Hemodynamic monitoring should be kept as simple as possible. But not simpler!

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ESICM Berlin 2007
Disclosure

The speaker cooperates with the following companies

Drager-Siemens
iMDsoft
InSightec
Pulsion*
The hemodynamic status of critically ill patients is characterized by:

- Often being complex (hypovolemia, myocardial depression or both).
- Rarely does the ‘diagnosis’ tell you what is the main physiological disturbance.
- Complicating co-morbidities.
- Therapeutic conflicts (e.g., hemodynamic instability and ARDS).
In such situations therapeutic decisions are often critical since:

- Occult hypoperfusion is associated with increased mortality.
- Volume overload may cause or worsen
  - heart failure
  - pulmonary and other organ edema
  - intra-abdominal hypertension
and is associated with increased mortality.
HEMODYNAMIC MONITORING

- The ‘minimalist’ approach
- The ‘consensus’ approach
- The ‘advanced cardiopulmonary monitoring’ approach
“72yo man with a significant cardiac history who underwent removal of massive renal cell carcinoma and a necrotic gallbladder.

Following 24 hrs - oliguric, hypotensive but responsive to fluids. **20L positive balance over 24h.** Blew up like a balloon, but interestingly urine output still 45ml/hr. On a bit of noradrenaline…

I came into the unit at 0400 to start CVVHD……..whilst I was scrubbing he arrested.”
Question: “Don’t you think that the patient may have been under-monitored?”

“We actually monitored metabolic function of the liver (lactate), skin perfusion (clinical assessment), urine output - all good measures of organ function and perfusion rather than simply arbitrary pressures, volumes or flows.

The biggest problem with ALL the fancy numbers (and even the non-fancy numbers like CVP & MAP), is that in the individual patient…you have NO idea what the "best" number is supposed to be….

So then we get back to old fashioned clinical examination, measurement of indices of tissue function, and careful therapeutic trials…”
It seems that the many negative reports on the inadequacy of advanced hemodynamic monitoring (mainly the PAC) have created confusion and misunderstandings that lead to many instances of unjustified insufficient monitoring of critically ill patients.

I call this the “Back to Nature” movement....
Clinical evaluation compared to pulmonary artery catheterization in the hemodynamic assessment of critically ill patients
Eisenberg PR et al, Crit Care Med 1984; 12: 349

Assessing hemodynamic status in critically ill patients: Do physicians use clinical information optimally?

Therapeutic impact of PAC in the ICU
Steingrub et al, Chest 1991; 99: 1451

PAC in critically ill patients: A prospective analysis of outcome changes associated with catheter-prompted changes in therapy

Hemodynamic and pulmonary fluid status in the trauma patient: are we slipping?
The PiCClin Study
Part I: Clinicians' prediction of advanced cardiopulmonary variables in critically ill patients.

Methods: Cardiopulmonary assessment was done in critically ill patients from 12 European ICU's just before the use of the PiCCO monitoring system (Pulsion, Germany).

One to four physicians per patient independently predicted the cardiac output (CO), systemic vascular resistance (SVR), preload (indexed global end-diastolic volume - GEDVi), and indexed extravascular lung water (EVLWi).
The patient population included 206 patients, which were evaluated by 166 residents and 146 specialists (total of 315 questionnaires).

The main reasons for using the PiCCO included:

- Unclear fluid status (136)
- Suspected sepsis / septic shock (89)
- Respiratory failure (59)
- Cardiogenic shock (24)
- Renal failure (32)
- Other (21)
The accuracy of predicted cardiopulmonary parameters

<table>
<thead>
<tr>
<th></th>
<th>CO (n=315)</th>
<th>SVR (n=312)</th>
<th>GEDVi (n=314)</th>
<th>EVLWi (n=304)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Underestimation &gt;20%</strong></td>
<td>170 (54%)</td>
<td>46 (14.7%)</td>
<td>97 (30.9%)</td>
<td>83 (27.3%)</td>
</tr>
<tr>
<td><strong>Within ± 20%</strong></td>
<td>110 (34.9%)</td>
<td>107 (34.3%)</td>
<td>154 (49%)</td>
<td>124 (40.8%)</td>
</tr>
<tr>
<td><strong>Overestimation &gt;20%</strong></td>
<td>35 (11.1%)</td>
<td>159 (51%)</td>
<td>63 (20.1%)</td>
<td>97 (31.9%)</td>
</tr>
</tbody>
</table>

