Lung Distention, Barotrauma and Mechanical Ventilation

M.R. Pinsky

Introduction

Mechanical ventilation usually entails the application of varying amounts of positive airway pressure in a cyclic fashion to force tidal breaths into the lungs and maintain distending pressure. The addition of supplemental O₂ to enrich the ventilating gas is universal, and is itself a separate factor of artificial ventilation. Positive-pressure ventilation supplies a force necessary to overcome elastic and resistive forces of the entire ventilatory apparatus. Only a portion of this force is actually directed at alveolar distention, and in subjects with severe bronchospasm or asynchrony of spontaneous ventilatory efforts with those of the ventilator, very little of the distended pressure may be sensed by the alveoli. However, if positive-pressure ventilation grossly overdistends the alveoli even once or induces repetitive degrees of lesser overdistention of the lungs because either the overall tidal volume is too high or the distribution of the delivered gas is such that only certain regions of the lung are distended, then alveolar injury may occur. These injuries are collectively referred to as barotrauma. Such injury actually reflects over-distention of the alveoli rather than over-pressure of the airways. Accordingly, the term “volutrauma” has been suggested to reflect this process [1]. Although overdistention of the lung at end-inspiration is a major cause of barotrauma, changes in end-expiratory volume also play a major role, especially if fixed tidal volumes are used to ventilate a subject. In fact, much of the end-inspiratory overdistention can be directly linked to sustained hyperinflation at end-expiration rather than to what would otherwise be normal tidal volumes. Thus, we will first describe normal physiology as it relates to end-expiratory volume, then progress through overdistention of normal lung to often inadvertent overdistention of diseased lung.

Normal ventilatory physiology and mechanical ventilation

Functional residual capacity (FRC) and hyperinflation

End-expiratory lung volume is the volume in the thorax at the end of expiration, immediately proceeding inspiration. Under normal conditions, that is when:
1) expiratory time is adequate to allow all alveolar gas under pressure to exhale, and 2) airways obstruction does not either limit exhalation or does not prematurely collapse the terminal airways trapping alveolar gas which would otherwise exhale, end-expiratory volume reflects functional residual capacity (FRC). FRC represents an equilibrium intrathoracic volume between the elastic forces of the lung parenchyma, which tend to collapse the lung inward towards itself and the chest wall and respiratory muscles, which through resting muscle tone, bony stiffness and gravitational effects pulling on the diaphragm tend to increase intrathoracic volume. Thus, at FRC resting pleural pressure is negative as it balances these two opposing forces. FRC is a dynamic volume in normal conditions, varying by over one liter in normal 70 kg subjects by changes in body position (recumbancy decreases FRC by reducing the gravitational effects on the diaphragm), exercise and breathing pattern. FRC will also vary relative to pleural pressure as a function of lung compliance. The degree of lung distention is a function of both lung compliance and the distending pressure, which is the transpulmonary pressure. Transpulmonary pressure can be approximated as airway pressure minus pleural pressure. Accordingly, FRC may exceed resting FRC values if pleural pressure were to become more negative, airway pressure more positive, lung compliance increase or chest wall stiffness increase. Pleural pressure often becomes more negative during expiration because of expiratory muscle breaking (maintenance of respiratory muscle tone during expiration) as occurs in exercise and asthma. Furthermore, external negative body pressure (iron lung) may increase FRC. Increases in airway pressure at end-expiration include air trapping due to terminal airway collapse as occurs in patients with chronic airflow obstruction when end-expiratory airway pressure is intentionally increased by the application of either positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP). In fact, almost all of the gas exchange effects of both PEEP and CPAP can be explained by their ability to increase FRC. Lung compliance may vary acutely if the subject develops increased lung water or even overt pulmonary edema. Finally, chest wall stiffness may vary via expiratory breaking, changes in intra-abdominal pressure or body position. As FRC varies, the level to which end-inspiratory lung volume will also vary for fixed tidal volume ventilation. Accordingly, a major factor inducing marked overdistention of the lungs at end-inspiration is hyperinflation at end-expiration.

**Tidal ventilation**

Cyclic increases in transpulmonary pressure induce the pressure gradient between the alveoli and the airway opening necessary to generate airflow into the lungs. The degree of inflation will be primarily a function of the distending pressure and, at high flow rates, the time for inspiration. Exhalation is usually passive, with air flowing out of the alveoli as a function of driving pressure and airways resistance. The product of lung compliance and airway resistance is called the respiratory time constant and reflects the overall expiratory flow characteristics of the lung. During spontaneous inspiration, pleural pressure