm</td>
<td>BREAK</td>
</tr>
<tr>
<td>3:00pm – 3:40pm</td>
<td>Giraffes &amp; Zebras in the ICU</td>
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<tr>
<td></td>
<td>- Steven Ritter, MD, Sleep Medicine, Critical Care, Internal Medicine and Pediatrics, Medical Associates of Hudson Valley; Medical Director, Northern Dutchess Sleep Center</td>
</tr>
<tr>
<td>3:40pm – 4:30pm</td>
<td>Ultrasound Guided Resuscitation for Undifferentiated Shock</td>
</tr>
<tr>
<td></td>
<td>- Stephanie Midgley, MD, Director of Emergency Medicine Ultrasound, EOS Medical Group, Emergency Medicine Attending, Team Health, PC, Vassar Brothers Medical Center, Putnam, Northern Dutchess and Ellenville</td>
</tr>
</tbody>
</table>
1st Annual ResusCon Conference

General Information

Course Objectives
Following the completion of this conference, attendees should be able to:

1 - Consider the new roles for ultrasound in the resuscitation of the critical patient.
2 - Aggressively manage crashing patients using ECMO.
3 - Consider the risk vs. benefits for the use of thrombolytics for massive PE.
4 - Improve awareness to critical considerations and interventions post ROSC to ensure the best possible outcomes.
5 - Rational and techniques for the use of push does pressors and push dose nitro in the emergency setting.
6 - Consider the history, theory and technique of damage control resuscitation for the critically injured patient.
7 - Decipher the role stroke severity plays in identifying large vessel occlusion.
8 - Improve awareness of genetic variability associated with increased susceptibility and potential outcomes for severe sepsis.

Accreditation
Vassar Brothers Continuing Medical Education is accredited by the Medical Society of the State of New York to provide continuing medical education for physicians.

AMA Credit Designation
Vassar Brothers Continuing Medical Education designates this live education activity for a maximum of 6.50 AMA PRA Category 1 Credit(s)™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Nursing Accreditation
6.50 CME = 6.50 Contact Hours http://www.nursecredentialing.org/RenewalRequirements.

AAPA Accreditation
AAPA accepts Certificates of Participation for educational activities certified for Category 1 from AOACCME or a recognized state medical society. Physician assistants may receive a maximum 6.50 of Category 1 credit for completing this program.

Physical Therapy Accreditation
Vassar Brothers Medical Center is recognized by the New York State Education Departments State Board for Physical Therapy as an approved provider of physical therapist assistant continuing education. This activity has been approved for 6.50 hours.

EMS
8.00 HVRMESCO Medical Control Hours
Occupational Therapy
6.50 CME = 6.50 Contact Hours. To claim units, you must submit your certificate of attendance with the activity brochure to NBCOT. [https://www.nbcot.org/](https://www.nbcot.org/)

Americans with Disabilities Act
We encourage participation by all individuals. If you have a disability, Advance notification of any special needs will help us to better serve you. Please notify us of your needs in advance of the program. Thank you.

Acknowledgements
Special Thanks to:
• The Grandview
• Vassar Brothers Medical Center for sponsoring this event
• The staff of Vassar Brothers CME

Disclosure
In accordance with the disclosure policies of Vassar Brothers CME, the effort is made to ensure balance, independence, objectivity, and scientific rigor in all educational activities. These policies include resolving all conflicts of interest between faculty and commercial interest that might otherwise compromise the goal and educational integrity of this activity.

Dr. A. Reshad Garan is an unpaid consultant for Abiomed. He will support his presentation and clinical recommendations with the “best available evidence” from the medical literature. He will refrain from making recommendations, regarding products or services, e.g., limit presentation to pathophysiology, diagnosis, and/or research findings.

All of the other faculty members participating in this activity have disclosed all significant relationship -financial or otherwise - with the manufacturers or providers of products or services mentioned in the activity. The planners of this activity have reviewed these disclosures and have determined that the faculty relationships are not inappropriate in the context of their respective presentations and are consistent with the educational goals and integrity of the activity. Unless otherwise noted, the planners and faculty participants do not have any financial arrangements or affiliations with any commercial entities whose products, research or services may be discussed in these materials.

This activity has not been funded by and unrestricted educational grant

Vassar Brothers gratefully acknowledges the support of the following exhibitors: Health Care Technologies, Hudson Valley Hospice
Upcoming CME Teaching Days

23rd Annual GI Teaching Day
The Grandview
Wednesday, September 26, 2018 | 8:00AM – 3:00PM

6th Annual Orthopedic Education Day
The Grandview
Thursday, October 4, 2018 | 7:30AM – 4:00PM

39th International Conference on Screening for Lung Cancer & 8th Conference on Research for Early Lung Cancer Treatment
The Culinary Institute of America
Tuesday, October 16, 2018 | 9:00AM – 4:45PM
Wednesday, October 17, 2018 | 9:00AM – 4:00PM

48th Annual Cardiology Teaching Day
The Grandview
Wednesday, October 24, 2018 | 7:30AM – 4:00PM

The Dr. Ian Portelli Memorial Trauma Conference
The Grandview
Wednesday, November 14, 2018 | 7:30AM – 4:00PM

For more information call 845-483-6013
TTY for the hearing impaired 800.421.1220
Register online at http://cmetracker.net/HQ/Catalog

Vassar Brothers Medical Center
HealthQUEST
During the conference, the full digital syllabus will be available on the conference webpage: https://vbmc.libguides.com/ResusCon1

You can view and take notes on this PDF syllabus on your mobile device through the free Adobe Reader app downloadable at http://www.adobe.com/products/reader-mobile.html.

Keep up-to-date on what other Vassar Brothers CME activities are offered on our website: http://vbmc.libguides.com/CME.

On staff with Health Quest? Need research or information? Check out the Health Quest Knowledge Resources website: infoDispensary at http://vbmc.libguides.com.

For more information call 800.123.4567 or register online at healthquest.org. TTY/Accessibility: 800.421.1220
Sepsis: Forecasting and Eliminating End Organ Dysfunction

Timothy Buchman, PhD, MD, FACS, FCCP, MCCM, Professor of Surgery, Emory University School of Medicine; Professor of Anesthesiology, Emory University School of Medicine; Chief, Critical Care Services, Emory Healthcare
Sepsis
from Surviving, to Forecasting, to Eliminating the Transition from Infection to Organ Dysfunction

Timothy G. Buchman, PhD, MD, FACS, FCCP, MCCM
Professor of Surgery, Anesthesiology, and Biomedical Informatics
Emory University
Medical Director, Emory eICU Center, Emory Critical Care Center
Editor-in-Chief, *Critical Care Medicine*
External Faculty, Santa Fe Institute
Cell: 404-561-3557
Email: tbuchma@emory.edu
Disclosure

Timothy G. Buchman

I do not have any relevant financial relationship(s) with any commercial interest that pertains to the content of my presentation.

Disclosure

Timothy G. Buchman is Editor-In-Chief of Critical Care Medicine

Opinions expressed in this lecture are personal, and may not reflect the position of the journal or of the Society of Critical Care Medicine.
Sepsis: An Approach

- Predicting Sepsis
- Preempting (Preventing) Sepsis
- Recognizing Sepsis
- Mitigating (Treating) Sepsis
- Recovering From Sepsis

Sepsis: This Talk

- Predicting Sepsis
- Preempting (Preventing) Sepsis
- Recognizing Sepsis
- Mitigating (Treating) Sepsis
- Recovering From Sepsis

1 2

"EMORY CRITICAL CARE CENTER"
Sepsis: This Talk

1. Predicting Sepsis
2. Preempting (Preventing) Sepsis
3. Recognizing Sepsis
4. Mitigating (Treating) Sepsis
5. Recovering From Sepsis

Sepsis-a confusing label

National Issues – 1990s
- Prevalence
- Cost
- (Lack of) Identifiability
  - Among caregivers
  - Among lay public
  - Among policy makers
  - Among funding agencies

Sepsis at a Glance

- IT CAUSES A LOT OF DEATHS
  - 3rd Leading Cause of Death
  - Contributes to 1 in every 3 hospital deaths

- IT COSTS A LOT
  - Most Expensive Condition Treated in U.S. Hospitals
  - $24 billion

- IT CAN PROGRESS QUICKLY
  - 7.6% drop in chance of survival each hour until antibiotics are begun

Sepsis at a Glance

What Does It Take to Beat Sepsis?
- Surveillance and early detection
- Antimicrobial stewardship
- Education and training
- Enhanced communication

Source: www.ecri.org, Dec. 2015
Dear SIRS, I'm sorry to say that I don't like you
Vincent, Jean-Louis MD, PhD, FCCM, Critical Care Medicine (1997), 25:372

• You’re too sensitive – more than 2/3 of ICU patients
• You don’t help us understand the physiology—jogging, e.g.
• You’re not helping our clinical trials—better scores (SOFA)
• You’re not helping us in our practice—so common, no clinical implications

Dear SIRS, I'm afraid we don’t need you
SIRS sensitivity—Contemporary Complaints

SIRS is an appropriate response to infection – and to many other stimuli that activate inflammation:
“Is it Sepsis—or not?”

Conclusions: Almost half of patients hospitalized on the wards developed SIRS at least once during their ward stay. Our findings suggest that screening ward patients using SIRS criteria for identifying those with sepsis would be impractical.

But ... is it sepsis?

Benchmarking the Incidence and Mortality of Severe Sepsis in the United States*

Four different ways to identify sepsis; four different sets of results

Number of cases

Total mortality

Crit Care Med 2013; 41: 1167-1174

Angus     Wang     Dombrovskiy     Martin     Mean Weighted
Admin coding *doesn’t* line up with the clinical data

---

**Incidence and Trends of Sepsis in US Hospitals Using Clinical vs Claims Data, 2009-2014**

Chen CH, N. W., Raymond D., M. S. N. Y, Lauren E. N., M. S. E. (et al)

Figure 2. Sepsis Trends From 2009-2014: Incidence, In-hospital Sepsis Mortality, and In-hospital Mortality or Discharge to Hospital.

Explicit codes: Severe Sepsis, Septic Shock

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**GO BACK AND START AGAIN**
Sepsis: issues, needs

Each year
- 250,000 American deaths
- 5.3 million deaths worldwide

Sepsis treatment is costly
- Most expensive hospital condition
- US hospital costs alone, $24 B/year
- Costs will rise proportionate to aging of population

From Sepsis-(1,2) to Sepsis-3: Sepsis (Re)Defined

Sepsis is (now) defined as:
(a) life-threatening
(b) organ dysfunction caused by a
c) dysregulated host response to
(d) infection

SEPSIS CLINICAL CRITERIA

INFECTION

CHANGE IN:
SEPSIS-RELATED
ORGAN FAILURE
ASSESSMENT

≥2

PaO₂/FIO₂
HYPOTENSION OR
VASOPRESSORS
GLASSOW
COMA SCALE
PLATELETS
BILIRUBIN
CREATININE,
OLIGURIA
From Sepsis-(1,2) to Sepsis-3: Sepsis (Re)Defined

Sepsis is (now) defined as:
(a) life-threatening
(b) organ dysfunction caused by a
c(c) dysregulated host response to
(d) infection

But are these measureable?
(a)Yes
(b)Yes
(c)Indirectly
(d)Sometimes

The measurement of organ (dys)function

<table>
<thead>
<tr>
<th>System</th>
<th>Score</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td></td>
<td>≥400 (53.3)</td>
<td>&lt;400 (53.3)</td>
<td>≥300 (40)</td>
<td>&lt;200 (26.7) with respiratory support</td>
<td>&lt;100 (13.7) with respiratory support</td>
</tr>
<tr>
<td>Coagulation</td>
<td></td>
<td>≥150</td>
<td>&gt;150</td>
<td>≥100</td>
<td>&lt;50</td>
<td>&gt;20</td>
</tr>
<tr>
<td>Liver</td>
<td></td>
<td>≥1.2 (10)</td>
<td>&gt;1.2-3.9 (20-32)</td>
<td>2.0-5.9 (100-101)</td>
<td>6.0-11.9 (102-204)</td>
<td>&gt;12.0 (204)</td>
</tr>
<tr>
<td>Cardiopulmonary</td>
<td></td>
<td>MAP ≥70 mm Hg</td>
<td>MAP &lt;70 mm Hg</td>
<td>Dopamine &lt;5 or diabeteric (any dose)</td>
<td>Dopamine 5-15 or epinephrine (0.5) or norepinephrine (0.1)</td>
<td>Dopamine &lt;1.5 or epinephrine &gt;0.5 or norepinephrine &gt;0.1</td>
</tr>
<tr>
<td>Central nervous system</td>
<td></td>
<td>Glasgow Coma Scale score *</td>
<td>15</td>
<td>10-14</td>
<td>10-12</td>
<td>6-9</td>
</tr>
<tr>
<td>Creatinine, mg/dl</td>
<td></td>
<td>&lt;1.2 (11)</td>
<td>1.2-3.9 (130-170)</td>
<td>2.0-3.4 (170-209)</td>
<td>3.5-4.9 (330-440)</td>
<td>&gt;5.0 (440)</td>
</tr>
<tr>
<td>Urea nitrogen, mg/dl</td>
<td></td>
<td>&lt;500</td>
<td>&gt;500</td>
<td>&gt;500</td>
<td>&gt;100</td>
<td>&gt;200</td>
</tr>
</tbody>
</table>

Abbreviations: Fio2, fraction of inspired oxygen; MAP, mean arterial pressure; PaO2, partial pressure of oxygen.

*Glasgow Coma Scale scores range from 3-15; higher score indicates better neurological function.
Making this work at the **inpatient bedside**...

1. **Do you suspect INFECTION?**
   
   In other words, did you culture + prescribe antibiotics?

1. **If you suspect INFECTION, is the patient **REALLY SICK**?**
   
   In numerical terms,
   
   (a) is the SOFA ≥ 2; or
   
   (b) is there a change ≥ 2, ΔSOFA?

---

**Definitions were data driven**

*Infection suspicion: First episode of cultures, antibiotics*

- **Derivation** - 1.3 million EHR records from UPMC
  - 148,000 with suspected infection

- **Validation** – almost 6 million records
  - KPNC
  - VA
  - “ALERTS” database from Germany
  - Kings County (Seattle) EMS
  - > 700,000 with suspected infection
How did that SOFA/delta SOFA come to represent “really sick”? 

- Clinical review committees
- Death in the hospital (countable)
- Prolonged stay in the ICU (measurable)
- Discharge diagnosis of sepsis
- Positive microbiologic cultures

Definitions were data driven

**SOFA**

- **Derivation cohort**
  - Surviving Sepsis Campaign Database (SSC) 2005-2010,
    - n = 28,150

- **Validation cohort**
  - 12 hospitals in Pennsylvania (UPMC), 2010-2012;
    - n = 1,309,025
  - 20 Hospitals (Kaiser Permanente Northern California, KPNC), 2009-2013;
    - n = 1,847,165
Details here, free:

**Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)**

JAMA 2016; 315: 801-10

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But…what about “in the field”?

- Studied 21 variables from Sepsis-2
- Multivariable logistic regression for in-hospital mortality

- Respiratory rate ≥ 22 bpm
- Altered mentation
- Systolic blood pressure ≤ 100 mmHg
### The Criteria—getting to SOFA and qSOFA

<table>
<thead>
<tr>
<th>Criteria</th>
<th>SIRS</th>
<th>SOFA</th>
<th>LODS</th>
<th>qSOFA</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICU</td>
<td>0.64 (0.62, 0.66)</td>
<td>&lt;0.01</td>
<td>0.74 (0.73, 0.76)</td>
<td>0.66 (0.64, 0.68)</td>
</tr>
<tr>
<td>SOFA</td>
<td>&lt;0.01</td>
<td>0.76 (0.75, 0.77)</td>
<td>0.75 (0.73, 0.76)</td>
<td>0.66 (0.64, 0.68)</td>
</tr>
<tr>
<td>LODS</td>
<td>&lt;0.01</td>
<td>0.20</td>
<td>0.75 (0.73, 0.76)</td>
<td>0.66 (0.64, 0.68)</td>
</tr>
<tr>
<td>qSOFA</td>
<td>0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>0.66 (0.64, 0.68)</td>
</tr>
</tbody>
</table>

**AUROC in-hospital mortality**

**ICU encounters**
- N = 7,932
- 0.76 (0.75, 0.77) < 0.01
- 0.79 (0.78, 0.80) < 0.01
- 0.81 (0.80, 0.82) < 0.01

**Outside the ICU encounters**
- N = 66,522
- 0.72
- 0.81 (0.80, 0.82)

SOFA and LODS superior in the ICU

qSOFA similar to complex scores outside the ICU

### Lactate does ***not*** make a difference with qSOFA

**All KPNC encounters**
- N = 321,380

<table>
<thead>
<tr>
<th>Sensitivity vs. 1-specificity</th>
<th>0.00</th>
<th>0.25</th>
<th>0.50</th>
<th>0.75</th>
<th>1.00</th>
</tr>
</thead>
<tbody>
<tr>
<td>qSOFA baseline</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>qSOFA + lactate 12.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>qSOFA + lactate 20.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>qSOFA + lactate 32.0</td>
<td></td>
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*EMORY CRITICAL CARE CENTER*
Septic shock

2016 septic shock definition
Subset of sepsis in which
• underlying circulatory, cellular and metabolic abnormalities are associated with
• a greater risk of mortality
• than sepsis alone
Hypotension after fluids

Vasopressors

Lactate>2

Prevalence Surviving Sepsis Campaign

Mortality

<table>
<thead>
<tr>
<th>Group</th>
<th>Hypotension after fluids</th>
<th>Vasopressors</th>
<th>Lactate&gt;2</th>
<th>Prevalence Surviving Sepsis Campaign</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>45.2%</td>
<td>42.3%</td>
</tr>
<tr>
<td>Group 2</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>21.2%</td>
<td>30.1%</td>
</tr>
<tr>
<td>Group 3</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>1.2%</td>
<td></td>
</tr>
<tr>
<td>Group 4</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>17.3%</td>
<td>25.7%</td>
</tr>
<tr>
<td>Group 5</td>
<td>Never (pre)</td>
<td>No</td>
<td>Yes</td>
<td>14.3%</td>
<td>29.7%</td>
</tr>
<tr>
<td>Group 6</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>0.8%</td>
<td></td>
</tr>
</tbody>
</table>

Patient groups defined by easily accessible variables

Bedside criteria for septic shock

- **Despite** adequate fluid resuscitation
  - **vasopressors needed** to maintain MAP ≥65 mmHg
  - **AND**
  - lactate >2 mmol/l
Confusion and Confirmations – Sepsis-3

• The Sepsis-3 criteria perform as expected
  • Minimal incremental improvement adding lactate, ...
  • SIRS remains more sensitive, low specificity
  • Sepsis-3 works in diverse practice settings
  • Picks out a sicker population
  • Confirmations worldwide

Recognizing Sepsis remains a “Work in Progress”...

Rev. bras. ter. intensiva vol.29 no.1 São Paulo Jan./Mar. 2017
http://dx.doi.org/10.5935/0103-507x.20170002

Dear Sepsis-3, we are sorry to say that we don’t like you.

António Henrques Carneiro1
Pedro Póvoa2 3
José Andrade Gomes4

Details and rebuttal
are the subject of
a different talk...
Sepsis: This Talk

Predicting Sepsis
Preempting (Preventing) Sepsis
Recognizing Sepsis
Mitigating (Treating) Sepsis
Recovering From Sepsis

From classifiers to guidelines

Classifiers serve one purpose (Sepsis-3 is a Classifier)
- To differentiate or distinguish
  - Diagnoses
  - Syndromes
  - Treatments
  - Prognoses

Guidelines serve a different purpose (Surviving Sepsis is a Guideline)
- To recommend one or more practices
  - For the general population
  - For a (classified) subpopulation
- Intended to realize one or more specific outcomes
Guidelines change in response to evolving knowledge (evidence) and practice (“usual care”)

ACLS “knowledge”
- “Acidotic patients die!”
- Acidosis is a marker for inadequate perfusion

ACLS “practice”
- Administer Sodium Bicarbonate!
- CAB! Push hard! Push fast!

