Key words: cardiogenic shock, inotrope, intra-aortic balloon pump, ventricular assist device

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Severe heart failure, cardiogenic shock (CS), and pericardial tamponade are life-threatening conditions, constituting the most severe forms of acute heart failure (AHF). In contrast to patients with chronic heart failure, AHF patients have recourse to very few effective treatments to improve clinical outcomes. This could be explained by the lack of large randomized trials in this setting over the last 2 decades. Recently, guidelines have become available to address the management of AHF, and surveys and registries have generated important information concerning the clinical characteristics and prognoses of patients with AHF syndrome (including CS).

EPIDEMIOLOGICAL CHARACTERISTICS OF CARDIOGENIC SHOCK

The main cause of CS is ischemic cardiomyopathy. Mortality rates for CS vary from 25% to 60%.

Moreover, CS remains the leading cause of death in patients hospitalized with acute myocardial infarction (AMI).

Table 1 on page 154 synthesizes reported incidence and management of CS in heart failure.
Table 1.
Reported Incidence and Management of Cardiogenic Shock in Heart Failure Registries

<table>
<thead>
<tr>
<th>Registry</th>
<th>Sample Size</th>
<th>Type of Patients</th>
<th>Incidence of CS, %</th>
<th>In-Hospital Mortality Rate of CS, %</th>
<th>Use of IABP, %</th>
<th>Use of LVAD, %</th>
<th>Use of IV Inotropes, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALARM-HF</td>
<td>4,953</td>
<td>All patients with AHF</td>
<td>11.7</td>
<td>NR</td>
<td>4.8</td>
<td>NR</td>
<td>39</td>
</tr>
<tr>
<td>EuroHeart Failure survey</td>
<td>11,327</td>
<td>Chronic HF and AHF</td>
<td>&lt;1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>7.2</td>
</tr>
<tr>
<td>EFICA&lt;sup&gt;5&lt;/sup&gt;</td>
<td>599</td>
<td>All patients with AHF admitted to CCU/ICU</td>
<td>29</td>
<td>58</td>
<td>NR</td>
<td>NR</td>
<td>53</td>
</tr>
<tr>
<td>Italian survey&lt;sup&gt;4&lt;/sup&gt;</td>
<td>2,807</td>
<td>All patients with AHF</td>
<td>7.7</td>
<td>25</td>
<td>1.2</td>
<td>NR</td>
<td>24.6</td>
</tr>
<tr>
<td>OPTIMIZE-HF</td>
<td>48,612</td>
<td>Chronic HF and AHF</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>7</td>
</tr>
<tr>
<td>SHOCK Trial Registry&lt;sup&gt;2&lt;/sup&gt;</td>
<td>1,190</td>
<td>CS complicating acute MI</td>
<td>100</td>
<td>60</td>
<td>53</td>
<td>0.8&lt;sup&gt;b&lt;/sup&gt;</td>
<td>70.1</td>
</tr>
<tr>
<td>EHFS-II&lt;sup&gt;45&lt;/sup&gt;</td>
<td>3,580</td>
<td>All patients with AHF</td>
<td>4</td>
<td>40</td>
<td>2.2&lt;sup&gt;c&lt;/sup&gt;</td>
<td>NR</td>
<td>10.2&lt;sup&gt;d&lt;/sup&gt; (dobutamine only)</td>
</tr>
<tr>
<td>ADHERE&lt;sup&gt;24&lt;/sup&gt;</td>
<td>105,388</td>
<td>Chronic HF and AHF</td>
<td>NR</td>
<td>NR</td>
<td>&lt;1&lt;sup&gt;e&lt;/sup&gt;</td>
<td>NR</td>
<td>NR</td>
</tr>
</tbody>
</table>

AHF, acute heart failure; CCU, cardiac care unit; HF, heart failure; IABP, intra-aortic balloon pump; LVAD, left ventricular assist device; MI, myocardial infarction; NR, not reported; SC, cardiogenic shock.

