Cardiogenic shock: invasive and non-invasive monitoring

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Classification of AHF: ESC 2008

Modified from Filippatos G and Zannad F. Heart Failure Rev 2007

Hypertensive AHF or Vascular AHF or De Novo AHF

Normotensive AHF or Cardiac Failure or Acutely Decompensated Chronic HF

PULMONARY EDEMA

Right Heart Failure

“Hypotensive AHF”/Cardiogenic shock

ACS with Heart Failure
Frequency of CS in AHF registries

EHS HFII
- AdHF: 65%
- Hypertensive HF: 16%
- Right HF: 11%
- Pulmonary oedema: 4%
- Cardiogenic shock: 3%

ALARM-HF
- AdHF: 39%
- Hypertensive HF: 37%
- Right HF: 12%
- Pulmonary oedema: 7%
- Cardiogenic shock: 4%
- High cardiac output failure: 1%

Cardiogenic shock (4% vs. 12%) was significantly different between the two studies.

ALARM-HF vs EHS-HF II: In-Hospital Mortality according to ESC Classification of AHFS

Sample = EHS HF II (3,580), All ALARM-HF patients (4,953)

EuroHeart Survey HFII: Long-term mortality in the different clinical classes of AHF

Harjola et al. Eur Journal Heart Fail 2010 12, 239–248
Classic Criteria for Diagnosis of Cardiogenic Shock

1. **Systemic Hypotension**
   - systolic arterial pressure < 90 mmHg

2. **Persistent Hypotension**
   - at least 30 minutes

3. **Reduced Systolic Cardiac Function**
   - Cardiac index < 2.2 x L/min/m² with support or < 1.8 without support

4. **Tissue Hypoperfusion**
   - Oliguria, cold extremities, confusion

5. **Increased Left Ventricular Filling**
   - Pulmonary capillary wedge pressure > 18 mmHg
Spectrum of Clinical Presentations

Mortality

- Respiratory Distress: 5.6%
- Hypotension: 1.4%
- Hypoperfusion: 65%

- 21%
- 22%
- 70%
- 60%
Table 1. Causes of cardiogenic shock

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute myocardial infarction</td>
<td>Pump failure&lt;br&gt;Large infarction&lt;br&gt;Smaller infarction with preexisting left ventricular dysfunction&lt;br&gt;Infarction extension&lt;br&gt;Severe recurrent ischemia&lt;br&gt;Infarction expansion</td>
</tr>
<tr>
<td>Mechanical complications</td>
<td>Acute mitral regurgitation caused by papillary muscle rupture&lt;br&gt;Ventricular septal defect&lt;br&gt;Free-wall rupture&lt;br&gt;Pericardial tamponade&lt;br&gt;Right ventricular infarction</td>
</tr>
<tr>
<td>Other conditions</td>
<td>End-stage cardiomyopathy&lt;br&gt;Myocarditis&lt;br&gt;Myocardial contusion&lt;br&gt;Prolonged cardiopulmonary bypass&lt;br&gt;Septic shock with severe myocardial depression&lt;br&gt;Left ventricular outflow tract obstruction&lt;br&gt;Aortic stenosis&lt;br&gt;Hypertrophic obstructive cardiomyopathy&lt;br&gt;Obstruction to left ventricular filling&lt;br&gt;Mitral stenosis&lt;br&gt;Left atrial myxoma&lt;br&gt;Acute mitral regurgitation (chordal rupture)&lt;br&gt;Acute aortic insufficiency&lt;br&gt;Acute massive pulmonary embolism&lt;br&gt;Acute stress cardiomyopathy&lt;br&gt;Pheochromocytoma</td>
</tr>
</tbody>
</table>
Trends in incidence of CS

Figure 1. Trends in the incidence rates of cardiogenic shock in patients with AMI.

