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Effects of anesthesia on the respiratory system

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Most anesthetics cause a loss of muscle tone that is accompanied by a fall in the resting lung volume. The lowered lung volume promotes cyclic (tidal) or continuous airway closure. High inspired oxygen fractions cause rapid absorption of gas behind closed airways, resulting in atelectasis. This chapter deals with these mechanisms in more detail, and it addresses possible measures to keep the lung open with the use of recruitment maneuvers, continuous and/or end-expiratory positive pressure, as well as the interaction with different oxygen concentrations. The effects on ventilation/perfusion matching and pulmonary gas exchange are also discussed.

Introduction

Anesthesia causes a respiratory impairment, whether the patient is breathing spontaneously or is ventilated mechanically. Thus, most anesthetics cause a fall in functional residual capacity (FRC) because of loss of muscle tone [1]. The fall in FRC promotes airway closure and gas resorption behind occluded airways, leading to atelectasis [2]. To prevent hypoxemia, it has become routine to add oxygen to the inspired gas so that the fraction of inspired O_2 (F_IO_2) is around 0.3–0.4. Even as high F_IO_2 as 0.8

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has been suggested with the assumption that it prevents wound infection [3], full agreement does not exist.

Airway and alveolar collapse may trigger inflammatory responses [4,5]. Mechanical ventilation per se may have adverse effects by increasing stress and strain of the lung although the duration and magnitude of exposure should play a role [6,7]. The effects of anesthesia and mechanical ventilation may remain up to the postoperative period, and pulmonary complications are frequent, ranging from a few percent to as much as 40% depending on patient, surgery, and definition of complications [8]. This will be discussed in more detail in another chapter. It should be emphasized that this chapter describes effects of anesthesia during mechanical ventilation to a major extent. The effect of each of them cannot always be separated. Thoracic anesthesia with one-lung ventilation will not be covered in this chapter, but the interested reader is referred to recent reviews [9].

Respiratory muscle tone and lung volume

In the adult human, the resting lung volume (FRC) is reduced by 0.8–1.0 l by changing body position from upright to supine, and there is a further decrease by 0.4–0.5 l with the induction of general anesthesia [1] (except with ketamine, see subsequent text [10]). As a result, the end-expiratory lung volume is reduced from approximately 3.5 upright, awake to 2 l supine, anesthetized, the latter being close or equal to residual volume. The anesthesia per se causes a fall in FRC despite the maintenance of spontaneous breathing [11], and the drop in FRC occurs whether the anesthetics are inhaled or given intravenously [12]. Muscle paralysis and mechanical ventilation cause no further decrease in FRC. The reduction in FRC may contribute to an altered distribution of ventilation and impaired oxygenation of blood, as will be discussed later.

The decrease in FRC seems to be related to the loss of respiratory muscle tone, shifting the balance between the elastic recoil force of the lung and the outward forces of the chest wall to a lower chest and lung volume. Maintenance of muscle tone, as during ketamine anesthesia, does not reduce FRC [10]. The influence of age and body position (upright vs. supine) and anesthesia on FRC is shown in Fig. 1. As can be seen in the figure, FRC increases with age if weight and height remain unaltered over the years, which might not always be the case. Any weight gain will lower the FRC and thus oppose the effect of losing elastic tissue in the lung.

The major cause of the fall in FRC during anesthesia is a cranial displacement of the diaphragm with only a minor contribution by a decreased transverse area of the thorax [13]. There seem to be different motions of the diaphragm whether the diaphragm is acting as a respiratory muscle or is a passive membrane separating the thoracic cavity from the abdomen. During an active inspiration, the dorsal

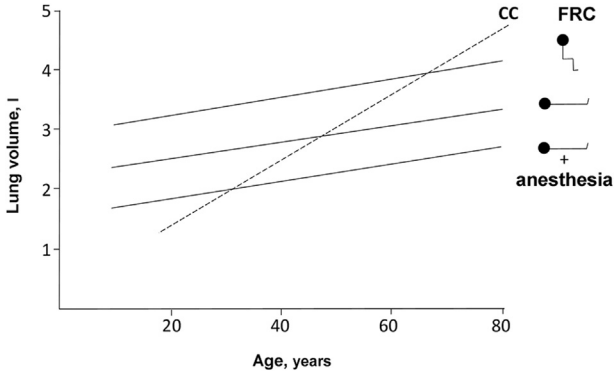

Fig. 1. Influence of age on FRC awake in different body positions (sitting and supine) and during anesthesia (supine). Closing capacity (CC), the lung volume at which airways begin to close during expiration, is also shown. Note the increase in FRC with increasing age, provided that body height and weight are constant. Note also the decrease in FRC by approximately 0.7–0.8 l when lying down from upright and the further decrease by another 0.4–0.5 l during anesthesia. CC increases faster with age so that a certain amount of airway closure occurs above FRC in an upright position at ages above 65 years and at around 50 years in the supine position. During anesthesia, most patients older than 30 years will suffer from airway closure.