<sup>a</sup> Thirty-two percent of men but only 15% of women had left ventricular systolic dysfunction reported as severe.

<sup>b</sup> Value is based on 856 patients with available data.

<sup>c</sup> Value corresponds to 30.9% of patients with cardiogenic shock.

<sup>d</sup> Value corresponds to 44.6% of patients with cardiogenic shock.

<sup>e</sup> Value corresponds to 2% among the 19,754 patients admitted to CCU/ICU.
Figure 1.
Mortality of patients with cardiogenic shock according to cause.

In theory, inotropic agents can improve hemodynamic parameters by increasing cardiac output and reducing left and right ventricular filling pressures. Figure 2 on page 156 depicts the rationale for using inotropic agents in AHF, as reported in the 2005 European Society of Cardiology (ESC) guidelines. Inotropic agents are indicated in the presence of peripheral hypoperfusion (hypotension and decreased renal function) with or without congestion and in cases of pulmonary edema refractory to diuretics and vasodilators at optimal dose (class IIa recommendation; level of evidence: C). Despite their hemodynamic benefits and ability to improve mitochondrial function of noninfarcted myocardium, inotropes increase oxygen demand in a failing heart with limited supply and therefore may provoke arrhythmias and lead to cellular disruption and necrosis. Thus, the lowest dose of inotropes should be used in CS. Moreover, the ALARM-HF registry suggests that the use of inotropic agents in AHF
Figure 2.
Rationale for use of inotropic drugs in acute heart failure.

ACEI, angiotensin-converting enzyme inhibitor; AHF, acute heart failure; BNP, brain natriuretic peptide; CPAP, continuous positive airway pressure; NTG, nitroglycerine; PDEI, phosphodiesterase inhibitor; SBP, systolic blood pressure.

Adapted with permission from European Heart Journal.

syndromes may increase mortality regardless of the patient's severity of disease.

Evidence is lacking to support recommendations for the use of novel inotropic agents in CS. It is increasingly suggested that dopamine should be avoided in patients with CS.12,13 Only one small observational study showed that administration of vasopressin in patients with CS due to AMI increased mean arterial pressure without negative impact on cardiac output.14 Levosimendan could be of interest for patients with CS,15,16 but further study is needed. The phosphodiesterase inhibitors enoximone and milrinone have been shown to increase cardiac index in patients with CS, but there are no data supporting superiority of these agents in comparison to catecholamines.

Of note, the updated ESC guidelines19 do not recommend vasopressors (norepinephrine) as first-line agents except in the case of CS when the combination of an inotropic
agent and fluid challenge fails to restore the systolic blood pressure to more than 90 mm Hg and there is inadequate organ perfusion despite an improvement in cardiac output. Patients with sepsis complicating AHF may require vasopressors. Because systemic vascular resistance is usually high in cases of CS, all vasopressors should be used with caution and discontinued as soon as possible. Norepinephrine can be used with any of the above-mentioned inotropic agents in CS, ideally via a central line.

REVASCULARIZATION IN CARDIOGENIC SHOCK COMPLICATING ACUTE MYOCARDIAL INFARCTION

Over the last 2 decades, the morbidity and short-term mortality of AMI have been substantially reduced by the use of reperfusion therapy. However, CS remains the leading cause of death in patients hospitalized with AMI. Several controlled trials have attempted to resolve the issue of revascularization in CS complicating AMI. The SHOCK trial demonstrated that in patients with CS, emergency revascularization did not significantly reduce overall mortality at 30 days; however, after 6 months there was a significant survival benefit. This finding led the authors to recommend early revascularization for patients with AMI complicated by CS. A follow-up study of the patients included in the SHOCK trial showed that a strategy of early revascularization resulted in a 13.2% absolute and a 67% relative improvement in 6-year survival compared with initial medical stabilization. Another recent article reported results from the National Registry of Myocardial Infarction (NRMI) in the United States. The study showed that among 293,633 patients included in the registry, CS occurred in 2.3% of patients younger than 75 years and in 3.1% of those 75 years or older. In this population, the use of percutaneous coronary intervention for patients with CS was associated with improved in-hospital survival (27.4% vs 54.4%).