The PiCClin Study
<table>
<thead>
<tr>
<th></th>
<th>Residents (n=165)</th>
<th>Seniors (n=144)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Underestimation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;20%</td>
<td>87</td>
<td>80</td>
</tr>
<tr>
<td><strong>Within ± 20%</strong></td>
<td>59</td>
<td>50</td>
</tr>
<tr>
<td><strong>Overestimation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;20%</td>
<td>19</td>
<td>14</td>
</tr>
</tbody>
</table>
Conclusions PiC Clin (part I):

Intensivists’ predictions of advanced cardiopulmonary parameters based on clinical evaluation and conventional monitoring alone is inaccurate and is not improved by experience.

Intensivists often tend to assume that CO is lower and that SVR is higher than the actually measured values. This may be due to a common misinterpretation of hypotension.
In the absence of further hemodynamic information, what would be your therapeutic decision?

| Fluid loading | Red blood cells | Inotropic agent | Vaso-constrictor | Diuretic | Dialysis/filtration | Other |
|---------------|-----------------|-----------------|------------------)|----------|-------------------|-------|

The PiCClin Study
Part II: Change of therapeutic plan following advanced cardiopulmonary monitoring in critically ill patients
## The PiCClin Study

**Part II: Change of therapeutic plan following advanced cardiopulmonary monitoring in critically ill patients.**

<table>
<thead>
<tr>
<th>(n=315)</th>
<th>Original therapeutic plan</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pursued</td>
</tr>
<tr>
<td>Fluids</td>
<td>67.6%</td>
</tr>
<tr>
<td>Inotropes</td>
<td>78.4%</td>
</tr>
<tr>
<td>Vasoconstrictors</td>
<td>77.5%</td>
</tr>
<tr>
<td>Diuretics</td>
<td>86.1%</td>
</tr>
</tbody>
</table>
Conclusions (part II):

The measurement of advanced cardio-pulmonary parameters caused both specialists and residents to make considerable changes in therapeutic decisions that were made based on clinical judgment and conventional monitoring alone.

The PiCClin Study
So,

which parameters should we monitor?

The ‘consensus’ approach
Should we monitor preload and fluid responsiveness in shock?

How and when should we monitor stroke volume or cardiac output in shock?

What is the evidence for using hemodynamic monitoring to direct therapy in shock?
We do not recommend the routine use of the PAC for patients in shock.

We recommend that preload measurement alone not be used to predict fluid responsiveness.

We recommend that in shock, low values of commonly used static measures of preload such as CVP, RAP, PAOP (for example < 4 mm Hg) and ventricular volumes, should lead to immediate fluid resuscitation with careful monitoring.
Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge* 

David Osman, MD; Christophe Ridel, MD; Patrick Ray, MD; Xavier Monnet, MD, PhD; Nadia Anguel, MD; Christian Richard, MD; Jean-Louis Teboul, MD, PhD  

CCM 2007 35:64-8

➢ The significance of PAOP and the CVP to predict fluid responsiveness was poor. (AUC of 0.58 and 0.63, respectively).

➢ A CVP of <8 mm Hg and a PAOP of <12 mm Hg predicted volume responsiveness with a positive predictive value of only 47% and 54%, respectively.

Figure 2. Individual values (open circles) and mean ± sd (closed circles) of pre-infusion central venous pressure (CVP) (both expressed in millimeters of mercury) in responders (R) and nonresponders (NR).

Figure 3. Individual values (open circles) and mean ± sd (closed circles) of pre-infusion pulmonary artery occlusion pressure (PAOP) (both expressed in millimeters of mercury) in responders (R) and nonresponders (NR).
Global end-diastolic volume as an indicator of cardiac preload in patients with septic shock.
The very frequent question “Will my patient respond to fluids?” cannot be accurately answered by any ‘preload’ parameter.

*F Michard et al, Chest 2003*
Functional hemodynamic parameters (SPV, PPV, SVV) are the most sensitive parameters for the assessment of fluid responsiveness in mechanically ventilated patients.

