Management of Acute Cardiogenic Pulmonary Oedema

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Causes of LV Failure

- Hypertension
- Ischaemia
- Muscle (CMO, drugs, toxins, infiltrative, nutritional, endocrine, HIV, peripartum)
- Valvular
- Congenital
Prognosis

- 50% are dead at 4 yrs
- 40% of those admitted with HF are dead or readmitted within 4 years
<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
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<tbody>
<tr>
<td><strong>New onset</strong></td>
<td>First presentation</td>
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<tr>
<td></td>
<td>Acute or slow onset</td>
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<tr>
<td><strong>Transient</strong></td>
<td>Recurrent or episodic</td>
</tr>
<tr>
<td><strong>Chronic</strong></td>
<td>Persistent</td>
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<td>Stable, worsening, or decompensated</td>
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Diastolic HF: HFPEF

- Symptoms ± signs of HF & preserved LVEF (40–50%) (no consensus on cut-off for “preserved EF”)
- Clinical diagnosis alone inadequate, esp in ♀, elderly, obese
- HFPEF is present in 50%
- Prognosis similar to systolic HF

Remes Eur Heart J 1991
Wheeldon Q J Med 1993
Diagnosis of HFPEF

- Requires 3 conditions
  - Signs and/or symptoms of chronic HF
  - Normal or mildly abnormal LV systolic function
  - Diastolic dysfunction (abnormal relaxation or diastolic stiffness)
Diagnosis of Heart Failure

Clinical examination, ECG, Chest X-ray, Echocardiography

Natriuretic peptides

- BNP <100 pg/mL
  - NT-proBNP < 400 pg/mL
  - Chronic HF unlikely

- BNP 100–400 pg/mL
  - NT-proBNP 400–2000 pg/mL
  - Uncertain diagnosis

- BNP >400 pg/mL
  - NT-proBNP > 2000 pg/mL
  - Chronic HF likely

Dickstein ESC Guidelines: Eur J Heart Failure 2008
Therapeutic Modalities:

- Oxygen
- Non-invasive ventilation
- Loop diuretics
- Vasodilators
- Inotropic agents
- Cardiac glycosides
- Morphine
ACPO is Frequently not Associated with Systemic Congestion

Hypertensive AHF

PULMONARY ODEMA

ACS and HF

Cardiogenic shock

Right HF

Acutely Decompensated Chronic HF
Worsening/decompensated CHF

- Peripheral oedema/congestion:
- Systemic congestion with low BP
- Often both RV and LV dysfunction so LA pressure not increased
- Poor prognosis
- Pulmonary congestion less common
Pulmonary Congestion

RA → RV → LUNG → LA → LV

TV → PA → PV → Mitral → LV

INCREASED PRESSURE
Acute Pulmonary Oedema: Hypertensive HF

- High BP: systolic function mostly preserved
- Increased sympathetic tone, tachycardia, vasoconstriction
- No systemic congestion; Euvolaemic or mildly hypervolaemic
- Pulmonary congestion
Isolated right HF:

- Low output syndrome
- Increased JVP± hepatomegaly
- Low LV filling pressures
- No pulmonary congestion
ACS and HF

- 15% of patients with ACS have signs and symptoms of HF
- HF frequently associated with/precipitated by arrhythmia (bradycardia, AF, VT)
- Not hypervolaemic at onset of ischaemia
# Classification of HF severity in MI: Killip

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
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</table>
| Stage I | No heart failure.  
No clinical signs of cardiac decompensation |
| Stage II | Heart failure.  
Diagnostic criteria include rales, S3 gallop, and pulmonary venous hypertension.  
Pulmonary congestion with wet rales in the lower half of the lung fields. |
| Stage III | Severe heart failure.  
Frank pulmonary oedema with rales throughout the lung fields |
| Stage IV | Cardiogenic shock.  
Signs include hypotension (SBP <90 mmHg), and evidence of peripheral vasoconstriction such as oliguria, cyanosis and sweating |
Classification of HF severity in MI: Forrester

1. Normal perfusion and pulmonary wedge pressure (PCWP—estimate of left atrial pressure)
2. Poor perfusion and low PCWP (hypovolaemic)
3. Near normal perfusion and high PCWP (pulmonary oedema)
4. Poor perfusion and high PCWP (cardiogenic shock)
Tako-tsubo

