Review article

Respiratory function under anaesthesia

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Abstract

General anaesthesia can affect every aspect of ventilation. It produces dose dependent depression of control of respiration. Carbon dioxide response curve is shifted to the right. Muscle tone is reduced and the airway can get obstructed. One of the most prominent changes is reduction in functional residual capacity (FRC) with the adoption of supine function and induction of anaesthesia. The closing capacity increases with age. The atelectasis that occurs consequent to the fall in FRC and increasing age is compounded by the use of high concentrations of oxygen, cephalad movement of the diaphragm, residual effect of muscle relaxants and inadequate analgesia after thoracic or abdominal surgery. One of the important causes of ventilation perfusion mismatch during anaesthesia is atelectasis. This has been shown to contribute to hypoxia as well as increased postoperative pulmonary complications. A lot of research is being conducted as to reduce the effects of atelectasis during anaesthesia, including the best ventilator strategy during anaesthesia. Most inhalation anaesthetics are good bronchodilators. Inhalation anaesthetics depress hypoxic pulmonary vasoconstriction (HPV) beyond 1 MAC. The movement of ciliary movements is reduced with the use of dry gases. All these can increase postoperative pulmonary complications which are important causes of postoperative morbidity. Appropriate preoperative preparation has to be combined with optimal management of ventilation intraoperatively to minimise postoperative pulmonary complications. The role of lower tidal volume, use of positive end-expiratory pressure, limiting oxygen concentrations and the utility of recruitment manoeuvres are increasingly being recognised and have been the topics of recent research.

Keywords: Anaesthesia, atelectasis, lung injury, respiratory function

Introduction

The primary function of respiration is ventilation and gas exchange, i.e., uptake of oxygen and removal of carbon dioxide. Adequacy of gas exchange depends on optimal function of every aspect of respiration, namely the respiratory centre, nerves supplying the respiratory muscles, the neuromuscular junction, muscle power, respiratory rate and tidal volume, respiratory compliance and resistance, bronchial muscle tone, ciliary movement, adequate reflexes and finally normal pulmonary vascular pressures.

Induction of anaesthesia can affect many or all of these mechanisms irrespective of whether the respiratory function was normal or abnormal prior to induction. Various factors can affect the extent of effect on the respiratory system such as the type of anaesthetic given, spontaneous or controlled ventilation, the airway (endotracheal tube or supraglottic airway) in use.

Effect of supine position on ventilation

The adoption of supine position itself reduces functional residual capacity (FRC) and basal atelectasis. In the adult, the functional residual capacity (end-expiratory volume) reduces by 0.8 to 1 L when a person adopts supine position as

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against the upright position. This is mainly due to a decrease in expiratory reserve volume rather than any change in residual volume. When a person lies down supine, the abdominal contents push the diaphragm upwards, more so in the posterior aspect and produces atelectasis of the basal alveoli. The oxygen remaining in the FRC represents the oxygen reserve for the patient, and when it reduces, the risk of hypoxaemia during apnoea increases.

The relationship of closing volume to FRC is also important. Closing volume (CV) is the lung volume at which some alveoli collapse during expiration. This volume is less than FRC in the supine and upright position in young adults. By the age 44 years, the closing volume is higher than FRC in supine position. By age 60 years, the CV is higher than FRC in both supine and upright positions. Thus, some alveoli are always in a collapsed state in these patients, presdisposing them to hypoxia.

Effect of anaesthesia on ventilation

Respiratory centre: General anaesthesia can affect every aspect of ventilation, beginning with generation of respiratory impulse by the respiratory centres. General anaesthesia produces dose dependent depression of control of respiration by the cerebral cortex as well as depresses the respiratory centres situated in the medulla. The main stimulus to breathe is carbon dioxide in normal individuals. The carbon dioxide response curve is shifted to the right which means that the increase in ventilation in response to an increase in blood carbon dioxide levels is blunted, and the rate (with narcotics) and/or tidal volume (with inhalation anaesthetics) reduces. When the minute volume reduces, the alveolar ventilation also reduces and thus patients breathing spontaneously under general anaesthesia tend to be hypercarbic. The apnoeic threshold (the partial pressure of arterial carbon dioxide - $PaCO_2$ above which carbon dioxide stimulates to breathe) is also shifted to the right. Thus, patients emerging from anaesthesia tend to be hypercarbic.

Neuromuscular component: General anaesthesia reduces muscle tone of all voluntary muscles including the accessory muscles of respiration. If muscle relaxants have been used, the neuromuscular blockade thus induced paralyses the diaphragm and all spontaneous respiration is lost. The induction of anaesthesia produces a reduction in functional residual capacity by 0.5 L and causes ventilation-perfusion mismatch.