Responsive

Non-responsive
We do not recommend the routine use of dynamic measures of fluid responsiveness (including but not limited to pulse pressure variation, aortic flow changes, systolic pressure variation, respiratory systolic variation test, and collapse of vena cava).

There may be some advantage to these measurements in highly selected patients.
Predicting fluid responsiveness in patients undergoing cardiac surgery: functional haemodynamic parameters including the Respiratory Systolic Variation Test and static preload indicators

S. Preisman*, S. Kogan, H. Berkenstadt and A. Perel†
We recommend a fluid challenge to predict fluid responsiveness...with a goal of obtaining a rise in CVP of at least 2 mmHg.

A positive response includes measures of improved cardiac function and tissue perfusion.
## Hemodynamic response to fluid loading

<table>
<thead>
<tr>
<th>Patients</th>
<th>Definition of Responders</th>
<th>N</th>
<th>Challenge</th>
<th>Responders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preisman S (2005)</td>
<td>Cardiac surgery</td>
<td>&gt; 15% SV</td>
<td>18</td>
<td>250 mL colloids</td>
</tr>
<tr>
<td>Hofer CK (2005)</td>
<td>Cardiac surgery</td>
<td>&gt; 25% SVI</td>
<td>35</td>
<td>10 mL/kg (IBW) 6% HES</td>
</tr>
<tr>
<td>Swensen CH (2006)</td>
<td>Abdominal surgery</td>
<td>Increase in CO</td>
<td>10</td>
<td>25 mL/kg of Ringer</td>
</tr>
<tr>
<td>Tavernier B (1998)</td>
<td>Sepsis w. circulatory failure</td>
<td>&gt; 15% SVI</td>
<td>15</td>
<td>500-1000 mL HES</td>
</tr>
<tr>
<td>Michard F (2000)</td>
<td>Sepsis w. circulatory failure</td>
<td>&gt; 15% CI</td>
<td>40</td>
<td>500 mL HES</td>
</tr>
<tr>
<td>Michard F (2003)</td>
<td>Septic shock</td>
<td>&gt; 15% SVI</td>
<td>27</td>
<td>500 mL HES</td>
</tr>
<tr>
<td>Feissel M (2005)</td>
<td>Septic shock</td>
<td>&gt; 15% CI</td>
<td>20</td>
<td>8 ml/kg HES</td>
</tr>
<tr>
<td>Monnet X (2005)</td>
<td>Critically ill w. circulatory failure</td>
<td>&gt; 15% increase in ABF (Doppler)</td>
<td>38</td>
<td>500 ml NS</td>
</tr>
<tr>
<td>Vallee F (2005)</td>
<td>Critically ill w. circulatory failure</td>
<td>&gt; 10% increase in SVI</td>
<td>51</td>
<td>4 ml/kg colloid X 2</td>
</tr>
<tr>
<td>Heenan S (2006)</td>
<td>Critically ill w. circulatory failure</td>
<td>&gt; 15% in CO</td>
<td>21</td>
<td>1 L Ringer or 500 mL HES</td>
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<tr>
<td>Lafanechère A (2006)</td>
<td>Critically ill w. circulatory failure</td>
<td>&gt; 15% increase in ABF (Doppler)</td>
<td>22</td>
<td>PLR and 500 ml NS</td>
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<tr>
<td>Osman D (2007)</td>
<td>Septic</td>
<td>&gt; 15% in CO</td>
<td>96</td>
<td>500 mL HES</td>
</tr>
</tbody>
</table>

### 50% of critically ill patients are loaded with fluids unnecessarily!
“The effects of a typical rapid volume infusion on hemodynamics and LV areas is surprisingly small.”
In patients who did not increase their CO in response to fluid loading, volume kinetic analysis suggested that only 25% of the infused fluid resided in the central fluid space at the end of the infusion and only 3% at the end of the study.
We do not recommend routine measurement of CO for patients with shock.

In the absence of hypotension…, we recommend that a marker of inadequate perfusion be measured (decreased ScvO2, SvO2, increased blood lactate, increased base deficit, perfusion-related low pH).

We recommend instituting goal-directed therapy without delay, in patients presenting with septic shock, particularly where ScvO2 is below 70%.
Initial resuscitation

Begin resuscitation immediately in patients with hypotension or elevated serum lactate.