Guidelines seek to bridge and to reconcile evidence and care

Surviving Sepsis Guidelines 2016 published in *Critical Care Medicine*

- Dozens of Recommendations
  - 32 Strong recommendations: “*We recommend*”
  - 39 Weak recommendations: “*We suggest*”
  - 18 Best Practice Statements (seems reasonable)
Sepsis and septic shock are medical emergencies and we recommend that treatment and resuscitation begin immediately.

Best Practice Statement (no evidence!)

Antibiotics

• We recommend that administration of IV antimicrobials be initiated as soon as possible after recognition and within 1 h for both sepsis and septic shock.

(strong recommendation, moderate quality of evidence).

• We recommend empiric broad-spectrum therapy with one or more antimicrobials to cover all likely pathogens.

(strong recommendation, moderate quality of evidence).
Does pre-hospital antibiotic administration improve outcome in sepsis?

**Sepsis-2 (SIRS + Suspected Infection)**

- **3228 patients screened**
- **2698 randomized**
- **Treatment arm: Ceftriaxone 2g**
- **Primary outcome: all-cause mortality**
- **8% in both control and treatment groups**
Hemodynamic Recommendations

• Start resuscitation early with source control, intravenous fluids and antibiotics.

• Frequent assessment of the patients’ volume status is crucial throughout the resuscitation period.

• We suggest guiding resuscitation to normalize lactate in patients with elevated lactate levels as a marker of tissue hypoperfusion.

Lactate can help guide resuscitation

• We suggest guiding resuscitation to normalize lactate in patients with elevated lactate levels as a marker of tissue hypoperfusion.

(Weak recommendation; low quality of evidence)
The Surviving Sepsis Campaign Bundle: 2018 Update

- Measure lactate level. Remeasure if initial lactate is >2 mmol/L.
- Obtain blood cultures prior to administration of antibiotics.
- Administer broad-spectrum antibiotics.
- Begin rapid administration of 30ml/kg crystalloid for hypotension or lactate ≥4 mmol/L.
- Apply vasopressors if patient is hypotensive during or after fluid resuscitation to maintain MAP ≥65 mm Hg.

**“Time zero” or “time of presentation” is defined as the time of triage in the Emergency Department or, if presenting from another care venue, from the earliest chart annotation consistent with all elements of sepsis (formerly severe sepsis) or septic shock ascertained through chart review.**

Figure 1. Hour-1 Surviving Sepsis Campaign Bundle of Care.

### Table 1. Bundle Elements With Strength of Recommendations and Under-Pinning Quality of Evidence (12, 13)

<table>
<thead>
<tr>
<th>Bundle Element</th>
<th>Grade of Recommendation and Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measure lactate level. Remeasure if initial lactate is &gt;2 mmol/L</td>
<td>Weak recommendation, low quality of evidence</td>
</tr>
<tr>
<td>Obtain blood cultures prior to administration of antibiotics</td>
<td>Best practice statement</td>
</tr>
<tr>
<td>Administer broad-spectrum antibiotics</td>
<td>Strong recommendation, moderate quality of evidence</td>
</tr>
<tr>
<td>Rapidly administer 30mL/kg crystalloid for hypotension or lactate ≥ 4 mmol/L</td>
<td>Strong recommendation, low quality of evidence</td>
</tr>
<tr>
<td>Apply vasopressors if patient is hypotensive during or after fluid resuscitation to maintain mean arterial pressure ≥ 65 mm Hg</td>
<td>Strong recommendation, moderate quality of evidence</td>
</tr>
</tbody>
</table>
Insight

• Early Therapy
  • The useful parts have become standard care
  • Evidence remains weak, strengthening may be difficult
  • Controversy continues

• The Next Frontier
  • Identifying Sepsis-3 patients before they meet those criteria
  • (Earlier antibiotics are the only therapy consistently demonstrated to save lives.)

Sepsis: This Talk

Predicting Sepsis
Preempting (Preventing) Sepsis
Recognizing Sepsis
Mitigating (Treating) Sepsis
Recovering From Sepsis
Finding the septic patient: two approaches

Managing data:
Clinical assessment, data integration & synchronization

Intensive Care Unit (ICU)

Chart Review

Waveform Matching

Clinical Databases
Electronic Medical Records (EMRs)
The Emory data pipeline

- Philips eICU Adapter
- Cleanse & Transform
- Score Sepsis Model
- Raise Clinical Alert
- Philips eICU Adapter
- BedMasterEX Adapter
- Enrich & Summarize
- Generate Features
- BedMasterEX Adapter
- Ingest
- Analyze
- Predict & Respond

(Vendors named for identification purposes only)

Artificial Intelligence Sepsis Expert (AISE)

- Trained and validated on independent 30,000 and 50,000 patient cohorts, 1,000 miles apart.
- Predicting Sepsis (following Sepsis-III definition) 4-8 hours in advance with an AUC of 0.85.
- Reveals top causes per each prediction

An Interpretable Machine Learning Model for Accurate Prediction of Sepsis in the ICU

- Trained and validated on independent 30,000 and 50,000 patient cohorts, 1,000 miles apart.
- Predicting Sepsis (following Sepsis-III definition) 4-8 hours in advance with an AUC of 0.85.
- Reveals top causes per each prediction

in Critical Care Medicine
April 2018 - Volume 46 - Issue 4 - p 547–553
An Emerging Role: Sepsis Safety Officers
The Emory Program eICU Program:
- eRNs: 24 x 7 x 365
eMDs: nights weekends and holidays
Since Mar 2013
- >100,000 PATIENT DAYS
- 16 locations, 142 beds in 5 hospitals
  (2 university, 1 hybrid, 2 community)

What would Emory like to do next around sepsis?

Validation of this (and other) predictive tools without exchanging confidential health information

Two possible approaches:
1. Share the architecture and the algorithms
   run the analyses locally at collaborating hospitals

2. New strategies for data collaboration
   NOT data sharing
   Brokered third space

Wearable monitors following discharge to home—ensure reliable progress towards healthy state.
An Overview of
Areas of Interest

Timothy G. Buchman, PhD, MD
13 SEP 2018
BARDA Division of Research, Innovation, and Ventures (DRIVE)

DRIVE Mission: Transforming Health Security
Accelerate the research, development, and availability of transformative countermeasures to protect Americans from natural and intentional health security threats.

Areas of Interest (AOI)

• DRIVE wants to emphasize revolutionary approaches hyperfocused on the following AOIs:
• Early Notification to Act, Control, and Treat (ENACT)
• Solving Sepsis
• Other Innovative Products with potential to radically transform health security.
Sections of Interest:

- Health Signatures Discovery & Validation
- Diagnostic Technology & Development
- Novel Biosensing & Wearable Technologies
- Cloud-based reporting & data analytics
- Prediction & Artificial Intelligence
- In-home, near user deployment

DRIVE AOI #2: Solving Sepsis

BARDA’s mission is to develop MCMs against CBRN, influenza and emerging infectious disease threats, to minimize public health impact.

Sepsis is a secondary confounder that arises from primary insults – threatens our ability to protect our Nation.

Targeting pathogen or insult is critical, but not always sufficient.
Solving Sepsis

PATIENT → CLINIC VISIT → ICU CLINICAL MANAGEMENT

- Education and Awareness
- Diagnostic/Prognostic toolkits
- Continuous Monitoring/Integrated Feedback
- Individualized Treatment capabilities
- National sepsis database

*Virtual Community

Not expected to be funded via traditional BAA mechanism

PREVENT RE-ADMISSION

DRIVE

INVESTMENT TARGET AREAS

Enabling Technologies

Database: No central Sepsis repository exists
Continuous Monitoring: Need to monitor dynamic changes
Point-of-Care Prognostic/Diagnostic: Host vs. pathogen based

Biomarker discovery
Host-pathogen pathway analysis
Individualized treatment approaches to restore homeostasis
DRIVE AOI # 3: Other Innovative Products with potential to radically transform Health Security

What’s your moonshot idea?

DRIVE wants to hear your bold idea to radically transform Health Security

For additional information

https://drive.hhs.gov

My USG contact information (DRIVE business only):
Timothy G. Buchman, PhD, MD
Senior Advisor
IPA to the DRIVE (Division of Research, Innovation and Ventures)
Cell: 202-424-9181
Email: Tim.Buchman@hhs.gov
Thank you for the privilege of speaking this morning

stop sepsis save lives
Fluid Resuscitation in Trauma- Do No Harm

Ronald Simon, MD, Chief, Division Acute Care Surgery Maimonides Medical Center Brooklyn, Brooklyn, New York
Fluid Resuscitation: Does what and how much we use really make a difference

Ronald Simon, MD, FACS
Chief, Division of Acute Care Surgery
Maimonides Medical Center
Brooklyn, NY

Fluids: the double edged sword

• Fluid administration:
  • One of the most frequently performed interventions in the hospital (and probably one of the least understood)
  • To little can resultant in a persistent shock state
  • To much can lead to increased morbidity and mortality
  • No good measurement for the adequacy of volume resuscitation
  • Limited data on the types of fluids we use
How did we get here?

- Wiggers prep for hemorrhagic shock
  - 3:1 ratio of crystalloid to blood
- Shoemakers articles on resuscitation
  - Maximize oxygen delivery with massive crystalloid infusions for preload maximization
  - New epidemic of intraabdominal hypertension and abdominal compartment syndrome
- Original Rivers sepsis guidelines (Surviving sepsis guidelines 2001)
  - Start with 3L of crystalloid

Crystalloids will be the death of us all...

Aggressive early crystalloid resuscitation adversely affects outcomes in adult blunt trauma patients: An analysis of the Glue Grant database

- Prospective database of severe injured BLUNT trauma patients
- Blood transfusion within 24hrs, or ED SBP<90, or BD >-6
- 1,754 patients
- Divided into 4 groups based on volume of crystalloids within initial 24hrs

J Trauma Acute Care Surg. 2013;74: 1215-1222
<table>
<thead>
<tr>
<th></th>
<th>Value</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>43.5 (18.0)</td>
<td>18 to 90</td>
</tr>
<tr>
<td>Male sex, %</td>
<td>66.6</td>
<td></td>
</tr>
<tr>
<td>ED SBP, mean (SD), mm Hg</td>
<td>111.1 (30.6)</td>
<td>0 to 243</td>
</tr>
<tr>
<td>ED HR, mean (SD), beats per minute</td>
<td>109.0 (26.7)</td>
<td>0 to 200</td>
</tr>
<tr>
<td>GCS score, mean (SD)</td>
<td>11.3 (4.6)</td>
<td>3 to 15</td>
</tr>
<tr>
<td>WBC, mean (SD)</td>
<td>16.6 (7.7)</td>
<td>1 to 50.8</td>
</tr>
<tr>
<td>Hemoglobin, mean (SD), g/dL</td>
<td>11.5 (2.5)</td>
<td>2.7 to 18.7</td>
</tr>
<tr>
<td>BD, mean (SD)</td>
<td>-8.4 (4.5)</td>
<td>-30 to 11</td>
</tr>
<tr>
<td>Lactate, mean (SD), mmol/L</td>
<td>4.4 (2.6)</td>
<td>0.6 to 25</td>
</tr>
<tr>
<td>APACHE II, mean (SD)</td>
<td>28.1 (7.2)</td>
<td>5 to 49</td>
</tr>
<tr>
<td>ISS, mean (SD)</td>
<td>32.2 (13.4)</td>
<td>1 to 75</td>
</tr>
<tr>
<td>ISS &gt; 25, %</td>
<td>71.7</td>
<td></td>
</tr>
<tr>
<td>Charlson comorbidity index, mean (SD)</td>
<td>0.4 (1.0)</td>
<td>0 to 10</td>
</tr>
<tr>
<td>Major surgery in first 24 h, %</td>
<td>33.4</td>
<td></td>
</tr>
<tr>
<td>Ventilator days, mean (SD), d</td>
<td>10.4 (10.8)</td>
<td>0 to 135</td>
</tr>
<tr>
<td>ICU LOS, mean (SD), d</td>
<td>14.0 (12.6)</td>
<td>0 to 142</td>
</tr>
<tr>
<td>Hospital LOS, mean (SD), d</td>
<td>25.1 (22.8)</td>
<td>3 to 354</td>
</tr>
<tr>
<td>Mortality, %</td>
<td>9.1</td>
<td></td>
</tr>
</tbody>
</table>

HR, heart rate; SBP, systolic blood pressure; WBC, white blood cell count.

<table>
<thead>
<tr>
<th>24-h Crystalloid Resuscitation</th>
<th>&lt;5 L</th>
<th>5-10 L</th>
<th>10-15 L</th>
<th>&gt;15 L</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>87</td>
<td>475</td>
<td>574</td>
<td>618</td>
<td></td>
</tr>
<tr>
<td>Age, mean (SD), y</td>
<td>50.9 (21.4)</td>
<td>44.9 (19)</td>
<td>43.4 (17.3)</td>
<td>41.5 (17.1)</td>
<td>0.004</td>
</tr>
<tr>
<td>GCS score, mean (SD)</td>
<td>11.1 (4.9)</td>
<td>11.6 (4.4)</td>
<td>11.3 (4.6)</td>
<td>11.0 (4.8)</td>
<td>0.332</td>
</tr>
<tr>
<td>ED SBP, mean (SD), mm Hg</td>
<td>106 (32.8)</td>
<td>108 (27.5)</td>
<td>111 (31.5)</td>
<td>113 (31.5)</td>
<td>0.014</td>
</tr>
<tr>
<td>ED HR, mean (SD), beats per minute</td>
<td>103.2 (26.7)</td>
<td>104.1 (25.9)</td>
<td>109.5 (26.4)</td>
<td>113.3 (27.7)</td>
<td>0.007</td>
</tr>
<tr>
<td>BD, mean (SD)</td>
<td>-7 (5)</td>
<td>-7 (4.3)</td>
<td>-8 (4.1)</td>
<td>-9 (4.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hemoglobin, mean (SD), g/dL</td>
<td>11.4 (2.5)</td>
<td>11.6 (2.3)</td>
<td>11.5 (2.5)</td>
<td>11.3 (2.8)</td>
<td>0.105</td>
</tr>
<tr>
<td>ISS, mean (SD)</td>
<td>27.8 (12.9)</td>
<td>29.9 (12.6)</td>
<td>32.1 (13.6)</td>
<td>34.8 (13.4)</td>
<td>0.348</td>
</tr>
<tr>
<td>APACHE II, mean (SD)</td>
<td>23.9 (8)</td>
<td>26.1 (7.6)</td>
<td>27.6 (6.8)</td>
<td>30.6 (6.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Comorbidity index, mean (SD)</td>
<td>0.6 (1.4)</td>
<td>0.5 (1.1)</td>
<td>0.4 (0.9)</td>
<td>0.3 (0.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ventilator days, mean (SD)</td>
<td>7.3 (7.9)</td>
<td>8.4 (9.2)</td>
<td>10 (11.4)</td>
<td>12.8 (11.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ICU LOS, mean (SD), d</td>
<td>11.3 (8.7)</td>
<td>12.5 (12.1)</td>
<td>13.1 (12.2)</td>
<td>16.4 (13.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hospital LOS, mean (SD), d</td>
<td>19.6 (12.2)</td>
<td>21.6 (19.5)</td>
<td>23.8 (17.7)</td>
<td>30 (29.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ALI/ARDS, %</td>
<td>11.5</td>
<td>14.1</td>
<td>23.3</td>
<td>37.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>MOF, %</td>
<td>14.9</td>
<td>20.2</td>
<td>28.2</td>
<td>46.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Abdominal compartment syndrome, %</td>
<td>0</td>
<td>0.6</td>
<td>4.3</td>
<td>12.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Extremity compartment syndrome, %</td>
<td>1.1</td>
<td>1.7</td>
<td>3.6</td>
<td>5.7</td>
<td>0.003</td>
</tr>
<tr>
<td>BSI, %</td>
<td>8</td>
<td>9.9</td>
<td>12.9</td>
<td>20.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SSI, %</td>
<td>4.6</td>
<td>9.1</td>
<td>14.1</td>
<td>20.9</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

HR, heart rate; SBP, systolic blood pressure.
<table>
<thead>
<tr>
<th>Condition</th>
<th>OR (95% CI)</th>
<th>p</th>
<th>OR (95% CI)</th>
<th>p</th>
<th>OR (95% CI)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALI/ARDS</td>
<td>Reference</td>
<td>1.7 (0.71-3.9)</td>
<td>0.24</td>
<td>2.3 (1.5-5.4)</td>
<td>0.05</td>
<td>3.4 (1.5-7.9)</td>
</tr>
<tr>
<td>MOF</td>
<td>Reference</td>
<td>1.5 (0.7-3.3)</td>
<td>0.20</td>
<td>1.9 (0.9-4.1)</td>
<td>0.1</td>
<td>2.9 (1.3-6.1)</td>
</tr>
<tr>
<td>Abdominal compartment syndrome</td>
<td>Reference</td>
<td>NA</td>
<td>4.8 (1.4-16.4)</td>
<td>0.01</td>
<td>8.7 (2.6-28.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Extremity compartment syndrome</td>
<td>Reference</td>
<td>1.3 (0.2-10.5)</td>
<td>0.855</td>
<td>2.4 (0.3-18.9)</td>
<td>0.393</td>
<td>3.8 (0.5-29.3)</td>
</tr>
<tr>
<td>BSI</td>
<td>Reference</td>
<td>1.5 (0.6-4)</td>
<td>0.308</td>
<td>1.9 (0.7-4.8)</td>
<td>0.206</td>
<td>2.5 (1-6.6)</td>
</tr>
<tr>
<td>SSI</td>
<td>Reference</td>
<td>1.7 (0.6-5)</td>
<td>0.32</td>
<td>2.3 (0.8-6.6)</td>
<td>0.206</td>
<td>2.6 (1-8.2)</td>
</tr>
</tbody>
</table>

ORs controlled for age, GCS score, ISS and APACHE II score, concomitant co-factor and blood product administration over the same time frame.
CI, confidence interval, NA, not applicable.
Aggressive early crystalloid resuscitation adversely affects outcomes in adult blunt trauma patients: An analysis of the Glue Grant database

- Incidence of mortality and ARF were not impacted by volume (type of crystalloids used was not studied)
- Study suggests that increasing volume of crystalloid resuscitation worsens outcomes (except mortality/ARF) even when controlled for injury severity and physiologic markers of severity

Goal-directed resuscitation in the prehospital setting: a propensity-adjusted analysis

- Retrospective study of 1200 blunt trauma patients
- Divided into 2 groups depending on prehospital crystalloid volume
  - <500cc vs >500cc
- Prehospital hypotension captured

• No difference in mortality between volume groups in the **hypotensive subgroup**
• Mortality was greater in:
  • >500cc group in the never hypotensive group
  • Presence of coagulopathy present on admission(Incr INR)
• Suggests that at less volume during early resuscitation is at least as good as more, and may be better

**Fluids and the Macro vs Micro Circulation**

• **Macrocirculation**
  • Frank Starling relationship between preload and cardiac output
  • Fluid responsive vs Fluid non-responsive
  • Hard to assess fluid responsiveness prior to fluid administration
    • Need to have some measure of CO(minimum is an aline)
    • Heart rate and CVP have been shown to be misleading
Fluids and the Macro vs Micro Circulation

• Microcirculation
  • The part of the circulation that is responsible for tissue perfusion
• In health there is coherence between the macro and micro circulations
• The status of the microcirculation is effected by many factors
  • Blood flow
  • Vasoactive molecules released into the local circulation
  • Acidosis, oxygen tension
• In disease states this coherence can be lost

Fluids and the Macro vs Micro Circulation

• In disease states this coherence can be lost
• Nitric oxide is released in sepsis causing microcirculatioriy vasodilation
• Sepsis can produce a state of hypercoagulopathy that can result in local thrombosis and tissue ischemia
• Oxidative stress leads to endothelial dysfunction
Principles of fluid management and stewardship in septic shock: it is time to consider the four D’s and the four phases of fluid therapy

