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ISCCM Manual of Noninvasive Ventilation

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Chapter

2

Heart–Lung Interactions during Noninvasive Ventilation

Jigeeshu Divatia, Jacob George Pulinilkunnathil

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INTRODUCTION

Heart–lung interactions refer to the changes in cardiovascular physiology resulting from changes in lung volume and intrathoracic pressure (ITP) during respiration. The heart and lungs are enclosed in the thoracic cavity and are subjected to the surrounding intrathoracic pressure. Phasic changes in the intrathoracic pressure and lung volume during respiration affect the cardiac function. These interactions become more prominent with intermittent positive pressure ventilation (IPPV). This chapter describes physiological changes and the clinical applications of heart–lung interactions during noninvasive ventilation (NIV).

The major determinants of heart–lung interactions are:

- Changes in intrathoracic pressure
- Changes in lung volume
- Ventricular interdependence
- Changes in intra-abdominal pressure due to the movements of the diaphragm

EFFECTS OF CHANGES IN INTRATHORACIC PRESSURE

Venous Return

During spontaneous respiration, the intrathoracic pressure reduces with the onset of inspiration. The reduction in intrathoracic pressure is transmitted to the right atria. The gradient between mean systemic filling pressure (MSFP) and right atrial pressure (RAP) increases, resulting in increased venous return and an increase in RV preload **(Fig. 1)**. The increase in venous return is limited due to the collapse of the great veins at the root of the neck at extremes of ITP.

With positive pressure ventilation, the increase in ITP is transmitted to the right atrium and MSFP–RAP gradient reduces, resulting in a decreased venous return and reduced right ventricular (RV) output.[1] This effect is more significant in hypovolemic patients. Compensatory mechanisms include venoconstriction due to an increase in sympathetic tone secondary to lung inflation. This maintains the MSFP–RAP gradient and venous return. The descent of diaphragm during inspiration increases the intra-abdominal pressure, compresses the splanchnic circulation, and also aids in maintaining the venous return to thorax.[2]

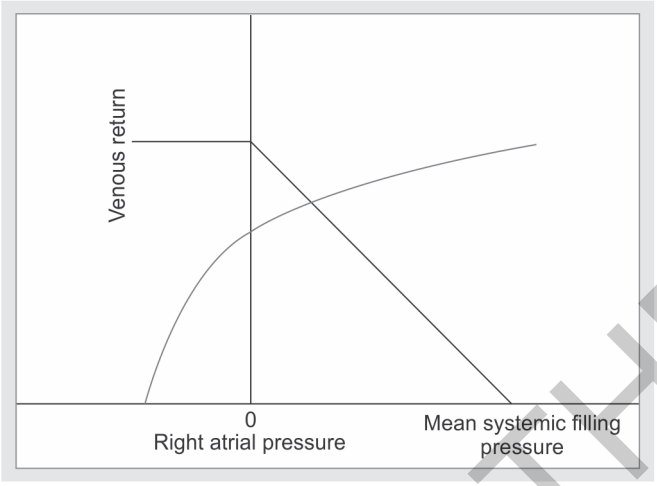
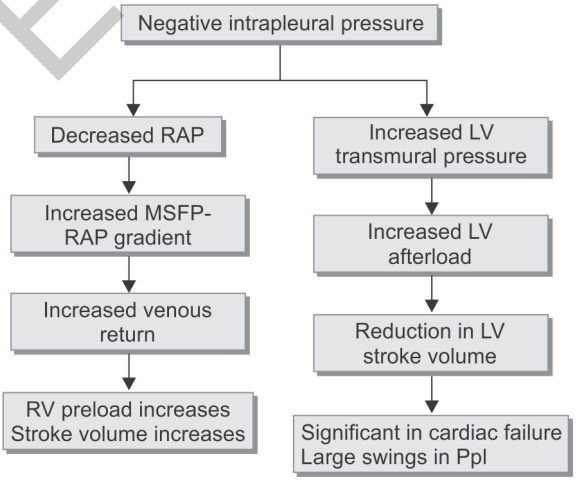


Fig. 1: Graph showing the relation between right atrial pressure, mean systemic filling pressure (MSFP), and venous return.

