Applied respiratory physiology

Derek Randles

Stuart Dabner

Abstract

Anaesthesia has many effects on respiratory physiology, the knowledge of which is relevant to clinical practice. Anaesthesia causes decreased muscle tone in the upper airway, which can lead to airway obstruction. Pulmonary hypoventilation occurs in the spontaneously breathing patient. There is a progressive decrease in the ventilatory response to CO_2 with increasing concentration of volatile agents, and even low doses of volatile have a profound effect on the ventilatory response to hypoxia. Functional residual capacity (FRC) is significantly reduced in the anaesthetized patient. Airway closure occurs when closing capacity exceeds FRC, with a reduced FRC this is more likely to happen especially in older patients or patients with coexisting lung pathology when closing capacity may be increased. The resulting atelectasis will affect oxygenation. Respiratory system compliance reduces very early during anaesthesia and there is little difference between the paralysed and spontaneously breathing patient. Alveolar dead space is decreased due to impairment of V/Q matching. During anaesthesia, venous admixture accounts for 10% of cardiac output due to increased shunt and changes in V/Q scatter. During anaesthesia and surgery patient position, type of surgery, smoking and obesity all have specific effects on respiratory physiology. Exercise physiology parameters such as anaerobic threshold have a role as a measure of cardiorespiratory fitness such as in cardiopulmonary exercise testing (CPX). CPX is increasingly used in risk stratification in patients undergoing major surgery. Anaerobic threshold is the point at which oxygen delivery mechanisms can no longer match the oxygen demand required in exercise.

Keywords Anaesthesia; applied respiratory physiology; exercise; obesity; smoking; surgery

Royal College of Anaesthetists CPD Matrix: 1A01

There are many areas of applied respiratory physiology which raise both fascinating and relevant issues. This article addresses the effects of anaesthesia, noting specific circumstances such as the smoker and types of surgery, and also explores the effects of exercise.

Derek Randles *MB BS FRCA is a Consultant in Anaesthesia and Intensive Care, University Hospital of North Durham, Durham, UK. Conflicts of interest: none declared.*

Stuart Dabner *MB BS FRCA is a Consultant Anaesthetist, University Hospital of North Durham, Durham, UK. Conflicts of interest: none declared.*

Learning objectives

After reading this article, you should be able to:

- describe how anaesthesia affects the mechanics of breathing and the implications on compliance, work of breathing and ventilation–perfusion matching
- recall the effects of positioning, obesity, abdominal surgery and smoking on such a patient's respiratory system
- describe the respiratory responses to exercise and applications in clinical practice

Anaesthesia

Anaesthesia has diverse effects on respiratory physiology. The knowledge of such effects is important in the safe and effective conduct of anaesthesia.

Effects on respiratory muscles

On induction of anaesthesia the nasopharynx is occluded by the soft palate falling against the posterior pharyngeal wall.[1] Pharyngeal dilator muscle tone is also reduced resulting in posterior movement of the tongue and epiglottis on inspiration. Together this leads to obstruction of the airway unless airway adjuncts or manoeuvres are used.

Although diaphragmatic muscular function is relatively unaffected during anaesthesia in a spontaneously breathing patient, both inspiratory intercostal and scalene muscle activity are often markedly reduced. This may result in a paradoxical pattern of ventilation with the drawing in of the ribcage and expansion of the abdomen on inspiration. This occurs especially in children, in whom chest wall compliance is increased, or in adults with respiratory pathology.

Control of breathing

General anaesthesia often results in pulmonary hypoventilation and hypercapnia in the spontaneously breathing patient owing to its effects on chemoreceptor reflexes. There is a progressive decrease in the ventilatory response to CO_2 with increasing end-expiratory volatile concentration of a number of agents (Figure 1).[2] However, not all agents are equal in this respect; for example, ether and ketamine have a much lesser respiratory depressant effect. This quality makes these agents particularly useful in field anaesthesia, or in the developing world, where effective monitoring is often unavailable.

The situation with low doses of anaesthetic agents is complex. Although there have been a number of conflicting reports in the literature, a recent systematic review[3] concluded that low doses of anaesthetic agents ($\leq$0.2 minimum alveolar concentration, MAC) had much more profound effects on the respiratory response to hypoxia than on that to hypercapnia. Furthermore, the depression of hypoxic ventilatory drive was dependent on the anaesthetic agent chosen so that, on average, halothane depressed acute hypoxic ventilatory response the most, followed by enflurane, then isoflurane, then sevoflurane.[4] Many patients will fail to mount an adequate response to hypoxia, especially in relative isolation from hypercapnia. The effect is compounded in the chronic respiratory patient with already impaired hypercapnic drive, and will persist postoperatively.[5]

Please cite this article in press as: Randles D, Dabner S, Applied respiratory physiology, Anaesthesia and intensive care medicine (2017), https://doi.org/10.1016/j.mpaic.2017.11.007