Goldberg Circulation 2009
Shock Categories

- Predominant LV Failure: 74.5%
- Acute Severe MR: 8.3%
- Ventricular Septal Rupture: 4.6%
- "Isolated" RV Shock: 3.4%
- Tamponade/Rupture: 1.7%
- Other: 1.7%

Shock Registry: Hochman, JACC 2000; 36: 1063
SHOCK Registry: Mortality by Shock Categories

- All (1422): 60.1%
- LVF (1116): 59.2%
- VSR (55): 87.3%
- MR (98): 55.1%
- RVF (40): 55.0%
- Tamp (20): 55.0%
- Other (95): 65.3%

p=0.001 6 Groups; VSR vs each p<0.01

Hochman, JACC 2000; 36: 1063
Potential Methods to Evaluate Cardiac Hemodynamics in Patients With Cardiogenic Shock

- Clinical evaluation
- Biomarkers
- Swan-Ganz Catheter
- Echocardiography
- Pulse waveform methods
- Bioimpedance
Acute pulmonary oedema: clinical characteristics, prognostic factors, and in-hospital management

John T. Parissis, Maria Nikolaou, Alexandre Mebazaa, Ignatios Ikonomidis, Juan Delgado, Fabio Vilas-Boas, Ioannis Paraskevaidis, Antony Mc Lean, Dimitrios Kremastinos, and Ferenc Follath

European Journal of Heart Failure
doi:10.1093/eurjhf/hfq138
Hemodynamic correlates of proportional pulse pressure

- Pulse Pressure
  Systolic BP - Diastolic BP

- Proportional Blood Pressure
  \[ \text{Systolic BP} - \text{Diastolic BP} = \leq 25\% \]

  [Systolic BP]

  \[ = \text{CI} \leq 2.2 \text{ L/min/M}^2 \]

  (JAMA 1989;261:884)
Rapid assessment of hemodynamic status

ADEQUATE PERFUSION?

+ Proportional pulse pressure
  Cool extremities
  Altered mentation
  ACE-I intolerance
  Worsening renal function

- Proportional pulse pressure

CONGESTION?

- Wet and Warm
  Orthopnea
  Rales
  Abnormal BP response to Valsalva maneuver
  Jugular venous pressure
  Abdominojugular reflux
  Hepatomegaly
  Ascites
  Edema

+ Dry and Warm

Dry and Cold
Acute Heart Failure: clinical evaluation

High jugular venous pressure

Peripheral Vasoconstriction

Pulmonary congestion/ oedema

Haemodynamic findings:
Low cardiac output (C.I < 2.2 L/min)
High PCW-pressure (>18 mmHg)
High systemic vascular resistance
Clinical assessment identifies hemodynamic profiles that predict outcomes in patients with AHF

ADEQUATE PERFUSION

CONGESTION

---
A
dry-warm
(N=123)

B
wet-warm
(N=222)

L
dry-cold
(N=16)

C
wet-cold
(N=91)

Nohria et al. JACC 2003;41:1797-1804
A severity scoring system for risk assessment of patients with cardiogenic shock: A report from the SHOCK Trial and Registry

Table III. Stage 1 (Clinical): scoring system without invasive hemodynamics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Points</th>
<th>Variable</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anoxic brain damage</td>
<td>30</td>
<td>Hypoperfusion</td>
<td>14</td>
</tr>
<tr>
<td>Shock on admission</td>
<td>6</td>
<td>Prior CABG</td>
<td>7</td>
</tr>
<tr>
<td>Noninferior MI</td>
<td>3</td>
<td>Creatinine $\geq 1.9$ mg/dL</td>
<td>5</td>
</tr>
<tr>
<td>Age, y†</td>
<td></td>
<td>Systolic BP, mm Hg†</td>
<td></td>
</tr>
<tr>
<td>≤ 55</td>
<td>12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>56-60</td>
<td>11</td>
<td>56-60</td>
<td></td>
</tr>
<tr>
<td>61-65</td>
<td>10</td>
<td>61-65</td>
<td></td>
</tr>
<tr>
<td>66-70</td>
<td>9</td>
<td>66-70</td>
<td></td>
</tr>
<tr>
<td>71-75</td>
<td>8</td>
<td>71-75</td>
<td></td>
</tr>
<tr>
<td>76-80</td>
<td>7</td>
<td>76-80</td>
<td></td>
</tr>
<tr>
<td>81-85</td>
<td>6</td>
<td>81-85</td>
<td></td>
</tr>
<tr>
<td>86-90</td>
<td>5</td>
<td>86-90</td>
<td></td>
</tr>
<tr>
<td>&gt;90</td>
<td>4</td>
<td>&gt;90</td>
<td></td>
</tr>
</tbody>
</table>