Ventilation perfusion (V/Q) relationship: The effect of anaesthesia is greatest on ventilation perfusion relationships. Normally, in an unanaesthetised upright person, ventilation and perfusion are greatest in the bases. In the supine position, the diaphragm moves cranially but the posterior diaphragm assumes a more convex position making it contract better. This is because the fibres of the diaphragm are longer and more convex posteriorly. They contract better in a way similar to that of myocardial muscle fibre (Starling's law) and better air movement is seen in the dependent regions where perfusion is higher as well. Once anaesthesia is induced, the tone of the diaphragm reduces, the convexity of the posterior diaphragm is lost and the dependent parts of the lungs tend to collapse. Thus, there will be unventilated alveoli in the bases, contributing to shunt. This effect is further exaggerated with muscle relaxation.[1]

One of the important causes of ventilation perfusion mismatch during anaesthesia is atelectasis. This has been shown to contribute to hypoxia as well as increased postoperative pulmonary complications.[2] A lot of research is being conducted as to reduce the effects of atelectasis during anaesthesia, including the best ventilator strategy during anaesthesia.

Traditionally, patients under general anaesthesia with muscle relaxation were being ventilated with a tidal volume of 10-12 ml/kg, believing that such tidal volumes would prevent any atelectasis that might occur. However, it has been shown that ventilating the lungs with such high tidal volumes even in patients with normal lungs is harmful. Lessons learnt from ARDSnet study[3] are applicable to patients with normal lungs and it is now increasingly recognized that ventilation with a tidal volume of 7-8 ml/kg predicted body weight is beneficial.[4-6]

Low tidal volume ventilation reduces the effect of overdistension of lungs but is associated with the risk of development of atelectasis. The use of high

concentrations of oxygen during preoxygenation, apnoea and during the surgery is another important factor promoting atelectasis. Preoxygenation with 100% oxygen is done with the intention of filling up the functional residual capacity with oxygen to act as reserve in the face of difficulty in securing the airway but this leads to the commencement of absorption atelectasis. The use of 45 degree head up position during induction might reduce this problem. One way of reversing this atelectasis would be to use a recruitment manoeuvre after intubation. Although this may help to some extent, it has been shown that it is necessary to use positive end-expiratory pressure (PEEP) to keep the alveoli open after the recruitment manoeuvre.[7] Recruitment can be achieved in many ways but sustained inflation at 30 cm H_2O for 30 s is one of them. No standard procedure has been described.

The exact amount of PEEP to be applied is still being studied. It is proposed that 5-6 cm H_2O is required in patients with normal lungs and body habitus. Obese patients are likely to require much higher, may be up to 10 cm H_2O to avoid atelectasis. The use of at least 5 cm H_2O PEEP has also become a standard practice perioperatively. There is some evidence that 5 cm H_2O PEEP may be insufficient to prevent atelectasis and recruitment-derecruitment of the alveoli which can contribute to shear injury. However, high PEEP can cause haemodynamic disturbances and possibly more harm. It is suggested that one must individualise the PEEP based on the intratidal lung compliance or the driving pressure.[8,9]

Recent literature suggests that prophylactic lung-protective mechanical ventilation using lower tidal volume (6–8 ml/kg of predicted body weight), moderate PEEP (6–8 cm H_2O), and recruitment maneuvers is associated with improved functional or physiological and clinical postoperative outcome in patients undergoing abdominal surgery.[10] Further studies will be required to determine whether a strategy consisting of low V_T combined with PEEP and recruitment maneuvers reduces PPCs in obese patients and other types of surgery (*e.g.*, laparoscopic and thoracic), compared to low V_T with low PEEP.[11]

Similarly, the use of high concentrations of oxygen before and just after extubation can also lead to atelectasis. It is now advocated that the use of less than 50% oxygen by using a mixture of air and oxygen is desirable if no airway problems are anticipated at extubation. One must be wary of the possibility of diffusion hypoxia when nitrous oxide is used concurrently with oxygen. It may also be appropriate to use continuous positive airway pressure or even a brief period of noninvasive ventilation after extubation in patients who are prone to large amounts of atelectasis, for e.g., morbidly obese patients after bariatric surgery.[2]

Laparoscopy and respiratory function: Induction of capnoperitoneum increases the intra-abdominal pressure, reducing chest wall and lung compliance further increasing basal atelectasis.[12] This is reflected in the ventilator parameters such as an increase in peak and plateau airway pressures if the patient is on volume controlled ventilation or reduction in the delivered tidal volume if on pressure controlled ventilation. Oxygenation may deteriorate and carbon dioxide absorption will need an increase in minute ventilation to maintain normocapnia. Investigations have shown that lower abdominal pressures (8 mmHg as opposed to 12 mm Hg) are associated with less compromise in respiratory function and less inflammatory mediators are released into the circulation. However, when CO_2 is insufflated into the abdomen, peritoneal acidosis occurs with hypercarbia. The increase may be managed by an increase in tidal volume but it should be limited to 10 ml/kg. A combination of increase in rate along with tidal volume is recommended. These effects are rapidly reversible once the capnoperitoneum is released. One or two recruitment manoeuvres may be required in some patients. The use of PEEP is a standard technique in these patients.

The strain on the lung can be calculated as V_T/EELV, where V_T is the tidal volume and EELV is the end-expiratory lung volume. This should be less than 2, *i.e.*, the tidal volume should be less than twice the end-expiratory lung volume.[13] While in most normal patients, this is not a problem, in patients (for e.g., obese) where the end-expiratory lung volume is grossly reduced, the strain on the lungs may be quite high. Similar problem may be seen in patients

with pre-existing lung disease.