- “Stress-induced CMO”, “broken heart” or “transient LV apical ballooning syndrome”
- 0.7–2.5% patients with apparent ACS
- Preceding hypertension in 43-76%
- Primarily older ♀ (90%), acute emotional/physiologic stress; also younger patients and ♂, not always with stressful events
Tako-tsubo: Diagnostic Criteria

- Transient hypo-, akinesia in LV mid segments ± apical involvement in >1 vessel distribution
- Absence of OCAD or plaque rupture on angio
- New ECG abnormalities/ modest Tn elevation
- Absence of myocarditis or phaeo

Prasad Am Heart J 2008
Tako-tsubo: Mechanisms

- Myocardial stunning: ? due to catecholamine or ischaemia-mediated multivessel, epicardial or microvascular spasm, aborted MI, or focal myocarditis.

- Why selective apical LV dysfunction is unknown; possibly increased responsiveness of apical myocardium to SNS stimulation.
Tako-tsubo

- Symptoms: ACS or ACPO
- Minimal ST elevation in precordial leads
- Small Tn elevation (peak < 24h), low for the extensive wall motion abnormalities:

Ramaraj Exp Clin Cardiol 2009
Sharkey Am J Cardiol 2008
Tako-tsubo: Systole: No contraction in apical region and apical ballooning

Zeb Postgrad Med J 2011
ACPO: Diuretics

- ACPO without pre-existent systemic congestion generally causes intravascular volume depletion
- Diuretics may cause further dehydration and exacerbate renal failure
<table>
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<tr>
<th>Condition</th>
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<tbody>
<tr>
<td>Hypertensive heart failure</td>
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<tr>
<td>Acute ischaemia/MI</td>
</tr>
<tr>
<td>HFPEF</td>
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<tr>
<td>Tako Tsubo</td>
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<tr>
<td>HOCM</td>
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<tr>
<td>Negative pressure pulmonary oedema</td>
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**ACPO: Conditions where diuretics may not be valuable**
Therapy: Oxygen

- Recommended as early as possible to achieve saturation $\geq 95\%$ (less in COPD)
NIV: Haemodynamic Response

- RV preload reduced by IPPV
- RV afterload determined by effect on PVR
  - Alveolar vessels: degree of alveolar overdistension
  - Extra alveolar interstitial vessels: atelectasis or recruitment

Magder: ICM 1998

Pinsky: JAP 1984; Crit Care Clin 1990
Marini: ARRD 1987
LV preload

Influenced by

- RV preload
- RV afterload
- Ventricular interdependence
LV Afterload

Determined by:

- Ventricular work: SVR and BP
- Cavity size $\text{Tension} \propto \text{radius}$ (Laplace)
- Transmural pressure (tension)

IPPV reduces: cavity size and transmural tension
Hemodynamics

- Preload
- Cavity size
- Muscle shortening
- Afterload
- Contractility
- Stroke volume
- SVR
- BP
- C.O.
- HR
- ±
- ITP
LV Afterload: Transmural Tension

IPPV increases ITP reducing transmural tension

Mean Arterial Pressure 60
Mean Airway Pressure +20

\[ T_T = \text{Transmural tension} = 60 + 20 = 80 \]
Afterload: Negative Pressure Pulmonary Oedema

- Markedly negative swings in ITP from spontaneous ventilation increases afterload and may precipitate ACPO particularly with LV dysfunction
- This can also occur in well compensated patients. Young fit males

Stalcup: NEJM 1977
Sofer: Chest 1984
Beach: CCM 1973
Therapy: NIV with PEEP

- Consider as early as possible with ACPO
- Improves clinical parameters and LV function by reducing LV afterload
- 3 meta-analyses showed reduced intubation and short-term mortality

Masip Heart Fail Rev 2007 & JAMA 2005
Peter Lancet 2006
Clinical Effects of NIV

- 30 extubated low risk patients:
- 4 intervals: spontaneous ventilation, NIV, spontaneous, NIV
- CI & $S_{mv}O_2$ increased significantly with NIV
- UO increased significantly