*When LVEF is added to this system, noninferior MI has 0 point, and LVEF has 10 points if ≤15%, 7 points if 16% to 25%, 5 points if 26% to 35%, 2 points if 36% to 45%, and 0 point if >45%.
†When dichotomized to assess elderly risk, patients ≥75 years were assigned 9 points and patients <75 years were assigned 0 point.
‡On support measures, including vasopressors, inotropes ± IABP. Obtained before or without IABP support in 76%.

Sleeper et al. Am Heart J 2010;160:443-50
## Evaluation of BNP levels in AHF clinical scenarios

<table>
<thead>
<tr>
<th></th>
<th>No Congestion at Rest</th>
<th>Congestion at Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adequate perfusion at rest</td>
<td>Warm and dry</td>
<td>Warm and wet</td>
</tr>
<tr>
<td></td>
<td>BNP 100–400</td>
<td>BNP ≥600</td>
</tr>
<tr>
<td>Low perfusion at rest</td>
<td>Cool and dry</td>
<td>* Cool and wet</td>
</tr>
<tr>
<td></td>
<td>BNP 400–1000</td>
<td>BNP ≥1000</td>
</tr>
</tbody>
</table>

* Except Acute MR

Omland T. Crit Care Med 2008;36:S17
Novel biomarkers under investigation in AHF and CS

- Biomarkers of cardiac stress: MR-proANP, MR-proADM, copeptin
- Biomarkers of cardiac injury: hs-troponins
- Biomarkers of renal injury: N-GAL, cystatin KIM-1
Usefulness of echocardiography in cardiogenic shock

- Evaluate left ventricular function and myocardium at risk
- Evaluate remote myocardial segments
- Screen for ventricular septal rupture
- Screen for severe mitral regurgitation and proceed to transoesophageal echocardiography as needed
- Look for tamponade/rupture
- Assess right ventricular function
- Look for aortic dissection

Menon V and Hochman J. Heart 2002;88:531–537
Diagnostic & treatment algorithm

Assess volume status
- Treat sustained arrhythmias - brady or tachy
- Mechanical ventilation, as needed
  - correct acidemia
  - correct hypoxemia
- Inotropic/vasopressor support (dopamine)

Acute massive ST↑/extensive evolving Q's or new LBBB

Cath lab immediately available

Yes

Cath lab

ST↑
   ↓
Lytic

No

ST↑
   ↓
Gp Iib/IIia

Aspirin, heparin

Rapid IABP

Coronary angiography ±
left ventricular angiography
Pulmonary artery catheterisation

PTCA for 1, 2 or moderate 3 vessel CAD
GP Iib/IIia antagonist
Coronary stent

No

Emergency echo/colour flow Doppler

Emergency echo/colour flow Doppler

Pump failure
RV, LV, both

Acute severe MR
VSR
Critical AS/MS

Operating room ±
coronary angiography

Aortic dissection tamponade

Cardiac surgery
CABG for severe 3 vessel or left main LAD
Correct mechanical lesions ± CABG

Hochmann Heart 2002
Acute MR after inferior MI as a cause of CS

Female 58 yrs, chest pain, acute dyspnea - satO2 83%
BP 83/65 mmHg
Rales – Killip IV
2-D Method