4 phases of fluid therapy in septic shock
1. Resuscitation – when to start
2. Optimization – how much to give
3. Stabilization – when to stop
4. Evacuation – active fluid removal

How to we balance the need for fluid resuscitation with the evolving reality that:
• Too much fluid is bad
• Which fluid we give can also effect outcome

### Composition of Common Isotonic Crystalloids

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Na</th>
<th>K</th>
<th>Cl</th>
<th>Ca</th>
<th>Mg</th>
<th>Lact</th>
<th>Acet</th>
<th>Glucon</th>
<th>Dext</th>
<th>Osmol mOsm/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.9% N Saline</td>
<td>154</td>
<td>0</td>
<td>154</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>308</td>
</tr>
<tr>
<td>Lactated Ringers</td>
<td>131</td>
<td>5</td>
<td>11</td>
<td>2.7</td>
<td>0</td>
<td>29</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>273</td>
</tr>
<tr>
<td>Hartmanns</td>
<td>129</td>
<td>5</td>
<td>109</td>
<td>4</td>
<td>0</td>
<td>29</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>278</td>
</tr>
<tr>
<td>Plasma Lyte</td>
<td>140</td>
<td>5</td>
<td>98</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>27</td>
<td>23</td>
<td>0</td>
<td>280</td>
</tr>
</tbody>
</table>
Saline vs Plasma-Lyte 148 for ICU fluid Therapy (SPLIT) Trial

- Multicenter study in New Zealand
- ICU patients (70% elective surgical)
- Comparing NS vs Plasma-lyte
- 2278 patients enrolled

Young, P et al. JAMA 2015 314( 16)

---

Saline vs Plasma-Lyte 148 for ICU Fluid Therapy (SPLIT) Trial

- Median volume infused during initial 24hrs – 2,000cc
- No difference in mortality
- No difference for need for RRT or development of AKI
- Problems with trial
  - Only 15% came from the ED
  - Only 4% had sepsis
  - Fluid volumes for entire stay in the ICU were small
  - Inadequately powered to show small differences

Young, P et al. JAMA 2015 314( 16)
SALT-ED Trial
Saline vs LR in the ED

- In **non-critically ill** patients in the ED, does fluid mgmt with NS vs BSS make a difference?
- Pragmatic, single center study over 16 mths looking at pts given either NS or LR in the ED and were admitted to a non-ICU setting
- Choice of fluids varied by month
- Primary outcome – hospital LOS
- Secondary outcomes – adverse kidney events: death, new RRT, or a doubling of baseline Cr


---

SALT-ED Trial
Saline vs LR in the ED

- 13,347 patients enrolled
- Median volume infused – 1,079cc while in the ED
- 32% of pts received >2L while in the ED
- 88.3% of pts received exclusively assigned fluid
- Medical 77%, Surgery 23%

SALT-ED Trial
Saline vs LR in the ED


Table 3. Clinical Outcomes According to Assigned Treatment Group in the Intention-to-Treat Analysis.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Balanced Crystalloids (N=6708)</th>
<th>Saline (N=6639)</th>
<th>Adjusted Odds Ratio (95% CI)</th>
<th>Adjusted P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median hospital-free days to day 28 (IQR)</td>
<td>25 (22–26)</td>
<td>25 (22–26)</td>
<td>0.98 (0.92–1.04)</td>
<td>0.41</td>
</tr>
<tr>
<td>Major adverse kidney event within 30 days</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>— no. (%)</td>
<td>315 (4.7)</td>
<td>370 (5.6)</td>
<td>0.82 (0.70–0.95)</td>
<td><strong>0.01</strong></td>
</tr>
<tr>
<td>Death — no. (%)</td>
<td>94 (1.4)</td>
<td>102 (1.5)</td>
<td>0.89</td>
<td></td>
</tr>
<tr>
<td>New renal-replacement therapy — no./total no. (%)</td>
<td>18/6582 (0.3)</td>
<td>31/6530 (0.5)</td>
<td>0.56</td>
<td></td>
</tr>
<tr>
<td>Final serum creatinine ≥200% of baseline — no./total no. (%)</td>
<td>253/6582 (3.8)</td>
<td>293/6530 (4.5)</td>
<td>0.84</td>
<td></td>
</tr>
<tr>
<td>Stage 2 or higher acute kidney injury — no./total no. (%)</td>
<td>528/6582 (8.0)</td>
<td>560/6530 (8.6)</td>
<td>0.91 (0.80–1.03)</td>
<td>0.14</td>
</tr>
<tr>
<td>In-hospital death — no. (%)</td>
<td>95 (1.4)</td>
<td>105 (1.6)</td>
<td>0.88 (0.66–1.16)</td>
<td>0.36</td>
</tr>
</tbody>
</table>

SALT-ED Trial
Saline vs LR in the ED

- No difference in hospital free days (25 vs 25 days)
- BSS resulted in lower incidence of adverse kidney events (4.7 vs 5.6%)
  - New RRT, doubling of baseline serum Cr, death
- Mortality – 1.4 v 1.5% (NS)
- Subgroup analysis showed those with renal dysfunction on admission or hyperchloremia had the greatest benefit.
- Conclusion: no major difference in outcomes in this group of pts that received limited fluid resuscitation.


Balanced Crystalloids vs Saline in Critically Ill Adults
(Smart Study)

- Randomized study in 5 ICU’s in one hospital(Vanderbuilt)
- Occurred over 2 year period
- Fluids given were determined by which ICU they were in
- Each ICU rotated fluids used by month
- Compared NS vs LR or Plasma-lyte

Balanced Crystalloids vs Saline in Critically Ill Adults
(Smart Study)

- Primary outcome: any adverse kidney event within 30 days
  - Death
  - Beginning of renal replacement therapy
  - Doubling of baseline creatinine
- Secondary outcomes
  - In hospital death
  - ICU, pressor and vent free days
  - Days free of RRT
  - Creatinine level at DC


Balanced Crystalloids vs Saline in Critically Ill Adults
(Smart Study)

- 15,802 pts enrolled (7,942 BSS, 7,860 NS)
- Median age: 58
- Male: 57.6%

### Table 1. Participant Characteristics at Baseline.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Balanced Crystalloids (N = 7942)</th>
<th>Saline (N = 7660)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong> — yr</td>
<td>58</td>
<td>58</td>
</tr>
<tr>
<td>Median</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>44.69</td>
<td>44.69</td>
</tr>
<tr>
<td>Male sex — no. (%)</td>
<td>4546 (57.2)</td>
<td>4557 (58.0)</td>
</tr>
<tr>
<td>White race — no. (%)</td>
<td>6381 (80.4)</td>
<td>6322 (80.4)</td>
</tr>
<tr>
<td>Weight — kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>80</td>
<td>79</td>
</tr>
<tr>
<td>Interquartile range</td>
<td>65-96</td>
<td>68-95</td>
</tr>
<tr>
<td><strong>Concomitant medical conditions — no. (%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic kidney disease of stage 2 or higher</td>
<td>1285 (17.5)</td>
<td>1290 (17.7)</td>
</tr>
<tr>
<td>Previous receipt of renal replacement therapy — no. (%)</td>
<td>384 (4.8)</td>
<td>402 (5.3)</td>
</tr>
<tr>
<td>Source of admission to ICU — no. (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emergency department</td>
<td>3975 (50.1)</td>
<td>3997 (50.9)</td>
</tr>
<tr>
<td>Operating room</td>
<td>1732 (21.8)</td>
<td>1649 (21.4)</td>
</tr>
<tr>
<td>Transfer from another hospital</td>
<td>1018 (13.1)</td>
<td>1018 (13.0)</td>
</tr>
<tr>
<td>Hospital ward</td>
<td>792 (9.9)</td>
<td>792 (9.9)</td>
</tr>
<tr>
<td>Outpatient</td>
<td>363 (4.6)</td>
<td>359 (4.6)</td>
</tr>
<tr>
<td>Another ICU within hospital</td>
<td>46 (0.6)</td>
<td>57 (0.7)</td>
</tr>
<tr>
<td>Diagnosis on ICU admission — no. (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Septic or septic shock</td>
<td>1567 (14.9)</td>
<td>1669 (14.9)</td>
</tr>
<tr>
<td>Traumatic brain injury</td>
<td>499 (8.8)</td>
<td>665 (8.5)</td>
</tr>
<tr>
<td>Mechanical ventilation — no. (%)</td>
<td>2723 (34.3)</td>
<td>2723 (34.7)</td>
</tr>
<tr>
<td>Vasopressor — no. (%)</td>
<td>2194 (26.4)</td>
<td>2198 (26.2)</td>
</tr>
<tr>
<td>Mean predicted risk of in-hospital death — % (95% CI)</td>
<td>6.4 (9.0-9.5)</td>
<td>9.6 (9.2-10.0)</td>
</tr>
<tr>
<td>Baseline creatinine level — mg/dl</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>0.19</td>
<td>0.89</td>
</tr>
<tr>
<td>Interquartile range</td>
<td>0.74-1.10</td>
<td>0.74-1.30</td>
</tr>
</tbody>
</table>

**Figure 1. Volume of Intravenous Isotonic Crystalloid Administered According to Group.**
Figure 2. Plasma Chloride and Bicarbonate Concentration According to Group.

Table 2. Clinical Outcomes.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Balanced Crystalloids (N=7942)</th>
<th>Saline (N=7860)</th>
<th>Adjusted Odds Ratio (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary outcome</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major adverse kidney event within 30 days — no. (%)</td>
<td>1139 (14.3)</td>
<td>1211 (15.4)</td>
<td>0.90 (0.82 to 0.99)</td>
<td>0.04</td>
</tr>
<tr>
<td><strong>Components of primary outcome</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In-hospital death before 30 days — no. (%)</td>
<td>818 (10.3)</td>
<td>875 (11.1)</td>
<td>0.90 (0.80 to 1.01)</td>
<td>0.06</td>
</tr>
<tr>
<td>Receipt of new renal-replacement therapy — no./total no. (%)</td>
<td>189/7358 (2.5)</td>
<td>220/7458 (2.9)</td>
<td>0.84 (0.68 to 1.02)</td>
<td>0.08</td>
</tr>
<tr>
<td>Among survivors</td>
<td>106/6787 (1.6)</td>
<td>117/6657 (1.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Final creatinine level ≥200% of baseline — no./total no. (%)</td>
<td>487/7558 (6.4)</td>
<td>494/7458 (6.6)</td>
<td>0.96 (0.84 to 1.11)</td>
<td>0.60</td>
</tr>
<tr>
<td>Among survivors</td>
<td>259/6787 (3.8)</td>
<td>273/6657 (4.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Among survivors without new renal-replacement therapy</td>
<td>215/6681 (3.2)</td>
<td>219/6540 (3.3)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Secondary outcomes

**In-hospital death — no. (%)**

<table>
<thead>
<tr>
<th></th>
<th>Before ICU discharge</th>
<th>Before 60 days</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>528 (6.6)</td>
<td>572 (7.3)</td>
</tr>
<tr>
<td></td>
<td>928 (11.7)</td>
<td>975 (12.4)</td>
</tr>
</tbody>
</table>

**ICU-free days¶**

<table>
<thead>
<tr>
<th></th>
<th>Median</th>
<th>Interquartile range</th>
<th>Mean</th>
<th>Mean ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25.3</td>
<td>22.1 to 26.6</td>
<td>21.8 ± 8.3</td>
<td>21.7 ± 8.6</td>
</tr>
</tbody>
</table>

**Ventilator-free days¶**

<table>
<thead>
<tr>
<th></th>
<th>Median</th>
<th>Interquartile range</th>
<th>Mean</th>
<th>Mean ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>28.0</td>
<td>26.0 to 28.0</td>
<td>24.2 ± 8.6</td>
<td>23.9 ± 8.9</td>
</tr>
</tbody>
</table>

**Vasopressor-free days¶**

<table>
<thead>
<tr>
<th></th>
<th>Median</th>
<th>Interquartile range</th>
<th>Mean</th>
<th>Mean ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>28.0</td>
<td>27.0 to 28.0</td>
<td>24.7 ± 8.3</td>
<td>24.4 ± 8.8</td>
</tr>
</tbody>
</table>

**Renal-replacement therapy-free days¶**

<table>
<thead>
<tr>
<th></th>
<th>Median</th>
<th>Interquartile range</th>
<th>Mean</th>
<th>Mean ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>28.0</td>
<td>28.0</td>
<td>25.0 ± 8.6</td>
<td>24.8 ± 8.9</td>
</tr>
</tbody>
</table>

### Secondary renal outcomes¶

**Stage 2 or higher AKI developing after enrollment — no./total no. (%)**

<table>
<thead>
<tr>
<th></th>
<th>807/7558 (10.7)</th>
<th>858/7458 (11.5)</th>
</tr>
</thead>
</table>

**Creatinine — mg/dl**

<table>
<thead>
<tr>
<th></th>
<th>Median</th>
<th>Interquartile range</th>
<th>Change from baseline to highest value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest before discharge or day 30</td>
<td>0.99</td>
<td>0.78 to 1.53</td>
<td>0.04 to 0.31</td>
</tr>
<tr>
<td>Change from baseline to highest value</td>
<td>0.98</td>
<td>0.78 to 1.53</td>
<td>-0.08 to 0.31</td>
</tr>
</tbody>
</table>
Results

- Overall study showed that there was a small but significant improvement in outcomes with BSS
- Primary outcome: 14.3 vs 15.4% (p<0.04)
- In hospital mortality: 10.3% vs 11.1% (p<0.06)
- These benefits was more prominent in:
  - Sepsis
  - Higher volumes of fluid
Why?

Hyperchloremia can cause:

• Renal vasoconstriction
• Causes metabolic acidosis
• Hyperkalemia via acidosis
• ?Impairment of coagulation
• Proinflammatory state

What we do and don’t understand about Hemostatic Resuscitation

• Trauma Induced coagulopathy is real
• It is present on ADMISSION in 25-30% of severely injured patients
• Although the data suggests a benefit to higher ratios, the data is limited though getting better
• Most met-analysis show a benefit
• There are two prospective studies showing benefit
• The best FFP: RBC ratio is probably somewhere between 1:1 and 1:2.
What we do and don’t understand about Hemostatic Resuscitation

- Are people dying because they are bleeding or, are they bleeding because they are dying?
- Is there a best ratio?
- Are high ratios favored because only the patients that survive have the opportunity to get more FFP?
- Is 1:1 worth shooting for because most of us never get there, but close is probably good enough.

Trauma Induced Coagulopathy

- Retrospective study of 1088 traumatized patients
- Reviewed PT/PTT/Thrombin Time on admission
- 24.4% of pts had evidence of TIC on admission
- Increased ISS correlated with incidence of TIC
- Mortality was higher in the TIC group (46 vs 11%)
Goals of Hemostatic Resuscitation
1. Treat TIC if present
2. Avoid causing TIC – cold, acidosis, hypotension and crystalloid overload
   “It is better to stay out of trouble then to get out of trouble”

What is the optimal ratio of FFP:RBC?

The Ratio of Blood Products Transfused Affects Mortality in Patients Receiving Massive Transfusions at a Combat Support Hospital
Matthew A. Borgman, MD, Philip C. Spinella, MD, Jeremy G. Perkins, MD, Kurt W. Grafwehl, MD, Thomas Repine, MD, Alec C. Beeckley, MD, James Sebesta, MD, Donald Jenkis, MD, Charles E. Wade, PhD, and John B. Holcomb, MD

• Retrospective study looking at combat casualties receiving >10U PRBC in 24 hrs (2003-2005)
• 246 pts included
• Divided FFP:RBC ratios into 3 groups
  • Low (0:22 – 1:4)
  • Medium (1:3.9 – 1:2.1)
  • High (1:2 – 1:0.6)
### Fluids data - 1994

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>IMMEDIATE RESUSCITATION (N = 309)</th>
<th>DELAYED RESUSCITATION (N = 289)</th>
<th>P VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before arrival at the hospital</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ringer’s acetate (ml)</td>
<td>870±667</td>
<td>92±309</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Trauma center</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ringer’s acetate (ml)</td>
<td>1608±1201</td>
<td>283±722</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Packed red cells (ml)</td>
<td>133±393</td>
<td>11±88</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Operating room†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ringer’s acetate (ml)</td>
<td>6772±4688</td>
<td>6529±4863</td>
<td>0.31</td>
</tr>
<tr>
<td>Packed red cells (ml)</td>
<td>1942±2322</td>
<td>1713±2313</td>
<td>0.07</td>
</tr>
<tr>
<td>Fresh-frozen plasma or platelet packs (ml)</td>
<td>357±1002</td>
<td>307±704</td>
<td>0.45</td>
</tr>
<tr>
<td>Autologous-transfusion volume (ml)</td>
<td>95±486</td>
<td>111±690</td>
<td>0.76</td>
</tr>
<tr>
<td>Hetastarch (ml)</td>
<td>499±717</td>
<td>542±696</td>
<td>0.41</td>
</tr>
<tr>
<td>Rate of intraoperative fluid administration (ml/min)</td>
<td>117±126</td>
<td>91±88</td>
<td>0.008</td>
</tr>
</tbody>
</table>

FFP:RBC = 1:5 to 1:6 (VERY LOW)

---

**The Ratio of Blood Products Transfused Affects Mortality in Patients Receiving Massive Transfusions at a Combat Support Hospital**

Matthew A. Borgenst, MD, Philip C. Spintila, MD, Jeremy G. Perkins, MD, Kurt W. Grathwohl, MD, Thomas Regine, MD, Alex C. Beekley, MD, James Sebesta, MD, Donald Jenkins, MD, Charles E. Wade, PhD, and John B. Holcomb, MD

*J Trauma, 2007;63:805–813.*

- Retrospective study looking at combat casualties receiving >10U PRBC in 24 hrs (2003-2005)
- 246 pts included
- Divided FFP:RBC ratios into 3 groups
  - Low (0:22 – 1:4)
  - Medium (1:3.9 – 1:2.1)
  - High ( 1:2 – 1:0.6)
- Groups were well matched
  - MOI, ISS, SBP, Acidosis - NS
The Ratio of Blood Products Transfused Affects Mortality in Patients Receiving Massive Transfusions at a Combat Support Hospital

Matthew A. Borgman, MD, Philip C. Spinella, MD, Jeryn G. Perkins, MD, Kurt W. Graithwohl, MD, Thomas Repine, MD, Alec C. Beeckley, MD, James Sebesta, MD, Donald Jenkins, MD, Charles E. Wade, PhD, and John B. Holcomb, MD


The ratio of blood products transfused affects mortality in patients receiving massive transfusions at a combat support hospital. The bar chart shows the mortality rates for different plasma:RBC ratio groups:

- Low: 65% (p<0.001)
- Medium: 34%
- High: 19%

The table below provides the cause of death and time to death for each group:

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>Low</th>
<th>Medium</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemorrhage %</td>
<td>92.5*</td>
<td>78*</td>
<td>37*</td>
</tr>
<tr>
<td>Intraop %</td>
<td>5</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>Hypotension %</td>
<td>0</td>
<td>11</td>
<td>13</td>
</tr>
<tr>
<td>ARDS %</td>
<td>0</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Refractory C</td>
<td>2.5</td>
<td>0</td>
<td>23</td>
</tr>
<tr>
<td>Time to death (hrs)*</td>
<td>2 (1–4)^*</td>
<td>4 (2–16)^*</td>
<td>38 (4–135)^*</td>
</tr>
</tbody>
</table>

*Significant differences

Note: The table and bar chart provide a comprehensive view of the mortality rates and causes of death for patients receiving massive transfusions at a combat support hospital, showing a significant reduction in mortality with higher plasma:RBC ratio groups.
The Prospective, Observational, Multicenter, Major Trauma Transfusion (PROMMTT) Study