Hoffman Thorac Cardiovasc Surg 2003
CPAP vs. Conventional therapy

- 5 trials: significant improvement in gas exchange and intubation rates
  - Rasanen Am J Cardiol 1985
  - Bersten NEJM 1991
  - Lin Chest 1995
  - L’Her ICM 2004

- The latter demonstrated a reduction in mortality with CPAP
CPAP vs. BiPAP

80 patients conventional therapy; CPAP 11cm H₂O vs BiPAP 17/11

- CPAP/BiPAP significantly improved dyspnoea, vital signs, P/F ratios
- Study stopped due to significant intubation difference 42 vs 7% CPAP & BiPAP
- 15day mortality higher with O₂ alone p< .05
- Mortality hospital discharge not different

Park CCM 2004
CPAP: Practicalities

- 5–7.5cmH\(_2\)O titrated to 10cmH\(_2\)O
- FiO\(_2\) >0.4

Contraindications
- Poor cooperation
- Immediate need for intubation
- Caution with ++ secretions
Measure Fluid Status: SVV
Fluid Status: SVV/PPV

Meta-analysis of 29 studies during MV:
- >12% predicted fluid responsiveness
- Sensitivity, specificity: PPV: 0.89 and 0.88
  SVV: 0.82 and 0.86

PPV more accurate (direct vs. derived)
Reliable only if TV ≥8 mL/kg and no spontaneous breathing
Thermodilution Technique
Thermodilution Decay Curve

\[ \text{MTt: Mean transit time} \]
\[ \text{DSt: Downslope time} \]

\[ \text{ITTV} = \text{CO} \times \text{MTt} \]
\[ \text{PTV} = \text{CO} \times \text{DSt} \]

MTt: Mean transit time
DSt: Downslope time

Belda FJ 26\textsuperscript{th} ISICEM Brussels 2006
\[ \text{CO} \times \text{MTt} = \text{Vd of cold indicator} = \text{IT Thermal Volume} \]

\[ \text{CO} \times \text{DSt} = \text{Cold indicator mixing chamber} = \text{P Thermal Volume} \]

\[ \text{CO} \times (\text{MTt} - \text{DSt}) = \text{GEDV} \]

\[ \text{ITBV (PBV)} = 1.25 \times \text{GEDV} \]

\[ \text{EVLW} = \text{ITTV} - \text{ITBV} \]
Fluid Status: GEDV/ITBV

- Lower values indicate volume-depletion
- Confirmatory evidence of fluid depletion esp atrial fib, spontaneous ventilation
- Fluid responsive CI corresponds with an increase in GEDVI and ITBVI
- But increased CI from inotropes leaves GEDVI/ITBVI unchanged

Berkenstadt Anesth Analg 2001
Fluid Status: EVLW

- EVLWI associated with more severe ARDS/LIS and higher mortality
- No consensus on normals or indexing parameters
- Predicted BW may be superior to predict mortality, MOF and diagnose ARDS
- Valuable confirmatory evidence of fluid depletion

Chew Crit Care 2012  
Chung PLoS One 2010  
Maharaj Cardiol Res Pract 2012  
Camporota Critical Care 2012
“Normal Values”

CI 3.0 – 5.0 l/min/m²
GEDVI 680 – 800 ml/m²
ITBI(PBV) 850– 1000ml/m²
SVV≤ 10 %
ELWI 3.0 – 7.0ml/kg (Oedema: >10mL/kg)
Interface
## Case Study

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<tbody>
<tr>
<td>CVP</td>
<td>8</td>
</tr>
<tr>
<td>MAP</td>
<td>70</td>
</tr>
<tr>
<td>PEEP</td>
<td>12</td>
</tr>
<tr>
<td>P/F</td>
<td>68</td>
</tr>
<tr>
<td>CI</td>
<td>1.6</td>
</tr>
<tr>
<td>SVV</td>
<td>22</td>
</tr>
<tr>
<td>SVRI</td>
<td>2800</td>
</tr>
<tr>
<td>EVLW</td>
<td>18ml/kg</td>
</tr>
<tr>
<td>GEDV</td>
<td>400ml/m²</td>
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Male 56: Post extubation dyspnea
Pink frothy sputum
Confused

Post extubation Pulm oedema:
⁻⁻ LV dysfunction/MI
⁻⁻ Negative pressure pulmonary oedema

**Intervention:** Fluids: if response inadequate, dobutamine