ble. I am hopeful that this article acts as a catalyst to expand protocols and guidelines throughout ICUs around the world.

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Pulmonary hypertension in the intensive care unit: Critical role of the right ventricle*

Pulmonary hypertension is a commonly encountered and often challenging problem in the intensive care unit (ICU) (1). The severity may sometimes be mild, reflecting left heart dysfunction or acute respiratory failure, and will respond to therapies directed at the primary insult. However, consequences of pulmonary hypertension in the ICU can be catastrophic when it is severe enough to precipitate acute right ventricular (RV) pump failure, sometimes referred to as the *acute right heart syndrome* (2), manifested by increased right-sided filling pressure and diminished cardiac output. Common causes for this syndrome include massive thromboembolism, acute lung injury, heart or lung transplantation, and surgery for valvular or congenital heart disease. Recent advances in the long-term treatment of pulmonary arterial hypertension have also created a population of patients with advanced stages of RV dysfunction, who present to ICUs with acute deteriorations. The consequences of acute right heart syndrome are cardiovascular collapse and death, unless the situation can be promptly stabilized and reversed. These observations highlight the central role of RV function in determining the outcome of critically ill patients with pulmonary hypertension.

In this issue of *Critical Care Medicine*, Dr. Zamanian and colleagues (3) review the current state-of-the-art management of pulmonary hypertension and right ventricular dysfunction in the ICU. The article is well researched and thorough but exposes the enormous knowledge gaps we have with regard to optimal management strategies. Little is known about how best to monitor RV dysfunction in the failing right heart, and even less is understood about optimal therapies. What is known is that the normal RV easily tolerates increases in preload and filling volume but is intolerant of increases in afterload (1). The pressure-generating capability of the normal RV is far more limited than that of the left ventricle, and an acute rise in mean pulmonary artery pressure >40 mm Hg usually results in cardiovascular collapse. At the same time, gauging the severity of pulmonary hypertension by pulmonary artery pressure can be misleading; a seemingly mild elevation in mean pulmonary artery pressure can be dire if associated with the acute right heart syndrome. For this reason, Doppler echocardiography is often useful as a way to assess RV structure and function (4).

Dr. Zamanian and colleagues (3) emphasize the precarious balance that intensivists must attempt to maintain in treating the failing RV. Both inadequate and overzealous volume resuscitation can be detrimental. The need to maintain an elevated central venous pressure in RV failure is often under-appreciated. One of the few mechanisms that the RV has to maintain forward flow in the face of elevated afterload is to increase RV end-diastolic pressure and volume. On the other hand, excessive intravascular volume may interfere with both left ventricular diastolic and systolic function owing to the phenomenon of ventricular interdependence (5). Positive-pressure ventilation and lung volume can also have profound effects on RV function (6). Increased intrathoracic pressure not only decreases RV preload, but also can increase RV afterload if it overdistends the lungs. At least part of the improved survival seen with use of lung protective

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ventilator strategies to treat acute respiratory distress syndrome may be attributable to the avoidance of lung overdistention and better preservation of RV function (6). On the other hand, low lung volume can also increase pulmonary vascular resistance, so patients with restriction owing to pulmonary edema, atelectasis, or pneumonia may benefit from an appropriate increase in lung volume.

How best to titrate these interventions to optimize RV function has not been established. RV dilation and hypokinesis are frequent echocardiographic findings but may not provide a reliable indicator of RV output (4). Biomarkers, such as plasma brain natriuretic peptide, and troponin levels may be of value in differentiating causes of respiratory failure owing to cardiac or non-cardiac causes (7) but are not adequate to differentiate between right and left ventricular dysfunction. Systemic blood pressure, cardiac output, and right atrial pressure are commonly used indicators but are blunt measures that detect late manifestations of RV dysfunction. Indicators of RV perfusion or energetics using computed tomography or magnetic resonance imaging are currently unavailable but, if developed, could provide a means of detecting incipient RV dysfunction and permit earlier and better tailored therapy.

The prompt reduction of pulmonary vascular resistance is the primary goal of therapy for right heart failure from pulmonary hypertension, but this is often difficult to achieve in the critical care setting. Selective pulmonary vasodilators can be lifesaving in a patient with a responsive pulmonary vasculature, but in the ICU, these agents may become unselective systemic vasodilators, with little effect on the diseased pulmonary circulation, and can worsen RV function by increasing demand for forward flow while reducing coronary perfusion pressure and precipitating RV ischemia (1).

The ability of the RV to cope with increases in afterload becomes the final determinant of survival in the acute right heart syndrome. Yet, little is known about how best to preserve RV function, either pharmacologically or by mechanical means. Dr. Zamanian and colleagues (3) consider dobutamine "the superior choice" for pharmacologically supporting RV function, but they acknowledge that its systemic vasodilatory action may be problematic and often necessitates combination with pressors that can increase pulmonary vascular resistance and further compromise coronary perfusion. Intravenous prostacyclin may offer more selective vasodilator effects on the pulmonary circulation but can have vasodilatory effects on the systemic circulation and can disrupt ventilation/perfusion relationships, thus worsening oxygenation. These potential adverse effects can be avoided by using prostacyclins via the inhalational route. This approach has been shown to improve pulmonary hemodynamics in patients with acute right heart failure following cardiac surgery (8). Inhaled nitric oxide also has the advantages of virtually no systemic effects and improvement of oxygenation via selective vasodilation of ventilated lung regions but is limited by adverse hemodynamic effects that occur commonly during withdrawal and is very costly (2). Brain natriuretic peptide serves not only as a marker of ventricular dysfunction but also has possible therapeutic actions, lowering pulmonary artery occlusion pressure in addition to pulmonary artery pressure and may be useful for the management of biventricular failure. Its pulmonary vasodilatory action may be potentiated by combination with sildenafil (9).

Ultimately, in the acute right heart syndrome, the ability of the RV to sustain function in the face of increased afterload determines the patient's survival. An improved understanding of how to detect incipient RV failure and to intervene to preserve RV function, either with pharmacological agents or mechanical interventions, such as RV assist devices or atrial septostomy (10), is likely to yield a survival benefit. Although further investigation aimed at minimizing pulmonary vascular resistance and optimizing recovery of pulmonary vascular integrity is certainly needed, we should not ignore the potential value of enhancing RV function to improve patient outcomes.

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