Sepsis: The Silent Killer

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Objectives

- Understand the continuum of sepsis
  - Identify patients with sepsis

- Current treatments for sepsis

- Understand basic principles of shock
  - What is “occult shock”

- Why do I need to know this (pre-hospital care)

- Future trends for screening, triage, and treatment
VCU Medical Center

- Construction of new ICU hospital
- Opened in fall 2008
- Over 120 ICU beds

- Only level 1 Trauma Center in Central Virginia
- ED 70,000 visits/yr
Who and What is VCURES

- Multidisciplinary Center of Excellence
- Focus: Acute Care Research, and Education
- 8 Colleges, 30 Departments, 50 Investigators
- Nontraditional Partnerships: Engineering, Physics, Chemistry, Computer Science
- Taking the Cancer Center Approach to Acute Injury and Illness

Restoring life through innovation and collaboration

Turning the Golden Hour into the Silver Day

www.vcures.com
Current Trends in Emergency Medicine Critical Care
Trends in ED Visits

Annual visits increased: 23,704 to 27,457
Number of ED’s decreased: 4,005 to 3,934

Figure 1. Trend in emergency department visits: United States, 1997–2000
Patient Demographics – Age/Race

Figure 2. Annual rate of emergency department visits by patient age, race, and ethnicity: United States, 2004

McCaig 2006
ED LOS Times (mean)

![Chart showing ED LOS times for different patient acuity levels.]

- Immediate: Waiting time: 30 minutes, Treatment time: 210 minutes
- Emergent: Waiting time: 45 minutes, Treatment time: 180 minutes
- Urgent: Waiting time: 60 minutes, Treatment time: 150 minutes
- Semiurgents: Waiting time: 75 minutes, Treatment time: 120 minutes
- Nonurgent: Waiting time: 90 minutes, Treatment time: 90 minutes

**NOTE:** The total length of the emergency department visit is the waiting time plus the treatment time.

Figure 8. Mean waiting time and treatment time in emergency departments, by patient acuity level: United States, 2004

McCaig 2006
Levels of Acuity

Figure 5. Percent distribution of emergency department visits, by immediacy with which the patient should be seen: United States, 2004

McCaig 2006
Growth in Medicare ICU and Hospital Admissions Since 1994

Year

% Change

-4.00 -2.00 0.00 2.00 4.00 6.00 8.00 10.00 12.00 14.00 16.00


Change ICU Use
Change Hospital Use

Milbrandt, E.B. and Angus, D.C. Unpublished data from CRISMA laboratories, Pittsburgh, PA
EMS Data

- **Number of annual ED Visits**: 114 million
  - 26% increase since 1993
  - Avg. 2% increase/year
  - Number of ED decreased 12.3% over same time

- **Number arrived via EMS**: 16 million (14%)
  - > 1/3 are older than 65
  - This number WILL increase over the next 20 years

- **58% of EDs were urban**
  - Representing 82% of all visits
So what are all of us faced with?

- Older population
- Increasing number of ED visits
- Decreasing number of EDs - increased diversion
- More EMS transports (older)
- Higher acuity
- Longer ED length of stay for the sickest
- More patients being admitted to ICU
Can we make a difference?

Do we already?

- STEMI – Cath lab 90 min
- Stroke – TPA within 3 hours
- Trauma system
- Sepsis?
“Critical care is a continuum that begins with out-of-hospital care, continues with ED intervention, and culminates in ICU admission and management.”

- Peter Safar, MD
  1924-2003
  Father of CPR
Peter Safar, MD 1924-2003

Capt McMahon, Chief Baltimore Fire and Ambulance Service, 1957

Baltimore City Hospital
Resuscitation Experiment, July 13, 1957
Chest Pressure Arm-Lift Method

Baltimore City Hospital
Resuscitation Experiment, July 13, 1957
Chest Pressure Arm-Lift Method

Virginia Commonwealth University Reanimation Engineering Shock Center
Peter Safar, MD 1924-2003

“the most sophisticated intensive care often becomes unnecessarily expensive terminal care when the pre-ICU system fails.” – Peter Safar, MD
What do you think of when you hear “sepsis”?
What is sepsis?
Greek origin = putrefaction