Comparative Effectiveness of a Time-Varying Treatment With Competing Risks

John R. Holcomb, MD; Deborah J. del Junco, PhD; Erin E. Fox, PhD; Charles E. Wade, PhD; Mitchell J. Cohen, MD; Martin A. Schreiber, MD; Louis H. Marcom, MD; Ya Bai, MD; PhD; Karen J. Bresel, MD, MPH; Eileen M. Bulger, MD; Bryan A. Cotton, MD, MPH; Nena Matijevic, PhD; Peter Mutsch, MD; John G. Myers, MD; Herb A. Phelan, MD, MScS; Christopher E. White, MD; Juijie Zhang, PhD; Mohammad H. Rahbar, PhD; for the PROMMTT Study Group


- Looked at the ratios & timing of transfusion and effect on death
- Prospective trial
- 10 trauma centers, 905 enrolled
- Only included pts who received >3 units and survived >30min
- Looked at mortality at 19 intervals including 6, 12, 24hrs and 30d
- 25% mortality in cohort
- Divided cohort into 3 groups

The Prospective, Observational, Multicenter, Major Trauma Transfusion (PROMMTT) Study

Comparative Effectiveness of a Time-Varying Treatment With Competing Risks


- Divided cohort into 3 groups
  - Low FFP < 1:2 (FFP:RBC)
  - Moderate ≥ 1:2 to < 1:1
  - High FFP ≥ 1:1
9/11/2018

The Prospective, Observational, Multicenter, Major Trauma Transfusion (PROMMTT) Study

Comparative Effectiveness of a Time-Varying Treatment With Competing Risks

- 94% of hemorrhagic deaths occurred w/in 24hrs
- 60% of those occurred within 3hrs
- FFP:RBC and Plts:RBC ratios varied within initial 24hrs

---

The Prospective, Observational, Multicenter, Major Trauma Transfusion (PROMMTT) Study

Comparative Effectiveness of a Time-Varying Treatment With Competing Risks

Table 3. Multivariable Cox Regression Models Examining the Association of Plasma and Platelet Transfusion Ratios With In-hospital Mortality

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Categorical Transfusion Ratio Variables</th>
<th>Continuous Transfusion Ratio Variables</th>
<th>Low, &lt;1:2</th>
<th>Medium, 1:2-&lt;1:1</th>
<th>High, ≥1:1</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR (95% CI)</td>
<td>P Value</td>
<td>HR</td>
<td>P Value</td>
<td>HR</td>
</tr>
<tr>
<td>Early initial and time-varying plasma:RBC ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early initial and time-varying platelet:RBC ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-h cumulative plasma:RBC ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-h cumulative platelet:RBC ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24-h cumulative plasma:RBC ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24-h cumulative platelet:RBC ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

AVG RBC – 6U (4-11)
The Prospective, Observational, Multicenter, Major Trauma Transfusion (PROMMTT) Study

Comparative Effectiveness of a Time-Varying Treatment With Competing Risks

Data suggests:
• Ratios lower than 1:2 are 3-4X more likely to be associated with death than 1:1
• The improved mortality with higher FFP and Plt ratios are concentrated in the first 6 hrs.
• Mortality at 24 hrs and day 30 was unaffected by ratios
Hemostatic Resuscitation During Surgery Improves Survival in Patients With Traumatic-Induced Coagulopathy

Juan C. Duchesne, MD, Tareq M. Islam, MD, MPH, Lance Stuke, MD, MPH, Jeremy R. Timmer, MD, James M. Barbeau, MD, JD, Alan B. Marr, MD, John P. Hunt, MD, MPH, Jeffrey D. Dellavolpe, MD, Georgia Wahl, MD, NREMT-P, Patrick Greiffenstein, MD, Glen E. Steeh, MD, Clifton McGinnes, MD, Christopher C. Baker, MD, and Norman E. McSwain, Jr., MD

(J Trauma. 2009;67: 33–39)

<table>
<thead>
<tr>
<th>Variables</th>
<th>( p )</th>
<th>PR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ratio</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1:4</td>
<td>0.03*</td>
<td>1.89 (1.09–3.25)</td>
</tr>
<tr>
<td>1:3</td>
<td>0.46</td>
<td>1.61 (0.83–3.25)</td>
</tr>
<tr>
<td>1:2</td>
<td>0.61</td>
<td>1.52 (0.78–2.99)</td>
</tr>
<tr>
<td>Age</td>
<td>0.55</td>
<td>1.00 (0.58–1.82)</td>
</tr>
<tr>
<td>Gender</td>
<td>0.99</td>
<td>2.46 (0.87–6.98)</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>0.11</td>
<td>0.99 (0.08–1.00)</td>
</tr>
<tr>
<td>ISS</td>
<td>0.77</td>
<td>1.00 (0.56–1.82)</td>
</tr>
<tr>
<td>Penetrating vs. blunt trauma</td>
<td>0.12</td>
<td>0.71 (0.46–1.10)</td>
</tr>
</tbody>
</table>

* Significant \( p \) value.

---

Hemostatic Resuscitation During Surgery Improves Survival in Patients With Traumatic-Induced Coagulopathy

Juan C. Duchesne, MD, Tareq M. Islam, MD, MPH, Lance Stuke, MD, MPH, Jeremy R. Timmer, MD, James M. Barbeau, MD, JD, Alan B. Marr, MD, John P. Hunt, MD, MPH, Jeffrey D. Dellavolpe, MD, Georgia Wahl, MD, NREMT-P, Patrick Greiffenstein, MD, Glen E. Steeh, MD, Clifton McGinnes, MD, Christopher C. Baker, MD, and Norman E. McSwain, Jr., MD

(J Trauma. 2009;67: 33–39)

<table>
<thead>
<tr>
<th>Variables</th>
<th>( p )</th>
<th>PR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ratio</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1:4</td>
<td>&lt;0.01*</td>
<td>4.17 (1.48–11.7)</td>
</tr>
<tr>
<td>1:3</td>
<td>0.03*</td>
<td>3.76 (1.18–11.9)</td>
</tr>
<tr>
<td>1:2</td>
<td>0.15</td>
<td>2.47 (0.72–8.54)</td>
</tr>
<tr>
<td>Age</td>
<td>0.74</td>
<td>1.00 (0.87–1.02)</td>
</tr>
<tr>
<td>Gender</td>
<td>0.30</td>
<td>2.00 (0.54–7.49)</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>0.25</td>
<td>0.99 (0.98–1.01)</td>
</tr>
<tr>
<td>ISS</td>
<td>0.60</td>
<td>0.99 (0.05–1.02)</td>
</tr>
<tr>
<td>Penetrating vs. blunt trauma</td>
<td>0.68</td>
<td>0.87 (0.43–1.73)</td>
</tr>
</tbody>
</table>

* Significant \( p \) value.
What’s the right ratio, 1:1 or 1:2?
(or does it matter?)

Transfusion of Plasma, Platelets, and Red Blood Cells in a 1:1 vs a 1:1:2 Ratio and Mortality in Patients With Severe Trauma
The PROPPR Randomized Clinical Trial  Holcomb et al JAMA 2015

- Randomized, prospective, multisite
- Inclusion: required at least 1U PRBC prior to or within 1 hr of admission and predicted to need MTP
- 680 pts predicted to require massive transfusion
- Excluded moribund or opt’d out pts
- Groups were well matched
- Mean # units PRBC transfused – 9(5-15)

<table>
<thead>
<tr>
<th>Table 2. Trial Outcomes by Treatment Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>1:1:1 Group (n = 343)</td>
</tr>
<tr>
<td>-----------------------</td>
</tr>
<tr>
<td>24-h Mortality, No. (%) 43 (12.7)</td>
</tr>
<tr>
<td>30-d Mortality, No. (%) 75 (22.4)</td>
</tr>
<tr>
<td>Achieved hemostasis</td>
</tr>
<tr>
<td>Acute, median (IQR), min†</td>
</tr>
<tr>
<td>Hospital† free days, median (IQR)‡</td>
</tr>
<tr>
<td>Ventilator† free days§</td>
</tr>
<tr>
<td>Total No. of patients</td>
</tr>
<tr>
<td>Median (IQR)†</td>
</tr>
<tr>
<td>ICU† free days§</td>
</tr>
<tr>
<td>Total No. of patients</td>
</tr>
<tr>
<td>Median (IQR)†</td>
</tr>
<tr>
<td>Incidence of primary surgical procedure</td>
</tr>
<tr>
<td>Disposition at 30 d, No. (%)†</td>
</tr>
<tr>
<td>Home</td>
</tr>
<tr>
<td>Remaining hospitalized</td>
</tr>
<tr>
<td>Other§</td>
</tr>
<tr>
<td>Merges</td>
</tr>
<tr>
<td>Unknown</td>
</tr>
<tr>
<td>Glasgow Outcome Scale—Extended score</td>
</tr>
<tr>
<td>Total No. of patients§</td>
</tr>
<tr>
<td>Median (IQR)†</td>
</tr>
</tbody>
</table>
Table 3. Adjudicated Cause of Death by Treatment Group and Period From Randomization

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>No. (%)</th>
<th>1:1 Group (n = 338)</th>
<th>1:1-2 Group (n = 342)</th>
<th>Difference (95% CI), %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total No. of deaths</td>
<td>43</td>
<td>58</td>
<td>75</td>
<td>89</td>
</tr>
<tr>
<td>Exsanguination</td>
<td>7 (1.7)</td>
<td>5 (1.5)</td>
<td>12 (3.1)</td>
<td>10 (2.9)</td>
</tr>
<tr>
<td>Traumatic brain injury</td>
<td>12 (3.6)</td>
<td>17 (5.0)</td>
<td>4 (2.0)</td>
<td>9 (2.6)</td>
</tr>
<tr>
<td>Respiratory, pulmonary contusion, or tension pneumothorax</td>
<td>18 (5.4)</td>
<td>20 (6.0)</td>
<td>2 (1.0)</td>
<td>3 (0.8)</td>
</tr>
<tr>
<td>Septic</td>
<td>3 (0.9)</td>
<td>5 (1.5)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Multiple organ failure</td>
<td>0</td>
<td>0</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Type of cardiovascular event</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>1 (0.3)</td>
<td>1 (0.3)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Transfusion-related fatality</td>
<td>0</td>
<td>0</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
</tbody>
</table>

What about TXA?

THE LANCET Vol 376 July 3, 2010

Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial
Crash 2

- Randomized placebo controlled
- 274 hospitals in 40 countries
- Adults with or at risk for significant bleeding
- Given within 8 hours of injury
- 20,211 pts enrolled into the 2 groups

Crash 2 - results

- 28 day mortality reduced – 14.5 vs 16 (p=0.0035)
- If given >3hrs after injury – mortality increased
- No increase in thrombotic complications
- 60% of pts in 3rd world countries
- Are we comparing apples to oranges?
Protective Effects of Fresh Frozen Plasma on Vascular Endothelial Permeability, Coagulation, and Resuscitation After Hemorrhagic Shock Are Time Dependent and Diminish Between Days 0 and 5 After Thaw

Shibani Pati, MD, PhD, Nena Matijevic, PhD, Marie-Françoise Doursout, PhD, Tien Ko, MD, Yanna Cao, MD, Xiyun Deng, PhD, Rosemary A. Kozar, MD, PhD, Elizabeth Hartwell, MD, Jodie Conyers, PhD, and John B. Holcomb, MD
Summary

• Too much fluid is bad
• How to assess volume status still unclear
• BSS appear to have a small but real benefit over NS especially in certain clinical scenarios
• Blood is the best resuscitation fluid when indicated
• FFP should be given in a 1:1 ratio with blood
ECMO in the Setting of Cardiac Arrest

A. Reshad Garan, MD, Associate Director of the Cardiac Intensive Care Units and Medical Director of the Acute Circulatory Support Program at the Columbia University Medical Center; Assistant Professor of Medicine at Columbia University Medical Center
ECMO in the Setting of Cardiac Arrest

Arthur Reshad Garan, MD
Assistant Professor of Medicine
Associate Director, Cardiac Intensive Care Unit
Medical Director, Acute Circulatory Support Program
Columbia University Medical Center, New York, NY, USA

Disclosures

Supported by National Institute of Health Grant No. KL2TR001874

Abiomed, Honoraria (past); Advisory Board (unpaid, current)
Overview

- ECMO Overview
- Whom to support - Patient Selection
- When to Support
- Additional Considerations
ECMO Overview

Circuit = inflow cannula + centrifugal pump + membrane oxygenator + heat exchanger + outflow cannula

Central or peripheral
Multiple configurations
Flows dependent on cannula size

ECMO – Pros

- Supra-normal flows
- Easily deployed
- Biventricular support
- Gas exchange
1. The patient was generally healthy prior to the arrest. This requires an attempt at a global assessment of the patient’s pre-arrest condition and is a challenging concept that requires an adept emergency physician with good clinical judgment.

2. Overall goals of therapy are curative (as opposed to palliative).

3. The event that caused the arrest is thought to be reversible with a specific medical or surgical intervention.

**Premise of ECPR**

ECPR is NOT a cure – it is a means to maintain circulation while the underlying cause of the cardiac arrest is treated.

Bellezzo, J., et al. Indications and Contraindications of ECLS. edemo.org

---

**Outcomes**

![Graphs showing outcomes of ECPR and conventional therapy.](image-url)
Outcomes

But...

1. Adult OHCA: Survival to hospital discharge (one month)
   - Study: Apstein et al., 2011
     - Odds Ratio: 1.29 (95% CI: 1.04, 1.60)
   - Study: Kim et al., 2014
     - Odds Ratio: 0.76 (95% CI: 0.28, 2.10)
   - Study: Lee et al., 2015
     - Odds Ratio: 0.75 (95% CI: 0.34, 1.68)

2. Adult OHCA: Long-term survival
   - Study: Cesario et al., 2017
     - Odds Ratio: 0.18 (95% CI: 0.08, 0.39)
   - Study: Kim et al., 2016
     - Odds Ratio: 2.18 (95% CI: 0.81, 7.75)
   - Study: Mantey et al., 2013
     - Odds Ratio: 6.49 (95% CI: 1.29, 34.96)
   - Study: Salamone et al., 2014
     - Odds Ratio: 1.90 (95% CI: 0.89, 3.89)
   - Study: Tanaka et al., 2016
     - Odds Ratio: 2.96 (95% CI: 1.5, 5.29)
Overview

- ECMO Overview
- Whom to support - Patient Selection
- When to Support
- Additional Considerations

Patient Selection
Patient Selection: Age

If no durable destination exists for the ECPR patient (recovery, transplant, implantable LVAD, or transplant) the patient is effectively left with a bridge to nowhere.

Destination – Bridge to Nowhere
A Typical Call

Overview

- ECMO Overview
- Whom to support - Patient Selection
- When to Support
- Additional Considerations
Timing of ECPR Implementation

Overview

- ECMO Overview
- Whom to support - Patient Selection
- When to Support
- Additional Considerations
Neurologic Outcomes

Neurologic outcomes after extracorporeal membrane oxygenation assisted CPR for resuscitation of out-of-hospital cardiac arrest patients: A systematic review

Michael M. Beyea\textsuperscript{1,2,5,}, Bourke W. Tillmann\textsuperscript{5}, Alla E. Iansavichene\textsuperscript{5}, Varinder K. Randhawa\textsuperscript{5}, Kristine Van Aarsen\textsuperscript{5}, A. Dave Nagpal\textsuperscript{5}

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Events</th>
<th>Total</th>
<th>Total</th>
<th>Weight</th>
<th>Odd Ratio</th>
<th>M-H Random 95% CI</th>
<th>Odd Ratio</th>
<th>M-H Random 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Choi et al 2015</td>
<td>29</td>
<td>320</td>
<td>19</td>
<td>320</td>
<td>35.0%</td>
<td>1.00 [0.97, 1.03]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kim et al 2014</td>
<td>8</td>
<td>52</td>
<td>1</td>
<td>62</td>
<td>12.7%</td>
<td>0.92 [0.62, 1.37]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mani et al 2013</td>
<td>7</td>
<td>24</td>
<td>2</td>
<td>26</td>
<td>18.4%</td>
<td>4.02 [0.91, 17.5]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sakamoto et al 2014</td>
<td>32</td>
<td>260</td>
<td>3</td>
<td>194</td>
<td>22.1%</td>
<td>0.94 [0.69, 1.29]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Siao et al 2015</td>
<td>6</td>
<td>20</td>
<td>3</td>
<td>23</td>
<td>19.7%</td>
<td>8.22 [1.88, 35.0]</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Heterogeneity</strong>: Tau(^2) = 0.70; Chi(^2) = 10.85, df = 4 (P = 0.03); P = 0.03% Test for overall effect: Z = 3.18 (P = 0.001)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Conclusion: Although a trend towards improved survival with good neurologic outcome was reported in controlled, low-risk of bias cohort studies, a preponderance of low-quality evidence may ascribe an optimistic effect size of ECPR on survival among OHCA patients. Our confidence in a clinically relevant difference in outcomes compared to current standards of care for OHCA remains weak. In this state of equipoise, high-quality RCT data is urgently needed.

LV Venting
When to Stop?

- Hemorrhage
- Infection
- Hemolysis
- Limb Ischemia

Complications of (any) Mechanical Support
Management on Device: Constant Evaluation

- Frequent assessment of:
  - Adequacy of end-organ perfusion
  - Satisfactory cardiac unloading
    - Aortic valve opening?
  - Potential complications
    - Bleeding, limb ischemia, embolic events, hemolysis
  - Need for ongoing support: can MCS be weaned? Is long term heart replacement an option?

Shock Team

- Referring Cardiologist
- Interventional Cardiologist
- Heart Failure Cardiologist
- Severe Refractory Cardiogenic Shock Patient
- CT Surgeon
- Cardiac Intensivist

- 24 x7 availability
- Match device to patient needs
- Care from admission to ultimate destination
TEAM – BASED care does not end after the initial management decision!

Future Direction

- Upcoming Studies

Conclusions

- ECPR offers improved chance at survival for carefully selected patients

- ECPR is a not-curative but only offers a means of facilitating treatment of the underlying etiology of cardiac arrest

- Creation of a multi-disciplinary team which can be rapidly mobilized to deploy ECMO but also care for the patient after cannulation is essential to optimizing outcomes
Return of Spontaneous Circulation- Care in the CCU

Simon Gorwarra, MD, FACC, President of The Heart Center, Chief of Cardiology, Vassar Brothers Medical Center
RETURN OF SPONTANEOUS CIRCULATION – CARE IN THE CCU

Simon Gorwara MD FACC
President, The Heart Center
Chief of Cardiology, Vassar Brothers Medical Center

RETURN OF SPONTANEOUS CIRCULATION – CARE IN THE CCU HOSPITAL

Simon Gorwara MD FACC
President, The Heart Center
Chief of Cardiology, Vassar Brothers Medical Center
RETURN OF THE JEDI – BY FAR THE GREATEST STAR WARS MOVIE

Simon Gorwara MD FACC
President, The Heart Center
Chief of Cardiology, Vassar Brothers Medical Center
Jedi Master

TEAM APPROACH - COORDINATED CARE OF THE CARDIAC ARREST PATIENT WITH ROSC

• EMS Personnel
• ER Staff
• Respiratory Therapy
• Lab
• Cath Lab
• Intensive care unit team
HARSH REALITY

• Despite our best efforts, prognosis for meaningful survival remains poor in survivors of cardiac arrest
• However, with timely and appropriate interventions and coordination of care this is an area where we can potentially make a huge difference

THE GOOD NEWS

• Recent advances in resuscitation therapy for cardiac arrest victims have led to improved survival and neurologic outcomes
• Emergent coronary revascularization in appropriate patients combined with therapeutic hypothermia and hemodynamic support can lead to improved outcomes
• There is a high incidence of coronary artery disease in survivors of cardiac arrest
WHAT IS THE ROLE FOR CARDIAC CATHETERIZATION?