DEVICE THERAPY

Recent guidelines state that temporary circulatory assistance may be indicated (1) in patients with AHF who are not responding to conventional therapy and where there is a potential for myocardial recovery or (2) as a bridge to heart transplant or interventions that may result in significant recovery of heart function (class IIb recommendation; level of evidence: B).

Early mechanical device therapy may be useful in patients who have not responded to other therapies during the first 6 to 12 hours after presentation. Patients who may be candidates for device therapy include those with severe and persistent hypotension or hypoperfusion despite the use of inotropes, urine output less than 30 mL/h, decreasing oxygen saturation, persistent ischemia, or cold or mottled skin. Figure 3 on page 158 depicts the selection of candidates for LV assist devices as proposed by the 2005 ESC guidelines and updated in 2008. Moreover, expert opinion indicates that device therapy should be applied as soon as needed to shorten tissue hypoperfusion and to avoid the potential detrimental effect of catecholamines. There are 2 categories of devices: intra-aortic balloon pumps (IABPs) and ventricular assist devices (VADs).

Intra-Aortic Balloon Pumps

Intra-aortic balloon pumps are the first-line device. They can be placed rapidly in the cardiac catheterization laboratory or in the cardiac care unit or ICU. Use of an IABP improves coronary and peripheral perfusion via diastolic balloon inflation and augments LV performance via systolic balloon deflation with an acute decrease in afterload. The incidence of IABP use for AHF varies among the registries, from less than 1% to as much as 53%, as described in Table 1 on page 154. Data from the Acute Decompensated Heart Failure National Registry (ADHERE) indicate that less than 1% of patients hospitalized for AHF syndrome receive an IABP during the entire hospital stay; thus, the utility of the IABP during early AHF is relatively low and should be further defined. In the SHOCK trial registry, lower in-hospital mortality was observed in patients treated with IABP support,
Figure 3.
Selection of candidates for left ventricular assist devices.

Acute heart failure
  → Initiate therapy (a)
  → No response
  → Permanent end organ dysfunction (b)
  → No
  → Potential myocardial recovery or Potential intervention to recovery (c)
  → Yes → Conservative treatment
  → No → Intra-aortic balloon pump
  → No response (d)
  → Left ventricular assist device (e)
  → Eventual recovery
  → Intervention to recover ventricular function
    Or
    Transplant

(a) No response to conventional treatment of acute heart failure, including appropriate use of diuretics and fluids, intravenous inotropes, and vasodilators. (b) End-organ dysfunction, including severe systemic disease, severe renal failure, pulmonary disease, hepatic dysfunction, and permanent central nervous system injury. (c) Potential recovery of myocardial function or cardiac function: for example, acute myocardial ischemia, postcardiotomy shock, acute myocarditis, acute valvular heart disease, or candidate to heart transplant. (d) Absence of clinical improvement after intra-aortic balloon pump and mechanical ventilation. (e) Final indication may depend on availability of device and experience of cardiovascular team.

Adapted with permission from European Heart Journal.¹
particularly when combined with thrombolysis or early revascularization. A similar trend was observed in a large database of patients presenting with AMI; in-hospital mortality was lower (49%) in patients treated with IABP support than in those treated without it (67%). The most common indication for IABP is in the postoperative period of coronary artery bypass grafting. A recent Cochrane review stated that the IABP is integral to current postoperative management and is of undeniable efficacy, so it is not surprising that there have been no randomized controlled trials (RCTs) in this setting, as ethical permission would not be forthcoming. Within current clinical practice, the IABP is deployed preoperatively in a number of circumstances including unstable angina refractory to medical treatment and CS following coronary intervention. The Cochrane review also suggested that the IABP may be beneficial in terms of survival following the operation; however, the review pointed out many problems concerning the validity of the trials reviewed and concluded that a categorical answer to this question requires further RCTs.

