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Pulmonary Hypertension in the Intensive Care Unit

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Abstract Pulmonary hypertension, a condition that can lead to right ventricular failure and hemodynamic collapse, can be very challenging to manage in critically ill patients who require the intensive care unit. Because of the underlying structure of the right ventricle, significant increases in right ventricular afterload initiate a vicious cycle of degenerating right ventricular function, giving rise to right ventricular failure and cardiogenic shock. In patients with pulmonary hypertension, inciting factors such as sepsis and arrhythmias can exacerbate this process. Important management principles include close monitoring of hemodynamics with both noninvasive and invasive modalities, optimization of right ventricular preload, maintenance of systemic blood pressure, enhancement of right ventricular contractility, reduction of right ventricular afterload, and reversal of identifiable inciting factors. The goal of this review is to discuss these key concepts in managing this difficult patient population. (Prog Cardiovasc Dis 2012;55:187-198)

Keywords: Pulmonary hypertension; Right ventricular failure; Intensive care unit

Pulmonary hypertension (PH) in critically ill patients requiring the intensive care unit (ICU) is a complex and challenging disorder to manage. Whether the elevations of pulmonary arterial pressures are acute or preexisting, significant PH in the setting of acute illness can lead to rapid deterioration of right ventricular (RV) function, precipitating hemodynamic collapse and death. Outcomes of patients with PH who require the ICU are quite poor. In patients with underlying pulmonary arterial hypertension (PAH) or inoperable chronic thromboembolic PH (CTEPH) who are admitted to the ICU, mortality rates between 32% and 41% have been reported.[1,2] Given their fragile hemodynamic status, understanding the pathogenesis of RV failure secondary to PH is critical for RV rescue and successful treatment of these patients. In this review, we will briefly discuss normal RV structure and function, pathophysiology of RV failure, and management of PH and RV failure in the ICU. Other authors in this symposium will address additional concepts of RV development and support.

Normal structure and function of the RV

The RV should not be viewed as simply a smaller and weaker version of the left ventricle (LV); the two ventricles are embryologically, morphologically, and functionally distinct.[3,4] Although the RV contains helical fibers, unlike the LV, it lacks circumferential constrictor fibers and must therefore rely on longitudinal shortening. This results in a bellows-like contraction beginning near the apex of the heart and moving in a wave toward the outflow tract.[5]

The low pressure and high capacitance of the pulmonary vasculature give rise to pressure-volume relationships for the RV that differ markedly from the pressure-volume relationships for the LV. Under normal loading conditions, the RV has only brief periods of isovolumic contraction and relaxation and has sustained

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Abbreviations and Acronyms

CO = cardiac output

CPR = cardiopulmonary resuscitation

CTEPH = chronic thromboembolic pulmonary hypertension

ICU = intensive care unit

LV = left ventricle

NO = nitric oxide

PA = pulmonary artery

PAC = pulmonary artery catheterization

PAH = pulmonary arterial hypertension

PDE5 = phosphodiesterase-type 5

PH = pulmonary hypertension

PVR = pulmonary vascular resistance

RCA = right coronary artery

RV = right ventricle

SVR = systemic vascular resistance

VA-ECMO = venoarterial extracorporeal membrane oxygenation

ejection during pressure rise and fall. This circuit structure makes for a highly efficient right heart pump, with myocardial energy expenditure approximately one-fifth that of the LV despite similar cardiac outputs (COs). Increases in RV afterload significantly change this dynamic and lead to the development of prolonged periods of isovolumic contraction and relaxation, ultimately resulting in a decline in RV performance[5,6] (Fig 1).

Coronary perfusion also differs between the two ventricles. Myocardial perfusion in the LV occurs predominantly during diastole, when intramyocardial tissue pressure falls below aortic root pressure. Because RV intramyocardial tissue pressure remains below aortic root pressure throughout the cardiac cycle under normal loading conditions, the RV receives continuous coronary flow from the right coronary artery (RCA).[7,8]

Last, the RV and LV cannot be viewed as isolated chambers because they share the same visceral cavity (the pericardium), common myofibers, and the interventricular septum. As a result of this ventricular interdependence and the contractile relationships noted above, RV systolic function depends significantly on the LV (and vice versa), and changes in the condition of one ventricle can significantly impact the function of the other.[9,10] With these normal anatomical and physiologic characteristics in mind, the pathophysiologic derangements that occur in the ICU setting may be better understood.

Pathophysiology of RV failure

Right ventricular failure can be defined as low CO and systemic hypoperfusion despite high RV filling pressures.[11] Because the RV is very sensitive to increases in afterload, rapid elevations in RV afterload decrease RV ejection fraction and induce RV dilatation.[12] In contrast, when RV afterload rises more gradually, adaptive RV myocardial hypertrophy may occur, reducing wall stress to maintain adequate stroke volume.[13-17] In the acute setting, a pressure-overloaded RV dilates and RV end-diastolic volumes and pressures rise, increasing RV wall stress and placing the RV on the descending portion of the Frank-Starling curve.[18] The Laplace relation, which states that wall stress is inversely proportional to the thickness of the wall, helps explain why the thinner RV free wall experiences a greater rise in wall tension with incremental elevations in RV pressure compared with the thicker LV free wall.[7]

Higher wall stress in the RV increases myocardial oxygen demand and oxygen consumption.[19,20] As RV wall tension increases, RCA blood flow, normally continuous during both systole and diastole, occurs only during diastole, causing a further decrease in oxygen delivery to the RV.[8,21] The combination of increased myocardial oxygen demand and decreased oxygen supply leads to RV ischemia and decreased RV contractility.[19,22]

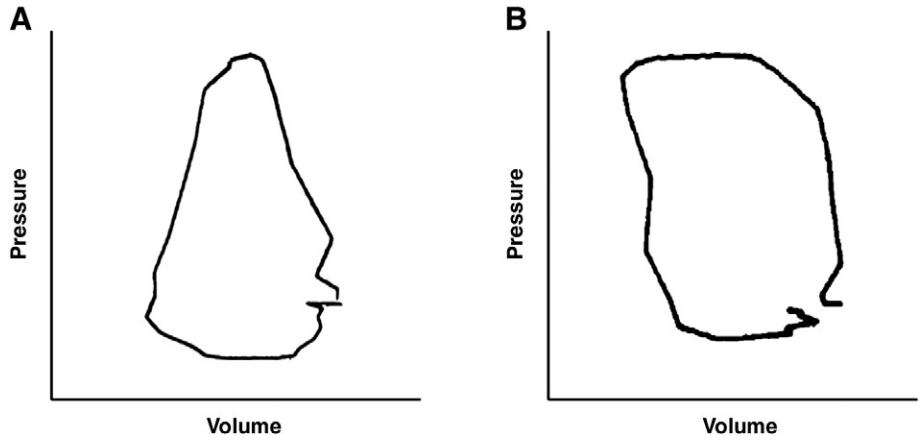

Fig 1. Right ventricular pressure-volume curve from a healthy subject (A) and from a patient with increased RV afterload (B). Adapted from Redington et al.[6]