Clinical scenario: trauma, new murmur, hemodynamic instability

Perioperative low cardiac output syndrome

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## Classification of heart failure

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>New onset</strong></td>
<td>First presentation, acute (AHF) or slow onset</td>
</tr>
<tr>
<td><strong>Transient</strong></td>
<td>Recurrent or episodic</td>
</tr>
<tr>
<td><strong>Chronic</strong></td>
<td>Persistent, Stable, worsening, or decompensated (ADHF)</td>
</tr>
<tr>
<td><strong>Etiology</strong></td>
<td></td>
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<tr>
<td><strong>Clinical presentation</strong></td>
<td></td>
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<tr>
<td><strong>Precipitating factors</strong></td>
<td></td>
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</tbody>
</table>

*ESC guidelines 2008*
**Clinical presentations in AHF**

<table>
<thead>
<tr>
<th>Presentation</th>
<th>Incidence</th>
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<tbody>
<tr>
<td><strong>Worsening or decompensated chronic heart failure:</strong> (evidence of systemic and pulmonary congestion, low blood pressure is associated with a poor prognosis)</td>
<td>70%</td>
</tr>
<tr>
<td><strong>Pulmonary edema:</strong> (severe respiratory distress, tachypnoea, orthopnoea, pulmonary rales, aO2 sat.&lt;90%,)</td>
<td>3-13%</td>
</tr>
<tr>
<td><strong>Hypertensive HF:</strong> HF accompanied by hypertension, relatively preserved LV systolic function, increased sympathetic tone, tachycardia, flash pulmonary edema</td>
<td>50%</td>
</tr>
<tr>
<td><strong>Cardiogenic shock:</strong> evidence of tissue hypoperfusion, reduced systolic blood pressure &lt;90 mmHg, low urine output &lt;0.5 ml/kg/hr</td>
<td>&lt;1-4%</td>
</tr>
<tr>
<td><strong>Right HF:</strong> low output syndrome, absence of pulmonary congestion, increased jugular venous pressure</td>
<td>?</td>
</tr>
<tr>
<td><strong>Acute new onset HF:</strong> evidence of ACS, cardiac and non-cardiac precipitating causes</td>
<td>25%</td>
</tr>
</tbody>
</table>
Patients eterogeneity: different haemodynamic profiles in acute setting

Cotter et al: Eur J Heart Fail, 2003; 5: 443-51
Perioperative Low output state and hypotension

• Hypovolemia
  – Replacement of fluids judiciously
• Right atrial hematoma/tamponade
• Vasodilatory hypotension:
  – Protamine reaction
  – Pitressin deficiency
  • Risk factors for Vasod Hyp=Preop use of achetylchelinesterase inh and low EF
Low output syndrome post cardiac surgery
Pericardial Pathology

• Pericardial effusion: almost 50% at 8 day post surgery
  – 30% moderate
  – 2% large

• Tamponade in 2% (more in valve surgery than CABG)

• Most effusion loculated (leak from right atrial cannulation)

• Post op tamonade: 60% localized posterior effusions
  – Right atrial collapse in 35%, right ventricular in 30% and left ventricular in 65%

• Fluids can be infused as temporal measure
DEFINITION OF LOW CARDIAC OUTPUT SYNDROME

Need for:

– postoperative intraaortic balloon pump
– or inotropic support for longer than 30 minutes in the intensive care unit

• In order to maintain:
  • the systolic blood pressure greater than 90 mm Hg
  • and the cardiac index greater than 2.2 L/min/m2.
Etiology of Cardiogenic Shock and In-hospital Mortality

N=1159

<table>
<thead>
<tr>
<th>Cause</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Predominant left ventricular failure</td>
<td>74.5%</td>
</tr>
<tr>
<td>Mechanical complication (VSD and MR)</td>
<td>12.5%</td>
</tr>
<tr>
<td>Isolated right ventricular failure</td>
<td>3.4%</td>
</tr>
<tr>
<td>Severe prior valvular disease</td>
<td>2.9%</td>
</tr>
<tr>
<td>Cardiac tamponade or rupture</td>
<td>2.3%</td>
</tr>
<tr>
<td>Other</td>
<td>6.3%</td>
</tr>
</tbody>
</table>

In-Hospital Mortality: 62.1%

Wong, JACC 2000;36:1077-83
Post cardiopulmonary bypass cardiac function

- Postoperative myocardial stunning in ~50%
- 25% post CABG: inotropic support due to myocardial dysfunction
- Vasodilatory shock

- Postcardiotomy cardiogenic shock: 2-6%
  - (adult cardiosurgery)
  - 40% have RV dysfunction
Survival post ICU heart failure: role of underlying pathophysiology
The overall prevalence of low cardiac output syndrome was 9.1%. The operative mortality rate was higher in low cardiac output syndrome (16.9% versus 0.9%).