**Principle**

Stroke volume = End diastolic volume – End systolic volume

LV volumes estimated by Simpson’s method, which is the summation of the volume of stacked cylinders within the LV at end-diastole and end-systole

150 ml - 52 ml = 98 ml
Doppler Method

**Principle**

Flow (stroke volume) = Area * Velocity  
CO = Stroke volume * Heart rate

**Area** of left ventricular outflow tract  
Obtain LVOT dimension in parasternal long axis view

**Simplified formula**

\[
\text{Flow Velocity at LVOT} = (2.1\text{cm})^2 \times 0.785
\]

\[
3.46\text{cm}^2 \times 25\text{cm} = 87\text{ cm}^3
\]

Flow Velocity at LVOT  
Pulsed wave Doppler at LVOT in apical 5 chamber view
Comparison of cardiac output measured with echocardiographic volumes and aortic Doppler methods during mechanical ventilation

Axler et al Intensive Care Medicine 2003;29:208:17
Echocardiography

Advantages
- Non-invasive
- Readily available in the ICU
- Can provide multiple information (etiology, filling pressures, venous pressures, cardiac output)

Disadvantages
- Volume Measurement Dependent Upon Endocardial Visualization
- Doppler Flow measurement less accurate if Aortic Regurgitation
- Not validated in patients with shock
Invasive Hemodynamic Monitoring in CS

Pinsky, Chest 2007;132:2020-2029

Invasive monitoring

Arterial catheterization
- Systolic BP, diastolic BP, MAP, HR, and pulse pressure
- Arterial blood gas analysis
  - pH, PaO₂, SaO₂, PCO₂, hemoglobin
- Arterial pressure waveform analysis
  - Stroke volume, cardiac output, PPV and SVV

Central venous catheterization
- Central venous pressure, venous pressure waveform
  - ("v" waves), respiratory variations
- Central venous blood gas analysis
  - pH, Pcvo₂, Scvo₂, Pcvo₂, hemoglobin
- Thermodilution indices (when coupled to an arterial thermal sensor)
  - Stroke volume, cardiac output, intrathoracic blood volume, global end-diastolic volume, and DO₂

Pulmonary artery catheter
- Systolic BP, diastolic BP, MAP, pressure waveform
  - ("v" waves), and Ppao
- Mixed venous blood gas analysis
  - pH, PνO₂, SνO₂, PνCO₂, hemoglobin
- Thermodilution cardiac output (by thermodilution either intermittent or continuous)
  - Stroke volume, cardiac output, RV ejection fraction, and RV end-diastolic volume

Esophageal Doppler echocardiographic monitoring
- Stroke volume, cardiac output, and SVV
The Pulmonary Artery Catheter

William Ganz and H.J.C. Swan

Hemodynamic Parameters

- Systemic Vascular Resistance (SVR)
- Cardiac Output (CO)
- Mixed Venous Oxygen Saturation (SvO2)
- Pulmonary Capillary Wedge Pressure (PCWP)
- Central Venous Pressure (CVP)
Swan-Ganz Catheter: Differentiating Types of Shock

<table>
<thead>
<tr>
<th>Physiologic variable</th>
<th>Preload</th>
<th>Pump function</th>
<th>Afterload</th>
<th>Tissue perfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical measurement</td>
<td>Pulmonary capillary wedge pressure</td>
<td>Cardiac output</td>
<td>Systemic vascular resistance</td>
<td>Mixed venous oxygen saturation</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>↑ (circled)</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Distributive</td>
<td>↓ or ↔</td>
<td>↑</td>
<td>↓ (circled)</td>
<td>↑</td>
</tr>
</tbody>
</table>
Vasodilators in Mitral Regurgitation

PCW and LV pressures

Changes in the magnitude of the peak 'V' wave and mean PCW during nitroprusside infusion.