**Ventricular Assist Devices**

There are 3 indications for VAD insertion: bridge to cardiac transplantation, bridge to recovery, and destination therapy.

Interestingly, left VADs (LVADs) allow partial or total support of the systemic circulation in cases of severe LV failure, whereas IABPs only decrease the preload and the afterload. Along with allowing the recovery of normal hemodynamic parameters, LVADs reduce ventricular strain and promote remodeling and thus may have beneficial effects on long-term outcome.

Left VADs are not generally used within the first 6 to 12 hours. However, VADs should be considered earlier rather than later, before end-organ dysfunction is evident. Types of LVADs are described in Table 2 on page 160. Several recently published reviews detailed the characteristics, advantages, and risks associated with specific LVADs.

A meta-analysis of controlled trials comparing percutaneous LVAD versus IABP for the treatment of CS was recently conducted. Three trials including 100 patients were included in the meta-analysis. The conclusion was that although percutaneous LVAD provides superior hemodynamic support compared with IABP in patients with CS, the use of these more powerful devices did not improve early survival (relative risk for 30-day mortality 1.06; 95% confidence interval, 0.68-1.66). Research results do not yet support percutaneous LVAD as the first choice in the mechanical management of CS. Larger RCTs using the most recent devices are needed to address this issue.

There is no evidence to guide the choice between surgically implanted versus percutaneous VAD. In a recent review, Cook and Windecker reported that the average 30-day survival rates of patients receiving VAD in case of CS due to AMI were 49% and 85% for surgically implanted versus percutaneous VAD, respectively. However, these results came from an observational study and no RCTs results support this difference.

**Extracorporeal Membrane Oxygenation**

Extracorporeal membrane oxygenation (ECMO) is performed percutaneously and is increasingly used for temporary mechanical circulatory support given the relatively low cost of the system and disposables as well as its broad availability (Table 2 on page 160). Extracorporeal membrane oxygenation is a simplified cardiopulmonary bypass using a centrifugal pump (5-6 L/min), allowing for augmentation of venous drainage despite relatively small cannulas and providing the option of taking over the full workload from the heart. There are several indications for ECMO, including intraoperative or perioperative low cardiac output syndrome, severe AMI, and cardiac resuscitation (all of which are types of ventricular failure). Another advantage is that ECMO can be useful in cases of associated respiratory failure and even as a renal support by addition of a hemofilter. The limitations of ECMO mainly stem from the necessity of permanent operator supervision and intervention and its relatively limited length of use (30 days).
Table 2.
Left Ventricular Assist Devices

<table>
<thead>
<tr>
<th>Percutaneous devices</th>
<th>Principle</th>
<th>Characteristics</th>
<th>Clinical Uses</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tandem Heart (Cardiac Assist Inc, Pittsburgh, Pennsylvania)</strong></td>
<td>Two cannulas: inflow cannula placed via the femoral vein into the atrium by a transseptal puncture approach and outflow cannula in the femoral artery.</td>
<td>Can generate flow up to 5 L/min.</td>
<td>Successfully used for high-risk percutaneous interventions, for postcardiomyotomy heart failure, bridge-to-bridge device, and as a bridge to transplantation.</td>
<td></td>
</tr>
<tr>
<td><strong>Impella Recover LP2.5 and LP5.0 (Abiomed Europe, Aachen, Germany)</strong></td>
<td>Placement via the femoral artery.</td>
<td>Can generate flow of 2.5 L/min (Impella 2.5) or 5 L/min (Impella 5.0).</td>
<td>Used successfully during high-risk coronary angioplasty and for patients with cardiogenic shock caused by MI. Used to treat AHF due to cardiac allograft rejection.</td>
<td>Lower-extremity ischemia and cannula dislodgement.</td>
</tr>
<tr>
<td><strong>Extracorporeal membrane oxygenation</strong></td>
<td>Two cannulas: one for the inflow, the other for the outflow. Performed percutaneously using femoral access.</td>
<td>Incorporates an oxygenator into the circuit.</td>
<td>Useful in patients with both heart failure and an inability to adequately oxygenate the blood.</td>
<td>Bleeding and distal leg ischemia.</td>
</tr>
</tbody>
</table>

PERICARDIAL TAMPOONADE

Pericardial tamponade is compression of the heart due to the pericardial accumulation of liquid, which can be fluid, pus, blood, clots, or gas. The cause could be effusion, trauma, or rupture of the heart. The management of pericardial tamponade includes symptom management and treatment of the underlying cause. Thus, prompt identification of the cause is critical.