Sepsis or Septic = Decomposition, Decay
Clinically, Confusion Reigns

- Wide range of mortality and morbidity
  - 35% - 75% mortality

- Unclear terminology commonly used

- No clear consensus on what “Sepsis” meant

- Bacteremia = septicemia = sepsis = sepsis syndrome = septic shock
Body’s response to an “attack”

The inflammatory response
Inflammatory Response - Local
The Endothelial System

Pan-endothelial Disruption
**SIRS**
(Systemic Inflammatory Response Syndrome)

- **SIRS Definition (at least 2)**
  - Fever or hypothermia (T > 38°C or < 36°C)
  - Tachypnea (RR > 20 bpm or PaCO2 < 32 mmHg)
  - Tachycardia (HR > 90 bpm)
  - Immune response (WBC > 12,000, or < 4,000 or > 10% immature (band) forms

ACCP/SCCM Consensus Conference, Critical Care Medicine 1992; 20:864-74
Sepsis

- At least 2 SIRS criteria PLUS a suspected infection
- Up to 30 - 60% may be “culture negative”
- Culture “positive” or “negative” have similar mortalities

ACCP/SCCM Consensus Conference, Critical Care Medicine 1992; 20:864-74
Relationship Of Infection, SIRS, Sepsis Severe Sepsis and Septic Shock

Bone et al. Chest 1992;101:1644
A clinical response arising from a nonspecific insult, including ≥2 of the following:

- Temperature ≥38°C or ≤36°C
- HR ≥90 beats/min
- Respirations ≥20/min
- WBC count ≥12,000/mm³ or ≤4,000/mm³ or >10% bands
- PaCO2 < 32mmHg

SIRS with a presumed or confirmed infectious process

Sepsis with ≥1 sign of organ failure:

- Global Tissue Hypoxia ?
- Renal
- Respiratory
- Hepatic
- Hematologic
- CNS
- Unexplained metabolic acidosis

Some Clinical Signs of Severe Sepsis
Identifying Acute Organ Dysfunction as a Marker of Severe Sepsis

- Altered Consciousness
  - Confusion
  - Psychosis

- Tachypnea
  - PaO₂ <70 mm Hg
  - SaO₂ <90%
  - PaO₂/FiO₂ ≤300

- Oliguria
  - Anuria
  - ↑ Creatinine

- Jaundice
  - ↑ Enzymes
  - ↓ Albumin
  - ↑ PT

- Tachycardia
- Hypotension
- Altered CVP
- Altered PAOP

- ↓ Platelets
  - ↑ PT/APTT
  - ↓ Protein C
  - ↑ D-dimer

How big of a problem is Sepsis?
Severe Sepsis:
A Significant Healthcare Challenge

Major cause of morbidity and mortality worldwide

- Leading cause of death in noncoronary ICU (US)*
- 11th leading cause of death overall (US) †§

More than 750,000 cases of severe sepsis in US annually with 215,000 deaths annually

- Costs $16.7 Billion‡

In the US, more than 500 patients die of severe sepsis daily.‡

*Sands KE et al. JAMA. 1997;278:234-40; †Based on data for septicemia.
§Murphy SL. National Vital Statistics Reports.
‡Angus DC et al. Crit Care Med. 2001;29:1303-1310; reflects hospital-wide cases of severe sepsis as defined by infection in the presence of organ failure.
Incidence of Severe Sepsis

- AIDS *
- Colon CA #
- Breast CA #
- CHF %
- Severe Sepsis **

# American Cancer Society, 2001
% American Heart Association, 2001
Comparative Mortality

- AIDS *
- Breast CA #
- Acute MI %
- Severe Sepsis **

# American Cancer Society, 2001
% American Heart Association, 2001
Is there a treatment?
New therapies for Sepsis

- Early antibiotic administration
- Low volume ventilation – ARDS
- Drotrecogin alpha (activated) Xigris®
- Corticosteroids
- Strict glycemic control
- Early goal-directed therapy
Common treatments for critically ill