- 2013 ACC AHA guidelines have a class I recommendation for immediate angiography and PCI in comatose patients after cardiac arrest with ST elevation myocardial infarction
- There are no ACC AHA guidelines for comatose cardiac arrest patients without ST segment elevation on EKG
- Mortality in post cardiac arrest patients with ST elevation myocardial infarction who are awake and undergo successful PCI is only 5%
- The mortality increases to 50% if patients are comatose on presentation

WHAT ABOUT HYPOTHERMIA?  
(TARGETED TEMPERATURE MANAGEMENT)

- Early initiation of targeted temperature management is critical in comatose patients post cardiac arrest
- This can limit tissue injury after ischemia reperfusion conditions occurring post cardiac arrest
- Mild therapeutic hypothermia can potentially improve survival and neurologic outcomes when combined with PCI in patients status post out of hospital cardiac arrest
• Post arrest patients with ST elevation MI who are comatose upon arrival and receive Targeted Temperature Management were found in one study to have a hospital discharge rate of 60% with 86% of these survivors being neurologically intact.

• Excess bleeding was seen in earlier studies of extreme hypothermia less than 28°C. This has not been seen with targeted temperature management recommended in cardiac arrest 32°C to 36°C.

12 LEAD EKG

• Ideally done in the field and transmitted to ER prior to patient arrival. This allows early activation of the cath lab team.

• If STEMI – Emergent cath is a Class I recommendation and the standard of care - unless there are multiple unfavorable risk features.

• If no STEMI – Culprit vessel is still found in about 33% of patients without STEMI.
EKG – LOCATION OF MI

Diagram showing the locations of MI on EKG:
- High Lateral
- Septal
- Anterior
- Inferior
- Lateral

Specific leads includes:
- aVR
- V1
- V4
- V2
- V5
- V3
- V6
- aVL
- aVF

Diagram showing an EKG with waveforms indicating possible MI locations.
AMI in the presence of LBBB

Figure 1. Electrocardiogram Meeting All Three Independent Criteria for the Diagnosis of Acute Myocardial Infarction in a Patient from the GUSTO Trial with Left Bundle-Branch Block.

The electrocardiogram shows ST-segment elevation of at least 1 mm that is concordant with theQRS complex in leads II, ST-segment depression of at least 1 mm in leads V_1 and V_6, and ST-segment elevation of at least 5 mm that is discordant with the QRS complex (leads III and aVF).
GIVEN THE HIGH INCIDENCE OF CORONARY DISEASE IN SURVIVORS OF CARDIAC ARREST SHOULD ALL PATIENTS WITH ROSC GO TO THE CATH LAB?

• This approach is actually followed and advocated by a few academic centers
• There is however a lack of consensus about the best approach to patients without STEMI on EKG and the role of revascularization. This can lead to some confusion when the patient arrives in the ER.
• Public reporting of data is a reality. These patients have a very high mortality rate
• Many of these patients may have incidental coronary disease unrelated to the cardiac arrest, or no coronary disease at all. In these patients, subjecting them to an unnecessary procedure can be harmful.
• In patients with multiple unfavorable resuscitation features – even if a culprit vessel is found there is unlikely to be any meaningful benefit of cath/revascularization
• At Vassar we have therefore developed a Cardiac Arrest algorithm to help streamline decision making
WHAT ARE UNFAVORABLE RESUSCITATION FEATURES

- Unwitnessed arrest
- Initial rhythm non VF/VT
- No bystander CPR (16.1% versus 3.9% survival)
- > 30 min to ROSC
- Ongoing CPR
- Severe acidemia pH < 7.2 lactate > 7 mmol/l
- Age > 85
- ESRD

PROCAT II REGISTRY
PARISIAN REGISTRY OUT OF HOSPITAL CARDIAC ARREST

- Between 2004 and 2013 – all patients with out of hospital cardiac arrest were admitted directly to the cardiac cath lab for angiography in the absence of an obvious non-cardiac cause of arrest (resp failure, stroke, hemorrhage etc.)
- 695 patients underwent emergent coronary angiography (excluding patients with STEMI)
- Culprit lesion was identified and treated by PCI in 29%
- Favorable neurologic outcome noted in patients who underwent PCI compared with those who did not
PREDICTIVE FACTORS OF GOOD NEUROLOGIC OUTCOME – LESSONS FROM PROCAT II REGISTRY

### TABLE 2: Predictive Factors of Good Neurological Outcome At Discharge (Multivariable Analysis)

<table>
<thead>
<tr>
<th>Factor</th>
<th>OR</th>
<th>95% CI</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (for each additional year)</td>
<td>0.97</td>
<td>0.95-0.99</td>
<td>0.002</td>
</tr>
<tr>
<td>Male</td>
<td>1.18</td>
<td>0.68-2.06</td>
<td>0.55</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.64</td>
<td>0.89-3.0</td>
<td>0.11</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.03</td>
<td>0.62-1.69</td>
<td>0.92</td>
</tr>
<tr>
<td>Current smoker</td>
<td>1.21</td>
<td>0.75-1.94</td>
<td>0.46</td>
</tr>
<tr>
<td>Public location of CA</td>
<td>1.27</td>
<td>0.78-2.07</td>
<td>0.34</td>
</tr>
<tr>
<td>Witnessed CA</td>
<td>3.43</td>
<td>0.89-13.26</td>
<td>0.07</td>
</tr>
<tr>
<td>Bystander CPR</td>
<td>1.35</td>
<td>0.85-2.15</td>
<td>0.20</td>
</tr>
<tr>
<td>Initial shockable rhythm</td>
<td>3.40</td>
<td>1.95-5.91</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Resuscitation length &lt;20 min</td>
<td>3.15</td>
<td>1.94-5.10</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Epinephrine &gt;2 mg</td>
<td>0.27</td>
<td>0.16-0.46</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>TTM</td>
<td>0.93</td>
<td>0.41-2.07</td>
<td>0.85</td>
</tr>
<tr>
<td>Post-CA shock</td>
<td>0.58</td>
<td>0.36-0.92</td>
<td>0.02</td>
</tr>
<tr>
<td>Successful PCI</td>
<td>1.80</td>
<td>1.09-2.97</td>
<td>0.02</td>
</tr>
</tbody>
</table>

CI = confidence interval; OR = odds ratio; other abbreviations as in Tables 1 and 2.

WHICH PATIENTS ARE MORE LIKELY TO REQUIRE PCI?

### TABLE 3: Baseline and Demographic Characteristics According to PCI

<table>
<thead>
<tr>
<th>Factor</th>
<th>Successful PCI (n=154)</th>
<th>Re-Crated Lesion (n=106)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yrs</td>
<td>67.4±12.2</td>
<td>58.7±14.2</td>
<td>0.05</td>
</tr>
<tr>
<td>Race</td>
<td>104 (67.6)</td>
<td>113 (106)</td>
<td>0.01</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>66 (42.7)</td>
<td>83 (78.3)</td>
<td>0.06</td>
</tr>
<tr>
<td>Hypertension</td>
<td>84 (54.7)</td>
<td>110 (104)</td>
<td>0.22</td>
</tr>
<tr>
<td>Current smoker</td>
<td>54 (35.4)</td>
<td>114 (104)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Initial shockable rhythm</td>
<td>19 (12.2)</td>
<td>28 (27)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Witnessed CA</td>
<td>180 (118)</td>
<td>201 (196)</td>
<td>0.20</td>
</tr>
<tr>
<td>Resuscitation length ≥20 min</td>
<td>90 (58.4)</td>
<td>223 (213)</td>
<td>0.40</td>
</tr>
<tr>
<td>Bystander CPR</td>
<td>192 (127)</td>
<td>244 (235)</td>
<td>0.28</td>
</tr>
<tr>
<td>Epinephrine &gt;2 mg</td>
<td>57 (37)</td>
<td>220 (214)</td>
<td>0.007</td>
</tr>
<tr>
<td>Post-resuscitation shock</td>
<td>132 (87)</td>
<td>304 (293)</td>
<td>0.07</td>
</tr>
<tr>
<td>TTM</td>
<td>175 (118)</td>
<td>418 (402)</td>
<td>0.07</td>
</tr>
<tr>
<td>at least 2 significant lesions</td>
<td>108 (70.3)</td>
<td>206 (198)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Onset to ROSC (median, IQR)</td>
<td>90 (130)</td>
<td>706 (217)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PCI by 2.2-fold discharge</td>
<td>85 (15)</td>
<td>166 (30)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Values are mean±SD or n (%). These values were subjected to two-tailed tests or chi square tests, as appropriate. CI: confidence interval; other abbreviations as in Table 1.
Until 2015 we had no clear algorithm for managing cardiac arrest. Each patient was evaluated individually and each time we would make calls back and forth between different team members and services trying to decide the best treatment plan.

Based on the new data with the potential to improve outcomes as well as in order to streamline the management process and coordinate care amongst different service lines, in 2015/2016 we developed an algorithm for managing the post cardiac arrest ROSC patient.

Our algorithm is a modification of the guideline developed by the Interventional Council of the American College of Cardiology and published in JACC in 2015.
CASE EXAMPLE

• 56 year old male no cardiac history presents with chest pain
• Develops VF – shock x 1
CASE NUMBER 2

- 66 year old female with sudden onset chest pain. Called EMS. On arrival found to have VF – shocked x 1 in field. Developed asystole and received CPR and epinephrine with ROSC.
- On arrival awake, in distress, pale diaphoretic, severe chest pain
- BP 80/67
- Started on Norepinephrine in ER

EKG ON ARRIVAL
• Brought emergently to cardiac cath lab.
• Impella device placed (percutaneous left ventricular assist device)
• Angiography performed
• Pressors weaned off
• Next day Impella removed
• Following day discharged home
• Returned last Thursday for elective outpatient PCI of the RCA
• Enrolled in ECLIPSE trial (atherectomy versus conventional balloon angioplasty for calcific coronary disease) Randomized to conventional balloon angioplasty arm
POST CARDIAC ARREST – MANAGEMENT OF CARDIOGENIC SHOCK

UNDERSTANDING CARDIOGENIC SHOCK – ITS NOT SO SIMPLE AS WEAK HEART MUSCLE = LOW BLOOD PRESSURE = SHOCK
SHOCK TRIALS

- Before routine use of early revascularization in-hospital mortality > 80%
- Shock trial showed significant 6 and 12 month mortality reduction (Only randomized cardiogenic shock trial to show benefit of intervention)
- Successful PCI 35% mortality, Unsuccessful PCI 80% mortality.
- Similar mortality benefit for patients who underwent CABG
- Shock II – No benefit of routine IABP placement (Downgraded to IIB in US and Class III recommendation in Europe)
Pulsatile

IABP

Axial-Flow

Impella CP

Centrifugal Flow

PHP *

TandemHeart

VA-ECMO

Intracorporeal

Extracorporeal

* Investigational

**Percutaneous Mechanical Circulatory Support Versus Intra-Aortic Balloon Pump in Cardiogenic Shock After Acute Myocardial Infarction**

Dagnino M., Ormeñez, MD,*, Gillel Eliakim, MD,*, Ashkan Kianfar, MD, PhD,* Ioan W. van Engelen, MD,* Alexander Schreiber, MD, PhD,*, Rinse D. Baan, MD, PhD,* Martijn Wijsman, MD, PhD,* Ramos I. Wittersley, MD, PhD,* Hart T. Koel, MD, PhD,* John Bana, MD, PhD,* Robert J. de Wijer, MD, PhD,* Ioan W. van Engelen, MD, PhD,* Wim R. Lempi, MD, PhD,*, Sir A. Charles de Belder, MD, PhD,* Jan C. T. 't Hart, MD, PhD,* Jose P. E. Bertoglio, MD, PhD,*

**ABSTRACT**

**BACKGROUND** Despite advances in treatment, mortality in acute myocardial infarction (AMI) complicated by cardiogenic shock (CS) remains high. Short-term mechanical circulatory support devices acutely improve hemodynamic conditions.

**OBJECTIVES** The aim of this study was to determine whether a new percutaneous mechanical circulatory support system (pMCS) vs Impella CP (Abiomed, Danvers, Massachusetts) decreases 30-day mortality when compared with a conventional balloon pump (BPP) in patients with severe shock complicating AMI.

**METHODS** In a randomized, prospective, open-label, multicenter trial, 49 patients with severe CS complicating AMI were assigned to pMCS (n = 24) vs BPP (n = 25). Severe CS was defined as systolic blood pressure < 90 mm Hg or the need for inotropic or vasopressor medication and the requirement for mechanical ventilation. The primary endpoint was 30-day all-cause mortality.

**RESULTS** At 30 days, mortality in patients treated with either pMCS or BPP was similar (56% vs 52%, respectively), based on Wilcoxon rank sum test (p = 0.18). At 4.6 months, mortality rate for both pMCS and BPP were 39% [95% confidence interval (CI): 0.43 to 0.65] vs 37% [95% CI: 0.32 to 0.48]; the difference was not statistically significant.

**CONCLUSIONS** In this randomized controlled trial involving mechanically ventilated patients with CS after AMI, routine treatment with pMCS was not associated with reduced 30-day mortality compared with BPP. **(IMPROVE** in Severe Shock, NCT00443962) U. Am Coll Cardiol 2018;70:78-87. © 2017 by the American College of Cardiology Foundation.
SO IS IMPELLA NO BETTER THAN BALLOON PUMP?

cholamines before randomization. Although we did not aim to include resuscitated patients, the inclusion criteria resulted in 92% of enrolled patients having a cardiac arrest prior to randomization. In addition, almost one-half (48%) of the patients had time to ROSC longer than 20 min. Traumatic injuries due to cardiac arrest were frequently present (15%). These criteria identified a unique patient population that is usually excluded from randomized CS clinical trials.

Figure 3

Kaplan-Meier curve survival to 30 days. CS, cardiogenic shock; DBP, diastolic blood pressure; MAP, mean arterial pressure; MV, mechanical ventilation; NSTEMI, non-ST elevation myocardial infarction; PVD, peripheral vascular disease; SBP, systolic blood pressure.
WHAT ABOUT PATIENTS WITH REFRACTORY CARDIOGENIC SHOCK FROM RV INFARCT

- Since the Impella device only pump blood from the left ventricle to the aorta it is completely dependent on the preload coming from the right ventricle. If the right ventricle is failing then the left sided impella will not work.

- In those cases we may need to place a percutaneous right sided pump – called an RP Impella
RIGHT SIDED IMPELLA

Actual Vassar patient 8/2018

SURVIVAL IN AMI CARDIOGENIC SHOCK HAS REMAINED UNCHANGED FOR DECADES
DETOUR CARDIOGENIC SHOCK INITIATIVE

ALGORITHM

INCLUSION CRITERIA
- Acute Myocardial Infarction
- SUV and/or troponin evidence of AMI (STEMI or NSTE)
- Cardiogenic Shock
- Hypotension (<90/60) or the need for vasopressors or inotropes to maintain systolic blood pressure >90
- Evidence of end-organ hypoperfusion (cool extremities, oliguria, lactic acidosis)

EXCLUSION CRITERIA
- Evidence of Anoxic Brain Injury
- Unsuccessful out of hospital cardiac arrest or any cardiac arrest in which ROSC is not achieved in 30 minutes
- IABP placed prior to Impella
- Septic, sepsis, hypercoagulable, and nonischemic causes of shock
- Non-ischemic causes of shock (Pulmonary Embolism, Pericardititis, Aneurysms, Tamponade, etc.)
- Active Bleeding
- Recent major surgery
- Mechanical Complications of AMI
- Visible left main coronary occlusion
- Patient who did not receive revascularization
- Mechanical aortic valve

ACCESS & SUPPORT
- Obtain femoral arterial access (via direct visualization with use of ultrasound and fluoroscopy)
- Obtain venous access (Femoral or Internal IJ)
- Obtain either Fick calculated cardiac index or LVHPD

IF LVEDP >15 or Cardiac Index < 2.2 AND anatomy suitable, place IMPELLA

Coronary Angiography & PCI
- Attempt to provide TIMI III flow in all major epicardial vessels other than CTO
- If unable to obtain TIMI III flow, consider administration of intra-coronary vasodilators

Perform Post-PCI Hemodynamic Calculations
1. Cardiac Power Output (CPO): \( \frac{MAP \times CO}{451} \)
2. Pulmonary Artery Pulsatility Index (PAPI): \( sPAP - dPAP \)

Wean OFF Vasopressors and Inotropes
- If CPO is >6.0 and PAPI >0.9, operators should wean vasopressors and inotropes and determine if Impella can be weaned and removed in the Cath Lab or left in place with transfer to ICU

Escalation of Support
- If CPO remains <6.0 operators should consider the following options:
  - PAPI is >0.9 consider right sided hemodynamic support
  - PAPI <0.6 consider for additional hemodynamic support
- Local practice patterns should dictate the next steps:
  - Placement of more robust MCS device(s)
  - Transfer to LVAD/Transplant center

Vascular Assessment
- Prior to discharge from the Cath Lab, a detailed vascular exam should be performed including femoral angiogram and Doppler assessment of the affected limb.
- If indicated, external bypass should be performed.
Acute Myocardial infarction mortality remains high and is essentially unchanged despite major advances in Cardiac Care in past 20 years.

Acute Myocardial infarction complicated by cardiogenic shock (AMICS) is a deadly condition with an in-hospital survival rate of only 50%.*

Vassar Brothers Medical Center Cardiogenic/ Refractory Shock patients receiving PCI in 2016 and 2017

<table>
<thead>
<tr>
<th>Year</th>
<th>Total number of PCI cases</th>
<th># of AMI with Cardiogenic shock</th>
<th>Alive at discharge</th>
<th># of AMI with Refractory shock</th>
<th>Alive at discharge</th>
<th>Rate of AMICS survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016</td>
<td>873</td>
<td>20 (75%)</td>
<td></td>
<td></td>
<td></td>
<td>50%</td>
</tr>
<tr>
<td>2017</td>
<td>1036</td>
<td>17 (76%)</td>
<td></td>
<td></td>
<td></td>
<td>50%</td>
</tr>
</tbody>
</table>

Rate of AMICS survival: 50%

Note: Shock Trial NEJM 1999 – 62% survival with successful revascularization

Definition of Cardiogenic Shock: Hypotension (<90/60) or the need for vasopressors or inotropes to maintain systolic blood pressure >80.

Definition of Refractory Shock: Episode of systolic blood pressure <80 mmHg determined to be secondary to cardiac dysfunction despite the use of parenteral inotropic or vasopressor agents or mechanical support (e.g., Intra Aortic Balloon Pump, Impella)
SHOCK TEAM APPROACH

1. Interventional Cardiologist
2. Cardiac Surgeon
3. Critical Care / Intensivist (MD)
4. Advanced HF Specialist
5. Critical Care Nursing Team
6. Perfusion Team
7. Respiratory Specialists
8. Physical and Occupational Therapy
9. Palliative Care

Acute MI Cardiogenic Shock

Advanced HF Cardiogenic Shock

1. Advanced HF Specialist
2. Interventional Cardiologist
3. Cardiac Surgeon
4. Critical Care / Intensivist (MD)
5. Critical Care Nursing Team
6. Palliative Care
7. Perfusion Team
8. Respiratory Specialists
9. Physical and Occupational Therapy

VASSAR PERCENT OF PATIENTS RECEIVING PCI IN 90 MINUTES AND RISK ADJUST MORTALITY OUTCOMES IN PCI (A SHAMELESS PLUG)

PROPORTION OF STEMI PATIENTS RECEIVING IMMEDIATE PCI WITHIN 90 MINUTES

PCI IN-HOSPITAL RISK-ADJUSTED MORTALITY (EMERGENT AND NON-EMERGENT)

<table>
<thead>
<tr>
<th>PCI in-hospital risk adjusted mortality (STEMI patients excluded)</th>
</tr>
</thead>
<tbody>
<tr>
<td>My Hospital</td>
</tr>
<tr>
<td>--------------</td>
</tr>
<tr>
<td>0.37</td>
</tr>
</tbody>
</table>

Your hospital’s PCI in-hospital risk adjusted mortality rate for patients with other diagnoses (not STEMI) using the NCOR® risk adjustment model.