**Independent predictors of low output syndrome**
- (1) left ventricular ejection fraction less than 20% (27%, odds ratio 5.7);
- (2) repeat operation (25%, odds ratio 4.4);
- (3) emergency operation (27%, odds ratio 3.7);
- (4) female gender (16%, odds ratio 2.5);
- (5) diabetes (13%, odds ratio 1.6);
- (6) age older than 70 years (13%, odds ratio 1.5);
- (7) left main coronary artery stenosis (12%, odds ratio 1.4);
- (8) recent myocardial infarction (16%, odds ratio 1.4);
- (9) triple-vessel disease (10%, odds ratio 1.3).
Predictors of difficult weaning from CPB

• Prolonged aortic cross clamp time > 60 minutes
• Prolonged bypass time
• Poor myocardial preservation during CPB
• Left ventricular ejection fraction (LVEF) < 0.3
• Pre bypass delta PCO$_2$ > 6 mmHg (veno-arterial and regional difference in the partial pressure of CO$_2$)
• Pre operative diastolic dysfunction
High Risk groups for perioperative diastolic dysfunction

**LV/RV diastolic dysfunction** are associated to difficult discontinuation from cardiopulmonary bypass

(65.5% and 72% of patients with moderate/severe LVDD and RVDD respectively, in contrast to 40.9% and 48% of patients with normal/mild LVDD/RVDD).

<table>
<thead>
<tr>
<th>Source</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVH</td>
<td>In patients with AS, preoperative DD is attributable to hypertension, myocardial hypertrophy-fibrosis, and/or to ischemia [64].</td>
</tr>
<tr>
<td>CAD</td>
<td>Patients with CAD are prone for the development of postoperative myocardial diastolic dysfunction [39]. Left ventricular filling abnormalities have been detected in as many as 90% of patients [39]. Possible related factors that were considered were ischemia, hypertrophy, and hypertension [79].</td>
</tr>
<tr>
<td>DM</td>
<td>All insulin-dependent diabetes mellitus patients with left diastolic dysfunction had evidence of definite autonomic neuropathy [80]. Moreover, diabetic patients with autonomic neuropathy form a subgroup of particularly high mortality and cardiovascular event risk [81,82].</td>
</tr>
<tr>
<td>Age</td>
<td>Aging is correlated to DD through an increase upon wall thickness (secondary to enlargement of cardiac myocytes), and changes in the vasculature, the diameter, and vascular stiffness of the aorta and large arteries [83]. Up to 60% of geriatric patients with normal EF, following non-cardiac surgery, had been postoperatively diagnosed with diastolic dysfunction [35].</td>
</tr>
</tbody>
</table>
Clinical review: Practical recommendations on the management of perioperative heart failure in cardiac surgery

Alexandre Mebazaa¹, Antonis A Pitsis², Alain Rudiger³, Wolfgang Toller⁴, Dan Longrois⁵, Sven-Erik Ricksten⁶, Ilona Bobek⁷, Stefan De Hert⁸, Georg Wieselthaler⁹, Uwe Schirmer¹⁰, Ludwig K von Segesser¹¹, Michael Sander¹², Don Poldermans¹³, Marco Ranucci¹⁴, Peter CJ Karpati¹⁵, Patrick Wouters¹⁶, Manfred Seeberger¹⁷, Edith R Schmid¹⁸, Walter Weder¹⁹ and Ferenc Follath²⁰
Inotropic support

- Patients likely to benefit from catecholamine support are those with low cardiac output (CI \( < 2.0 \text{ L.min}^{-1} \text{.m}^{-2} \)), with optimized heart rate, rhythm, ventricular preload, and afterload, and without evidence of acute cardiac tamponade.

- Dopamine and dobutamine enhance heart rate and cardiac output equally, but dobutamine produces greater reductions in left ventricular preload and afterload.

- Dobutamine augments myocardial coronary blood flow more than dopamine.
Pharmacological treatment of left ventricular dysfunction after cardiac surgery

Group recommendations

- In case of myocardial dysfunction, consider the following three options either alone or combined:
  - Among catecholamines, consider low-to-moderate doses of dobutamine and epinephrine: they both improve stoke volume and increase heart rate while PCWP is moderately decreased; catecholamines increase myocardial oxygen consumption
  - Milrinone decreases PCWP and SVR while increasing stoke volume; milrinone causes less tachycardia than dobutamine
  - Levosimendan, a calcium sensitizer, increases stoke volume and heart rate and decreases SVR

- Norepinephrine should be used in case of low blood pressure due to vasoplegia to maintain an adequate perfusion pressure. Volaemia should be repeatedly assessed to ensure that the patient is not hypovolaemic while under vasopressors

