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Pathophysiology and Clinical Impact of Heart-Lung Binomen in ICU

Coniglione F., Huti G., Bajraktari M., Abdyli A., Xhaja A. and Drishti A.
University of Medicine, UHC "Mother Teresa", Tirana, Albania

The heart-lung relationship represents a critical duality in the intensive care unit (ICU). This interrelationship gives rise to several "conflicts," such as reno-cardiac, renal, reno-pulmonary syndromes, mechanical ventilation-brain issues, and anticoagulants for cardiac reasons causing hemorrhages (gastrointestinal, cerebral). Recent literature provides valuable insights into the heart-lung duality, helping to understand the primary problems and side effects on each system during therapeutic interventions targeting the other system (1). The heart-lung circulation interconnects these organs, necessitating consideration of one organ's effects on the other to prevent decompensation of related chronic diseases.

Chronic pulmonary diseases (asthma, emphysema, chronic obstructive pulmonary disease) increase pulmonary resistance, leading to pulmonary hypertension (2). Pulmonary hypertension raises right ventricular afterload and pressure in the pulmonary artery, which can decompensate the right ventricle. During asthma, forced inspiratory pressure generates a significant negative intrathoracic pressure, enhancing venous return and overfilling the right ventricle. Right ventricular failure and increased filling pressures shift the interventricular septum leftward, reducing left ventricular filling volumes, leading to low cardiac output and pulmonary congestion. Spontaneous tachypneic respiration results in increased respiratory effort, fatigue and hypoxia (3). Hypoxia disrupts myocardial oxygen balance, negatively impacting cardiac output, arterial pressure and systemic organ perfusion.

Mechanical ventilation traps the heart between the lungs, which can increase diastolic filling pressures, decrease end-diastolic filling volumes, cause significant diastolic dysfunction, and further reduce stroke volume and cardiac output. Mechanical ventilation disrupts normal physiology, affecting heart function and hemodynamics (4). Under normal conditions, diaphragmatic movements during inspiration create negative pressure, enhancing the pressure difference between the mean systemic pressure and right atrial pressure. This pressure difference determines preload and venous return, which are components of cardiac output. Negative intrathoracic pressure directs venous blood into the heart, increasing end-diastolic filling. However, mechanical ventilation can overload and dilate the right ventricle, compressing the left ventricle. Under mechanical ventilation/PEEP/recruitment maneuvers, abnormal positive pressure inspiration reduces

venous return, cardiac output, and causes hypotension/ hypoperfusion. These phenomena are more pronounced under preexisting hypovolemia.

Lung hyperinflation increases right ventricular afterload and decreases right ventricular stroke volume, predisposing to hypotension via intraventricular septum shift and reduced left ventricular preload (5). Mechanical ventilation also stretches pulmonary vasculature, contributing to pulmonary hypertension.

During the weaning process, spontaneous breathing-induced negative intrathoracic pressure increases left ventricular afterload, left ventricular overfilling, left ventricular ischemia, and pulmonary edema, especially in patients with impaired left ventricular function.

Therapeutic maneuvers involve several strategies. Strict hemodynamic monitoring is essential, particularly in ventilated patients with septic shock and/ or ARDS. TEE, TTE, Doppler examination, PICCO, and pulmonary artery catheterization provide valuable information about hemodynamic changes during mechanical ventilation. Monitoring right ventricular filling is crucial. Excessive ventilation stretches pulmonary vessels, causing pulmonary hypertension and further reducing right ventricular afterload and ejection. Right ventricular overfilling leads to right ventricular ischemia and failure, reducing diastolic filling of the left ventricle. Right ventricular function can be supported by optimizing volume, inotropism, and reducing afterload. Drugs that reduce afterload include inodilators (dobutamine, milrinone) and pure dilators such as NO. ECMO can help mitigate the side effects of mechanical ventilation.

In conclusion, intensive care physicians must possess a thorough understanding of the interrelated adverse effects of the heart-lung pathophysiological relationship (6) to optimize patient’s outcomes.

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