[Data Link:3063]
CONCLUSIONS

• Cardiac arrest remains a condition associated with a poor prognosis
• With early intervention, use of therapeutic hypothermia, cardiac catheterization and revascularization in appropriate candidates and use of mechanical circulatory support in cardiogenic shock these outcomes can be improved
• To do so however requires coordination of care and a team approach. Having pre-identified algorithms for managing patients with cardiac arrest and cardiogenic shock can be helpful in streamlining this process
• Without a doubt Return of the Jedi was the best in the Star Wars series

THANK YOU!
Discussion Panel:
Integrating the Links in the Chain of Survival- Out of Hospital Cardiac Arrest

A. Reshad Garan, MD
Simon Gowara, MD, FACC
Timothy Collins, DO, FCCP
tPA for Massive Pulmonary Embolus

Timothy Collins, DO, FACCPC, Director, Pulmonary/Critical Care/Sleep Medicine; Adult Director Sleep Lab, Adult Director, Cystic Fibrosis Program Vassar Brothers Medical Center, Health Quest Medical Practice, P.C.
Massive Pulmonary Embolism

Timothy P. Collins, DO, FCCP
Chief, Division of Pulmonary/Critical Care/Sleep Medicine
Vassar Brothers Medical Center

Goals and Objectives

- Presenting signs and symptoms of massive PE
- Diagnosis of PE
- Treatment options for massive PE
- Pulmonary Embolism Response Teams
- Cases from VBMC
Nomenclature

- Acute, subacute, chronic
- Provoked vs unprovoked
- Massive vs submassive
  - SBP 90 or lower (persistent) = massive
  - Submassive- low, intermediate, high risk

<table>
<thead>
<tr>
<th>Early Mortality Risk</th>
<th>Risk Parameters and Scores</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Shock or Hypotension</td>
</tr>
<tr>
<td>High</td>
<td>+</td>
</tr>
<tr>
<td>Intermediate</td>
<td>−</td>
</tr>
<tr>
<td>High</td>
<td>−</td>
</tr>
<tr>
<td>Low</td>
<td>−</td>
</tr>
</tbody>
</table>
Mortality

- Low risk PE <1%
- Intermediate PE 5-20%
- Massive PE 35-65%

Outcomes in Pulmonary Embolism

100% -------------------------------------

Cardiac Arrest • Mortality 10%
Sudden Death & hemorrhagic by intimal dysplasia? Hemodynamically Stable RV Normal

Fig. 1. Outcomes in PE. (From Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. Chest 2002;121:877-905; with permission.)
Risk Stratification

- Shock?
- Biomarker leakage?
  - Troponin-I, BNP
- RV dysfunction?
  - RV/LV ration > 0.9 on CTA
  - Echo findings- McConnell's sign
- Bleeding risk?
- Clot size, location?

Diagnosis

- CT angiogram
- Ventilation perfusion scan
- Echocardiogram
McConnell's sign is highly specific for massive PE.

**Treatment**

**Goals**

- Improve RV function
- Decrease PA pressure
- Lessen clot burden
- Normalize hemodynamics
- Minimize long term morbidity
Treatment Options

• Systemic thrombolysis (TPA 100mg)
• Surgical embolectomy
• Catheter directed therapy (EKOS, Penumbra, Flowtriever)
• Angiovac
• ECMO

Systemic TPA, when given for massive or submassive PE decreases the risk of post thrombotic syndrome as well as CTEPH.
EKOS

Figure 1: Infusion of a thrombolytic through a mesh-enabled catheter results in rapid delivery of the drug through non-shadowed pathways [A]. A multi-drug infusion catheter is embedded within an embolus in the left lower lobe, allowing thrombolytics to be infused directly into the clot [B].
A New Era for Massive and Submassive PE

Acute Pulmonary Embolism Trial Confirms Safety and Efficacy of Ultrasound Accelerated Endovascular Thrombolysis

150 pts (31 massive PE, 119 submassive PE)
Ultrasound-facilitated catheter-directed low-dose thrombolysis
24 mg tPA 1mg/h for 24 hours

RESULTS:
1 death attributed to PE
No deaths in massive PE within 30 days follow-up
No intracranial hemorrhages
Catheter Directed Therapy with EKOS system

- Safe
- Improves RV function in first 24 hrs compared to standard therapy
- Less vascular complications of DVT/PE beyond 30 days
- No mortality benefit proven as of yet
- Can be used as salvage or first line therapy in select cases of high risk submissive and massive PE.
Surgical embolectomy

ECMO
Standard of Care

- Systemic TPA or surgical embolectomy Grade IIa evidence

- Catheter directed therapy, suction thrombectomy, IVC filters, ECMO, Angiovac are not evidence based (Grade IIb, IIc or III), and some are off label.
Contraindications to Thrombolysis

- Absolute
  - History of hemorrhagic CVA
  - Ischemic CVA within 3 months
  - Cerebral AVM
  - Recent CNS surgery
  - Recent head trauma with fracture or TBI
  - Active bleeding (excluding menses)
  - Known bleeding diathesis

- Relative
  - Minor head trauma ie syncope
  - Major non-cns surgery within 3 weeks
  - Recent puncture of non compressible vessel
  - Recent internal bleeding (2-4 weeks)
  - Platelets less than 100
  - Oral anticoagulation therapy
  - Chronic severe and poorly controlled HTN
  - SBP greater than 180 on presentation
  - Age greater than 75
  - Post partum less than 1 week
Pulmonary Embolism Response Team (PERT)

- 2012 MGH, 1st PERT
- 2015 PERT Consortium
- 2018 >100 programs nationwide
  - VBMC instituted PERT 2017
  - Average 25 pulmonary embolism cases per month
  - 1-2 massive PEs per month
  - Pulmonary/Critical Care, Cardiology, Interventional Radiology, Vascular Surgery, Hematology and CT surgery

Case #1

- 58 yo chemical engineer with HTN, and OSA BIBA with 2 days of worsening chest heaviness and progressive DOE. LLE pain noted 2 days earlier. Traumatic rib, humerus, pelvic fracture and splenic laceration s/p MVA 2 weeks prior. “Minor head trauma” with no ICH or cranial fracture. Recently discharged from acute rehab.

- Splenic laceration was managed conservatively, humerus and pelvic fractures required surgical stabilization.

- 76/30 hr 115 rr 24 spo2 81% RA

- Exam with tachycardic s1 s2, no JVD, no RV heave, clear lungs and mild LLE swelling from knee to groin
Case #1

- CT Brain negative
- Hb 13.5
- BNP 900
- Trop 3.
- BLE DVT from SFV to popliteal vein
- PERT discussion

Case #1
Management

- Systemic TPA full dose (100mg)
- Surgical thrombectomy
- EKOS with TPA (25mg)
- Catheter directed suction thrombectomy without TPA
Case #1

- IVC filter placed
- EKOS catheter placement with 1mg/hr TPA in each catheter infused over 12 hours
- Initial PA pressures 65/30
- 6 hours later BP normalized and he no longer required vasopressors.
- Oxygenation improved
- EKOS Catheters removed 12 hours later PA pressures 45/20

Case #2

- 29 yo, 8 days post partum for first child (normal vaginal delivery without complication) presents from OB office with 12 hours of worsening SOB and pleuritic chest pain.
- 80/50 hr 130 rr 24 spo2 92% on Venti mask
Case #2
Management

- Systemic TPA full dose (100mg)
- Surgical thrombectomy
- EKOS with TPA (25mg)
- Catheter directed suction thrombectomy without TPA

Case #2

- Bedside echo showed right heart strain
- Trop 1.8, BNP 665, Hb 9.4
- Vasopressor requirements 8 mcg norepi
- BiPAP added for work of breathing but she maintained mental status.
- 100mg IV TPA given over 60 minutes
- 1 hour later she was off pressors, hr 95, less oxygen requirement
Case #3

- 62 yo active man with HTN, hyperlipidemia presents from home with 24 hrs of progressive left upper extremity pain and swelling.
- In ER, 105/60, hr 118, rr22 98% Venti mask T100
- Absent LUE brachial and radial pulses
Case #3

- Admitted to ICU. Remained hemodynamically stable.
- Echo confirmed right heart strain. Normal LV. Trop 4
- Extensive b/l LE DVTs
- PERT discussion. Options
  - Systemic TPA
  - Catheter directed TPA to PA and LUE
  - Surgical LUE embolectomy followed by heparin and watchful waiting.
### Early Mortality Risk

<table>
<thead>
<tr>
<th></th>
<th>Shock or Hypotension</th>
<th>PESI Class or sPESI &gt;1</th>
<th>Signs of RV Dysfunction on an Imaging Test</th>
<th>Cardiac Laboratory Biomarkers</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High</strong></td>
<td>+</td>
<td>( + )</td>
<td>+</td>
<td>( + )</td>
</tr>
<tr>
<td><strong>Intermediate</strong></td>
<td>-</td>
<td>+</td>
<td>Both positive</td>
<td>Either 1 (or none) positive</td>
</tr>
<tr>
<td>Intermediate-high</td>
<td>-</td>
<td>+</td>
<td>Assessment optional: If assessed, both negative</td>
<td></td>
</tr>
<tr>
<td>Intermediate-low</td>
<td>-</td>
<td>+</td>
<td>Either 1 (or none) positive</td>
<td></td>
</tr>
<tr>
<td><strong>Low</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>


### Case #3

- OR for LUE axillary artery embolectomy followed by heparin drip. Intubated and sedated
- IVC filter placed
- Post op 4 hrs later in ICU 70/40 hr 120 and requiring escalating doses of vasopressors and increasing oxygen requirements on the vent
- Hb 8.3
- Repeat bedside echo shows worsening right heart strain
Case #3

- PERT discussion
- Options
  - Catheter directed therapy with EKOS
  - Systemic TPA
  - Surgical embolectomy
  - Catheter directed therapy with suction thrombectomy.

Case #3

- Tolerated catheter directed suction thrombectomy with Penumbra catheter well
- Vasopressors weaned off over the next 12 hours
- Extubated the next day
- Transitioned to Warfarin
Case #4

- 64 yo woman with HTN, lymphoma treated with chemo in 1999 presented to the ER from home for lightheadedness, pre syncope and SOB. Reports diarrhea and abdominal cramping intermittently for the last 3 days.

- Vitals in triage 120/60, hr 105, rr 20, 95% RA, sbp dropped to 105 and hr increased to 125 on standing.

- Complaining of lightheadedness with any movement in triage.
Case #4

- Syncope moving from chair to bed in the ER 5 hours later. IVF started.

- CTA ordered. Code blue en route from scanner to room.

- PEA arrest.
Case #4

- ACLS protocol for PEA for approximately 3-4 min
- 100mg TPA given by IV push
- ROSC, sinus tacycardia, mentating
- norepi 10mcg to maintain MAP
- FiO2 80% for PAO2 72
Case #4

- LE dopplers with RLE DVT from thigh to knee
- Over the next 12 hours norepi requirements slowly increased to 30mcg and epi was added for MAP 60-65
- Echo with dilated, hypokinetic RV + McConnell’s sign
- IVC filter placed
- FiO2 requirements increased to 100%
- PERT discussion. CTA repeated

Presentation 15 hours later
Case #3
Management

• Systemic TPA (100mg)
• Surgical thrombectomy
• EKOS with TPA (25mg)
• Catheter directed suction thrombectomy without TPA

Surgical embolectomy
Case #4

- Extubated post operatively with improved oxygenation and hemodynamics in the PACU
- Discharged 6 days later on 2l/min nasal cannula
- Outpatient follow up 3 weeks later: no oxygen requirements, returned to work, echo with near complete resolution of right heart strain. IVC filter removed.

Thank You
Questions?
References

• Seattle II Group JACC 2015 Aug;(8): 1382-1392
• Moheson et al. MOPPETT Am J Card 2012;9-27
• Tapson, VF Acute PE, NEJM 2008 March;358
• Wood, KE et al, CHEST 2012 March 121;877
• PERT Consortium Website www.pertconsortium.org
   _____2018
Advancing Care in New York State, How Developing Protocols Can Work for Our Patients

Michael Dailey, MD, FACEP, Chief Division of Pre Hospital and Operational Medicine, Albany Medical Center; Medical Director, Regional Emergency Medical Organization
NYS Collaborative Protocols

Advancing care in New York State – How our Protocols can work for our patients

Michael W. Dailey, MD FACEP FAEMS
Regional EMS Medical Director – REMO
Professor of Emergency Medicine
Albany Medical College

Conflicts?

- Nope.
- Albany Medical College employee
- Town of Colonie employee
- Member, EMS World Advisory Board
- Member, NYS SEMAC, STAC, etc.
- Unpaid advisor to many
- Paid advisor to few... (not on purpose)
I. ADMINISTRATION PROCEDURES

1. There are no standing orders for medication administration in the REMO program. Medications may be given only:
   A. On the verbal order of a REMO certified physician.
   B. By a REMO certified paramedic.
   C. When selected from an up-to-date REMO Medication Schedule.
   D. When administered according to REMO procedures and protocols.

5. Upon receipt and confirmation of an order, the paramedic will:
   A. Write down the order, including the time.
   B. Remove package from medication box and read the container. Always be sure the medication ordered is the one in hand, and make sure the medication is not outdated.
   C. Administer the medication as ordered.
Preface to the 2002 REMO Protocols

• Patients, however, do not always fit into a “cookbook treatment” approach. Therefore, PROTOCOLS ARE NOT A SUBSTITUTE FOR GOOD CLINICAL JUDGEMENT, especially when a situation occurs which does not fit these guidelines.

• As an Advanced Emergency Medical Technician (AEMT), your field treatment must be carefully balanced with the knowledge of when your capabilities fall short of what the patient needs.

• When treating a patient you must ask yourself, what does this patient need as opposed to what you can provide?

---

M-13 PAIN MANAGEMENT/ ANALGESIA

NO STANDING ORDERS EXIST FOR PAIN MANAGEMENT/ ANALGESIA

1. Manage ABC’s as necessary.
2. Establish IV access, draw blood samples and infuse Normal Saline at KVO.
3. Contact Medical Control while enroute to the hospital and the AAREMS Physician will consider the following options:
   - Option A: Morphine, 2-5 mg, slow IV push.
   - Option B: Morphine, 2 mg, slow IV push every 10 minutes until an endpoint* occurs or a maximum of 10 mg is given.

CONTINUED ON NEXT PAGE

IV. PATIENTS WITH ADVANCED RENAL DISEASE

NO STANDING ORDERS EXIST FOR PAIN MANAGEMENT/ ANALGESIA

1. Manage ABC’s as necessary.
2. Establish IV access, draw blood samples and infuse Normal Saline at KVO.
3. Contact Medical Control while enroute to the hospital and the AAREMS Physician will consider the following options:
   - Option A: Morphine, 2-5 mg, slow IV push.
   - Option B: Morphine, 2 mg, slow IV push every 10 minutes until an endpoint* occurs or a maximum of 10 mg is given.

CONTINUED ON NEXT PAGE
C-1 ANALGESIA AND TREATMENT FOR SUSPECTED AMI

1. PARAMEDIC AND CRITICAL CARE TECHNICIAN STANDING ORDERS:
   A. Four (4) chewable baby aspirin, 81 mg each (324 mg total), PO,* (may be given before NTG, or in between doses of NTG)
   B. Three (3) doses of Nitro Spray (or tablets), 0.4 mg. five (5) minutes apart, provided the patient’s systolic B/P is greater than 120 and typical cardiac pain is present. If systolic B/P drops below 100, administer a 250 cc bolus of Normal Saline.**
   C. For agencies with 12 lead capabilities, consider obtaining and transmitting a 12 Lead EKG. However, DO NOT DELAY TRANSPORTATION.
   D. Complete the REMO Thrombolytic Checklist. (see page 166)
   E. Nitroglycerine Paste, 1-2 inches, to the chest wall after resolution of chest pain (as long as the systolic blood pressure remains above 90 mm Hg).

2. If pain/symptoms continue, contact Medical Control and the REMO Physician will consider the following options:
   Option A: Repeat doses of Nitro Spray (or tablets), 0.4 mg. every 5 minutes, checking B/P
   Option B: Morphine Sulfate, 2-3 mg. slow IV push. May repeat every 5 minutes, checking B/P, respiratory and mental status between doses.
   Option C: Additional Nitroglycerine Paste, 1-2 inches, to the chest wall
   Option D: Repeat 12 Lead EKG.
   Option E: Metoprolol (lapros) 5 mg IV every 5 minutes. X 3 doses (provided the heart rate remains above 60 BPM and the systolic blood pressure remains above 100 mm Hg).
Overview of Collaborative Process

• Amazing effort by a great group of EMS providers, physicians, educators and others

• REMO
  + Mtn Lakes + AAREMS
  + SREMS
  + Hudson Valley + Westchester

• Western

• CNY + Midstate + NorthCountry
  + Western + etc!

= COLLABORATIVE!
2006 – REMO Pain Management

EMS Program Agencies

1. Lake Placid Community Care Network
   Andrew Field, 306-747-1111
2. University Emergency Medical Services
   Scott Mancini, 518-694-5128
3. Southern Tier Emergency Medical System
   Home Rooter, 716-218-4148
4. Finger Lakes Regional EMS
   Home Rooter, 607-768-1010

CCT

- No standing orders

CCT STOP

PARAMEDIC

- Morphine 0.05 mg/kg IV or IM (SEE KEY POINTS BELOW)
- Morphine may be repeated once after 5 minutes, with a maximum total given not to exceed 10 mg
- Promethazine (Phenergan) 12.5 mg IV or 25 mg IM, if patient becomes nauseous

PARAMEDIC STOP
- Morphine 0.05 mg/kg IV or 0.1 mg/kg

**MEDICAL CONTROL CONSIDERATIONS**

- Additional morphine IV or IM
- Additional fentanyl IV, IM, or IN
- Ketamine* 25 mg IV over 5 minutes or 50 mg IM
  
  minutes; maximum total dose of 200 mcg

* Additional morphine IV or IM
* Additional fentanyl IV, IM, or IN
* Ketamine* 25 mg IV over 5 minutes or 50 mg IM
  
  May consider weight-based dosing
- Vascular access
- If glucose level is below 60 mg/dL and the patient cannot swallow on command, administer dextrose 10%, up to 25 grams (250 mL) IV; may redose if
  - If glucose level is below 60 mg/dL, and the patient cannot swallow on command, administer dextrose 10%, up to 25 grams (250 mL) IV; may redose if hypoglycemia recurs
  - If unable to obtain vascular access, administer glucagon 1 mg IM
(1-6) Cardiac Arrest: Termination of Resuscitation

EMT

ADVANCED

- See “Cardiac Arrest: Determination of Obvious Death” protocol

EMT AND ADVANCED STOP

CC

PARAMEDIC

- Patients who do not meet the “Cardiac Arrest: Determination of Obvious Death” protocol, but are in cardiopulmonary arrest, must meet ALL of the following requirements for termination of resuscitative efforts to be considered without a medical control order:
  - Age 18 or older
  - Arrest not witnessed by a bystander or by EMS
  - No bystander-administered CPR
  - No automated external defibrillator or manual shock delivered
  - No return of spontaneous circulation up to the time termination is considered
  - At least 20 minutes of resuscitation has been provided

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Key to ANY new protocol or procedure

• First do no harm

• Call and talk to a doctor if you have any questions

• DO NOT perform any procedures unless:
  – APPROVED by your medical director and
  – AUTHORIZED by scope of practice and protocol

• Ambulances credentialed as, “Ambulance Transfusion Services,” may transport patients with blood products initiated at the hospital, but must have orders for the blood products and orders for response to complications, written by the sending physician.

- The crew must determine if the patient's current condition is appropriate for the provider’s level of training, experience, and available equipment.
- If there are any changes in the patient’s condition that are not covered by the prescribed orders or agency protocols.
### Why reduce a patella dislocation?

- If not reduced it creates a significant need for pain medication
  - In many cases, should be an ALS call
  - In rural or understaffed regions, this may not be possible
- If not reduced this can cause significant ongoing pain
- Less complication with early reduction

---

### Why reduce a patella dislocation?

- It is good medicine!
- Is there potential harm? No
- If reduced there is no pain or very little pain
- Risk – none
- Benefit – huge
- Time for a new protocol.
Demonstration by EMS and a physician
Demonstration by EMS and a physician

Basic info

- 24 cases
  - Oldest 88
  - Youngest 8
  - Average 24 (if three oldest removed, average 17)

- Pain scores
  - Initial 9.2
  - Final 2.8
In bed with boyfriend

Successful on my first attempt!

