Management of Right Heart Failure in the Critically Ill

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KEYWORDS

• Right ventricular failure • Right ventricular dysfunction • Pulmonary hypertension • Acute respiratory distress syndrome

KEY POINTS

- Right ventricular failure complicates a number of commonly encountered conditions in the critically ill and is generally associated with worsened outcomes.
- An understanding of the pathophysiologic changes seen in the failing right ventricle is essential for developing an appropriate treatment strategy.
- Echocardiography is the screening test of choice for right ventricular failure. Focused critical care echocardiography can facilitate timely diagnosis by the bedside clinician.
- Timely diagnosis and treatment of the cause of right ventricular failure is essential.
- Reduction of right ventricular afterload and optimization of right ventricular preload and contractility form the principles of management. Oftentimes this requires combined use of vasopressors, inotropes, and pulmonary vasodilators.

INTRODUCTION

The critical importance of the right ventricle (RV) has long been underestimated, as classic teaching of cardiac physiology has emphasized left ventricular (LV) structure and function. Once thought a relatively unimportant conduit facilitating the flow of blood to the pulmonary vasculature, the RV is now recognized as a dynamic structure intricately linked to LV systolic and diastolic function. Likewise, research and clinical experience continue to demonstrate the importance of RV function in a variety of clinical conditions, including heart failure, myocardial infarction, congenital heart

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disease, pulmonary embolism, and pulmonary hypertension. Critically ill patients in the intensive care unit (ICU) with RV failure have increased morbidity and mortality compared with those patients with preserved RV function, and clinical management of these patients remains a formidable challenge.[1] Despite advances in technology, support of the failing RV, whether acute or chronic, has lagged behind that of the failing LV.

In this review, we describe the anatomy and physiology of the healthy RV and contrast it with the maladaptive responses of the failing one. We provide a conceptual framework for the etiology of RV failure, discuss basic techniques for diagnosing RV dysfunction, and provide general management strategies for the critically ill patient with RV failure. Finally, the article focuses on the treatment of conditions frequently seen in the critically ill patients in the ICU, including decompensated severe pulmonary arterial hypertension (PAH), massive pulmonary embolism (PE), and RV infarction.

ANATOMY AND PHYSIOLOGY ON THE HEALTHY RV

The healthy RV serves 2 roles: to pump venous blood to the lungs and to fill the systemic LV. In the normal heart, the RV fills with blood from the inferior and superior vena cava and pumps it into the pulmonary arteries. During LV diastole, oxygenated blood returns from the lungs by the pulmonary veins. The RV and LV are pumps in series, with roughly equivalent cardiac outputs, although each is characterized by the vasculature they are connected to. The pulmonary vasculature is composed of thin-walled and large-diameter vessels, contrasting sharply with the high-resistance, muscular arteries of the systemic vasculature. Under normal conditions, the pulmonary vasculature is a low-impedance, high-capacitance system, with lower vascular resistance and greater distensibility than the systemic vasculature.[2] Accordingly, the myocardium of the RV is thin, approximately one-third the thickness of the LV, and is more compliant, allowing the RV to accommodate large variations in venous return without significantly altering end-diastolic pressures.[3] Compared with the LV, the RV has increased sensitivity to changes in afterload. Under normal conditions, the systolic pressure of the RV is approximately 25 mm Hg, less than one-fifth the systolic pressure generated by the LV.[2]

The RV appears triangular on longitudinal section and crescent-shaped in cross section.[4] The RV relies primarily on longitudinal shortening during systole whereas the LV uses circumferential constrictor fibers for contraction.[5] This results in a “peristaltic” contraction that moves in a wave from the RV apex to the outflow tract.[3,5] Under normal circumstances, the RV follows the Frank-Starling mechanism by which increases in preload improve myocardial contractility. Factors that influence RV filling include intravascular volume, RV compliance, heart rate and rhythm, LV filling, and abnormalities of the pericardium. Excessive RV volume loading can result in constraint by the pericardium, compression of the LV, and an increase in ventricular interdependence.

The RV has increased resistance to ischemic injury compared with the LV. Besides a lower rate of oxygen consumption, the RV has a more extensive system of collateral vessels. In most individuals, the right coronary artery (RCA) perfuses the RV free wall and the posterior third of the interventricular septum, whereas the anterior two-thirds of the interventricular septum and apex of the RV are supplied by the left anterior descending artery (LAD).[3] Because the RV tissue pressure is lower than aortic root pressure under normal conditions, the RV receives continuous perfusion throughout both systole and diastole.[5] Although patients with acute RV ischemic injury tend to be hemodynamically challenging to manage, those who recover typically do well because of the absence of permanent RV ischemic injury.