- Optimal use of inotropes or vasopressors in the perioperative period of cardiac surgery is still controversial and needs further large multinational studies
Vasodilators

Nitroglycerin
- Reduces preload
- Relieves ischaemia
- Improves symptomatic HF

Nitroprusside
- Reduces afterload
- Relieves blood pressure
- Increases cardiac output

Nesiritide
- Reduces preload & afterload
- Increases cardiac output
- Decreases neurohormonal activation
- Relieves dyspnoea

None of the above have been shown to improve mortality for ADHF in randomised controlled clinical trials
Ultrafiltration

- Great potential for the use of UF in patients with decompensated HF either resistant to diuretics or [who] demonstrate unfavourable side effects
- An early application of this technology in such patients should allow effective therapy, prevention of adverse events, and improvement of after-discharge outcome
- Whether UF should replace standard IV diuretic therapy as a broadly applicable first-line therapy in acute HF remains to be shown in larger safety, efficacy, and cost-effectiveness studies
Management of perioperative diastolic dysfunction

- Appropriate increase of volume load

- Phosphodiesterase inhibitors seem to be beneficial for diastolic dysfunction improvement, and should be used in perioperatively.

- Levosimendan may be used in perioperative management of diastolic dysfunction.
  - It increases cardiac output and decreases pulmonary capillary wedge pressures. This mode of enhanced contractile force generation is achieved without an increase in myocardial oxygen consumption, intracellular calcium concentrations, or an adverse effect on diastolic function.
In Hospital Management
ESC Guidelines Algorithm of treatment strategy
- according to SBP and LV filling pressure -

Treatment strategy in AHF according to systolic blood pressure

- Oxygen/NIV
- Loop diuretic +/- vasodilator
- Clinical evaluation

- SBP > 100 mmHg
- SBP 90-100 mmHg
- SBP < 90 mmHg

- Vasodilator (NTG, nitroprusside, nesiritide, levosimendan)
- Vasodilator and/or inotrope (dobutamine, PDEI, levosimendan)
- Consider preload correction with fluids
- Inotrope (dopamine)

- Good response
  - Stabilise and initiate diuretic, ACEI/ARB, beta-blocker
- Poor response
  - Inotrope
  - Vasopressor
  - Mechanical support
  - Consider PAC

Treatment strategy in AHF according to LV filling pressure

- Pulmonary congestion and SBP > 90 mmHg
- Adequate filling pressure
- Adequate CO
  - Reversal of acidosis
  - SvO₂ > 65%
  - Adequate organ perfusion

- NO
- YES
- Reassess frequently

- YES
- Vasodilators, diuretics if volume overload
- Fluid challenge
- Inotrope
- Vasodilator
- Mechanical support
- Consider PAC

- If volume overload with peripheral or pulmonary congestion: consider diuretic and vasodilators.

- If hemodynamic compromise with hypoperfusion and hypotension is predominant: consider positive inotropic support.

ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008
## Derive haemodynamic state

<table>
<thead>
<tr>
<th></th>
<th>Volume</th>
<th>Function</th>
<th>LAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Empty</td>
<td>↓</td>
<td>N</td>
<td>↓</td>
</tr>
<tr>
<td>Diastolic Failure</td>
<td>↓</td>
<td>N</td>
<td>↑</td>
</tr>
<tr>
<td>Systolic Failure</td>
<td>↑</td>
<td>↓</td>
<td>N</td>
</tr>
<tr>
<td>S + D Failure</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>RV Failure</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Vasodilated</td>
<td>↓</td>
<td>N</td>
<td>↓</td>
</tr>
</tbody>
</table>
Potential deleterious effects of diuretics and cardiorenal syndrome of HF

- Increased morbidity and mortality
- Pathological remodeling
- Congestion
- Neurohormonal activation
- Diuretic resistance
- Impaired renal function
- Diuretic therapy
- Neurohormonal activation
- Vasoconstriction
- Diminished blood flow
- Decreased renal perfusion
How to evaluate volume status

• Volume estimates of preload seem more predictive of volume status
  – utility of LV volumes

• Changes in circulating volumes do not correlate with changes in LV enddiastolic volume and stroke volume

• Gold standard hemodynamic technique is yet to be determined
Difficult to estimate volume status using hemodynamic measurements

- Pressure estimates (both central venous pressure and pulmonary capillary wedge pressure): insensitive indicators of volaemia

- Low values may reflect hypovolemia but high values do not necessarily indicate volume overload
  - Uncoupling between PCWP can be the consequence of elevated pulmonary vascular resistance, pulmonary vеноconstriction and reduction of transmural left ventricular compliance
LOW OUTPUT STATE
ROLE OF PERIOPERATIVE ECHO

