Respiratory function abnormalities in morbidly obese subjects and the risk of low lung volume injury

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SUMMARY. Morbidly obese subjects are characterized by important changes in respiratory function both during spontaneous breathing as well as during general anesthesia and mechanical ventilation. The most characteristic abnormalities consist of decreased functional residual capacity (FRC), reduced expiratory reserve volume, decreased compliance and increased resistance of the respiratory system. Breathing at low lung volume promotes airway closure in the dependent lung zones with consequent gas exchange abnormalities. Furthermore, the decreased expiratory reserve as a result of decreased FRC and the higher ventilatory requirements of these patients due to increased metabolic demands may promote expiratory flow limitation (EFL) in the tidal volume range. The presence of peripheral airway closure and EFL during tidal breathing promotes peripheral airway injury and may accelerate the abnormalities of lung function. The risk of injury is expected to be higher during mechanical ventilation due to the high pressure transients, which develop under this condition. Consequently, external positive end-expiratory pressure must be applied to these subjects in order to increase the end-expiratory lung volume above the closing volume as well as the flow limitation volume and thus, decrease the risk of peripheral airway injury. Pneumon 2007; 20(3): 230–234.

INTRODUCTION

Obesity, a modern epidemic of developed societies, is a metabolic disease characterized by an excessive accumulation of adipose tissue. It is a multifactorial disease involving the integration of social, behavioural, cultural, metabolic and genetic factors. Its impact on developed societies is rising and it is estimated that 97 million adults in the USA and 15-20% of the population in Europe are considered to be obese¹.

For the assessment of body fat in the clinical setting the most commonly used index currently is the body mass index (BMI), calculated as the quotient

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of body weight and height squared (Kg/m²). The upper limit of normal for the BMI is 25 Kg/m². Values of greater than 30 Kg/m² correspond to obesity and values in excess of 40 Kg/m² to severe obesity. Obesity is associated with a variety of acute and chronic morbidity conditions that may result in a decrease in life expectancy².

Pulmonary function alterations during spontaneous breathing and during anaesthesia and mechanical ventilation are among the most frequent complications of obesity. It is important to identify these abnormalities, since obese subjects are at a greater risk of developing further respiratory impairment when exposed to other factors associated with increased respiratory morbidity, such as cigarette smoking, respiratory infections, thoracic or intra-abdominal operations and mechanical ventilation.

**RESPIRATORY FUNCTION DURING SPONTANEOUS BREATHING**

The earliest and most prominent pulmonary function test abnormalities associated with obesity consist of decreased expiratory reserve volume and functional residual capacity (FRC), while the vital capacity and total lung capacity are essentially normal or reduced to a lesser degree⁴. The proposed mechanism for these reductions is related to changes in the static mechanics of the chest wall, which are usually attributed to mass loading.

Total respiratory system compliance is decreased in severe obesity, the reduction being due mainly to reduced chest wall compliance⁴. The causes of the reduced chest wall compliance are thought to be structural changes in the chest wall and rib cage, decreased thoracic volume and increased abdominal mass, which in turn result in a reduction of the relaxation volume of the respiratory system⁴. Slight reduction in lung compliance has been reported by some investigators, and attributed to increased pulmonary blood volume and airway closure in the dependent lung zones⁶. Relative to normal values, the total respiratory system compliance decreases more in the supine than in the sitting position, because of the greater degree of chest wall compression and cranial displacement of the diaphragm when lying⁶.

Morbidly obese subjects exhibit increased airway and respiratory system resistance (Rrs)⁶⁻⁸. The primary mechanism for the increased lung resistance and total Rrs was thought to be the reduced lung volume, given that a close correlation was found between airway conductance and lung volume. However, measurements using the forced oscillation technique by Yap and al. have shown that Rrs is increased to a degree greater than that accounted for by the reduction in FRC¹⁰. Similarly, in a recent study by King et al. on young adults it was found that in obese subjects, mainly males, the increased Rrs was explained only in part by the reduced lung volume¹¹. These findings suggest that other pathophysiological mechanisms may also lead to increased Rrs. Total Rrs rises when obese subjects shift from the upright to the supine position, probably as a result of further reduction in FRC. Even when lung resistance is increased, the ratio of forced expiratory volume in one second to forced vital capacity has been found to be normal⁹⁻¹⁰, implying that the increased lung resistance is present in the lung tissue and small airways, rather than in the large airways.

