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REVIEW

How to improve preoxygenation before intubation in patients at risk?

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SUMMARY

Intubation is the most common procedure performed in intensive care units and in operating rooms. Nevertheless, it can be associated with life-threatening complications when difficult airway access occurs or when performed in an emergency setting on critically ill patients. To improve its safety, several techniques have been developed, most of them combined in bundle. Among those techniques, preoxygenation is a major one. Preoxygenation consists in increasing the lung stores of oxygen, located in the functional residual capacity, and helps preventing hypoxia that may occur during intubation attempts. Although it has been incriminated to potentially cause atelectasis when high FiO_2 was delivered, its benefits outweigh this possible complication in patients at risk.

Recent studies in the field have indicated that in order to maximize the value of preoxygenation, patients at risk can benefit from the combination of breathing 100% oxygen and non-invasive positive pressure ventilation (NIV) with end expiratory positive pressure (PEEP) in the proclive position. Recruitment manoeuvres may be of interest immediately after intubation to limit the risk of lung derecruitment.

1. Introduction

Airway management is one of the most commonly performed procedures in operating rooms (OR), intensive care units (ICUs) and emergency departments (EDs). Hypoxaemia and cardiovascular collapse represent the initial and most serious life-threatening complications associated with difficult airway access, both in planned intubations (e.g. scheduled surgeries) and in emergency intubations of the critically ill patients.[1–3] To prevent and limit the incidence of hypoxaemia following intubation, several preoxygenation techniques have been entertained. Nonetheless, these techniques, which usually combine breathing manoeuvres and high inspired oxygen fraction (FiO_2), may be associated with some adverse effects and may be complicated by post-intubation atelectasis.[4] They may also provoke discomfort for patients and are time consuming on a daily practice. The objectives of the present review are to describe the rationale for optimizing preoxygenation and its existing manoeuvres, to discuss the evidence of performing these manoeuvres, and finally to propose an algorithm for secure airway management in patients considered “at risk” for life-threatening complications.

2. Preoxygenation: what is the rationale?

2.1. Cellular oxygenation

The aim of preoxygenation is ultimately to prevent and to limit cellular hypoxia during the intubation process, by increasing oxygen stores. Cellular oxygenation directly depends on the arterial delivery of oxygen (DO_2) to the cells and is calculated as follows (Eq. (1)):

$$DO_2 = 10 \times CO \times 1.33 \times [Hb] \times SaO_2 + 0.003{*}PaO_2 \quad (1)$$

(DO_2 is the oxygen delivery in mlO_2/min, CO the cardiac output in l/min, [Hb] the plasma haemoglobin concentration in g/l; SaO_2 the arterial oxygen saturation in percentage and PaO_2 the partial arterial pressure for O_2 in mmHg).

Even though blood product transfusions and drugs increasing cardiac output may promote oxygen delivery to cells, we do not consider these treatments “preoxygenation manoeuvres”.

Oxygen stores are limited to what is present in the blood and in the lungs. Since dissolved oxygen (non-linked to haemoglobin) represents a minor portion of the arterial oxygen content (Eq. (1)), the main blood oxygen store will vary based on the haemoglobin concentration and its affinity to oxygen (variables that cannot be simply modified). Even after oxygenation with 100% O_2, the blood stores of oxygen will only mildly increase, from about 850 ml to

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950 ml.[5] However, the lungs can store a much larger amount of oxygen. This amount depends on both the patient's functional residual capacity (FRC) and the maximal alveolar pressure of oxygen (PAO_2).

2.2. Functional residual capacity

The FRC is the volume of air (approximately 3–4 L in a healthy adult, smaller in women) that is present in the lungs after passive expiration, i.e. when the inward elastic recoil of the lungs is counterbalanced by the outward recoil of the chest wall and diaphragm. Indeed, if the lung elastic recoil decreases (age, chronic or acute obstructive lung disease), FRC will increase. If the lung elastic recoil increases (i.e. pulmonary fibrosis) FRC will decrease (Fig. 1).

Surgical procedures, anaesthesia, intubation and mechanical ventilation are associated with certain factors that may affect FRC, such as the supine position and anaesthetic drugs. Supine position decreases FRC (0.8–1.0 L on average) through multiple mechanisms, including the lungs' weights on a rigid chest wall, and the heart and chest wall weights decreasing the transverse thoracic diameter.[6] Abdominal contents are also moved upwards, causing a cranial shift of the diaphragm, thereby impairing lung volumes. Obesity is therefore also associated with a reduction in FRC.[7,8]

Anaesthetic drugs induce a decrease in muscular tone. This will result in a reduction in FRC (0.4–0.5 L on average) by shifting the balance between the inward elastic recoil force of the lung (unchanged) and the outward recoil of the chest wall (reduced)[6] towards smaller lung volumes.