Absolute Mortality Reductions

- **EGDT**
  - Rivers EP, NEJM 2001
- **Steroids**
  - Annane D, JAMA 2002
- **APC**
  - Bernard GR, NEJM 2001
- **Intensive Insulin**
  - Van den Berge G, NEJM 2001
- **Stk in STEMI**
  - ISIS-2, Lancet 1988
- **Il/b Il/a inhibit in NSTEMI**
  - Boersma E, Lancet 2002

- Sepsis
- ICU pts
- AMI
Early Goal Directed Therapy

- Based on recognizing EARLY Shock States
- Reverses oxygen debt
- Therapy aimed at improving delivery of oxygen to the tissues
- Works along with
  - Antibiotics
  - Steroids
  - Xigris®
  - Glucose control
  - Source control (abscess drainage, etc…)
SHOCK
What is Shock?
Inability to supply (or utilize) enough oxygen to meet the metabolic needs of the tissue.

Tissue Hypoperfusion!
Shock: An Outdated Definition

Normal perfusion $\rightarrow$ Global Tissue Hypoxia and O2 Debt HYPOPERFUSION $\rightarrow$ Traditional Shock

Normal Vitals $\rightarrow$ Normal Vitals $\rightarrow$ Abnormal Vitals (Hypotension)
Blood pressure fails to detect global tissue hypoxia

- Adequate pressure does not always mean adequate flow to tissues

\[
\text{MAP} \sim \text{SVR} \times \text{CO}
\]

(Schwaitzberg, J Ped Surg, 1988)
Cryptic Shock

**Patients with a Baseline MAP > 100**

Lactate > 4 mM/L  
Control n = 25  
EGDT n = 23

- A significant co-morbid variable:
  - 2-fold increase in sudden cardiovascular collapse, cause of early death.
  - 56.5% in early hospital mortality.
  - Natural selection process between the onset of severe illness and ICU arrival.

Patients with a Baseline MAP > 100 and Lactate > 4 mM/L:

- Control n = 25
- EGDT n = 23

<table>
<thead>
<tr>
<th>Time</th>
<th>Control</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hosp</td>
<td>61%</td>
<td>20%</td>
</tr>
<tr>
<td>30 Day</td>
<td>65%</td>
<td>20%</td>
</tr>
<tr>
<td>60 Day</td>
<td>70%</td>
<td>24%</td>
</tr>
</tbody>
</table>

*Control*  
*Treatment*
What’s really going on?
Oxygen Delivery

\[ CaO_2 = (1.34 \times Hb \times SaO_2) + (0.0031 \times PaO_2) \]

- Cardiac Output
- Heart Rate
- Stroke Volume
- Preload
  - CVP
  - PCWP
- Afterload
  - SVR (MAP)
- Contractility
Venous Oxygen Delivery
750 ml/min

Arterial Oxygen Content ($\approx$ SaO$_2$)
20 vol% = 20 ml/dl

Venous Oxygen Content
SvO$_2$ = 75%

Venous Extraction
25%

Oxygen Consumption

Arterial Oxygen Delivery

Oxygen Extraction
25%

Cardiac Output
5 L/minute

Oxygen Consumption

1000 ml/min

250 ml/min
What is going on at the tissue level?
Capillary system

- Lymph vessel
- Lymphatic capillaries
- Arterial capillaries
- Venule
- Arteriole
- Venous capillaries
- Tissue cells
Lactate

Hypoperfusion (shock)

Capillary

Red blood cell

Direction of blood flow

Body Cells
OPS: A Picture is Worth a Thousand Words?

Normal Sublingual Circulation
Normal Capillary Bed
Sublingual Circulation in Shock
Sublingual Circulation in Shock
The Balance of Life

Oxygen Extraction
(Metabolic needs of the tissues)

Oxygen Delivery

C.O.
SaO₂
Hgb

Lactate
ScvO₂

Microvascular Alterations

Oxygen Utilization

Metabolic needs of the tissues
Oxygen Delivery Basics

First 6 HOURS

- Delivery Dependent
  - Lactate
  - $S_{cv}O_2$

- Delivery Independent
  - $DO_2$ crit
  - or norm $S_{cv}O_2$
  - or norm Lactate

6 to 72 HOURS

Oxygen Delivery ($DO_2$)

Oxygen Extraction ($VO_2$)
How do we bring our septic shock patient back into “balance”?
Early Goal-directed Therapy