Severely obese subjects are often hypoxaemic, with a widened alveolar-arterial oxygen tension gradient¹²⁻¹⁴. Ventilation (V) and perfusion (Q) are mismatched. Direct evidence of V/Q mismatch due to peripheral airway closure in sitting obese subjects was provided by Holley et al who, with the use of radioactive Xe, found that during quiet breathing, the distribution of inspired gas was preferential to upper lobes in subjects with expiratory reserve volume <0.3 L, but more uniform in those with expiratory reserve volume >0.4 L¹². The extent of airway closure within the range of tidal breathing has been related to a reduction in PaO₂, thus providing a causal relation in obese subjects¹⁵.

Most obese subjects are eucapnic. To meet the increased demands, they increase their minute ventilation mainly by increasing the respiratory rate, while the tidal volume is either normal or increased¹⁶.

**Peripheral airway closure**

In otherwise healthy obese subjects, the relaxation volume is reduced because the elastic equilibrium between the elastic forces of the lung and chest wall occurs at a lower thoracic gas volume due to alterations in the static volume-pressure curve of the chest wall¹⁶⁻¹⁷. Tidal breathing at low lung volume promotes peripheral airway closure¹⁸. While in young normal adults the FRC is considerably larger than the closing volume, in young obese subjects closing volume is substantially increased in both the sitting and supine positions¹⁵⁻¹⁶, so that peripheral airway closure can occur during tidal breathing¹⁹. Airway closure during tidal breathing may be the cause of impairment of gas exchange and risk of injury to peripheral airways due to mechanical stresses related to their cyclic opening and closing²⁰⁻²¹.
Expiratory flow limitation

The term expiratory flow limitation (EFL) should be used to describe a condition in which expiratory flow cannot be augmented at a given lung volume by further increasing pleural and, therefore, alveolar pressure. The presence of EFL depends on the balance between expiratory flow reserve and ventilatory requirements. The expiratory flow reserve in the tidal volume range is provided by the difference between the maximal flows available and the flows developed during tidal expiration. In obese subjects, the decreased flow reserve in the tidal volume range due to reduced FRC, and the higher ventilatory requirements due to increased metabolic demands would be expected to promote EFL during tidal breathing. A high prevalence of EFL was found by Pankow et al. and Ferretti et al. in spontaneously breathing morbidly obese subjects in the supine position (88% and 59%, respectively). In the presence of predisposing conditions, such as shortening of expiration time due to rapid breathing, augmented minute ventilation due to high ventilatory demand, and increased airway resistance caused by reduced lung volume, EFL can readily induce dynamic hyperinflation and intrinsic positive end-expiratory pressure (PEEPi) by preventing the respiratory system from reaching its relaxation volume during expiration. PEEPi represents a significant threshold load that must be overcome by the inspiratory muscles during tidal breathing. In addition, hyperinflation is accompanied by a concomitant decrease in the effectiveness of the respiratory muscles as pressure generators. All the above factors may represent important mechanisms leading to dyspnoea in morbidly obese subjects. Furthermore, the presence of EFL entails cyclic heterogeneous dynamic compression and re-expansion of peripheral airways during tidal breathing, which may result in an increased risk of peripheral airway injury.