Ventricular Function

The ventricular output depends upon the preload, contractility, and afterload. The transmural pressure of the ventricle is the intraventricular pressure minus the intrathoracic pressure. During normal inspiration, the intrathoracic pressure is negative. During extreme tachypnea or removal of positive pressure support, the ITP becomes extremely negative, and the left ventricular (LV) and aortic transmural pressures increase, resulting in an increased afterload and impedance to left ventricular output. Simultaneously, a reduction in ITP increases venous return and intrathoracic blood volume.[3] Hence, during weaning from mechanical ventilation, as the spontaneous ventilation is being reduced, the venous return and the RV preload

Flowchart 1: Effects of spontaneous breathing on biventricular function.



(RAP: right atrial pressure; LV: left ventricle; MSFP: mean systemic filling pressure; RV: right ventricle; Ppl: intrapleural pressure)

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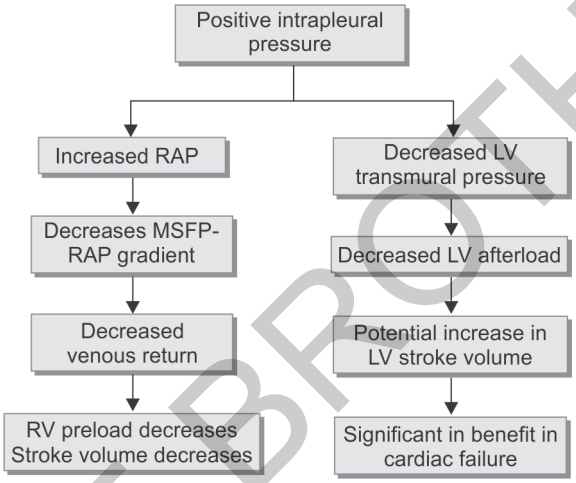
increase while the LV output is impeded by an increase in LV transmural pressure. This may result in weaning-induced pulmonary edema. The effects of intrathoracic pressure swings on biventricular function during spontaneous ventilation are summarized in **Flowchart 1**.

With IPPV, the venous return reduces and RV afterload increases, with a resultant reduction in right ventricular output and LV preload. The positive intrapleural pressure decreases the LV transmural pressure and afterload, thus improving ejection of the left ventricle.[3]

These physiological changes induced by IPPV are used to manage heart failure. The effects of IPPV on biventricular function are summarized in **Flowchart 2**.

Flowchart 2: Effects of intrathoracic pressure (ITP) during positive pressure ventilation on biventricular function.



(RAP: right atrial pressure; LV: left ventricle; MSFP: mean systemic filling pressure; RV: right ventricle; Ppl: intrapleural pressure)

EFFECTS OF CHANGES IN LUNG VOLUME

Changes in Autonomic Tone

Inflation of the lungs with normal tidal volumes has a vagolytic effect resulting in tachycardia, whereas lung inflation with tidal volume >10 mL/kg can result in bradycardia with vasodilation. Positive pressure ventilation causes increased prostaglandin synthesis and reduced atrial natriuretic peptide (ANP) synthesis resulting in salt and water retention.[4]

Effects on the Cardiovascular System

During IPPV, as the lungs expand, the pulmonary vessels are squeezed, resulting in a transient increase in blood volume entering the left atrium. This effect is overtaken by the reduction in venous return, with a net reduction in preload to both ventricles. The inflated lungs compress the heart in the cardiac fossa and reduce the cardiac volume resulting in a reduction in diastolic compliance.[5]

During spontaneous breathing, the dilation of pulmonary vessels results in pooling of blood in the pulmonary circulation and reduced LV preload. Over the next few beats, increased venous return leads to increased right ventricular output with resultant increase in LV preload.

