Managing acute heart failure in the intensive care unit

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The European Society of Cardiology and the European Society of Intensive Care Medicine classify patients with acute heart failure (HF) into six categories:[1]

- Hypertensive acute HF — signs and symptoms of HF are accompanied by high blood pressure and relatively preserved left ventricular function, with appearance on chest x-ray compatible with acute pulmonary oedema.
- Pulmonary oedema (verified by chest x-ray) accompanied by severe respiratory distress, with crackles over the lungs, orthopnoea, and O_2 saturation usually < 90% on room air before treatment.
- Acute decompensated HF (de novo or as decompensation of chronic HF) — signs and symptoms of acute HF are mild, and do not fulfil the criteria for cardiogenic shock, pulmonary oedema or hypertensive crisis.
- Cardiogenic shock — this is defined as evidence of tissue hypoperfusion induced by HF after correction of preload. There is no clear definition for haemodynamic parameters, but cardiogenic shock is usually characterised by reduced blood pressure (systolic blood pressure < 90 mmHg or a drop in mean arterial pressure > 30 mmHg) and/or low urine output (< 0.5 mL/kg per h), with a pulse rate > 60 beats per minute, with or without evidence of organ congestion. There is a continuum from low cardiac output syndrome to cardiogenic shock.
- High output failure — this is characterised by high cardiac output, usually with high heart rate (caused by arrhythmias, thyrotoxicosis, anaemia, Paget's disease, or iatrogenic or other mechanisms), with warm peripheries, pulmonary congestion, and sometimes low blood pressure, as in septic shock.
- Right HF — this is characterised by low output syndrome with increased jugular venous pressure, enlarged liver, and hypotension.

As in other critical illnesses, two key factors may markedly improve morbidity and mortality in acute HF syndromes: early initiation of treatment and tailored therapy. Early initiation aims to stop the negative cascade of heart dysfunction. Tailored therapy should be based on the level of systolic blood pressure at admission. Indeed, EFICA[2] and OPTIMIZE-HF[3] showed that patients with high systolic blood pressure are likely to have preserved left ventricular systolic function, while those with low systolic blood pressure have a lower left ventricular ejection fraction and frequent signs of organ hypoperfusion. Among the proposed treatments, non-invasive ventilation was the only treatment consistently proven to have a beneficial effect on morbidity and mortality in almost all types of acute HF syndrome. Concerning pharmacological agents, actions should be taken to increase the use of vasodilators, and reduce the use of diuretics.

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References

1 Zannad F, Adamopoulos C, Mebazaa A, Gheorghiade M. The challenge of acute decompensated heart failure. *Heart Fail Rev* 2006; 11: 135-9.
2 Zannad F, Mebazaa A, Juilliere Y, et al; EFICA Investigators. Clinical profile, contemporary management and one-year mortality in patients with severe acute heart failure syndromes: the EFICA study. *Eur J Heart Fail* 2006; 8: 697-705.
3 Adamopoulos C, Zannad F, Fay R, et al. Ejection fraction and blood pressure are important and interactive predictors of 4-week mortality in severe acute heart failure. *Eur J Heart Fail* 2007; 9: 935-41. ❑