2.3. Closing volume (CV)

Despite a maximum expiratory effort, the lungs will not completely empty. The remaining lung volume is called the residual volume (RV). There are two main mechanisms explaining why the lungs do not collapse at the end of expiration. First, the chest wall would need to be totally deformed, which is mechanically not possible. Second, in adults, at the end of a forceful expiration, the distal airways close before complete alveolar collapse.[9]

The volume at which airways begin to close during expiration is called the closing capacity (CC). The volume of air between the closing capacity and the residual volume is called the closing volume (CV) (Fig. 2).

It is important to note that the distal airways do not close at the same time throughout the lungs during a forceful expiration

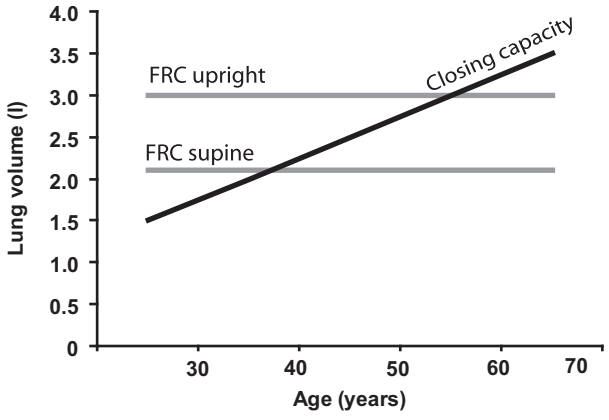

Fig. 1. Figure describing the expected changes with age, with regards to the closing capacity and Functional Residual Capacity (FRC). Note how the FRC is lower in the supine position. It is approached by the closing capacity around the age of 65 in an upright patient. Adapted from Ref. 9.

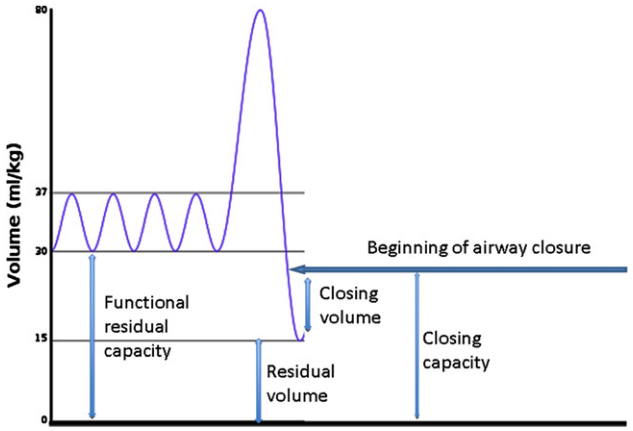

Fig. 2. Figure describing the spirometric nomenclature. Note that the closing capacity is the sum of the residual volume and the closing volume. By definition, at closing capacity, distal airways in the dependent areas start to close which impairs ventilation, possibly resulting in hypoxaemia. Adapted from Ref. 9.

manoeuvre. Airways in the dependent areas (located in the lower lobes when upright and in the lower/posterior lobes when supine) close first. This concept can be easily understood when the changes in pleural pressures within the chest are taken into account.[9]

In a normal subject at any lung volume, the pleural pressure is slightly more positive in the dependent areas of the thorax due to gravity. For instance, in a healthy standing adult at end-expiration (FRC), the pleural pressure is about -10 cmH_2O at the top of the thoracic cavity and about -2 cmH_2O at the bottom. During a forceful expiration, the pleural pressure is (voluntarily) increased and becomes positive (more so at the bottom of the chest). This pressure is transmitted to the alveoli and permits exhalation of air via a pressure drop over the course of the larger airways. At low lung volumes, the higher pleural pressure in the lower chest will preferentially compress the lower distal airways, causing some lower airway closure. Airways will be kept patent either due to the cartilage within their walls (larger airways) or due to the traction created by the adjacent parenchyma. Therefore, any process that would decrease lung recoil and parenchymal traction (emphysema, age, oedema), weaken the small airways (asthma, bronchitis) or exaggerate the pleural pressure gradient over the chest (obesity, pregnancy in the supine position) will promote earlier closure of the lower airways and thereby increase the closing capacity (Fig. 1).[9,10] In the above-mentioned cases, the work of breathing will necessarily include the work required to open the closed distal airways.

2.4. Alveolar pressure of oxygen (PAO_2)

PAO_2 depends on inspiratory pressure of oxygen, alveolar pressure of carbon dioxide and the respiratory quotient, which represents the ratio of carbon dioxide production per oxygen consumption (Eq. (2)):

$$PAO_2 = PIO_2 - PACO_2/R \quad (2)$$

(PAO_2 is the alveolar pressure of oxygen in mmHg, PIO_2 is the inspiratory pressure of oxygen in mmHg, $PACO_2$ is the alveolar pressure of carbon dioxide in mmHg and R the respiratory quotient which is usually 0.8 in most of the patients).

Equation(2) shows how hypoventilation, which may occur during anaesthesia due to difficult airway access and through the effects of anaesthetic agents, decreases PAO_2. One effective preoxygenation manoeuvre could include an increase in minute