RESPIRATORY FUNCTION DURING ANAESTHESIA AND MECHANICAL VENTILATION

It is well known that general anaesthesia impairs pulmonary function even in normal individuals with healthy lungs, resulting in decreased oxygenation. It also causes reduction in FRC of up to 50% of the pre-anaesthesia value. Computed tomography (CT) has shown that pulmonary atelectasis is common in anaesthetized subjects, occurring in 85-90% of healthy adults. Atelectasis develops within minutes of the induction of anaesthesia, and in normal subjects up to 15% of the entire lung may become atelectic, resulting in a 5-10% shunting of cardiac output. In morbidly obese subjects, general anaesthesia causes a much greater degree of atelectasis than in normal subjects.

As FRC becomes less than the closing volume, airway closure will be present during tidal breathing. Together with alveolar collapse, this leads to an abnormal V/Q distribution. Substantial impairment of oxygenation has been reported in anaesthetized, morbidly obese subjects, both pre- and postoperatively. Peripheral airway closure implies non-homogeneous lung emptying, since short-time constant units can start filling while the long-time constant units are still emptying, and hence expiration is not synchronous in all lung regions.

Increased abdominal mass and pressure in morbidly obese subjects cause a cephalad displacement of the diaphragm with reduction in the passive movements of its dependent part, resulting in a further decrease of end-expiratory volume and marked abnormalities in respiratory system mechanics. Respiratory system compliance has been shown to be reduced in postoperative morbidly obese subjects, mainly due to reduced lung compliance. In obese anaesthetized subjects, respiratory resistance values have been found to be significantly greater than in anaesthetized control subjects of normal weight.

Due to the fact that anaesthesia-paralysis causes a further decrease of FRC in obese subjects, a high prevalence of EFL and PEEPi should be predictable, and indeed, using the negative expiratory pressure technique, morbidly obese postoperative mechanically ventilated subjects have been demonstrated to exhibit expiratory flow limitation with concomitant PEEPi. Non-significant differences in respiratory mechanics and blood gases were found between EFL and non-EFL subjects.

Risk of low lung volume injury

Peripheral airway closure and EFL during tidal breathing commonly seen in morbidly obese subjects because of reduced FRC, together with alveolar collapse, constitute the basis for lung inhomogeneity during anaesthesia and mechanical ventilation. Peripheral airway closure implies cyclic opening and closing of peripheral airways, the presence of EFL implies dynamic compression and re-expansion of small airways and the presence of atelectasis implies regional increases in transmural pressures, leading to the development of shear stresses within the lung. All these factors may lead to mechanical injury of...
the peripheral lung units. It has been shown recently that in normal rabbits submitted to open-chest conditions low volume ventilation, which entails cyclic opening and closing of peripheral airways, leads to a persistent increase in airway resistance and damage of terminal and membranous bronchioles, characterized by denuding of the epithelium and sloughing. This may explain the observation that in supine spontaneously breathing morbidly obese subjects Rrs is abnormally high. In addition, a recent model analysis suggests that heterogeneous peripheral airways constriction, such as occurs during tidal EFL, amplifies airflow-related shear stresses within the peripheral airways, with risk of injury, even in the absence of airway closure. Although these effects may occur during long-standing spontaneous breathing at low lung volume, the risk of injury should, in this case, be lower than during mechanical ventilation. During mechanical ventilation with constant flow inflation, there is a rapid initial increase in airway pressure which, by snapping open the closed or compressed airways and generating high shear stresses, enhances peripheral airway injury. Although this has not been proved to occur in morbidly obese subjects during mechanical ventilation, it may be prudent to avoid the risk of low volume injury. Accordingly, positive end-expiratory pressure (PEEP) should be applied in order to increase the end-expiratory lung volume above the closing volume and the expiratory flow limitation volume. It has been shown that application of PEEP levels high enough to abolish EFL in mechanically ventilated morbidly obese subjects caused a significant reduction in PEEPi and in the elastance and resistance of the respiratory system, without however significantly improving gas exchange.

In order to improve oxygenation, researchers suggest the use of recruitment manoeuvres with end-inspiratory plateau pressure values higher than those used for normal weight subjects, with the aim of abolishing or markedly reducing atelectasis and decreasing intrapulmonary shunt.

REFERENCES