Reduction attempted prior to pain medication and was unsuccessful – patient could not tolerate any hands on her leg

Easiest procedure I have ever done – should be a BLS skill!!

No pain at all post-reduction!

Patient held leg tense and therefore it was difficult to straighten – the patella would not reduce

Reduction attempt prior to pain medication and was unsuccessful – patient could not tolerate any hands on her leg

It was a very simple procedure and made a big difference for our patient

Made me much less queasy than watching the procedure on the video!
Interesting questions
**Beware of public outcry**

*Objective:* To understand glucagon use and availability in the prehospital and outpatient setting.

*Methods:* We included cases from the National EMS Information System in which glucagon was administered. Response times longer than 60 minutes were eliminated as probable errors. Medicare Part D data from 2014 were filtered, retaining only those cases in which glucagon was prescribed. State EMS offices were contacted to review protocols for glucagon administration and blood glucose testing. Data from the National EMS Information System between 2013 and 2015 and from the Centers for Medicare & Medicaid Services data warehouse were analyzed in Alteryx, version 11 (Alteryx), and Tableau, version 10.2.1 (Tableau Software).

*Findings:* Across all states, glucagon was administered in 89,263 cases in the prehospital setting between 2013 and 2015. Dispatchers correctly coded only 44.67% of the calls as a “diabetic problem.” The patient experienced adverse effects in 39.44% of the events in which glucagon was administered.

https://www.goodrx.com/glucagon
Underutilization of Glucagon in the Prehospital Setting

Despite the favorable safety profile of glucagon, it is infrequently prescribed in the outpatient setting. The average cost to Medicare for a glucagon prescription is $212. Patients who filled glucagon prescriptions had fewer hypoglycemia-related emergency department visits, suggesting that providing this agent to patients may effectively reduce prehospital hypoglycemic complications (1).

All emergency personnel should have access to glucagon along with training to safely administer this agent to minimize unintended treatment accidents. Ensuring access is an important opportunity to reduce morbidity, mortality, and health care costs. Diabetes specialists should work with emergency medical personnel to design curricula for the safe and effective use of glucagon nationwide.
What would be the impact of glucagon for all?

- Cost of glucagon?
  
  $295 (GoodRx.com 3/7/18)

- Number of ambulances in NYS?
  
  4,605

- Total cost? $1.3 Million!
- How many uses would there be? No one knows...
Can a paramedic initiate TPA infusions?
  – Yes: It is in the National Model Scope of Practice for STEMI

Can a paramedic initiate TPA for stroke?
  – Nope. Neither can a doctor without a CT scan first.

Can a paramedic transport a patient with TPA hanging?
  – Maybe. Regional decision.
  – Must have a pathway for transport of stroke patients in a timely manner to endovascular centers.
Pulmonary edema

- Research led us to remove Lasix
  - Made patients worse
  - Wrong patients got it
- Nitro doses went up – more Ntg is better!
- Good medicine led us to remove Nitro paste
  - Sick patients don't absorb from the skin
- Patients are doing much better
  - Fewer intubations

Research

- Cardiac monitor
- Aggressive nitroglycerin 0.4 mg SL or equivalent, as needed:
  - One dose/tablet every 5 minutes if the patient’s systolic BP 120 – 160
Where are we going from here?

**Big changes...loss of the CC**

- Curriculum has not been updated since 1990s
- Anticipated expense of updating CC – a cool million
- No national standard
- Not really a statewide standard...
- Advanced EMT serves similar function across the rest of the country
Changes in EMS – loss of the CC

### Mtn. Lakes

<table>
<thead>
<tr>
<th>Year</th>
<th>CFR</th>
<th>EMT</th>
<th>AEMT</th>
<th>CC</th>
<th>Paramedic</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>2002</td>
<td>8,116</td>
<td>41,610</td>
<td>293</td>
<td>1,631</td>
<td>7,053</td>
<td>58,703</td>
</tr>
<tr>
<td>2016</td>
<td>13.83%</td>
<td>70.89%</td>
<td>0.5%</td>
<td>2.78%</td>
<td>12.02%</td>
<td></td>
</tr>
</tbody>
</table>

Total certified providers by level in NYS – 2016
### Skill-Medication Administration - Routes

<table>
<thead>
<tr>
<th>Skill-Medication Administration - Routes</th>
<th>AEMT</th>
<th>NYS AEMT</th>
<th>Paramedic</th>
<th>NYS Paramedic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inhaled – self-administered (nitrous oxide)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Intramuscular (epinephrine or glucagon)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Intranasal (naloxone)</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Intravenous push (naloxone, dextrose 50%)</td>
<td>L</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Intravenous push (epinephrine)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intravenous piggyback</td>
<td></td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Nasogastric</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Oral (glucose)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>

### Skill - Airway/Ventilation/Oxygenation

<table>
<thead>
<tr>
<th>Skill - Airway/Ventilation/Oxygenation</th>
<th>AEMT</th>
<th>NYS AEMT</th>
<th>Paramedic</th>
<th>NYS Paramedic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventilator – Automated transport (ATV)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
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</tbody>
</table>

### Skill - Cardiovascular/Circulation

<table>
<thead>
<tr>
<th>Skill - Cardiovascular/Circulation</th>
<th>AEMT</th>
<th>NYS AEMT</th>
<th>Paramedic</th>
<th>NYS Paramedic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac monitoring – multi-lead (interpretive)</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Cardiac monitoring – single lead (interpretive)</td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Cardiopulmonary resuscitation (CPR)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>
### BLS Protocols

**Criteria**
- Any specific information regarding the protocol in general

**EMR/CFR**
- EMR/CFR and EMT standing orders
  - **EMR/CFR STOP**

**EMT**
- EMT standing orders
  - **EMT STOP**

**MEDICAL CONTROL CONSIDERATIONS**
- Medical control may give any order within the scope of practice of the provider
- Options listed in this section are common considerations that medical control may choose to order as the situation warrants.

**Key Points/Considerations**
- Additional points specific to patients that fall within the protocol

### Next steps...

- Assure the educational standards meet the medical needs across the State
- Advance opportunities to make sure that interfacility transport can continue to exist...
- Assure that the changes in the provider level are made with limited negative impact to patient care
- Changes must be operationally and fiscally prudent
- Use data to drive protocol changes
Thank you

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Training the Limbic System for High Performance During Resuscitation

Faizan Arshad, MD, EMS, Medical Director, Vassar Brothers Medical Center; Medical Director, Regional Emergency Medical Organization
FINGERS
Front cortex
Human brain

THU B
Jic system
anima brain

WRIST
Brain stem
Lizard brain
Training gives us an outlet for suppressed energies created by stress and thus tones the spirit just as exercise conditions the body.

— Arnold Schwarzenegger —
The legacy of Hans Selye and the origins of stress research: A retrospective 75 years after his landmark brief "letter" to the Editor# of Nature

Sandor Szabo, Yvette Tache & Arpad Somogyi
Prozac Nation Is Now the United States of Xanax

By ALEX WILLIAMS | JUNE 10 2017
Growth of World Population and the History of Technology

- Agricultural Revolution
- Pottery
- Invention of Plow
- 1st Irrigation
- 1st Cities
- Metallurgy
- Writing
- Agriculture
- Phone
- Internet
- Nuclear Energy
- DNA Discovered
- Penicillin
- Automobile
- Telephone
- Germ Theory
- Railroads
- Mobile

Source: Milken Institute, Robert Fogel/University of Chicago
Human Emotion...
CHANGE YOUR STORY
CHANGE YOUR LIFE
State

Emotion  Perception
Happiness and freedom begin with a clear understanding of one principle. Some things are within your control. And some things are not.

—Epictetus
WHAT HAPPENS ONE HOUR AFTER DOING THE WIM HOF METHOD

1st 30 - 60 SECONDS
PUMPS YOUR BODY WITH VITAL OXYGEN
Deep breathwork for 30-50 cycles leads to vital oxygen rushing to your skin and tissues. Your cells and brain become more alert and positive.

1: 1 - 3 MINUTES
STILL BREATHING, STILL MIND, INNER PEACE
The retention phase lasting around 1-3 minutes, leaves the body feeling more relaxed and awake.

2 - 3 MINUTES
ENERGY RUSH
Your body signals that you now need more oxygen. Breathing deeply makes your oxygen up-take increase by 30% in every cell and tissue.

3 - 20 MINUTES
COLD IS YOUR NEW WARM FRIEND
Cold therapy using cold showers or ice baths, triggers your body to release endorphins and activate your survival mode.

20 - 25 MINUTES
VIVA LAS VAGUS
With further practice you can control your vagus nerve and autonomic nervous system.

30 - 60 MINUTES
ASTONISHING LONG-TERM BENEFITS
Control your immune system, change your body temperature, create superhuman strength and stamina, sleep deeply. It's a complete transformation, from mind to body.

IF YOU PRACTICE HARD ENOUGH YOU MAY BE ABLE TO BEAT ONE OF WIM HOF’S 26 WORLD RECORDS!

For the full article including citations please visit: therenegadepharmacist.com/WimHofMethod
Homework!

- Emotional Wellness
- Health and Fitness
- Romantic Relationship(s)
- Financial Fitness & Retirement Planning
- Your Career and Fulfilling your Purpose
- Spirituality and Faith
- Activities that lead to personal growth
- Fun & Leisure
- The quality of your environment and Surroundings

I am grateful for.........
“Our intention creates our reality.”

Wayne Dyer
F" TINC., ...
BE B\cK
500N!
Giraffes & Zebras in the ICU

Steven Ritter, MD, Sleep Medicine, Critical Care, Internal Medicine and Pediatrics, Medical Associates of Hudson Valley; Medical Director, Northern Dutchess Sleep Center
More Serious than Zebras

Just a few of the many humbling experiences in my arena

Critical Care

Bradycardia

68 yo h/o chronic RLE lymphedema, recurrent cellulitis, and chronic complaints of neck pain with seen SFH and Emergency 1 admit for RLE cellulitis 9/3/15

• Ortho consult: 3 months of neck pain following episode of angioedema

• Exam shows FROM, no sig pain on movement, strength 5/5, pain on palpation mid/lower Cspine
• Rapid response called for patient 9/4/15 0145 when she is found poorly responsive reportedly does well on venti-mask
• 0500 ED physician called emergently to intubate patient
• Stat CT head negative
• Noted to be recurrently bradycardic by cardiology concern for sick sinus

• Remains stable on MV
• Noted to have L hemiplegia no CVA noted
  – Seen by neurology
• CT soft tissue neck only shows cervical spondylosis CT head repeated 9/5
• Extubated 9/6 0930
• Became markedly bradycardic, then asystolic
• Had emergent re-intubation 9/6
  – Noted to have sinus tach
• Neurology: normal CT ST neck and CT head
  – Also noted ? Relation of bradycardia to turning
• Cardiology suggesting PPM placement
• Sent for CTA 9/7

• During CTA pt inadvertently extubated
• CTA findings:
  – Significant interval change in the C1-C2 relationship.
  – The dens of C2 is subluxed posteriorly and to the left and there is basilar invagination as well. This is possibly resulting in compression of the medulla or cord at the cervical medullary junction.
  – If there is instability here it could also be resulting in compromise of the vertebral arteries.
• Repeat Ortho consult notes R side weakness as well 9/7
  — Placed in C-collar
  — Notes previous CT neck showed some erosive changes C1-2 with widening
  — Re-evaluation (CTA) shows posterior displacement of C1-2 articulation.
  — Witheld steroids as may be infectious for incipient quadriplegia

• Transfer arranged to WCMC
  — Patient intubated prophylactically with inline stabilization to avoid decompensation during transport.

• Was standard intubating technique in setting of unstable C1-2 the subsequent emergency?
PRES

56 yo female h/o EtOH abuse, seizures, chronic anemia, and gastric bypass x 2, last WCMC 2/13/17 admit 2/25/17 with altered MS, BP 185/90 -202/145, L gaze preference, and anisocoria.

- CT head: Diffuse bilateral and symmetric low-attenuation involving bilateral cerebral and cerebellar hemispheres, predominantly involving the white matter, however extending to the gray matter in bilateral frontal and occipital lobes

- MRI 2/26
  There are a few punctate foci of increased signal in the medial right temporal lobe and right parietal lobes. Diffuse white matter edema is seen in the occipital lobes as well as the frontal lobes and less severely the anterior parietal lobe. Appearance consistent with posterior reversible encephalopathy syndrome(PRES) with possible superimposed few embolic microinfarcts on the right.
• Subsequently stabilized BP on Cardene gtt
• Mental status improved.
• Moved to telemetry bed on floor
• Remained essentially bedbound
• D/C to SAR
• Reportedly did not feel well at SAR but no acute changes.

• Found in respiratory distress, unresponsive, 3 days after discharge
• BIB EMS King airway, I/O in place
• Resuscitation failed
• Because of rapid decline and arrest and in general mortality <10% with PRES autopsy requested
  — Had not been on anticoagulation so concern for PE
• Findings were notable:
  ▪ Multiple small hemorrhagic infarcts throughout the brain
  ▪ Vegetation on the mitral valve composed of fibrin with acute inflammatory cells, no bacteria
  ▪ Old L frontal SAH

• Dx was marantic (Libman-Sachs) endocarditis
  – Multiple hemorrhagic infarcts due to emboli
• Notable would have been danger of any anticoagulation
• Patient did not have occult malignancy but never w/u for SLE
Arrest

• 58-year-old woman, with h/o A fib, MVR, on coumadin (followed WCMC cardio) witnessed arrest working as babysitter in ED 0828
  – EMS found in V fib, defib x 5, epi x 5, amio, intubated at scene, CPR cont’d enroute (35 min)
  – Defib in ED with ROSC
  – 7 min later arrests again
  – ROSC after approx 15 min

• Remained hypotensive on max dose of dopamine and levophed
• Also on Amio
• Echo showed EF<10% (previous EF normal)
• Head CT no ICH early cerebral edema
• CTA no PE, bilateral airspace disease
• Troponin 0.9 and lactic 11.9
• Hypothermia protocol was initiated
• Escalating pressors: levo, vaso, dopa, epi
• Serial labs revealed Hgb 12.8 => 5.8
• MTP initiated, hypothermia aborted
• After 2u PRBC and 2u FFP BP started to improve
• Repeat troponin 33
• At 1642 became profoundly bradycardic and failed to respond to ACLS algorithms
• Pronounced 1656

• ME initially deferred on the case
• Given cont’d cardiology f/u unclear COD
• Concern for malfunction/complication of MV
• Agreed to take the case
• Gross exam did not reveal cardiac issue
  – MV was intact and functional
  – No significant CAD
  – No myocardial findings
  – Some blood noted in pericardium expected with CPR

• ME concerned that there was no cardiac cause of death
• Elects to do sections of heart
• Notable finds ruptured aneurysm of RCA
Malignant HTN

38-year-old female with no significant past medical history except resection of the right breast nodule many years ago which as per the patient was benign presented to the hospital with chief complaint of frontal headache 10 x 10 in intensity associated with multiple episodes of nausea and vomiting at home. (6/1 1600)

- BP 216/128 => 158/104 after medication

- CT head negative initially, f/u revealed evolving infarct
- CTA chest negative
- CTA neck/head:

  A CT of the head and neck revealed extensive thrombus in the right internal jugular vein over 10 cm segment. The left vertebral artery at the C3-4 level possibly due to intraluminal thrombus or dissection.

  - Cerebral angio confirmed RIJ (10cm), PCA, AICA, PICA, thrombus and underwent unsuccessful attempt at extraction RIJ thrombus
• Seen by hematology for hypercoagulable workup.
  – Antithrombin III 65% and protein S 59 (compromised by heparin
  – Dx HIT made as well
• CT abd/pelvis 6/3/15
  – complex mass in the right para-aortic compartment partially
    effacing the IVC measuring 4.9 x 5.8 x 5.1 cm which may represent
    hematoma or partially necrotic neoplasm
• Urine metanephrine total was 1383. Urine normetanephrines
  1284., Urine metanephrine total was within normal limits at
  99. Serum normetanephrines is elevated at 5590, additionally
  patient has one copy of a 1298 CVA and of them TH MR DNA.

• Patient d/c to NEC on 9/11/15 (admit 6/1/15)
• Remained on TC and TF (trach 6/18 PEG 6/19)
• Concern about recurrence of malignant HTN due to possible
  Pheo
• Complicated by req’d continuation of Arixtra
• Also complicated by proximity ? Invasion of IVC
• Hypercoagulable w/u was negative
  – PAI-1 negative
  – Cardiolipin Ab neg
  – Phosphatidyl serine Ab neg
  – Factor V Leiden neg
  – AT III 65 (80-120)
  – Protein C 70 (70-180)
  – Protein S 59 (60-140)
  – PF4/heparin Ab (-)

• 24 hour Urine testing 10/2015
  – Chromogranin 47 ng (<15), cortisol 5.9 mcg per ml
  – Metanephrine 71 mcg, normetanephrine 1240 mcg per liter
  – Cortisol 33.6 (4-50) (24 hour)
  – 5-HIAA 32.1 (<6) mg/24hr
  – VMA 12 (1.8-6.7) mg/24hr
• Serum
  – Normetanephines 936 (148)
  – Metanephines 973 (<2015)
• Presented at Tumor Board:
  – Abdominal mass question pheochromocytoma. Metanephrines are high but in the setting of catastrophic illness this could be unreliable. Needed MIBG scan, but this is fraught with problems when patient is taking a number of medications
  – Need to define if invading IVC to add to complexity.
  – Complexity of withholding anticoagulation for operation

• CT abd/pelvis repeat 12/10/15
  – There is a stable mass lesion just anterior to the aorta and IVC that has a necrotic center and shows peripheral enhancement. This mass measures 5.9 x 5.1 x 3.5 cm in size (was 4.9 x 5.8 x 5.1 cm)

• MIBG scan 2/3/16 (note pt has remained stable)
  – There is a focus of radiotracer uptake within the mass anterior to the aorta and IVC consistent with a pheochromocytoma.
• Subsequently arranged second opinion Montefiore
  – BP well controlled on Phenoxybenzamine 10mg tid
  – Metanephrines elevated 891
  – Chromogranin A elevated 36
  – Unfractionated Heparin Serotonin Release Assay
    • Negative (sensitivity/specificity 88-100% for HIT)
  – PF4/Heparin Ab 0.492 OD (optical density) sl elevated
    • Interpretation is (+)

• Notable was that a family member has VHL variant
• People with VHL mutations have von Hippel-Lindau (VHL) disease. VHL can be divided into two primary subgroups: VHL type 1 and VHL type 2. VHL type 2 is further divided into 2A, 2B, and 2C
• increased risk for non-cancerous tumors and certain types of cancer depending on the type of VHL you have:
  • VHL type 1: kidney cancer, PNET, non-cancerous tumors of the eyes, brain, spine, and pancreas
  • VHL type 2A: PCC, non-cancerous tumors of the eyes, brain, and spine
  • VHL type 2B: PCC, kidney cancer (high risk), PNET, and non-cancerous
  • tumors of the eyes, brain, spine, and pancreas
  • VHL type 2C: PCC only

• Previously asymptomatic 15-year-old boy was admitted to a local hospital 1 month PTA with subacute progressive headache for 2 weeks and Sz x 2. Initial examination showed HTN and mild postictal confusion. On evaluation, he had a left frontoparietal hematoma. His magnetic resonance imaging (MRI) with MR venogram showed superior sagittal and transverse sinus thrombosis.

• Admission blood pressure was 200/120 mm of Hg. Blood work up for renal function tests, coagulopathy, vasculitis, connective tissue disease, and prothrombotic state were negative. His MTHFR, Factor V Leyden, and prothrombin gene mutations were also negative. Urine catecholamine products were high. Computed tomography (CT) abdomen and metaiodobenzylguanidine (MIBG) scan showed adrenal and para-aortic masses.