Resuscitation goals:

- Central venous pressure: 8-12 mm Hg
- Mean arterial pressure ≥ 65 mm Hg
- Urine output ≥ 0.5 mL.kg⁻¹.hr⁻¹
- Central venous or mixed venous oxygen saturation ≥ 70%

If central venous oxygen saturation or mixed venous oxygen saturation of 70% is not achieved with a central venous pressure of 8-12 mm Hg, then transfuse packed red blood cells to achieve a haematocrit of ≥ 30% and/or administer a dobutamine infusion of up to a maximum of 20 μg.kg⁻¹.min⁻¹.
Cardiac output 6.77 L/min

Is this CO adequate???

ScvO₂ is 60%!
Patient is given dobutamine

Conclusion: CO was high, but not high enough
A man with fever and shortness of breath

ScvO₂ 72%
CVP 9 mmHg
Lactate 48
PaO₂/FiO₂ 75 (PEEP 10)

Following noradrenaline...
- Blood pressure increased to 120/65
- CO increased to 6.5 LPM (CI 3.7)
- ScvO₂ increased to 76%
- SaO₂ increased to 98% and FiO₂ was decreased
- Urine output increased to 60 ml/h
The main reason to measure CO is to identify patients that have low (or high) CO values that are not evident clinically.

The CO and the ScvO$_2$ complement each other.

However, a low CO or a low ScvO$_2$, will tell you that something is wrong but not what is wrong and what should be done about it (fluids? inotropes?).
• The SvO\textsubscript{2} of septic shock patients is mainly normal or even supra-normal due to reduced oxygen extraction.

• Therefore, a normal or high SvO\textsubscript{2} does not necessarily indicate adequate tissue oxygenation.

\[ O_{2ER} \approx 1 - S_{v} O_{2} \]
34 yr female; Very severe respiratory failure; Hemodynamic collapse; On noradrenaline.

<table>
<thead>
<tr>
<th>BP</th>
<th>113 / 67 mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>91 bpm</td>
</tr>
<tr>
<td>Urine</td>
<td>Good</td>
</tr>
<tr>
<td>SaO₂</td>
<td>86% !!!</td>
</tr>
<tr>
<td>ScvO₂</td>
<td>80% !!!</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CI</th>
<th>2.7 l/min/m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>ITBVi</td>
<td>578 ml/m²</td>
</tr>
<tr>
<td>EVLWi</td>
<td>20 ml/kg</td>
</tr>
<tr>
<td>ICG PDR (LiMON)</td>
<td>6.7% (18-25%)</td>
</tr>
</tbody>
</table>

Have we achieved initial resuscitation goals in this patient?
### PICCO

<table>
<thead>
<tr>
<th>PICCOI</th>
<th>2.229</th>
<th>2.182</th>
<th>2.400</th>
<th>2.394</th>
<th>2.882</th>
<th>2.705</th>
<th>3.029</th>
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<table>
<thead>
<tr>
<th>ITEM</th>
<th>689</th>
<th>573</th>
<th>525</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>EVLWI</th>
<th>1228</th>
<th>1293</th>
<th>909</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Picco SVRI</th>
<th>571.6</th>
<th>417.6</th>
<th>411.8</th>
<th>376.5</th>
<th>352.9</th>
<th>352.9</th>
<th>382.4</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Picco SV</th>
<th>47.18</th>
<th>40.52</th>
<th>44.62</th>
<th>42.38</th>
<th>46.9</th>
<th>46.53</th>
<th>54.66</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Picco SWI</th>
<th>27.8</th>
<th>23.8</th>
<th>26.2</th>
<th>24.9</th>
<th>27.6</th>
<th>27.4</th>
<th>32.2</th>
</tr>
</thead>
</table>

### Common IV / Medication

- **Actrapid**: 0 IU
- **Dobutex (Dobutamine)**: 0 mg
- **Dormicum**: 0 mg
- **Heparin**: 0 IU
- **Hydrocortisone**: 300 mg
- **MgSO4**: 0 mg

### Graphs

- **EVLWI**
- **PICCOI**
- **SpO2**
**Sepsis**

44 septic patients

15 patients \( \text{ScvO2} < 70\% \)
29 patients \( \text{ScvO2} > 70\% \)

