Critical situations in Critical Care

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When dealing with critically ill patients we should obtain data to enable us to make informed decisions.
ScvO2

Blood pressure

CVP
The hypotensive patient

What is the mechanism for hypotension?
The basic information available

Is it enough?

CVP

Blood pressure

8-12mmHg

ScvO2

65-70%

History
Physical diagnosis
Pulse
Sometimes the clinical problem is not too complicated
A 55 YO man is brought to the ER
- Patient is known to have a lymphoma
- has finished a course of chemotherapy a week ago
- 3 days ago started having fever and cough
On examination he is short of breath with 24 breath/minute
Blood pressure is 100/55 mmHg
Heart rate is 120 bpm
He has a PICC line with the tip in the SVC
$\text{ScvO2} = 65\%$
Fluids?  Inotropes?
The patient is given two 500 ml boluses of colloid
- Blood pressure - 120/50 mmHg
- HR 100/min
- RR 24/min
- Urine output improves
ScvO2 = 73%
Does this make a difference?

The New England Journal of Medicine

EARLY GOAL-DIRECTED THERAPY IN THE TREATMENT OF SEVERE SEPSIS AND SEPTIC SHOCK

Emanuel Rivers, M.D., M.P.H., Bryant Nguyen, M.D., Suzanne Havstad, M.A., Julie Ressler, B.S., Alexandria Muzzin, B.S., Bernhard Knoblich, M.D., Edward Peterson, Ph.D., and Michael Tomlanovich, M.D., for the Early Goal-Directed Therapy Collaborative Group®
<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>STANDARD THERAPY (N=133)</th>
<th>GOAL-DIRECTED THERAPY (N=130)</th>
<th>RELATIVE RISK (95% CI)</th>
<th>P VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-hospital mortality†</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All patients</td>
<td>59 (46.5)</td>
<td>38 (30.5)</td>
<td>0.58 (0.38–0.87)</td>
<td>0.009</td>
</tr>
<tr>
<td>Patients with severe sepsis</td>
<td>19 (30.0)</td>
<td>9 (14.9)</td>
<td>0.46 (0.21–1.03)</td>
<td>0.06</td>
</tr>
<tr>
<td>Patients with septic shock</td>
<td>40 (56.8)</td>
<td>29 (42.3)</td>
<td>0.60 (0.36–0.98)</td>
<td>0.04</td>
</tr>
<tr>
<td>Patients with sepsis syndrome</td>
<td>44 (45.4)</td>
<td>35 (35.1)</td>
<td>0.66 (0.42–1.04)</td>
<td>0.07</td>
</tr>
<tr>
<td>28-Day mortality†</td>
<td>61 (49.2)</td>
<td>40 (33.3)</td>
<td>0.58 (0.39–0.87)</td>
<td>0.01</td>
</tr>
<tr>
<td>60-Day mortality†</td>
<td>70 (56.9)</td>
<td>50 (44.3)</td>
<td>0.67 (0.46–0.96)</td>
<td>0.03</td>
</tr>
<tr>
<td>Causes of in-hospital death‡</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sudden cardiovascular collapse</td>
<td>25/119 (21.0)</td>
<td>12/117 (10.3)</td>
<td>—</td>
<td>0.02</td>
</tr>
<tr>
<td>Multiorgan failure</td>
<td>26/119 (21.8)</td>
<td>19/117 (16.2)</td>
<td>—</td>
<td>0.27</td>
</tr>
</tbody>
</table>

*CI denotes confidence interval. Dashes indicate that the relative risk is not applicable.
†Percentages were calculated by the Kaplan–Meier product-limit method.
‡The denominators indicate the numbers of patients in each group who completed the initial six-hour study period.
But sometimes the clinical situation may not be that simple
## What are our options?

<table>
<thead>
<tr>
<th>Basic monitoring tools</th>
<th>Advanced monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Heart rate</td>
<td>• Flow</td>
</tr>
<tr>
<td>• Blood pressure</td>
<td>• Preload – volumetric indices</td>
</tr>
<tr>
<td>• Urine output</td>
<td>• Fluid responsiveness</td>
</tr>
<tr>
<td>• CVP</td>
<td>• SVR</td>
</tr>
<tr>
<td>• ScvO2</td>
<td>• EVLWI</td>
</tr>
<tr>
<td></td>
<td>• Beat by beat cardiac output</td>
</tr>
</tbody>
</table>
Cardiac output

EVLW

SVV, PPV

SVR

GEDV, ITBVI
A 24 year old woman is admitted to the ICU

- Previously healthy
- Week long fever, nausea, vomiting,
- maculopapular rash on trunk and limbs
In the ER

- Patient is conscious, weak complains of muscle pain
- Temp 38.9 C
- Blood pressure: 64/40 mmHg, HR 138 bpm
- RR 28/min, Sat 88%
- Maculopapular rash over trunk and hands
- History of recent tampon use
Patient is intubated
Mechanically ventilated
Transferred to the ICU
Patient is admitted to the ICU - After 2.5 liters LR

What should we do?

