# Cardiovascular Issues in Respiratory Care*

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**The hemodynamic effects of ventilation are complex but can be grouped under four clinically relevant concepts. First, spontaneous ventilation is exercise, and critically ill patients may not withstand the increased work of breathing. Initiation of mechanical ventilatory support will improve oxygen delivery to the remainder of the body by decreasing oxygen consumption. To the extent that mixed venous oxygen also increases, $Pao_2$ will increase without any improvement in gas exchange. Similarly, weaning from mechanical ventilatory support is a cardiovascular stress test. Patients who fail to wean also manifest cardiovascular insufficiency during the failed weaning attempts. Improving cardiovascular reserve or supplementing support with inotropic therapy may allow patients to wean from mechanical ventilation. Second, changes in lung volume alter autonomic tone and pulmonary vascular resistance (PVR), and at high lung volumes compress the heart in the cardiac fossa. Hyperinflation increases PVR and pulmonary artery pressure, impeding right ventricular ejection. Decreases in lung volume induce alveolar collapse and hypoxia, stimulating an increased pulmonary vasomotor tone by the process of hypoxic pulmonary vasoconstriction. Recruitment maneuvers, positive end-expiratory pressure, and continuous positive airway pressure may reverse hypoxic pulmonary vasoconstriction and reduce pulmonary artery pressure. Third, spontaneous inspiration and spontaneous inspiratory efforts decrease intrathoracic pressure (ITP). Since diaphragmatic descent increases intra-abdominal pressure, these combined effects cause right atrial pressure inside the thorax to decrease but venous pressure in the abdomen to increase, markedly increasing the pressure gradient for systemic venous return. Furthermore, the greater the decrease in ITP, the greater the increase in left ventricular (LV) afterload for a constant arterial pressure. Mechanical ventilation, by abolishing the negative swings in ITP, will selectively decrease LV afterload, as long as the increases in lung volume and ITP are small. Finally, positive-pressure ventilation increases ITP. Since diaphragmatic descent increases intra-abdominal pressure, the decrease in the pressure gradient for venous return is less than would otherwise occur if the only change were an increase in right atrial pressure. However, in hypovolemic states, positive-pressure ventilation can induce profound decreases in venous return. Increases in ITP decrease LV afterload and will augment LV ejection. In patients with hypervolemic heart failure, this afterload reducing effect can result in improved LV ejection, increased cardiac output, and reduced myocardial oxygen demand.**

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**Key words:** heart-lung interaction; mechanical ventilation; spontaneous respiration

**Abbreviations:** CHF = congestive heart failure; CPAP = continuous positive airway pressure; HPV = hypoxic pulmonary vasoconstriction; ITP = intrathoracic pressure; LV = left ventricle/ventricular; PEEP = positive end-expiratory pressure; PVR = pulmonary vascular resistance; RV = right ventricle/ventricular; $Svo_2$ = mixed venous oxygen saturation

**Learning Objectives:** 1. To review the complex physiologic interactions between the cardiovascular and respiratory systems as they apply to the critically ill patient. 2. To understand the effects of mechanical ventilation versus spontaneous respiration on cardiorespiratory responses. 3. To describe the impact of ventilator settings and weaning from mechanical ventilation on heart-lung interactions.

Ventilation can profoundly alter cardiovascular function via complex, conflicting, and often opposite processes. These processes reflect the interaction between myocardial reserve, ventricular pump function, circulating blood volume, blood flow distribution, autonomic tone, endocrinologic responses, lung volume, intrathoracic pressure (ITP), and the surrounding pressures for the remainder of the circulation. Clearly, the final response to ventilatory stress is dependent on the baseline cardiovascular state of the subject.