• Underwent a para-aortic dissection and right adrenalectomy. He was discharged after an uneventful postoperative period. Histopathology of the specimen showed a pheochromocytoma.
AMS

19-year-old female PMHx developmental delay, OCD behaviors, who presents to the hospital 1/3/17 because of 4 days illness. On New Year's Eve she developed some nausea, dizziness, actually went home early from a gathering on New Year's Eve. It did persist, became worse. She presented to the emergency room twice because of nausea and vertigo, was released both times. Parents report changes in behavior during that time.

Had begun having multiple loose watery bowel movements per day. She did faint at home, lost consciousness briefly and was brought back to the hospital. More than 4 weeks ago she was on an antibiotic for a "double ear infection" treated at an urgent walk-in center. She was also recently seen for a rash, also had an urgent care center, was given a topical cream and this resolved.

Tm 102.1
Admitted (+) C. diff.

• On 1/6 RRT called for dystonic movements, head bobbing, dec’d responsiveness
• MRI 1/7 1245: Diffuse severe right cerebral hemispheric cortical signal abnormality especially in the frontal and temporal lobe. There is mild right to left midline shift without obstructive hydrocephalus. Diffuse leptomeningeal enhancement noted..
• LP 1/7 1425: Pro 71 glu 52 MBP CSF ALB 52 (SERUM 3.7), MBP 11 WBC 164 RBC 19, HSV 6 IgM 1:20 IgG 1:80, hponatremic with Na 131
  – VZV, West Nile, HIV negative
• Amp/Ceftriaxone/Acyclovir/Vanco/Vanco NG
• Intubated, hypertonic saline
• EEG R sided PLED’s
• Transfer to CPMC
• Course at CPMC complicated by herniation, R hemicraniectomy (1/9/17) and subsequent cranioplasty (2/1/17). Noted to have b/l ACA and R MCA infarcts. Had placement of subdural shunt as well.
• Re-admit 3/2017 for worsened mental status. Noted to have subgaleal collection, s/p subgaleal shunt
• Remained at Blythedale Children’s Hospital but then on 1/22/18 her neurologist believed there were some subtle deteriorations in behaviour: hypersexuality, impulsivity, confusion
• Had repeat LP
  – Glu 56 prot 36
  – 4 WBC 12 RBC
  – NMDAr Ab 1:40
• Serum NMDAr 1:2560
• Subsequently treated 5d IVIG and solumedrol 1g daily
• Behavior did not improve had Rituximab 1g for 2 doses

• Acute Disseminated Encephalomyelitis (ADEM) (10% of pediatric EM 8/10^6)
• Hashimoto’s Encephalopathy
• Rasmussen’s Encephalitis
• LGI1/CASPR2-Ab encephalitis (VGKC encephalitis)
• Limbic Encephalitis
• anti-NMDAR encephalitis (Dr.’s Dalmau/Lancaster 2007)

All of these have come about in the last 10 years
• A wide variety of clinical syndromes has been associated with antibodies to voltage-gated potassium channels (VGKCs).

• Six years ago, it was discovered that patients do not truly have antibodies to potassium channels, but to associated proteins.
  – This enabled the distinction of three VGKC-positive subgroups: anti-LGI1 patients, anti-Caspr2 patients and VGKC-positive patients lacking both antibodies.
  – Patients with LGI1-antibodies have a limbic encephalitis, often with hyponatremia, and about half of the patients have typical faciobrachial dystonic seizures.
  – Caspr2-antibodies cause a more variable syndrome of peripheral or central nervous system symptoms, almost exclusively affecting older males.

• Immunotherapy seems to be beneficial in patients with antibodies to LGI1 or Caspr2, stressing the need for early diagnosis.

• Half of the VGKC-positive patients lack antibodies to both LGI1 and Caspr2. This is a heterogeneous group of patients with a wide variety of clinical syndromes, raising the question whether VGKC-positivity is truly a marker of disease in these patients.
– Data regarding this issue are limited, but a recent study did not show any clinical relevance of VGKC-positivity in the absence of antibodies to LGI1 and Caspr2.

– The three VGKC-positive subgroups are essentially different, therefore, the lumping term ‘VGKC-complex antibodies’ should be abolished.

• “Brain on Fire”

• In 2009, Cahalan was in a serious relationship and her career as a reporter at the New York Post was taking off. But suddenly, as she tells it in this engaging memoir, she began suffering from a bizarre amalgam of debilitating symptoms including memory loss, paranoia, and severe psychosis that left her in a catatonic state that moved her close to death

• even though the disease was discovered in 2007, some doctors I spoke to believe that it’s been around at least as long as humanity has
Ultrasound Guided Resuscitation for Undifferentiated Shock

Stephanie Midgley, MD, Director of Emergency Medicine Ultrasound, EOS Medical Group, Emergency Medicine Attending, Team Health, PC, Vassar Brothers Medical Center, Putnam, Northern Dutchess, and Ellenville
Ultrasound Guided Resuscitation for Shock
The RUSH Exam

Stephanie Midgley
MD Vassar
Brothers Medical Center Director
of Emergency Ultrasound

<table>
<thead>
<tr>
<th>I'M DYING! CALL 911!</th>
<th>DON'T WORRY, SENT THEM A TEXT.</th>
<th>MY PHONE WAS ON VIBRATE, SORRY, WHAT'S THE NATURE OF YOUR EMERGENCY?</th>
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<tbody>
<tr>
<td><img src="9/11/2018" alt="Image 1" /></td>
<td><img src="9/11/2018" alt="Image 2" /></td>
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Cyanide and Happiness © Explosm.net
Why use ultrasound?
Scope

Danger of death
CP/SOB

Hypotension
US GUIDED PROCEDURES

Central Line
pIV Line

Pericardiocentesis
Paracentesis

Thoracentesis
Lumbar Puncture
How does ultrasound work?

Piezoelectric
current-crystal-vibration

Transmission

Hyperchoic
Echogenicity
Conventions & Terminology

Planes
Patient Planes

- Coronal Plane
- Sagittal Plane
- Transverse Plane

Object Planes

- Longitudinal Axis
- Transverse Axis
Object Planes

Longitudinal - Long axis

Transverse - Short axis

Parasternal long axis view

Parasternal short axis view

PROBES
Probes

High Frequency
High Resolution
Less Penetration

Low Frequency
Less Resolution
More Penetration
High Frequency Linear Probe

More Resolution, Less Penetration

Medium Frequency Phased Array Probe

Less Resolution, More Penetration

Low Frequency Curvilinear Probe

High Frequency Linear Probe

More Resolution, Less Penetration
Probe Markers
Moving the Probe

Well, I wasn’t able to get the nail out of your head...

But I was able to Photoshop it out of your head!

How’s that supposed to help?

Cyanide and Happiness © Explosm.net
911 Caller:
I’m so weak and dizzy, I fell and I can’t get up

EMS notifies on triage . . .
25F syncopal fall from home, hypotensive, tachy
HR 140
BP 60/30
RR 30
Accucheck 112

RUSH EXAM
Rapid Ultrasound in Shock and Hypotension
**RUSH EXAM**
Rapid Ultrasound in Shock and Hypotension

- **Heart**
- **I VC**
- **Morrison’s Pouch/FAST**
- **Aorta**
- **Pulmonary**

---

**RUSH(ed) Exam**

- **Heart**
- **I VC**
- **Morrison’s Pouch/FAST**
- **Aorta**
- **Pulmonary**
PUMP

TANK

- IVC
- Peritoneal Space
- Pleural Space
PIPES

• Aorta
• Veins

The Probe
**PUMP, TANK, PIPES**

Using US to determine type of shock

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RUSH(ed) Exam Sequencing

I  VC
M  Morrison's Pouch/FAST
A orta
P  pulmonary
Parasternal Long

- Parasternal
- Nipple level
- Index to right shoulder
- Slices heart in long axis
Parasternal Long
PSL

• Septum and LV posterior wall are parallel and horizontal as possible

• AV and MV are in the center of image
Apical 4 chamber

- Apex of the heart or PMI
- Aim probe horizontally
- Visualizes both atria and ventricles

Apical Four Chamber
Apical Four Chamber

- All four chambers are seen
- Septum is vertical and centered
Apical Four Chamber
Function

Estimation of EF

• BestViews:
  • PSL, PSS

• Visual Estimation of EF:
  • Wall thickening and motion
  • Changes in LV size during systole
Normal EF

- Ventricle contracts symmetrically towards center
- Myocardium thickening
- MV opens normally during diastole
Hyperdynamic EF

- LV with near complete obliteration of chamber size in systole
Low EF

- Minimal change in wall thickness in systole
- Minimal change in chamber size between diastole and systole
Cardiac Standstill

- No motion of LV or RV
- May see valve fluttering
  - Usually due to positive pressure ventilation
BACK TO OUR PATIENT...
Our Pt...
Pump - hyperdynamic
### PUMP, TANK, PIPES

#### US determination of shock type

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RUSH(ed) Exam Sequencing

H eart

M orrison’s Pouch/FAST

A orta

P ulmonary

Inferior Vena Cava
IVC US Anatomy

- Abuts the liver
- Hepatic veins drain into IVC
- See it pass through the diaphragm and into RA
- Empties into RA
Pathology

- Normal IVC (<50% collapse with respirations)
- Collapsing IVC (>50% collapse with respirations)
- Non-collapsing IVC (minimal respiratory changes)
Collapsing IVC

- Greater than 50% collapse with respirations
- Interpretation: hypovolemia
- Seen in Hypovolemic Shock, Distributive Shock
Collapsing IVC

Non-collapsing IVC

- Little to no respiratory variation of IVC
- Interpretation: outflow obstruction
- Seen in Obstructive Shock, Cardiogenic Shock
Non-collapsing IVC

BACK TO OUR PATIENT...
Our Pt...

Pump - hyperdynamic
IVC - collapsing
### PUMP, TANK, PIPES

#### US determination of shock type

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RUSH(ed) Exam Sequencing

H  eart

I  VC

A  orta

P  ulmonary

Morison's Pouch

Perisplenic

Subcostal

Suprapubic
Morison’s
Most sensitive view

Probe Position
Morison's Pouch

Perisplenic

Suprapubic

Subcostal

Splenorenal View
Probe Position
Bladder View

Probe Position
BACK TO OUR PATIENT...
Pump - hyperdynamic
Tank - IVC collapsing
- Peritoneal FF

PUMP, TANK, PIPES
US determination of shock type

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RUSH(ed) Exam Sequencing

H eart

I V C

M orrison's Pouch/FAST

P ulmonary

PUMP, TANK, PIPES
Using US to determine type of shock

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Anatomy

Prox Ao
Mid Ao
Distal Ao

Normal aorta
Aorta with large abdominal aneurysm
Transverse View
Probe Marker to Right
Gentle Compression
Start Subxyphoid and end below navel

Prox Ao

Portal vein

IVC

Celiac artery (with branches)

Aorta (transverse)
Distal Ao

Longitudinal View
Probe Marker to Head
Gentle Compression
Start Subxyphoid and End below navel
Measurements

- Measure **OUTSIDE to OUTSIDE**
- Measure **prox, mid, distal**
  - But SEE every centimeter
- Measure in **transverse ONLY**
- Measure Anterior-Posterior
Number

3 cm

ProxAo

1.8 cm
MidAo
2cm

Distal Ao
2cm
Number

3 cm

BACK TO OUR PATIENT...
Our Pt...

- Pump - hyperdynamic
- Tank - IVC collapsing
- Peritoneal FF
- Pipes - normal aorta
PUMP, TANK, PIPES
US determination of shock type

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RUSH(ed) Exam Sequencing

H eart
I VC
M orison's Pouch/FAST
A orta
### PUMP, TANK, PIPES

#### Using US to determine type of shock

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**PULMONARY VIEWS**

*Image of cartoon lungs with a message*
Normal Anatomy

Muscle

Rib

Pleural Line

Rib Shadow

Rib Shadow
Normal Anatomy

Normal Anatomy: Lung Sliding

Ants Marching

Comet tails
BACK TO OUR PATIENT...
We Interrupt This Program
To Bring You An Important Message...
Our Pt...

- Pump - hyperdynamic
- Tank - IVC collapsing
- Peritoneal FF
- Lungs normal
- Pipes - Normal
Diagnosis

Hypovolemic due to Shock
Ruptured ectopic
911 Caller:
My mom fell and she can’t get up

EMS notifies on triage . . .

75F fall from home, hypotensive had pacer placed yesterday
HR 140
RR 30
BP 60/30

RUSH(ed) Exam

Heart
VC
Morrison's Pouch/FAST
Aorta
Pulmonary
Pericardial Effusion

- Anechoic
- Dependent
- Between myocardium and pericardium
- Confirm with 2 views
Effusion

anechoic

dependent

between myocardium and pericardium
Effusion

Liver

anechoic

dependent

between myocardium and pericardium

Effusion?
• Effusions should be circumferential

• Mistaking fat pad for effusion

• Mistaking pleural effusion for pericardial effusion

• Pericardial Effusions are anterior to DTA and crosses midline
Tamponade

- Clinical diagnosis!
- Pericardial effusion, hypotension, tachycardia, and JVD
- US confirms pericardial effusion
- Rate of accumulation determines tamponade
US Findings of Tamponade

- Pericardial effusion
- Scalloping of RV and/or RA during diastole
- **RV collapse when MV is open**
- Plump IVC
ECHO FEATURE OF TAMPONADE

RA diastolic collapse

RV diastolic collapse

Pericardial effusion
RV diastolic collapse

RV FreeWall

Anterior Leaflet of MV
BACK TO OUR PATIENT...
RUSH(ed) Exam

H  eart
I  VC
M  orrison’s Pouch/FAST
A  orta
P  ulmonary
# PUMP, TANK, PIPES

**US determination of shock type**

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**Diagnosis**
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Tamponade
I THINK I HAVE AN ILLNESS

WHAT ARE YOUR SYMPTOMS?

I STAY UP ALL NIGHT DANCING

SEEMS LIKE YOU'VE GOT A CASE OF BOOGIE FEVER

HAHAHAHA!

IT'S FATAL

PARDON ME STRANGER
I'VE FALLEN AND CAN'T GET UP. MY NEIGHBORS ARE IGNORING MY SCREAMS FOR HELP

Cyanide and Happiness © Explosm.net
EMS notifies on triage …

80M with fall, hypotensive, SOB on CPAP, hx of CHF

HR 55, sinus brady
BP 60/30
RR 50 sating 80% on RA
Accucheck 112
RUSH(ed) Exam

H  eart
I  VC
M  orrison’s Pouch/FAST
A  orta
P  ulmonary
Pulmonary Edema

B Lines

- Vertical lines extending from the pleural line thru sono window
- Obliterates A Lines
- Moves with respirations
- Artifact is from fluid in interstium of lungs
- B Lines = Lung pathology
B Lines

• 3 or more constant B Lines in a lung field = Pulmonary Edema

• Studies have correlated the more B lines on US the more severe the plum edema

• B lines on US will appear earlier than B Lines on CXR

Orient Yourself

[Image of an ultrasound scan with labeled structures: rib, soft tissue, pleura, lung]
B Lines

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**Diagnosis**
Cardiogenic Shock

THANK GOD! A DOCTOR!
COULD YOU HEAL MY GAPPING WOUND?
HERE IS YOUR PROBLEM.

YOU HAD A BANDAGE ON YOUR WOUND!
NOW IT WILL BE JUST FINE!

Cyanide and Happiness © Explosm.net
Help.

I've fallen and I can't get up.

EMS notifies on triage . . .
80M fall from home, hypotensive, SOB on CPAP, hx of COPD
HR 120
RR 50
BP 70/30
Pox 85% RA

RUSH(ed) Exam

H eart
I VC
M orrison’s Pouch/FAST
A orta
P ulmonary
PTX

Findings:
1. No lung sliding
2. Loss of comet tails
3. Stratosphere sign
PTX
no pretty comets

M Mode: Normal Lung

Chest Wall/Muscle
Pleural line
Expanding & Contracting Lung

Aaahh, the beach
Out at sea, no beach in sight....
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OBStructive due to Shock
tension ptx

Well according to my clipboard, you've got a fractured skull.

Uh, I think that's a mistake. I came here because my stomach hurts.

I'm the doctor!
Help!

Life Alert!

I've fallen and I can't get up!

EMS notifies on triage . . .
80M fall from home, hypotensive, SOB on CPAP, hx of COPD
HR 120
RR 50
BP 70/30
Pox 85% RA

OH NO
NOT THIS AGAIN

RUSH(ed) Exam
H eart
I VC
Morrison’s Pouch/FAST
A orta
P ulmonary
RV Assessment

• Normal RV<2/3 LV
• RV pressure overload results in
  • Interventricular septal flattening (D shaped LV)
  • RV enlargement
RV Strain on US

- RV ≥ LV (A4C/PSS/subxiphoid)
- Acute tricuspid regurgitation (A4C)
- Dilated RA (subxiphoid view)
- D shaped LV (PSS)
- Underfilling of LV in systole
Tricuspid Regurgitation

RA Dilation

RA
LA
D Shaped LV

- Akinesia of the RV free wall with normal contraction of the apex
- In acute increases in pressure, the RV free wall loses the ability to contract while the apex's ability is preserved
- Apical 4 chamber or subxiphoid
McConnell’s Sign

Under Filling of LV
Fake Outs for RV Strain

- Chronic RV dilation due to:
  - Chronic right sided elevated pressures
  - COPD
  - Pulmonary HTN
Signs of Chronic RV Dilation

- Thickened RV free wall
- Absence of D shaped LV in setting of enlarged RV

Absence of RV strain points you away from PE as source of hypotension
Corroborative Studies

- LE US (DVT)
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Diagnosis

OBStructive due to Shock
pulmonary embolism

SEIZURE MAN! DO YOU NEED SOME HELPP?

ACTUALLY, YOU CAN JUST CALL ME "MAN."

I'VE BEEN USING MEDICINAL MARIJUANA TO TREAT MY SEIZURES.

THAT'S VERY INTERESTING.

SO WHY ARE YOU LAYING ON THE FLOOR DROOLING?

I'M SO GID DAMN HIGH.
Help! I've fallen

and I can't get up!

EMS notifies on triage ... 80M fall from home, hypotensive, SOB on CPAP, hx of CHF
HR 120
RR 50
BP 70/30
Pox 75% RA

OH NO, NOT AGAIN!

RUSH(ed) Exam
H eart
I VC
M orrison's Pouch/FAST
A orta
P ulmonary
Pneumonia

- Hepatization of Lung
- Trace pleural effusion
- Air bronchograms (branching echogenic structures which move with breathing)
- Multiple hyperechogenic spots (air trapped in the small airway)
Hepatization of Lung

Hepatization of Lung
Air Bronchograms

Pleural effusion

consolidations

Loss of A Lines

Air Bronchograms

Lung Consolidation

Loss of A Lines

Sonographic Air Bronchograms
Hyperechoic

Liver
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Diagnosis

Distributive due to Shock
Pneumonia

911 call
CPR in progress
EMS notifies on triage . . .

80M CPR in progress

OH NO NO NO NO NO NO NO

NO NO NO NO NO NO NO
RUSH(ed) Exam

H  eart
I  VC
M orrison's Pouch/FAST
A  orta
P  ulmonary
Abdominal Aortic Aneurysm

Number

3 cm

Ignore Thrombus

Outside to

Outside

Transverse View
EVERYBODY STAND BACK

I GOT THIS.
Cordis/A Lines placed
Massive transfusion protocol started
Pulses return
Vascular notified/OR activated
Epi gtt started

Pt to OR ...

...and survived!
Diagnosis
survived
ruptured
AAA

OK, WHAT's THE PROBLEM?

I'VE GOT A SONG STUCK IN MY EAO.

YEP! LOOKS LIKE IT'S PRETTY DEEP IN THERE!

Cyanide and Happiness © Explosm.net
WHY DOES MY MEDICAL RECORD SAY I'M DECEASED?

Danger of death
RUSH(ed) Exam

- Heart
- Intra-abdominal
- Morrison’s Pouch/FAST
- Aorta
- Pulmonary
PUMP

TANK

- IVC
- Peritoneal Space
- Pleural Space
PIPES

- Aorta
- Veins

To guide your resuscitation in shock
Thank You!