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Effects on Pulmonary Vascular Resistance

At low lung volumes, extra-alveolar vessels get kinked with resultant increase in pulmonary vascular resistance (PVR). At high-lung volumes, PVR is increased as overdistended alveoli compress the alveolar capillaries. The net PVR is least at functional residual capacity (FRC) and increases above and below the FRC.[6] RV afterload is determined mainly by the PVR.

VENTRICULAR INTERDEPENDENCE

Both ventricles are enclosed together in the pericardium and separated by the interventricular septum. The phenomenon by which volume or pressure overloading of either ventricle affects the function of the other ventricle is called ventricular interdependence.

Series Effect

During IPPV, the phasic changes in lung volume and intrathoracic pressure result in changes in the RAP, venous return, and intrathoracic blood volume. During inspiration, there is a reduction in RV preload and RV stroke volume, which results in reduced LV preload after about three heart beats. The LV output is also reduced transiently provided it is preload-responsive. These phasic variations in LV stroke volume (stroke volume variation—SVV) or its surrogate arterial pulse pressure [systolic pressure variation (SPV) and pulse pressure variation (PPV), respectively] during ventilation can help to identify patients are fluid responsive **(Fig. 2)**.

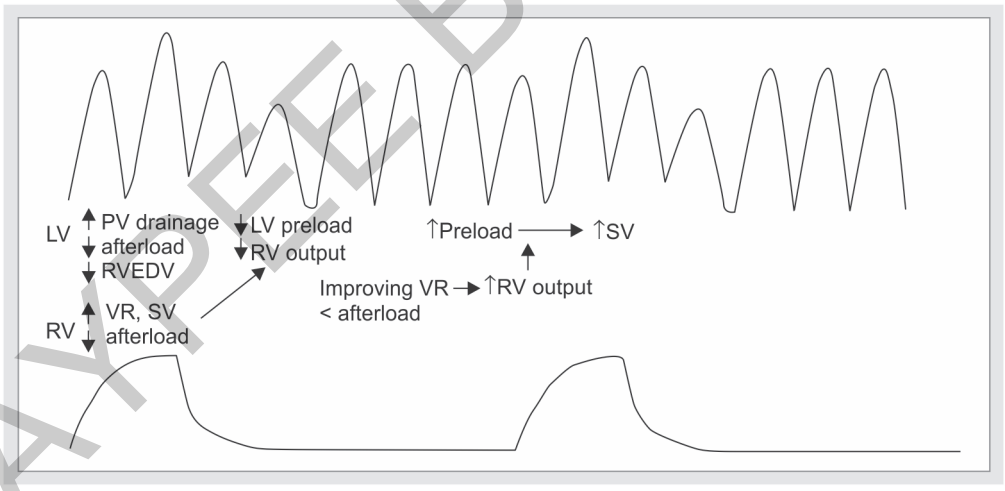

Fig. 2: Hemodynamic changes due to ventricular interdependence in series. (LV: left ventricle; RV: right ventricle; RVEDV: right-ventricular end-diastolic volume)

Parallel Effect

Normally, the LV pressure exceeds the RV pressure and the interventricular septum is usually central. During spontaneous breathing, in deep inspiration, the negative intrathoracic pressure results in increased venous return to the RA and preload of the RV.[7] If the RV is dilated, there

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is a shift of the septum into the LV cavity, reducing the left-ventricular end-diastolic volume (LVEDV) and stroke volume. If the there is a pericardial effusion that prevents expansion of the volume of the cardiac chambers, the LV diastolic compliance, LV filling, LVEDV, and LV output decrease, especially during inspiration, when the venous return and RV filling increase. This is manifested at bedside as an exaggerated drop in systolic pressure during inspiration in a spontaneously breathing patient, called pulsus paradoxus **(Fig. 3)**.