Supplemental oxygen ± endotracheal intubation and mechanical ventilation → Central venous and arterial catheterization → Sedation and/or paralysis (if intubated)

- CVP:
  - <8 mm Hg: Crystalloid
  - 8-12 mm Hg
  - >90 mm Hg: Vasoactive agents

- MAP:
  - <65 mm Hg: Vasoactive agents
  - >90 mm Hg

- ScvO2:
  - <70%: Transfusion of red cells to hematocrit ≥30%
  - ≥70%: Inotropic agents

Goals achieved:
- ≥70%
- <70%

No → Yes → Hospital admission

## Mortality

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>EGDT</th>
<th>RR (95% C.I.)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-hospital</td>
<td>46.5</td>
<td>30.5</td>
<td>0.58 (0.38-0.87)</td>
<td>0.009</td>
</tr>
<tr>
<td>28-day</td>
<td>49.2</td>
<td>33.3</td>
<td>0.58 (0.39 – 0.87)</td>
<td>0.01</td>
</tr>
<tr>
<td>60-day</td>
<td>56.9</td>
<td>44.3</td>
<td>0.67 (0.46-0.96)</td>
<td>0.03</td>
</tr>
</tbody>
</table>
## EGDT Therapy Comparison – Timing is Everything!

<table>
<thead>
<tr>
<th></th>
<th>0 – 6 hr</th>
<th>7 – 72 hr</th>
<th>0 – 72 hr</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Fluid (cc)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EGDT</td>
<td>4,981*</td>
<td>8,625</td>
<td>13,443</td>
</tr>
<tr>
<td>Control</td>
<td>3,499</td>
<td>10,602*</td>
<td>13,358</td>
</tr>
<tr>
<td><strong>Transfusion</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EGDT</td>
<td>64%*</td>
<td>11%</td>
<td>68%*</td>
</tr>
<tr>
<td>Control</td>
<td>19%</td>
<td>33%*</td>
<td>45%</td>
</tr>
<tr>
<td><strong>Vasopressor</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EGDT</td>
<td>27%</td>
<td>29%</td>
<td>37%</td>
</tr>
<tr>
<td>Control</td>
<td>31%</td>
<td>43%*</td>
<td>51%*</td>
</tr>
<tr>
<td><strong>Inotrope use</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EGDT</td>
<td>14%*</td>
<td>15%</td>
<td>15%</td>
</tr>
<tr>
<td>Control</td>
<td>1%</td>
<td>8%</td>
<td>9%</td>
</tr>
<tr>
<td><strong>Ventilator use</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EGDT</td>
<td>53%</td>
<td>3%</td>
<td>56%</td>
</tr>
<tr>
<td>Control</td>
<td>54%</td>
<td>17%*</td>
<td>71%*</td>
</tr>
</tbody>
</table>

*Significant difference compared to Control.
Is this practical?
EGDT Experiences at Two Hospitals

- **Brown University**
  - Primarily ED identifies and EGDT team does the resuscitation

- **Beth Israel Deaconess Medical Center**
  - ED identifies and ICU team does the resuscitation
Brown University ED Trigger to Start EGDT

- Hypotension: 43%
- Elevated Lactate: 40%
- Both: 17%
BIDMC ED Trigger to Start EGDT

- Both: 33%
- Hypotension: 31%
- Elevated Lactate: 36%
Time for Sepsis Therapies in ED – Brown University

Reduction
- Antibiotics: 45%
- Catheter: 50%
- ICU Transfer: 58%

Baseline
- Antibiotics: 185
- Catheter: 318
- ICU Transfer: 318

5 months
- Antibiotics: 102
- Catheter: 157
- ICU Transfer: 200

Time (Minutes)
Time for Sepsis Therapies in ED – BIDM

Baseline 3 months

- Antibiotics: 69%
- Catheter: 23%
- ICU Transfer: 7%

Reduction in Time (Minutes): 247 to 77 (69%), 200 to 77 (23%), 445 to 413 (7%)
Time to Catheter Insertion (EGDT starting)

- Both centers combined and mean times used
  - Before teams formed
    - 254 minutes (4.2 hours)
  - After teams formed
    - 157 minutes (2.6 hours)
Common Findings

