Pulmonary System and Obesity

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• Mechanical ventilation • ARDS

There are several challenges in the management of respiratory failure in the obese population. Pulmonary physiology is significantly altered leading to reduced lung volumes, decreased compliance, abnormal ventilation and perfusion relationships, and respiratory muscle inefficiency.[1] These complications lead to a prolonged requirement for mechanical ventilation and increased intensive-care-unit (ICU) length of stay.[2,3]

LUNG VOLUMES

Lung volumes are appreciably affected by obesity. Most reported is a decrease in functional residual capacity (FRC) and expiratory reserve volume (ERV).[4,5] An inverse relationship between body mass index (BMI) and FRC has been established[4,5] with modest increases in BMI leading to decreased FRC and ERV.[4] Vital capacity (VC), total lung capacity (TLC), and residual volume (RV) can also be reduced but to a lesser extent. Jones and Nzekwu[4] studied the effect of obesity on lung volumes in association with BMI and noted a 0.5% decrease in VC, TLC, and RV for each unit increase in BMI as compared with a 3% and 5% decrease in FRC and ERV, respectively. The increase in adipose tissue around the rib cage and abdomen leads to decreased chest wall compliance and increased resistance, which in turn increases the mass load on the respiratory musculature and significantly reduces FRC.[6] These changes are even more notable in the supine position because of the impedance of the diaphragm by the abdomen.[7] As the FRC decreases it approaches the closing capacity, which can lead to airway closure within the range of tidal breathing. Areas of lung may be underventilated leading to intrapulmonary shunting and hypoxemia.[8,9]

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LUNG MECHANICS

Another important alteration in respiratory physiology of patients who are obese is an overall stiffening of the respiratory system. Pulmonary compliance is significantly reduced in these patients and can be reduced up to 42% in patients with obesity hypoventilation syndrome.[10] This loss of compliance has been demonstrated in multiple studies and appears to be exponentially related to BMI.[5] There are several mechanisms contributing to the decrease in lung and chest wall compliance.[1] Reduced lung compliance is in part caused by an increase in the volume of blood within the pulmonary circulation,[11] but mostly is a function of decreased volumes. The lower the FRC the more it approaches the closing capacity of the smaller airways causing collapse and atelectasis.[9] These lower volumes also increase alveolar surface tension leading to decreased compliance.[12]

Several authors report that increased adipose tissue around the ribs, diaphragm, and abdomen leads to a decrease in chest wall compliance[1,5,6] and has been reported to be more pronounced in the supine position.[6] It has also been postulated that the reduction in compliance is caused by lower volumes and that obese subjects are breathing over a less compliant area of the thoracic volume-pressure curve.[10] The data regarding chest wall compliance remains variable because several studies have shown little or no effect on overall pulmonary compliance, stressing more of a reduction in lung compliance as the major factor.[5,13,14] Despite the controversy in the literature, which may be secondary to differences in technique, it seems logical that increased mass loading on the chest affects respiratory mechanics negatively, possibly because of irreversible closure or collapse of alveolar units leading to decreased lung compliance.[10]

Further compounding the increased work of breathing in obese individuals is increased airway resistance. The cross-sectional area of the upper airway is reduced because of increased parapharyngeal fat deposition. These airways are also more prone to collapse, especially at lower lung volumes as is seen with increased BMI.[15,16] The smaller airways are also affected. Rubinstein and colleagues[17] found higher levels of pulmonary resistance, a decrease in forced expiratory volume in the first second of expiration (FEV_1), and decreased flow rates in obese versus nonobese individuals. This finding suggested that change in airway caliber may be representative of remodeling from inflammatory adipocytokines or damage from repeated opening and closing of the airways during tidal respirations.[18]

VENTILATION AND PERFUSION

Obesity has been associated with changes in ventilation and perfusion (V/Q) matching. In normal pulmonary physiology the distribution of regional ventilation and perfusion is predominantly in the lower, dependant lung zones. In obesity the lower lung zones are also predominantly perfused, but ventilation is preferentially distributed to the upper lung zones.[19,20] It is thought that these changes are likely caused by small airway closure caused by lower ERV[19] and possibly related to extrinsic factors affecting chest wall and diaphragm function,[20] which can lead to V/Q mismatching and hypoxemia. These changes are more pronounced in the supine and lateral decubitus positions, and an inverse relationship between BMI and the partial pressure of oxygen in the inferior pulmonary vein while in the supine position has been identified.[21]

RESPIRATORY MUSCLE INEFFICIENCY

Obesity leads to decreased endurance of the muscles of respiration and increased oxygen consumption (VO2) by those muscles. It is estimated that the maximum