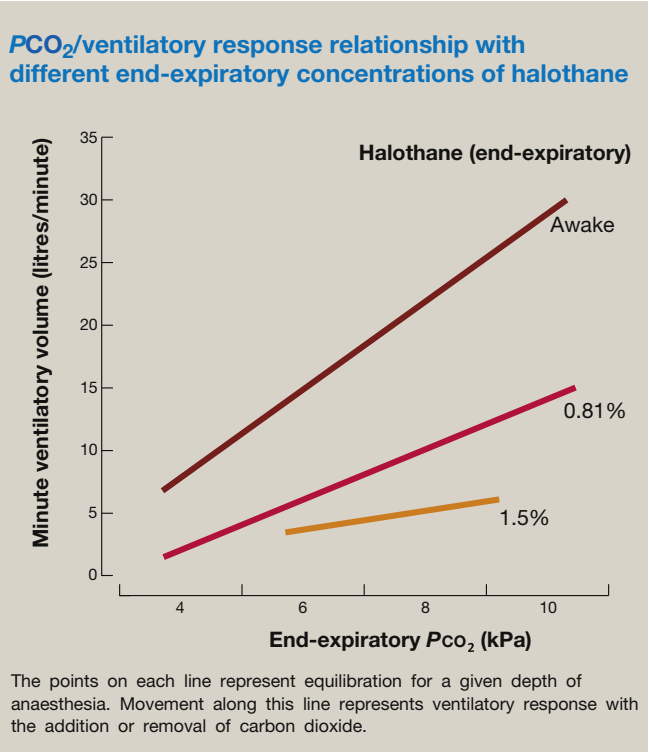

Figure 1

Functional residual capacity

Functional residual capacity (FRC) is the lung volume at the end of normal expiration. It is significantly reduced in the anaesthetized patient, and, depending on factors such as position (e.g. head down), obesity, late pregnancy, and restrictive lung pathology, can be reduced by up to 50%. This happens on induction and very early in anaesthesia,[6] and continues well into the postoperative period, whether or not the patient has been paralysed.

This reduction in FRC is due to both a change in chest wall shape secondary to impairment of respiratory muscle activity and reduction of normal end-expiratory diaphragmatic muscle tone with loss of forces which oppose the elastic recoil of lung tissue and cephalad movement of abdominal viscera into dependent regions when supine.[7] This results in a decreased alveolar volume, decreased ventilation–perfusion ratios and makes airway closure and alveolar collapse more likely.

Airway closure and atelectasis

Airway closure affects oxygenation when closing capacity exceeds FRC and small airways close during normal tidal ventilation, trapping gas in distal airways. This is particularly apparent in older patients or in those with diffuse lung pathology who have an increased closing capacity through loss of supporting elastic tissue. Airway closure occurs in these patients when they are awake and supine. It is exaggerated by general anaesthesia. Even without complete obstruction, airway narrowing creates areas with low ventilation–perfusion ratios and impaired gas exchange. The bronchodilating effects of anaesthesia attenuate this to some degree. Gas trapped distal to obstruction in an airway is rapidly absorbed. The resulting atelectasis in these dependent areas occurs in up to 90% of patients during general anaesthesia and may persist postoperatively. This is particularly the case in patients with poor lung function and sputum retention, and when chest movement is reduced, for example as a result of pain after upper abdominal surgery.[7]

Prevention of atelectasis[8]

High inspired oxygen concentrations promote atelectasis, most notably during induction and emergence when inhalation of 100% oxygen is routine. If 60% oxygen is used, collapse of alveoli is reduced significantly.[9] The absorption of oxygen is rapid owing to alveolar–arterial (A–a) O_2 pressure gradients of 80 kPa (600 mmHg) resulting in alveolar collapse in a matter of minutes. Nitrogen is less soluble, with a tension slightly below that of mixed venous blood, resulting in slow absorption and collapse over a course of hours. As such preoxygenation and emergence could in theory be considered with an F_iO_2 of 0.8 or even 0.6 rather than 1.0, to markedly reduce atelectasis, and not significantly increase the risk of hypoxia during periods of apnoea. However, it must be stressed that this is not currently a widely accepted practice, although it could potentially improve oxygenation well into the recovery period.

Positive airway pressures during induction and maintenance of anaesthesia reduce the occurrence of atelectasis and the rapidity of desaturation during apnoeic episodes.[10] High levels of positive end-expiratory pressure (PEEP) are required to expand pre-existing atelectasis, and may cause further ventilation/perfusion (V/Q) mismatch and cardiovascular disturbance at high inflation pressures. These manoeuvres are generally more effective in obese patients.

Respiratory mechanics

Airways resistance increases exponentially with a reduction in lung volume. Although there is an increase as the patient lies supine, much of the further increase expected with general anaesthesia is offset by bronchodilatational effects of inhalational anaesthetic agents. They cause suppression of airway vagal reflexes, directly relax airway smooth muscle and inhibit release of bronchoconstrictor mediators.

Total respiratory system **compliance** is reduced very early during anaesthesia[11] (Figure 2). This is illustrative of the change in position on the compliance curve reflecting a reduction in FRC. Smaller alveoli require greater forces of expansion despite a relatively thicker layer of surfactant. A major contribution is also due to the pulmonary atelectasis described above. Relative hypoventilation during anaesthesia without periodic large breaths corresponding to sighs may exacerbate poor compliance. Many patients take high-volume breaths on emergence, possibly reducing the insult in the recovery period. There is very little difference between anaesthesia with or without paralysis because the major changes are in lung rather than chest wall compliance.[11]

Gas exchange

Alveolar **dead space** is increased during anaesthesia owing to impairment of V/Q matching. Apparatus dead space is important. When including connections, dead space can be 50% of tidal volume – more in face mask anaesthesia, forcing hypercarbia or increased work of breathing. Some compensation occurs in reduced anatomical dead space at lower tidal volumes as

Please cite this article in press as: Randles D, Dabner S, Applied respiratory physiology, Anaesthesia and intensive care medicine (2017), https://doi.org/10.1016/j.mpaic.2017.11.007