Lung volume increases in a tidal fashion during both spontaneous and positive-pressure inspiration. However, ITP decreases during spontaneous inspiration owing to the contraction of the respiratory muscles, whereas ITP increases during positive-pressure inspiration due to passive lung expansion to increasing airway pressure. Thus, changes in ITP and the metabolic demand needed to create these changes represent the primary determinants of the hemodynamic differences between spontaneous and positive-pressure ventilation.[1]

## Spontaneous Ventilation Is Exercise

Although ventilation normally requires < 5% of total oxygen delivery,[2] in lung disease states the work of breathing is increased, such that its metabolic demand for oxygen may reach 25% of total oxygen delivery. If cardiac output also is limited, blood flow to other organs can be compromised, causing tissue hypoperfusion, ischemic dysfunction, and lactic acidosis.[3] Mechanical ventilation will decrease the work of breathing, even if delivered by noninvasive ventilation mask continuous positive airway pressure (CPAP).[4] The resultant cardiovascular effects will be increased oxygen delivery to other organs, decreased serum lactate levels, and increase mixed venous oxygen saturation ($Sv_{O_2}$). The obligatory increase in $Sv_{O_2}$ will result in an increase in the $Pa_{O_2}$ if fixed right-to-left shunts exist, even if mechanical ventilation does not alter the ratio of shunt blood flow to cardiac output. Finally, if cardiac output is severely limited, respiratory muscle failure develops despite high central neuronal drive, such that many heart failure patients die a respiratory death prior to cardiovascular standstill.[5]

Ventilator-dependent patients who fail to wean from mechanical ventilation may display impaired baseline cardiovascular performance[6] but routinely only have signs of heart failure during weaning. The transition from positive-pressure to spontaneous ventilation can be associated with pulmonary edema,[6] myocardial ischemia,[7,8] tachycardia, and gut ischemia.[9] Jubran et al[10] demonstrated that although all subjects increase their cardiac outputs in response to a weaning trial, consistent with the increased metabolic demand, those who subsequently fail to wean also display a decrease in $Sv_{O_2}$. Since the increased work of breathing may come from the endotracheal tube flow resistance,[11] failure to wean may reflect ventilator work rather than innate respiratory system resistance. Thus, some patients who fail a t-tube trial pass an extubation trial. Since weaning from mechanical ventilatory support is a cardiovascular stress, it is not surprising that weaning-associated ECG and thallium cardiac blood flow scan-related signs of ischemia have been reported in both subjects with known coronary artery disease[7] and in otherwise normal patients.[8] Similarly, initiating mechanical ventilation in patients with severe heart failure and/or ischemia can reverse myocardial ischemia.[12]

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## Inspiration Increases Lung Volume

### *Autonomic Tone*

Inflation induces immediate changes in autonomic output,[13] causing cardiac acceleration otherwise known as respiratory sinus arrhythmia,[14] which implies normal autonomic responsiveness.[15] Loss of respiratory sinus arrhythmia is seen in diabetic peripheral neuropathy, and its reappearance precedes the return of peripheral autonomic control.[16] Lung inflation to larger tidal volumes (> 15 mL/kg) decreases heart rate by sympathetic withdrawal.[17] Reflex arterial vasodilatation can also occur with lung hyperinflation.[13,18] Since patients with ARDS often ventilate a small amount of lung, regional hyperinflation of aerated lung units may develop and lead to reflex cardiovascular depression. Although humoral factors released from pulmonary endothelial cells during lung inflation may also induce this depressor response,[19] these interactions do not appear to be relevant clinically because unilateral lung hyperinflation does not alter systemic hemodynamics.[20]

### *Humoral Factors*

Positive-pressure ventilation and sustained hyperinflation induce fluid retention via right atrial stretch receptors that increase plasma norepinephrine, plasma renin activity,[21] and decrease atrial natriuretic peptide activity.[22] Congestive heart failure (CHF) patients receiving nasal CPAP decrease their plasma atrial natriuretic peptide activity inversely with the associated increase in cardiac output.[23]

### *Pulmonary Vascular Resistance*

Lung inflation primarily affects cardiac function and cardiac output by altering right ventricular (RV) preload and afterload.[24] Changes in ITP that occur without changes in lung volume, as may occur with obstructive inspiratory efforts or a Valsalva maneuver, will not alter pulmonary vascular resistance (PVR). Lung volume must change. The mechanism by which ventilation alters PVR is complex. If regional alveolar $P_{O_2}$ decreases below 60 mm Hg, local