- BP - 75/40
- HR - 130/min
- Sat - 92% on FiO2-0.6
- RR - 30/min
- Lactate – 5.6 mmol/L

More fluids?

Inotropes?

Vasoconstrictors?
If we give more fluids.....
Blood pressure is still low
If we measure hemodynamics

- CI - 3.55 LPM/M2 (3-5 LPM/M2)
- CVP - 7 mmHg (8 mmHg)
- ITBVI - 870 ml/M2 (800-1000 ml/M2)
- EVLWI - 17 ml/kg (<10 ml/kg)
- SVR – 660 dynes/cm5/sec
- Hb – 10 G/dl
What is the etiology of the patient’s hypotension?

- Preload is normal **No point in giving more fluids**
- Cardiac output is adequate
- SVR is low **Use a vasoconstrictor**
- EVLWI is high **Be very careful with fluids**
- DO2 is 440 ml/min/m2
Noradrenaline is started

- Blood pressure and cardiac output increase and lactate improves
- Patient is also given diuretics
- Over the next few days EVLWI decreases and the patient is weaned from mechanical ventilation
S. Aureus is cultured from a vaginal culture.

Ten days after admission, skin begins to peel, establishing diagnosis of toxic shock syndrome.
A 60 YO man is admitted to the trauma service after a MVA
- PMH is notable for CAD,
- Has undergone CABG with MV repair.
- Fractured ribs, pulmonary contusion
- On the 5th day develops fever, hypotension, tachycardia
- Severe oliguria
- Blood pressure: 100/51 mmHg
- HR: 80 /min
- Lactate: 4.6 mmol/L
- CVP: 7
- No urine output
- ScvO2: 72%
**Fluids**

When we measure his hemodynamics with a PiCCO monitor:

<table>
<thead>
<tr>
<th>Metric</th>
<th>Value</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO</td>
<td>6.45 LPM</td>
<td>Seems adequate</td>
</tr>
<tr>
<td>ITBVI</td>
<td>1070 ml/M2</td>
<td>High</td>
</tr>
<tr>
<td>EVLWI</td>
<td>9 ml/Kg</td>
<td>Normal</td>
</tr>
<tr>
<td>SVR</td>
<td>660 dyne/cm5/sec</td>
<td>Low</td>
</tr>
</tbody>
</table>
Patient is also placed on CVVH to decrease edema
Seeing real time beat by beat cardiac output allows better understanding of hemodynamics
You are called to the bedside of a patient with MOF because of an acute arrhythmia.
What do you think happened to her CO?

1. It decreased – look at the HR
2. It is unchanged
3. It increased – look at the HR
4. The BP indicates that the CO decreased
Cardiac output decreased substantially
What is the mechanism for the reduced CO?

1. The SVR is reduced which led to this effect
2. The stroke volume is decreased
3. The blood pressure reduction led to the reduced CO
1. The stroke volume decreased
With amiodarone, heart rate decreases, and so does stroke volume and cardiac output.
Another patient with an arrhythmia
What happened to this patient’s CO?

- It is unchanged, since blood pressure is not significantly changed
- It probably decreased significantly because of the atrial fibrillation
- It probably increased because of the increase in heart rate
What about the patient’’s SV?

1. It is unchanged since the patient’s CO did not decrease
2. It is increased to compensate for the high heart rate
3. It must have decreased
Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008

R. Phillip Dellinger, MD; Mitchell M. Levy, MD; Jean M. Carlet, MD; Julian Bion, MD; Margaret M. Parker, MD; Roman Jaeschke, MD; Konrad Reinhart, MD; Derek C. Angus, MD, MPH; Christian Brun-Buisson, MD; Richard Beale, MD; Thierry Calandra, MD, PhD; Jean-Francois Dhainaut, MD; Horwig Gerlach, MD; Maureen Harvey, RN; John J. Marini, MD; John Marshall, MD; Marco Ranieri, MD; Graham Ramsay, MD; Jonathan Sevransky, MD; B. Taylor Thompson, MD; Sean Townsend, MD; Jeffrey S. Vender, MD; Janice L. Zimmerman, MD; Jean-Louis Vincent, MD, PhD; for the International Surviving Sepsis Campaign Guidelines Committee
Advantage to volumetric indices

Technologies exist for measurement of flow, volumetric parameters, fluid responsiveness, and EVLW. They exist in the ICU and should be more accessible during the early resuscitation period and should be more accessible during the early resuscitation period.
A complete picture of hemodynamic status cannot be obtained by incomplete data

We cannot obtain enough information using blood pressure, CVP and ScvO2
Thank you for your attention