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Advances in Ventilation Management for Patients with Acute Respiratory Distress Syndrome

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DESCRIPTION

A ventilator has several effects on lung mechanics. When the diaphragm is pushed down during inspiration, it creates negative pressure in the chest cavity, creates negative pressure in the airways, and draws air into the lungs. The same negative intrathoracic pressure reduces pressure in the right atrium (RA), aspirates the Inferior Vena Cava (IVC), and increases venous return. The use of positive pressure ventilation changes this physiology. The positive pressure generated by the ventilator is transmitted to the upper airways, ultimately to the alveoli, to the alveolar cavity and the thoracic cavity, creating a positive pressure (or at least a lower negative pressure) in the pleural cavity. Increased RA pressure and reduced venous return reduce preload. This has a double effect on reducing cardiac output. With less blood in the right ventricle, less blood reaches the left ventricle, less blood can be pumped out, and cardiac output decreases. Less preload means the heart is working at a less efficient point on the Frank-Starling curve, and not doing a more efficient job, resulting in a further decrease in cardiac output and an average arterial Decreased when there is no compensatory response to increased pressure (MAP). This is a very important consideration for patients who may not be able to increase their SVR, such as those with distributive shock (septic, neurogenic, or anaphylactic shock). On the other hand, mechanical ventilation with positive pressure can significantly reduce the work of breathing. This reduces blood flow to the respiratory muscles and redistributes it to more vital organs. Reducing the work of respiratory muscles also reduces the production of CO_2 and lactic acid from these muscles, helping to improve acidosis. The effect of positive pressure ventilation on venous return may be beneficial in patients with cardiogenic pulmonary edema. In these patients with volume overload, reducing venous return directly reduces the amount of pulmonary edema caused by reduced right cardiac output. At the same time, a reduction in regurgitation may improve left ventricular hyperextension, placing it in a more favorable position on the Frank-Starling curve and improving cardiac output. An understanding of lung pressure and compliance is also required. Normal lung compliance is approximately 100 ml/cm H_2O. This means that in normal lungs, 500 ml of air delivered *via* positive pressure ventilation raises alveolar pressure by 5 cm H_2O. Conversely, a positive pressure of 5 cm H_2O increases lung volume by 500 ml. Abnormalities in the lungs can lead to significantly higher or lower compliance. Diseases that destroy the lung parenchyma, such as emphysema, increase compliance. Lung stiffening diseases (ARDS, pneumonia, pulmonary edema, pulmonary fibrosis) reduce lung compliance. The problem with stiff lungs is that a small increase in volume can cause a large increase in pressure, causing barotrauma, which causes problems in patients with hypercapnia or acidosis. This is because an increase in minute ventilation may be required to correct these problems. Increased respiratory rate can accommodate increased minute ventilation, but if this is not possible, increased tidal volume increases plateau pressure and can lead to barotrauma.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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