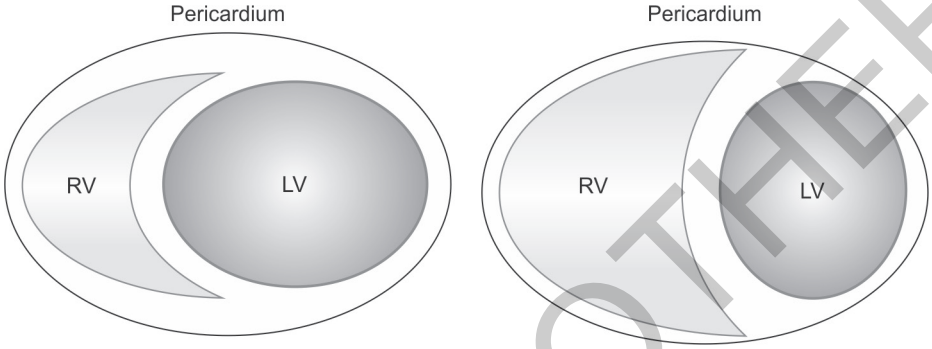

Fig. 3: Ventricular interdependence in parallel. (LV: left ventricle; RV: right ventricle)

CLINICAL APPLICATIONS OF HEART–LUNG INTERACTION

Pulsus Paradoxus

Pulsus paradoxus is an exaggerated physiological phenomenon based on ventricular interdependence. As ITP becomes negative during inspiration, the venous return increases and volume overload of the right ventricle results in a shift of the interventricular septum toward the left ventricle. This reduces the diastolic compliance of left ventricle, and reduces LVEDV and a fall in left ventricle stroke volume that manifests as a fall in systolic pressure. This systolic reduction in blood pressure during normal inspiration is further accentuated in patients with active breathing efforts with great negative swings of intrathoracic pressure and can be a marker of disease severity, e.g., status asthmaticus and pericardial diseases.[8]

Reverse Pulsus Paradoxus

A paradoxical increase in systolic pressure during inspiration followed by a decrease in systolic pressure during expiration is called reversed pulsus paradoxus. This is due to an augmentation in left ventricular function due to a significant reduction in left ventricular afterload in patients with relative hypervolemia such as LV systolic failure.

Kussmaul's Sign

During spontaneous breathing, inspiration results in reduced intrathoracic and right atrial pressure, increased MSFP–RAP gradient, and increased venous return. In conditions of poor RV compliance and increased RV afterload (pulmonary embolism), an increased venous return during inspiration will increase the right atrial pressure and central venous pressure (CVP). This paradoxical rise of CVP during inspiration is called Kussmaul's sign.[9]

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Negative Pressure Pulmonary Edema

In cases where there is a markedly negative intrathoracic pressure—such as breathing against an obstructed glottis—there is a marked increase in the venous return. The left ventricular transmural pressure will increase significantly, and the left ventricular outflow is impeded. This combination of increased venous return associated with an increased impedance to left ventricular ejection results in increased intrathoracic blood volume, increased venous hydrostatic pressure, and pulmonary edema, called negative pressure pulmonary edema.[10]

Predicting Fluid Responsiveness

During respiration, there is a cyclic change in the intrathoracic pressure causing cyclic changes in right ventricular and left ventricular function. These are identified at the bedside as systolic pressure variation (SPV), stroke volume variation (SVV), and pulse pressure variation (PPV), and are increasingly used to determine fluid responsiveness.[11] Both PPV and SVV can be calculated at bedside by commercially available minimally invasive monitors using inbuilt mathematical algorithms.

However, during spontaneous breathing, as during noninvasive ventilation, these cyclical changes in intrathoracic pressure and lung volume are not of the same magnitude. PPV and SVV may remain falsely negative even in preload responders and cannot be used as predictors of fluid responsiveness. Similarly, the variation in vena cava diameter in accordance with respiration measured by bedside echocardiography, either at superior vena cava (SVC) or inferior vena cava (IVC) distal to the entry of portal vein, is unreliable in spontaneously breathing patients receiving noninvasive ventilation.