- Both institutions had difficulties –
  - Most common
    - Patient identification
    - Line insertion

- Lactate used as a trigger = 73% of the cases!
  - Lactate alone up to 40%
  - Lactate with hypotension up to 33%

- TIME is TISSUE
  - Early detection of tissue hypoxia is ESSENTIAL
EGDT Pitfalls - Survey

- ED Physicians unfamiliarity with continuum of Sepsis
- Unfamiliarity with cryptic shock, only follow vital signs
- Screening in triage not sensitive enough to identify cryptic shock
- ED Physicians may have limited resources
- Many hospitals don’t routinely test for Lactate or it is delayed several hours
- Possibly patients with cryptic shock are admitted to floor or even discharged home without addressing oxygen debt
- Even in prestigious medical centers with an EGDT system in place, a delay of over 2\(\frac{1}{2}\) hours occurs before EGDT can be started
Can we do better?

Can we detect hypoperfusion in the field?
Lactate = the biochemical vital sign for hypoperfusion
Lactate = First biochemical vital sign!!

- Lactate is a familiar marker
  - Well shown lactate predicts mortality in 60’s
  - Compare to BNP or Troponin where clinicians are still getting used to interpreting the results and various assays
  - Mortality outcome prediction has held the test of time
  - Does not matter what causes elevated lactate (topic of debate)
  - Smaller studies validate usage in several disease states
    - Cardiac
    - Infectious
    - Trauma
    - Abdominal pain
Lactate is not a new marker


Lactate still correlates with severity of illness


Lactate and Outcome

(Mizock, Dis Mon, 1989) (Weil, Circulation, 1970)
Lactate Clearance and Mortality

(Knoblich and Rivers, Acad Emerg Med, 1999)
Cryptic Shock

Outcomes of Patients with a Baseline MAP > 100, Lactate > 4 mM/L
Control n = 33 and Treatment n = 30

% Mortality

Hosp 30 Day 60 Day

Control Treatment

P<0.001
Why should we detect hypoperfusion in the field

- Start therapy early (IV fluids)
- Triage to appropriate ED
- Monitor resuscitation (lactate clearance)
- Start definitive care in the ED earlier
- Improve outcome?
How do you check lactate in the field?
Abbott i-Stat
CLIA Mod-complex

Lactate Pro
CLIA Waived

Roche Accutrend Lactate
CLIA Mod-complex
Why is it so hard to get adequate resources to treat sepsis?
Damn Marketing!!!

- **Heart** – Thrombolytics (< 6hr onset): 1.5% ARR
  - PTCA “Door to balloon time” < 1 hr vs up to 90 min: 4% ARR

- **Brain** – rTPA: 90 min, 30% increase in functional neurologic outcome up to 90 days after. *(Tissue plasminogen activator for acute ischemic stroke. N Engl J Med 1995)*
  - 3X the rate of ICH vs control

- **Trauma** – “Golden Hour” *R Adams Cowley, MD* coined this phrase in the 1970’s
  - Trauma centers, trauma teams

- **Severe Sepsis** – Early Goal Directed Therapy: 6 hours
  - ARR of 16% - Best results of any sepsis trial to date
  - ?????? How do you make Pus and Sepsis Sexy?!
Time is Tissue!

- Need to identify hypoperfusion as early as possible
- Need to start treating the hypoperfusion
- Transfer to definitive care center
  - Trauma Center
  - Cardiac Cath Center
  - Stroke Center
  - ?Sepsis Center?
  - ?Shock Center?
What’s New?
Possible Future Direction

EMS
Lactate!
Why should we detect hypoperfusion in the field?