Chatterjee et al, Circulation 1973; 48:684-690
Clinical Application of Pulmonary Artery “Bedside” Monitoring in High Risk Patients

- Pulmonary artery catheters have been used in ICUs to:
  Determine the hemodynamic causes of hypotension and shock
- Differentiate left heart failure from increased permeability pulmonary edema
- Optimizing oxygen delivery through increasing cardiac output
- Optimizing volume status
- PA catheters became used indiscriminately, often by inexperienced personnel
Use of PA Catheters in Shock or ARDS (French PA Catheter Study Group)

676 patients with shock (septic), ARDS or both

"Even if the purpose of monitoring with PAC is ultimately to save lives, it would be unrealistic to believe that the prognosis of patients could be improved by its presence alone".

Richard et al, JAMA 2003; 290:2713-2720
Critically ill Patients in Intensive Care (PAC-Man Study)

n=1,041

Extremely Poor Prognosis

Log rank test (stratified by stratum): $\chi^2=0.77, p=0.381$

Lancet 2005; 366:472-477
Evaluation Study of CHF and PA Catheterization Effectiveness (ESCAPE)

Patients with Severe Symptomatic CHF

JAMA 2005; 294:1625-1633
Cardiac power is the strongest independent hemodynamic correlate of in-hospital mortality in patients with cardiogenic shock. Increasing age and female gender are independently associated with lower cardiac power.

### Hemodynamic variables and mortality in cardiogenic shock: a retrospective cohort study


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**Adjusted multivariate logistic regression models to detect independent associations between hemodynamic variables and 28-day mortality**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Wald</th>
<th>RR</th>
<th>95% Con Int</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model I - including cardiac index</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CI time integral (l/m²/h)</td>
<td>6.658</td>
<td>0.914</td>
<td>0.854-0.979</td>
<td>0.01*</td>
</tr>
<tr>
<td>CVP time integral (mmHg*min/h)</td>
<td>4.010</td>
<td>0.995</td>
<td>0.99-1</td>
<td>0.06</td>
</tr>
<tr>
<td>SVRI time integral (dyne*s/cm²/m² *min/h)</td>
<td>3.832</td>
<td>1</td>
<td>1-1</td>
<td>0.06</td>
</tr>
<tr>
<td>MAP time integral (mmHg*min/h)</td>
<td>2.914</td>
<td>1.003</td>
<td>1-1.006</td>
<td>0.09</td>
</tr>
<tr>
<td>HR time integral (bpm/min/h)</td>
<td>1.833</td>
<td>1.001</td>
<td>1-1.001</td>
<td>0.18</td>
</tr>
<tr>
<td>SvO₂ time integral (%*min/h)</td>
<td>0.069</td>
<td>1</td>
<td>0.998-1.002</td>
<td>0.79</td>
</tr>
<tr>
<td>MPAP time integral (mmHg*min/h)</td>
<td>0.001</td>
<td>1</td>
<td>0.998-1.002</td>
<td>0.97</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Wald</th>
<th>RR</th>
<th>95% Con Int</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model II - including cardiac power index</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPI time integral (W/m² *min/h)</td>
<td>6.281</td>
<td>0.648</td>
<td>0.462-0.91</td>
<td>0.01*</td>
</tr>
<tr>
<td>MAP time integral (mmHg*min/h)</td>
<td>4.106</td>
<td>1.003</td>
<td>1-1.007</td>
<td>0.08</td>
</tr>
<tr>
<td>CVP time integral (mmHg*min/h)</td>
<td>3.076</td>
<td>1.006</td>
<td>1-1.001</td>
<td>0.09</td>
</tr>
<tr>
<td>SVRI time integral (dyne*s/cm²/m² *min/h)</td>
<td>2.736</td>
<td>1</td>
<td>1-1</td>
<td>0.1</td>
</tr>
<tr>
<td>HR time integral (bpm/min/h)</td>
<td>2.072</td>
<td>1.001</td>
<td>1-1.001</td>
<td>0.15</td>
</tr>
<tr>
<td>MPAP time integral (mmHg*min/h)</td>
<td>0.086</td>
<td>1</td>
<td>0.998-1.002</td>
<td>0.77</td>
</tr>
<tr>
<td>SvO₂ time integral (%*min/h)</td>
<td>0.002</td>
<td>1</td>
<td>0.998-1.002</td>
<td>0.97</td>
</tr>
</tbody>
</table>