Management of Cardiogenic Pulmonary Edema

The pathophysiology of cardiogenic pulmonary edema (CPE) includes:

- Reduced left ventricular contractility, hypervolemia, and an increased afterload
- A low cardiac state resulting in tissue hypoxia that in turn increases myocardial stress and ischemia
- Wide swings of intrathoracic pressure with tachypnea that increases the venous return to the thorax. Positive pressure effectively acts on each step:
  - An increased intrathoracic positive pressure decreases the venous return and the preload of the heart
  - An increased intrathoracic positive pressure reduces the left ventricular transmural pressure, thereby reducing afterload and decreasing the impedance to the left ventricular ejection
  - As the respiratory muscles are also supported, the work of breathing reduces resulting in a reduction in the wide fluctuations of intrathoracic pressure. IPPV and PEEP maintain alveoli patent, augmenting oxygenation and reducing hypoxia-induced vasoconstriction and PVR.

In a large multicenter trial, 1,069 patients presenting to the emergency departments in the United Kingdom were randomized to receive standard oxygen therapy, continuous positive airway pressure (CPAP) (5–15 cm H_2O), or NIV (inspiratory pressure, 8–20 cm H_2O; PEEP, 4–10 cm H_2O). There was earlier improvement in symptoms of respiratory distress with CPAP and NIV compared to oxygen therapy; however, there was no difference in survival.[12]

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Systematic reviews[13-17] have concluded that NIV decreases the need for intubation and hospital mortality, and although an early trial suggested greater risk of myocardial infarction with NIV than with CPAP,[18] there is no increase in the occurrence of acute myocardial infarction with NIV. The current European Respiratory Society/American Thoracic Society (ERS/ATS) clinical practice guidelines on noninvasive ventilation for acute respiratory failure[19] recommend either bi-level NIV or CPAP for patients with ARF due to cardiogenic pulmonary edema (strong recommendation, moderate certainty of evidence).

Pulmonary Edema of Weaning

When patients with borderline cardiac reserve are planned for weaning, as spontaneous breathing is resumed, the left ventricular afterload increases and the work of left ventricle increases. The reduction in intrathoracic pressure increases the venous return to the heart and the patient is at risk of pulmonary edema **(*see* Flowchart 1)**. The net effect is an increased intrathoracic blood volume and decreased left ventricular function, both resulting in increased pulmonary hydrostatic pressure and pulmonary edema in patients with poor cardiac function. Development of pulmonary edema is associated with a drop in the lung volume and lung compliance, requiring greater respiratory effort for inspiration. These efforts in virtue of deep inspiratory efforts reduce the ITP and decrease the transmural pressure, thereby increasing the afterload. The increased afterload causes more pulmonary edema and the vicious cycle continues. In such patients, weaning or attempts to weaning cause an increased myocardial oxygen demand, increased respiratory muscle load, and increased oxygen demand due to pulmonary edema.[20] Such patients, if identified early, will benefit from reduction in preload and afterload or will require positive pressure support even after extubation, such as with NIV.

Cardiopulmonary Interactions that may Contribute to Lung Injury during NIV

The negative intrapleural pressure during a spontaneous breath results in higher alveolar transmural pressures with potential for volutrauma. Simultaneously, the transmural pressure across the pulmonary capillaries becomes more negative, resulting in increased pulmonary capillary blood flow and fluid shift from the capillaries into the pulmonary interstitium.[21]

In animal experiments, it has been shown that with a high-tidal volume, pulmonary blood flow and right ventricular cavity can be obliterated during inspiration and restored during expiration. This high flow–no flow–high flow cycle causes vascular shearing and endothelial injury. If, during NIV, high respiratory drive results in high tidal volumes then there is potential for lung injury by this mechanism. This may be mitigated, if NIV reduces work of breathing with a consequent reduction in tidal volume, by the maintenance of diaphragmatic tone that results in a counterbalancing PEEP.[22]

CONCLUSION

The heart–lung interactions occurring due to noninvasive ventilation can be beneficial or detrimental. These interactions can be successfully used for benefit in select cases such as cardiac failure. Early identification of patient's disease states and prediction of the possible heart–lung interactions are important for further hemodynamic monitoring and to optimize ventilatory management in critically ill patients.

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