- Triage to appropriate ED
- Treatment
  - Start therapy early (IV fluids)
- Monitor resuscitation (lactate clearance)
- Transport to the appropriate ED
- Start definitive care in the ED earlier
- Improve outcome?
Abbott i-Stat
CLIA Mod-complex

Lactate Pro
CLIA Waived

Roche Accutrend
Lactate
CLIA Mod-complex
Lack of correlation between lactate and hemodynamics

<table>
<thead>
<tr>
<th>Lactate Tiers</th>
<th>Total ( n = 857 )</th>
<th>Normal (&lt; 2.5) ( n = 599 )</th>
<th>Intermediate ( 2.5 - 3.9) ( n = 140 )</th>
<th>High ( \geq 4.0) ( n = 118 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (BPM)</td>
<td>( r = 0.15 ) (0.08 to 0.21)</td>
<td>( r = 0.09 ) (0.01 to 0.17)</td>
<td>( r = -0.15 ) (-0.31 to 0.01)</td>
<td>( r = 0.13 ) (-0.05 to 0.30)</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>( r = -0.08 ) (-0.14 to -0.01)</td>
<td>( r = -0.01 ) (-0.09 to 0.07)</td>
<td>( r = -0.23 ) (-0.07 to -0.038)</td>
<td>( r = -0.01 ) (-0.19 to 0.18)</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>( r = 0.14 ) (-0.21 to -0.07)</td>
<td>( r = -0.04 ) (-0.12 to 0.05)</td>
<td>( r = -0.26 ) (-0.41 to -0.09)</td>
<td>( r = -0.08 ) (-0.26 to 0.11)</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>( r = -0.15 ) (-0.21 to -0.08)</td>
<td>( r = -0.03 ) (-0.10 to 0.06)</td>
<td>( r = -0.26 ) (-0.41 to -0.10)</td>
<td>( r = -0.10 ) (-0.28 to 0.08)</td>
</tr>
<tr>
<td>Shock Index HR/SBP</td>
<td>( r = 0.20 ) (0.13 to 0.26)</td>
<td>( r = 0.07 ) (-0.01 to 0.15)</td>
<td>( r = 0.09 ) (-0.08 to 0.25)</td>
<td>( r = 0.10 ) (-0.08 to 0.28)</td>
</tr>
</tbody>
</table>

# Cryptic Shock?

<table>
<thead>
<tr>
<th>Lactate Tier (mmol / L)</th>
<th>Systolic Blood Pressure (mm / Hg)</th>
<th>≤ 90</th>
<th>&gt; 90</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 2.5 (n = 599)</td>
<td>14 (2.3%)</td>
<td>585 (97.7%)</td>
<td></td>
</tr>
<tr>
<td>2.5 – 3.9 (n = 140)</td>
<td>11(7.9%)</td>
<td>129 (92.1%)</td>
<td></td>
</tr>
<tr>
<td>≥ 4.0 (n = 118)</td>
<td>12 (10.2%)</td>
<td>106 (89.8%)</td>
<td></td>
</tr>
</tbody>
</table>

Table 1. Number of patients in each lactate level tier and their association with hypotension (defined by a SBP ≤ 90 mm/Hg). Total n = 857

*14% of all transports had HIGH lactate levels (≥ 4)

Lactate as a predictor of Admission and type of admission

- 2,034 patients transported to VCU Med Center or community hospitals in Richmond area
- RAA and Chesterfield Fire and EMS
- Common EMS vital signs used (HR, BP, GCS, RR, Glucose level, Age) and LACTATE
- Logistic regression model
- Only independent predictors of admission
  - Age
  - Lactate
- Lactate was better at predicting admission by more than 40% than all other variables
Pre-hospital Lactate as an independent predictor of admission

Lactate O.R. = 1.43
P < 0.001

Age O.R. = 1.03, P < 0.001
HR O.R. = 1.0, P = 0.61
RR O.R. = 1.0, P = 0.93
SBP O.R. = 1.0, P = 0.44
Pre-hospital lactate as an independent predictor of death

Lactate O.R. = 2.14  
P = 0.011

Age O.R. = 1.04, P = 0.001
HR O.R. = 0.97, P = 0.008
RR O.R. = 0.94, P = 0.11
SBP O.R. = 1.0, P = 0.659
Does this slow down our paramedics?

