Right ventricular failure: is it relevant for the intensivist?

D. van Dijk
Department of Intensive Care Medicine, University Medical Center, Utrecht, the Netherlands

Correspondence
D. van Dijk - d.vandijk@umcutrecht.nl

For a long time, the right heart has been perceived as a more or less passive accessory to its strong muscular neighbour, with minimal functional meaning, and no meaning at all for intensivists working in a general intensive care unit (ICU). Over the last decades, however, it has become clear that the failing right heart is a cause of insufficient cardiac output in a substantial proportion of ICU patients.[1,2] The board of the NJCC has therefore invited two groups of intensivists, both from centres with significant experience, to write a comprehensive review on right heart failure. In this issue of NJCC, they express their views on the pathophysiology, diagnosis and treatment of this condition.[3,4]

Roughly, the causes of right ventricular failure can be divided into three main categories: 1) too much preload, 2) impaired right ventricular contractility and 3) too much afterload. As an illustration, imagine a septic patient admitted to your ICU: the patient receives large amounts of fluids in the initial phase of resuscitation, eventually leading to a volume-overloaded right ventricle (too much preload). The patient develops septic cardiomyopathy, and systemic hypotension leads to insufficient right coronary artery perfusion (both causes of impaired right ventricular contractility). Poor oxygenation, acidosis, and positive pressure ventilation increase the pulmonary vascular resistance (too much afterload).

Some patients with refractory right heart failure should be transferred to specialised centres, where advanced treatment options such as inhaled nitric oxide and mechanical assist devices are available. However, many patients with right ventricular failure can be treated in your own ICU, with relatively simple measures. As a recipe, I would suggest the following:

- Make the diagnosis! Obtain a transthoracic or transoesophageal echocardiogram. A distended right ventricle, sometimes in combination with an empty left ventricle, a D-shaped left ventricle on short-axis view, massive tricuspid regurgitation or poor tricuspid annular plane systolic excursion (TAPSE) are indicators of right heart failure. An increased pulmonary artery pressure may also be diagnosed with echocardiography. Don’t forget to obtain a right-sided electrocardiogram (ECG) to diagnose additional right ventricular infarction in patients with an inferior wall myocardial infarction.
- If possible, treat the underlying cause. Administer thrombolysis for a pulmonary embolism, perform angioplasty of an occluded right coronary artery, treat arrhythmias.
- Optimise preload. This is a difficult one. Some patients (especially those with normal pulmonary vascular resistance) may benefit from more fluids. The blood then passively flows through the right heart (Fontan circulation) and cardiac output may be restored. In contrast, (often in the longer term) when the goal is to re-compensate the right heart, diuretics are indicated.
- Improve contractility. A very important determinant of right ventricular contractility is systemic blood pressure. An appropriate blood pressure enhances right coronary artery blood flow. It also opposes the right-to-left shifting of the intraventricular septum, and thus improves both right and left ventricular performance. Agents such as dobutamine and milrinone may be indicated, but not when they lead to refractory systemic hypotension.
- Reduce afterload. The most simple measures to treat an elevated pulmonary vascular resistance are easily forgotten: ensure good oxygenation. Apply a bit more FiO2 and a bit less PEEP to reduce mean airway pressure. Avoid hypercapnia and other causes of acidosis. Consider the use of vasodilators as nitroglycerin or milrinone, but only when the systemic blood pressure can be maintained.
And there is more that can be done. The two reviews by Hermens and Schuuring in this issue of NJCC will tell you all about it. Enjoy!

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References