ScvO2 vs. CO - no correlation (Pearson 0.20)
The influence of early hemodynamic optimization on biomarker patterns of severe sepsis and septic shock*

Emanuel P. Rivers, MD, MPH; James A. Kruse, MD; Gordon Jacobsen, MS; Kant Shah, MD; Manisha Loomba, MD; Ronny Otero, MD; Ed W. Childs, MD
Severe Global Tissue Hypoxia:
Lactate \(\geq 4\) mmol per liter and \(\text{ScvO}_2 < 70\%\)

Moderate Global Tissue Hypoxia:
Lactate \(\geq 2\) and \(< 4\) mmol per liter and \(\text{ScvO}_2 < 70\%\)

Resolved Global Tissue Hypoxia:
Lactate \(< 2\) mmol per liter and \(\text{ScvO}_2 \geq 70\%\)
The end-points of hemodynamic management of the surviving sepsis guidelines are not applicable to critically ill patients in the ICU
A Perel, M Maggiorini, M Malbrain, JL Teboul, J Belda, E Fernández-Mondéjar, M Kirov, J Wendon
(submitted to SCCM 2008)

Study population: 112 critically ill patients from 12 European ICU's who had central venous blood drawn at the time that a PiCCO catheter was inserted (on ICU day 1 in 53 patients, and on day 2 in 21, median=1).

Patients were then divided into 2 groups:
• Group A - All EGDT end-points (MAP≥65 mmHg, CVP≥8 mmHg, ScvO2 ≥70%) present.
• Group B – At least one EGDT end-point below its target value.
The end-points of hemodynamic management of the surviving sepsis guidelines are not applicable to critically ill patients in the ICU.

\( \text{ScvO}_2 \) was \( \geq 70\% \) in 76\% of the patients with lactate >4 mmol/L (n=21).

\( \text{ScvO}_2 \) was \( \geq 70\% \) in 66\% of the patients with lactate <4 mmol/L (n=89).

No correlation was found between lactate levels and \( \text{ScvO}_2 \) \( (r^2=0.0021) \) or between CI and \( \text{ScvO}_2 \) \( (r^2=0.0786) \).

Lactate, pH, heart rate, SVR, GEDV, EVLW, \( \text{PO}_2 /\text{FiO}_2 \) and hemoglobin were not different between the 2 groups.
<table>
<thead>
<tr>
<th>Treatment</th>
<th>Group A (n=54) Goals present</th>
<th>Group B (n=58) Goals absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluid loading</td>
<td>31</td>
<td>30</td>
</tr>
<tr>
<td>RBC</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>Inotropes</td>
<td>12</td>
<td>15</td>
</tr>
<tr>
<td>Vasoconstrictors</td>
<td>19</td>
<td>15</td>
</tr>
<tr>
<td>Diuretics</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>Dialysis/ filtration</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>Pt's with lactate &gt;4</td>
<td>12</td>
<td>9</td>
</tr>
</tbody>
</table>
Would anyone argue that iatrogenic fluid overload is safe?

Would it be more sensible to give guidelines as to when to use more sophisticated hemodynamic monitoring to better titrate fluid input, rather than react post-drowning?
Cumulative fluid balance and EVLW during the resuscitation of a septic patient with chronic heart failure.
More than half of the patients with severe sepsis but without ARDS had increased EVLW, possibly representing sub-clinical lung injury.
Negative fluid balance, if tolerated hemodynamically, is associated with improved end-points, such as reduced EVLW, less ventilator days and possibly better outcome.

Conservative strategy of fluid management improved lung function and shortened the duration of mechanical ventilation and intensive care without increasing non-pulmonary-organ failures.

These results support the use of a conservative strategy of fluid management in patients with acute lung injury.
Not all ARDS patients have high EVLW

35% of ARDS patients had EVLW<7 ml/kg

Some ARDS patients had EVLWI values only slightly increased above normal

25% of ARDS patients had normal EVLW
Hemodynamic monitoring should be kept as simple as possible. 

*But not simpler!*

The hemodynamic management of critically ill patients, especially those who present significant therapeutic conflicts (e.g., heart vs. lungs), necessitates monitoring of a combination of parameters.
“The hottest places in hell are reserved for those who, in times of great crisis, do nothing”

- Dante