Cine loops:
side by side comparison of different phases during surgery

=> Recognition of changes in:
   LV cavity size (preload changes)
ejection indices

Hypovolemia:

=> LV area --------- decreases
=> LV systolic function---increases
EVALUATION OF PRELOAD

FLUID CHALLENGE:

2.5% change in blood volume (200 ml) =>

change in LV end-diastolic size:

=> 6.5% (normal LV function)

=> 3.5% (depressed LV function)

Both linear relationship for 6 times X 200 ml blood removal
Doppler Evaluation of Elevated LV Filling Pressures

Mitral:
- DT < 200ms
- E/A > 1.5

Pulmonary vein:
- PVa dur > MVa
- PVa > .35 m/sec
- PVs < 50% of PVd

Mitral annular:
- E/Ea > 10

Estimating LAP

Diastole
- FC
- LOC
- SEPTUM
- LA
- RA

Systole
- FC
- SR
- SR
- SB

(high, normal, low)
Normal Doppler Velocities

- Pulm. vein
- Mitral
- Mitral annulus

- E/A > 1
- PVs = PVd
- Pva < .35 m/s
- Ea > .1 m/s
- E/Ea < 7
ECHO RV VOLUME semiquantitatively

ratio RV/LV
(normal <2/3) ➔

RV=LV: moderate dilatation

RV>LV: severe dilatation

Maximum cross sectional diameter
(inlet- 4 ch apical view) ➔

Area RV (4C): (automatic acoustic quantification, AQ)
Ejection fraction
RV

Sa<11.5 cm/sec

RVEF<45%

EHJ Feb 2001

Figure 1  Correlation between right ventricular ejection fraction (EF) and the peak tricuspid annular systolic velocity (Sa).
Ventricular assist devices
HOW TO MANAGE

A: Conventional
B: LV-apex – descending aorta
Ventricular assist devise
The role of right ventricle

• 9%–33% of patients have severe RV failure after LVAD insertion and may require an RVAD

• A LVAD can have on RV:
  – beneficial effect by reducing afterload
  – detrimental effect by increasing preload through leftward septal shift during LVAD support.

• A preoperatively dilated RV with increased RV preload and afterload predisposes to RV dysfunction after LVAD implantation
  – RV fractional area change <20% predisposes RV failure after LVAD insertion.
Ventricular assist devices
HOW TO MANAGE
Recommendations for reporting perioperative transoesophageal echo studies

Robert Feneck¹*, J. Kneeshaw², K. Fox³, D. Bettex⁴, J. Erb⁵, F. Flaschkampf⁶, F. Guarracino⁷, M. Ranucci⁸, M. Seeberger⁹, E. Sloth¹⁰, H. Tschernich¹¹, P. Wouters¹², and J. Zamorano¹³ on behalf of the European Association of Cardiothoracic Anaesthesiologists (EACTA) and the European Association of Echocardiography (EAE)
Post-cardiopulmonary bypass study

Every perioperative TEE should include a study following corrective surgery. This is one of the most important aspects of perioperative TEE, and the study should be fully reported. We would recommend the following.

(i) Report when the study was performed. In the cardiac surgical patient this might be immediately following CPB, following protamine, before or after chest closure. The findings at these times may be markedly different, and it is therefore important to note when the study was recorded.

(ii) Report the state of the LV function. This should include a note of any concurrent supportive treatment, i.e. inotropic drug, mechanical device support, etc. In patients undergoing revascularization attention should be directed to, and a note made of significant regional wall motion abnormality in the relevant areas.

(iii) Surgical outcome

(a) In valve replacement surgery the prosthetic valve function should be noted, any regurgitant jets including normal ‘washing’ jets should be noted.

(b) In valve repair, the severity and nature of any residual regurgitation should be noted. The method of assessment
Contemporary workflow in clinical scenarios / How to do? 
The Onassis Center Experience
Dynamic obstruction of LV outflow

1. Small LV size
2. Mitral valve repair / ±10%
Post Mitral valve repair
dynamic LV outflow obstruction
Rocking aortic bioprosthesis post early post op endocarditis
Rocking aortic bioprosthesi
post endocarditis: more to see?
left to right communication
Dysfunction of the MVR, due to surgical suture over the valve’s strand
Rupture of the LV

MI post CABG
/low output:
Initially perceived
as tamponade
CASE 1
Question for cannula position

CASE 2
LV unloading/volume added

CASE 3: LV collapsed:
add fluids
+ reduction of VAD rate

POST VAD LOW OUTPUT CASES
Post Transfemoral/Transapical AVR
Low Output Cases
Patient in shock!!!!
INITIAL AVR

AVR inside the initial AVR
The Odyssey of an AVR

LOW OUTPUT CASE??