All models were adjusted for age, admission year, mean catecholamine (epinephrine, norepinephrine, dobutamine, and milrinone) dosages and SAPS II (excl. the systolic arterial blood pressure and heart rate count). Variables are ranked (top to bottom) according to the value of the Wald statistics. * significant association with 28 day-mortality; 192 (77.2%) patients were monitored with a pulmonary arterial catheter. CI = cardiac index; Con Int = confidence interval; CPI = cardiac power index; CVP = central venous blood pressure; HP = hourly portion; HR = heart rate; MAP = mean arterial blood pressure; MPAP = mean pulmonary arterial blood pressure; RR = relative risk; SvO₂ = mixed venous oxygen saturation; SVRI = systemic vascular resistance index.

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**Key messages**

- Despite the key role of hemodynamic goals, there are few data addressing the question of whether hemodynamic variables are associated with patient mortality or should be used as treatment goals in cardiogenic shock.
- During the first 24 hours after intensive care unit admission, cardiac index and cardiac power index are the most important hemodynamic variables separately associated with 28-day mortality in cardiogenic shock patients.
- A cardiac index of 3 L/min/m² and a cardiac power index of 0.8 W/m² were best predictive of 28-day mortality.
- Randomized controlled trials are required to evaluate whether targeting these levels as early resuscitation endpoints can improve mortality in cardiogenic shock.
### Table. Current Indications for Use of the Swan-Ganz Catheter

<table>
<thead>
<tr>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not indicated as routine pulmonary artery catheterization in high-risk cardiac and noncardiac patients</td>
</tr>
<tr>
<td>Indicated in patients with cardiogenic shock during supportive therapy</td>
</tr>
<tr>
<td>Indicated in patients with discordant right and left ventricular failure</td>
</tr>
<tr>
<td>Indicated in patients with severe chronic heart failure requiring inotropic, vasopressor, and vasodilator therapy</td>
</tr>
<tr>
<td>Indicated in patients with suspected “pseudosepsis” (high cardiac output, low systemic vascular resistance, elevated right atrial and pulmonary capillary wedge pressures)</td>
</tr>
<tr>
<td>Indicated in some patients with potentially reversible systolic heart failure such as fulminant myocarditis and peripartum cardiomyopathy</td>
</tr>
<tr>
<td>Indicated for the hemodynamic differential diagnosis of pulmonary hypertension</td>
</tr>
<tr>
<td>Indicated to assess response to therapy in patients with precapillary and mixed types of pulmonary hypertension</td>
</tr>
<tr>
<td>Indicated for the transplantation workup</td>
</tr>
</tbody>
</table>

*Chatterjee et al, Circulation 2009; 119:147-152*
ESC Guidelines for PAC use in AHF

• Usually unnecessary for diagnosis

• Distinguish between a cardiogenic and non-cardiogenic mechanism in complex patients with concurrent cardiac and pulmonary disease

• In hemodynamically unstable patients not responding to traditional treatment,

• In patients with hypoperfusion (to exclude hypovolemia)

• Level IIa, LoE C
The validity of trans-esophageal Doppler ultrasonography as a measure of cardiac output in critically ill adults

