Hemodynamics of Acute Right Heart Failure in Mechanically Ventilated Patients with Acute Respiratory Distress Syndrome

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KEYWORDS

• Right heart dysfunction • Cor pulmonale • Pulmonary hypertension

KEY POINTS

- The study of right ventricular (RV) dysfunction in intensively ill, mechanically ventilated, and in particular patients diagnosed with ARDS is a new field.
- The RV is significantly susceptible to an increase in afterload (pulmonary vascular resistance), decreased contractility, and primary or secondary alterations in preload (volume and compliance) when challenged with high intrapulmonary pressures.
- In the face of hypoxemic hypoxia, high mean airway pressures, increased shunt, and aggressive volume resuscitation, one should question the validity of monitoring the pulmonary artery (PA) pressures and cardiac output/cardiac index as indicative of isolated LV failure.
- For patients with diagnosed RV dysfunction and ARDS, echocardiogram needs to be used to evaluate the effects of positive pressure on RF performance and LV filling.

INTRODUCTION

In critically ill patients with circulatory shock, the role of the left ventricle (LV) has long been appreciated and the object of measurement and therapeutic targeting. The right ventricle (RV), because of its thinner walls and often nonspecific ejection targets, is often undervisualized and overlooked. Generally, the RV operates passively, with little work or tension required to support the ejection of the diastolic volume. A loss of ventricular wall compliance (diastolic dysfunction) results in an alteration in the normal pressure-volume relationship. Traditional right heart filling indices (right atrial [RA] pressure and/or central venous pressure [CVP]) may increase because of the decreasing compliance. This elevation is frequently incorrectly interpreted as overall

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volume overload or indicative of LV dysfunction, when left ventricular end diastolic volume may actually be critical (secondarily to the RV volume alterations). The elevation in RV filling pressures may also indicate significant obstruction to flow through the pulmonary vascular bed (from positive pressure ventilation or pulmonary embolism). Pulmonary vascular dysfunction in acute respiratory distress syndrome (ARDS) is particularly related to the effects of a mean airway pressure strategy (positive end-expiratory pressure [PEEP], high opening airway pressures) and may contribute to acute cor pulmonale (ACP).[1–3]

The study of RV dysfunction in intensively ill, mechanically ventilated, and in particular, patients diagnosed with ARDS is a new field and knowledge has been built based on the comparison of arterial and pulmonary arterial pressures and calculated cardiac outputs,[4–6] and echocardiographic proof of significant effects on the RV.[3] This article presents a concise clinical perspective on acute respiratory distress and progressive positive mean airway pressure strategies that may induce ACP and RV failure.

ACUTE RIGHT VENTRICULAR FAILURE

The right heart, comprising the right atrium and RV, ejects about 75% of the total volume received by the end of diastole (RV end diastolic volume typically estimated with CVP) into the pulmonary circulation. The RV normally operates below its unstressed volume (ie, increasing filling volume does not raise pressure because the RV is relatively compliant under normal circumstances) and therefore the measured RA pressure may not reflect true volume load.[7] **Fig. 1**A highlights the filling pressures of RV and LV and the corresponding diastolic pressures in the exiting arteries (pulmonary and aorta). The compliant pulmonary vasculature is typically characterized by low resistance, therefore the work of the RV is significantly less than that of the LV, requiring little tension or muscular development to overcome that afterload.

In critically ill patients, the RV is significantly susceptible to an increase in afterload (pulmonary vascular resistance), decreased contractility, and primary or secondary alterations in preload (volume and compliance) (**Fig. 1**B). All three of these factors may be significantly affected by the introduction of positive pressure ventilation strategies focused on the mean airway pressure with little to no regard to the compromise placed

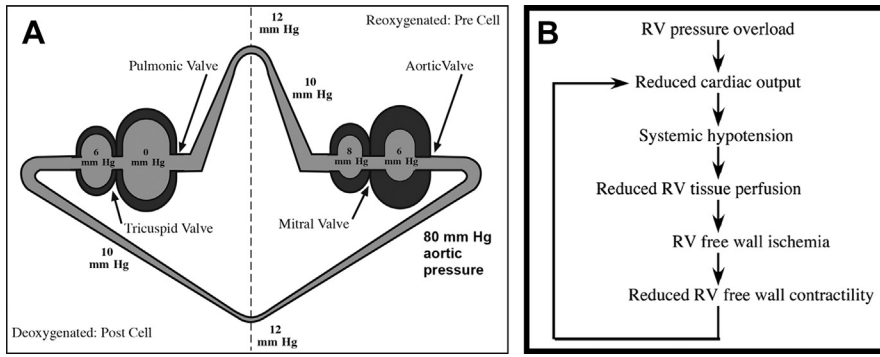

Fig. 1. (*A*) Visualizing the normal pressure relationships associated with right and left ventricular function. (*B*) The pathway to right ventricular dysfunction.