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# Acute Right Ventricular Dysfunction
## Real-Time Management With Echocardiography

*Sundar Krishnan, MBBS; and Gregory A. Schmidt, MD, FCCP*

VIDEO

In critically ill patients, the right ventricle is susceptible to dysfunction due to increased afterload, decreased contractility, or alterations in preload. With the increased use of point-of-care ultrasonography and a decline in the use of pulmonary artery catheters, echocardiography can be the ideal tool for evaluation and to guide hemodynamic and respiratory therapy. We review the epidemiology of right ventricular failure in critically ill patients; echocardiographic parameters for evaluating the right ventricle; and the impact of mechanical ventilation, fluid therapy, and vasoactive infusions on the right ventricle. Finally, we summarize the principles of management in the context of right ventricular dysfunction and provide recommendations for echocardiography-guided management. CHEST 2015; 147(3):835-846

**ABBREVIATIONS:** LV = left ventricular; LVAD = left ventricular assist device; PEEP = positive end-expiratory pressure; PVR = pulmonary vascular resistance; RAP = right atrial pressure; RV = right ventricular; RVFAC = right ventricular fractional area change; S′ = peak systolic velocity; TAPSE = tricuspid annular plane systolic excursion; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography

In critically ill patients with circulatory shock, the role of the left ventricle has long been appreciated. The right ventricle, in contrast, is considered "forgotten" perhaps because it is thinner walled, more difficult to image, and coupled indirectly to the systemic circulation. The ascendance of intensivist-conducted echocardiography has forced us to revise this view.[1] New evidence shows acute right ventricular (RV) dysfunction to be common, readily detected with simple bedside imaging, amenable to basic ICU interventions, yet often lethal. We review the epidemiology of RV dysfunction; the crucial role both transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) play to reveal shock due to acute RV dysfunction; and how ventilator changes, fluid therapy, and vasoactive drug infusions can be titrated based on real-time imaging. Although some patients with acute RV dysfunction have preexisting pulmonary hypertension, we restrict this review to patients without preexisting disease.

Knowledge of RV physiology is essential to the intensivist because several supportive therapies, including mechanical ventilation and fluid management, interact with RV dysfunction, potentially exacerbating shock. We briefly review the epidemiology of acute RV dysfunction and echocardiographic

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**AFFILIATIONS:** From the Department of Anesthesia (Dr Krishnan) and Department of Internal Medicine (Dr Schmidt), University of Iowa, Iowa City, IA.

**CORRESPONDENCE TO:** Gregory A. Schmidt, MD, FCCP, Department of Internal Medicine, University of Iowa, 200 Hawkins Dr, Iowa City, IA 52242; e-mail: gregory-a-schmidt@uiowa.edu



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parameters for judging RV function and conclude with principles and recommendations for using echocardiography to guide management. Strong evidence for the use of this echocardiographic-based approach is currently lacking; thus, the majority of our recommendations are based on experience, physiologic reasoning, and logical extension of the existing literature.

## Epidemiology

Acute RV dysfunction causes and exacerbates many common critical illnesses (Table 1). The right side of the heart, comprising the right atrium and right ventricle, accepts the entire cardiac output and pumps it through the pulmonary circulation. Because the pulmonary vasculature is characterized by low resistance and high compliance, pulmonary artery pressures are quite low, typically 25/10 mm Hg. Moreover, the right ventricle operates below its unstressed volume (ie, increasing its volume does not raise its pressure) so that atrial pressure does not reflect preload.[2] The thin-walled right ventricle is much more sensitive to increases in afterload than the left ventricle, making it vulnerable to systolic failure in disease states that raise RV afterload, a condition known as acute cor pulmonale.

ARDS is one of the most common conditions to challenge the right ventricle. The incidence of acute RV failure is around 25% with lung protective ventilation, a value corroborated by several studies, although it can be much higher depending on the severity of lung injury and the chosen ventilator strategy.[3-6] Acute RV dysfunction (defined as a dilated right ventricle with septal dyskinesia) was found in 22% of 226 patients studied by TEE within the first 3 days after diagnosis of moderate to severe ARDS,[7] with acute RV dysfunction an independent risk factor for 28-day mortality. Another survey revealed similar findings, with 22.5% of 204 patients with ARDS who were ventilated showing acute RV dysfunction.[8]

In unselected patients with pulmonary embolism, echocardiographic features of RV strain or dysfunction are present in between 29% and 56%.[9,10] The presence of RV dysfunction imparts a sixfold increase in the risk of in-hospital mortality. Even among patients with pulmonary embolism who are hemodynamically stable at presentation, the presence of acute RV dysfunction correlates with an increased risk of developing shock and dying in the hospital.[11]

Acute RV dysfunction is the hallmark of RV infarction, complicating roughly one-third of cases of ST-elevation infarction of the left ventricular (LV) inferior wall. In patients with inferior myocardial infarction, RV infarction increases the risk of complications and death. Distinguishing RV infarction from other causes of acute RV dysfunction can be challenging.

The pathophysiology of postcardiotomy RV failure is related to ischemia and myocardial depression during cardiopulmonary bypass and aortic cross-clamping, alteration of RV size and shape due to LV unloading after left ventricular assist device insertion (LVAD),[12] and donor heart ischemia and preexisting pulmonary vascular disease in heart transplant recipients.[13] On occasion, allograft rejection or mechanical obstruction at the pulmonary artery anastomosis in heart transplants, preexisting pulmonary hypertension, coronary embolism or graft occlusion, arrhythmias, pulmonary hypertension related to protamine or acute lung injury, postoperative pulmonary embolism, or sepsis could also contribute to postcardiac surgery RV dysfunction. Acute RV dysfunction after cardiothoracic surgery is a major cause of morbidity and mortality in specific patient populations. Although the overall incidence of RV failure after cardiotomy is 0.1%, it occurs in 2% to 3% of patients after heart transplantation and up to 30% of patients after LVAD insertion. Significantly, acute RV dysfunction after cardiac surgery is associated with mortality rates as high as 80%.[14] Excellent reviews on the topic have been published.[15,16]

Patients undergoing lung resection are at postoperative risk for increased pulmonary vascular resistance (PVR) due to loss of pulmonary tissue. When combined with the effects of atelectasis, hypoxia, and hypercarbia, acute RV dysfunction may follow.[17] Preexisting pulmonary vascular disease or RV dysfunction and the extent of pulmonary parenchymal resection predict the severity of postoperative RV dysfunction in these patients.

## Diagnosis of Acute RV Dysfunction

Clinical clues to acute RV dysfunction include elevated right-sided filling pressures, a right-sided third heart sound, tricuspid regurgitation, a pulsatile liver, and peripheral edema. Systemic hypotension, alterations in liver function tests,[18] creatinine level, urine output, neurologic function, mixed and central venous saturation, and lactate and natriuretic peptide levels provide insight into the degree of venous congestion and tissue hypoperfusion and are clues about the clinical impact of RV dysfunction. ECG signs of RV dysfunction are varied and inconsistent.[19] Especially in light of the fading relevance of the pulmonary artery catheter,[20] diagnosis typically relies on echocardiography. Intensivist-performed point-of-care echocardiography has become particularly