A key diagnostic finding of cardiac tamponade is pulsum paradoxus, conventionally defined as an inspiratory systolic decrease in arterial pressure of 10 mm Hg or more during normal breathing. However, pulsum paradoxus is not always
Table 2. (cont.)

Left Ventricular Assist Devices (cont.)

<table>
<thead>
<tr>
<th>Surgically implanted devices</th>
<th>Principle</th>
<th>Characteristics</th>
<th>Clinical Uses</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First-generation VADs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- PVAD (Thoratec Corporation, Pleasanton, California)</td>
<td>Pulsatile flow via large pericorporeal consoles. Device inserted via a traditional sternotomy.</td>
<td>Can generate cardiac output between 5 and 7 L/min.</td>
<td>Mainly used as bridge to transplant or bridge to recovery.</td>
<td>High risk of infections and bleeding. Perioperative mortality rate is 15%-20%, and overall survival until device explantation is 60%-70%.</td>
</tr>
<tr>
<td>- BVS 5000/AB5000 (Abiomed Corporation, Danvers, Massachusetts)</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>Second-generation VADs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- HeartMate IP/XVE LVAS (Thoratec)</td>
<td>Device inserted via a traditional sternotomy.</td>
<td>Can generate cardiac output between 5 and 7 L/min.</td>
<td>Mainly used as bridge to transplant or bridge to recovery. Sometimes used as destination therapy.</td>
<td>Improved patient mobility leading to decreased infection rates and neurocognitive complications.29</td>
</tr>
<tr>
<td>- Novacor (WorldHeart Corporation, Salt Lake City, Utah)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ArrowLionHeart (Arrow International, Reading, Pennsylvania)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Third-generation VADs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- HeartMate II (Thoratec)</td>
<td>Fully implantable, axial flow impeller pumps with lack of percutaneous lines and implantation within the pericardium, obviating a need for a pump pocket.</td>
<td>Can generate cardiac output up to 7 L/min.</td>
<td>Used as destination therapy.</td>
<td>Decrease in complications, specifically infections.</td>
</tr>
<tr>
<td>- Jarvik 2000 FlowMaker (Jarvik Heart, New York, New York)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- MicroMed DeBakey Pump (Micromed Inc, Houston, Texas)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>- LVAD (Thoratec)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Berlin Incor (Berlin Heart, The Woodlands, Texas)</td>
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</tr>
</tbody>
</table>
present; for example, it could be absent in cases of severe hypotension.

Electrocardiogram usually shows signs of pericarditis, but the only quasi-specific sign of tamponade is electrical alternation. Echocardiography is the principal tool for diagnosing pericardial effusion and cardiac tamponade. Among echocardiographic signs, the most characteristic (although not entirely specific) is chamber collapse, most always of the right atrium and ventricle.

The treatment of cardiac tamponade is drainage of the pericardial contents, preferably by needle pericardiocentesis with the use of echocardiography. Surgical drainage is preferable in case of intrapericardial bleeding, clotted hem pericardium, or thoracic conditions that make needle drainage difficult or ineffective. The goal of drainage is to relieve compression and not to entirely empty the pericardial space, which could be deleterious because of the risk of myocardial injury.

**SUMMARY**

Acute heart failure, especially secondary to CS and pericardial tamponade, represents a complex and challenging situation affecting a heterogeneous population at high risk of short-term morbidity and mortality. Diagnosis should be made early, and goal-directed treatment strategies should be initiated promptly to attain the best outcomes.

**REFERENCES**


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