RAA EMS run times
Priority 1

Before Lactate Study: 34.25 minutes
During Lactate Study: 35.00 minutes

Gunnerson KJ, et al. EDOCS prelim data
EMS SBP overestimates ED SBP

Correlation coefficient $r = 0.6388$

$N=1115$

Gunnerson KJ, et al. EDOCS prelim data
What do we do after you drop them off?
Surviving Sepsis Campaign

- Launched in Fall 2002 as a collaborative effort of European Society of Intensive Care Medicine, the International Sepsis Forum, and the Society of Critical Care Medicine

- Goal: reduce sepsis mortality by 25% in the next 5 years

- Guidelines revealed at SCCM in Feb 2004 and updated in 2008
  - Website: survivingsepsis.org
Background

- The Surviving Sepsis Campaign’s mission is to increase awareness and improve outcome in severe sepsis
- Guidelines developed by a group of international experts representing 11 organizations
- Developed under unrestricted industry educational grants
- Published in March 3, 2004 issue of *Critical Care Medicine*

Sponsoring Organizations

- American Association of Critical-Care Nurses
- American College of Chest Physicians
- American College of Emergency Physicians
- American Thoracic Society
- Australian and New Zealand Intensive Care Society
- European Society of Clinical Microbiology and Infectious Diseases
- European Society of Intensive Care Medicine
- European Respiratory Society
- International Sepsis Forum
- Society of Critical Care Medicine
- Surgical Infection Society

Key Components

- Fluid resuscitation
- Appropriate cultures prior to antibiotic administration
- Early targeted antibiotics and source control
- Use of vasopressors/inotropes when fluid resuscitation optimized
Cases
Case 1

- 80 y/o Nursing home transfer with mental status changes and decreased urine output
- HR 110
- RR 28
- Temp 102
- BP 110/50
- Pulse ox 95% 2l
Central line
Catheter Complications
What is the problem?

🌟 Is your patient septic?

🌟 Hypoperfusion? Does BP matter?
   - Lactate in ER 9.5 mmol/L

🌟 What are you going to do?
Case 2

- 28 y/o male recently discharged from the hospital for an abscess in upper arm
- He is in severe pain and wants you to take him to the ER for pain meds
- HR 130
- BP 100/40
- RR 22
- Temp 96
- Pulse ox 92% on RA
*Clostridium septicum* of upper arm after IV injection of cocaine
Sub-Cutaneous emphysema of soft tissue
Sub-Cutaneous emphysema of soft tissue in chest wall
Case 3

- 60 yo female with family, c/o SOB, fever. Family: pt confused
- HR 130, T=39.8, RR 26, BP=80/30, MAP 55
- PE: No retractions, no ST infections, Lungs: decreased BS, ABD: s/nt/nd, Ext: no edema, Neck: no JVD
Petechiae

Ecchymoses / Purpurae
Typical EMS Encounter

32 F with hx of asthma

CC: Difficulty Breathing
Typical EMS Encounter

HR = 86
RR = 16
BP = 106/82

LACTATE = 9.6
Hospital Course

- Admitted for “Asthma”
- Condition worsened and moved to stepdown - severe sepsis
- E. Coli grew out in the urine
- Pseudomonas grew out of old chest tube sites
- Enterococcus grew out in blood cultures
- Hospitalized for 7 days – 3 in step-down.
- Discharged home on home IV antibiotics
32 male with hx of paraplegia, wheelchair bound,

PICC line for long term antibiotics for decubitus ulcer,

Suprapubic catheter in place.

C/O of fever and “feeling cold” to visiting nurse.
HR 120,  
RR 22,  
BP 134/68,  
Temp 100.8 (ER)  
WBC 12.2

Lactate 6.9  
SIRS Criteria?  
Potential infectious source

PICC Line  
Decubitus ulcer  
Suprapubic Catheter

- Only on vancomycin
Hospital Course

- Taken to community hospital
- “Vital signs” not much different than EMS
- Rapid Urine Screen (+) for UTI
- Final culture Candida albicans
- Discharged back home
- No lactate available for follow-up
Sepsis conclusions

- Does my patient have sepsis?
  - 2 of 4 SIRS Criteria (HR, Temp, WBC, Resp Rate)
  - Infection causing this?

- Is there hypoperfusion (organ dysfunction)
  - Altered mental status
  - Jaundice
  - Petechiae
  - Elevated Lactate, > 4 mmol/l?

- Start treating the hypoperfused state
  - IVF
  - Tell the ED doc

- Definitive care
  - Sepsis Teams
  - Shock Centers
Thank you for your attention

Questions?

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