Effect of anaesthetics on respiratory function

Inhalational anaesthesia with isoflurane has been shown to be associated with less PaO_2 and higher $PaCO_2$ as compared to propofol.[14] Sevoflurane has been shown to protect against lung injury by ischaemic preconditioning mechanism similar to myocardial protection. The combination of low tidal volume and PEEP has been proven to decrease the blood or lung levels of inflammatory mediators, incidence of pulmonary infections and to prevent pulmonary coagulopathy in patients undergoing surgery.[15-17]

Respiratory function is also dependent on the patient position. A head-up position favours better gas exchange. Occasionally it may be necessary to adopt the concept of permissive hypercapnia.

Bronchial muscle tone: Most inhalation anaesthetics are good bronchodilators. Ketamine and halothane are actually used in the treatment of status asthmaticus. However, if there is any airway obstruction due to induction of general anaesthesia, it can precipitate bronchospasm in susceptible individuals.

Airway obstruction: The induction of general anaesthesia produces a loss of respiratory muscle tone and can predispose to airway obstruction. Occasionally, this may be severe enough to produce laryngospasm. Such an obstruction can also occur at extubation when the patient may still be under the influence of low levels of inhalation anaesthetics. This can precipitate laryngospasm and if severe and not dealt with immediately, can lead to negative pressure pulmonary oedema because of the wide swings in airway pressure with no gas movement. While blunting of respiratory reflexes can produce airway obstruction and aspiration, increased respiratory reflexes in a patient with irritable airways can lead to laryngospasm and bronchospasm and possible hypoxia.

Other respiratory injury: Inhalation anaesthetics depress hypoxic pulmonary vasoconstriction (HPV) beyond 1 MAC. The movement of ciliary movements is reduced with the use of dry gases. Inappropriate ventilation or other iatrogenic causes such as central venous cannulation or dissection at the root of the neck can produce a pneumothorax, which can have disastrous consequences on the respiration.

Mechanical ventilator strategies in patients with comorbidities

The effects of ventilator strategies are not uniform among all population, even if the lungs are normal because of various comorbidities. A variety of patient characteristics and comorbidities have been identified, which increase the risk of postoperative pulmonary complications (PPCs), including smoking, age, chronic obstructive pulmonary disease, pulmonary hypertension, obstructive sleep apnea, cardiac and neurologic diseases as well as critical illness. In contrast to the variety of conditions, evidence for specific intraoperative ventilation strategies to reduce PPC is very limited for most comorbidities.[18]

Chronic obstructive pulmonary disease: Patients with known COPD must have their disease controlled before being scheduled for elective surgery. Generally, patients with FEV1 > 60% will tolerate surgery well. Those having acute exacerbations must not undergo surgery until their disease is controlled. Regional anaesthesia is preferred and if general anaesthesia is required, it is best combined with a regional technique such as an epidural analgesia, which reduces postoperative pneumonia by four fold and halves 30-day mortality.

The ideal ventilator strategy in these patients is not very clear. The use of low tidal volumes will necessitate the use of higher respiratory rates. This in turn will promote development of more air-trapping and ventilation – perfusion disturbances. It is quite likely that this will also produce more haemodynamic disturbances. The effect of extrinsic PEEP is also unpredictable in these patients.

Asthma: A well-controlled asthma is not a predictor of increased postoperative pulmonary complications.

Pulmonary hypertension: The risk of PPCs is increased 15 fold in patients with known pulmonary hypertension. They are at such a high risk because any hypoxia, hypercarbia or acidosis increases

pulmonary vascular resistance exaggerating pulmonary hypertension. Thus, hypercapnia should be avoided as also increased intrathoracic pressures. Lower levels of PEEP reduces venous return and right ventricular preload but higher PEEP can increase right ventricular afterload also. Low levels of PEEP may be used considering the risk benefit ratio. The use of recruitment manoeuvres, however, may be associated with severe increases in pulmonary vascular pressures and are probably better avoided. There are no prospective studies that can be referred to recommend appropriate ventilator strategy in these patients.

Obstructive sleep apnoea: Avoid general anaesthesia, if possible. No specific ventilator strategies are recommended for intraoperative management but they are likely to benefit from the use of continuous positive airway pressure or even brief periods of noninvasive ventilation.

Patients with cardiac disease undergoing noncardiac surgery: Ventilation with low tidal volumes can cause hypercapnia which in turn can increase propensity for cardiac arrhythmias. Patients with intracardiac shunts also will tolerate hypercarbia poorly. Recruitment manoeuvres can cause hypotension and should be used with caution in these patients.

To conclude, postoperative pulmonary complications are important causes of postoperative morbidity. Respiratory system is one of the primarily affected systems with anaesthesia. Appropriate preoperative preparation has to be combined with optimal management of ventilation intraoperatively to minimize postoperative pulmonary complications. The role of lower tidal volume, use of positive end-expiratory pressure, limiting oxygen concentrations and the utility of recruitment manoeuvres are increasingly being recognised and have been the topics of recent research.

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