Dark and Singer, Intensive Care Med 2004

Abstract Objective: To determine the validity of the esophageal Doppler monitor (EDM) and echo-esophageal Doppler (Echo-ED) in measuring cardiac output in the critically ill. Design: Systematic search of relevant international literature and data synthesis. Search strategy: Literature search (1989–2003) using Ovid interface to Medline, Embase and Cochrane databases aimed at finding studies comparing EDM or Echo-ED cardiac output with that derived from simultaneous pulmonary artery thermodilution (PAC_TD) with Bland Altman measures of validity. Patients: Critically ill adults in operating departments or intensive care units. Data synthesis: Summary validity measures synthesized from Bland Altman analyses included pooled median bias and the median percentage of clinical agreement (PCA) derived from the limits of agreement. Main results: Eleven validation papers for EDM (21 studies) involving 314 patients and 2,400 paired measurements. The pooled median bias for PAC_TD versus EDM was 0.19 l/min (range -0.69 to 2.00 l/min) for cardiac output (16 studies), and 0.6% (range 0–2.3%) for changes in cardiac output (5 studies). The pooled median percentage of clinical agreement for PAC_TD versus EDM was 52% (interquartile range 42–69%) for cardiac output and 86% (interquartile range 55–93%) for changes in cardiac output. These differences in PCA were significant (p=0.03 Mann-Whitney) for bolus PAC_TD as the clinical “gold standard”. We found an insufficient number of studies (2 papers) to assess the validity of Echo-ED. Conclusions: The esophageal Doppler monitor has high validity (no bias and high clinical agreement with pulmonary artery thermodilution) for monitoring changes in cardiac output.

Keywords Cardiac output · Trans-esophageal Doppler ultrasonography · Esophageal Doppler monitoring
Reliability of a new algorithm for continuous cardiac output determination by pulse-contour analysis during hemodynamic instability.

Godge et al Crit Care Med 2002;30:52-8
Comparison of cardiac output measurements in critically ill patients: Flotrac/Vigileo vs transthoracic Doppler echocardiography

McLean et al. Anaesth Intensive Care 2011; 39: 590-598
Pulse Waveform Methods

Advantages
• Less-Invasive Than Thermodilution
• Real Time/ Repetitive Monitoring

Disadvantages
• Needs Usually Recalibration (except some devices e.g Vigileo)
• Dependent on Compliance of Arterial Tree
• Little Validation in Patients with Shock
Accurate, Noninvasive Continuous Monitoring of Cardiac Output by Whole-Body Electrical Bioimpedance

Figure 1. Recordings of the interrelation between impedance variations and hemodynamic parameters, according to Epstein et al. and Xue et al.

Figure 2. Wrist-ankle configuration of the electrodes in WBBI. I = electric current; V = electric voltage.

Figure 3. Plot of CI values measured by the NICO and thermodilution.

Cotter et al. Chest 2004;125;1431-1440
Comparison of Cardiac Output Determined by Bioimpedance, Thermodilution, and the Fick Method

Figure 1 Bland-Altman plot (top) of bias and precision. Linear regression line (bottom): $y = 0.6976x + 1.2475$, $r^2 = 0.5711$.

Abbreviations: BI, bioimpedance; CO, cardiac output; TD, thermodilution.

Figure 2 Cardiac outputs determined by the Fick, thermodilution, and bioimpedance methods for each patient in whom all 3 methods were used.

Abbreviations: BI, bioimpedance; CO, cardiac output; TD, thermodilution.
Bioimpedance

Advantages
• Less Invasive
• Can perform repetitive measures

Disadvantages
• Not routinely available in the intensive care unit
• Multiple competing methodologies
• Potential limitations: obesity, pacemakers, AR
• Little Validation in Patients with Shock
Is There Ideal Cardiac Output Monitoring Technique?

- Precise
- No bias
- Non-invasive
- Readily available in the ICU
- Leads to treatment changes/improvement in outcome

There is still no ideal method
Take home messages

• CS is an urgent condition with high short- and long-term mortality.
• Recognition of etiology is very essential for the effective management.
• Clinical and hemodynamic evaluation remain the gold standard methods for patient monitoring.
• ECHO is very useful non-invasive technique to recognize early etiology and guide the treatment.
• New techniques and biomarkers are promising as non-invasive evaluation methods but their clinical use is limited in CS.
• More prospective trials are needed in this field.