Ventilatory management during routine general anaesthesia

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Intraoperative hypoxaemia and postoperative respiratory complications remain the challenges of modern anaesthetic practice. Anaesthesia causes both depression of respiratory centres and profound changes of respiratory mechanics. Most anaesthetized patients consequently require mechanical ventilation and supplemental oxygen. Recent data suggest that intraoperative respiratory management of a patient can affect postoperative outcome. In this review, we briefly describe the mechanisms responsible for the impairment of intraoperative gas exchange and provide guidelines to prevent or manage hypoxaemia. Moreover, we discuss several aspects of mechanical ventilation that can be employed to improve patients' outcome. ***Eur J Anaesthesiol*** **26:1–8 © 2009 European Society of Anaesthesiology.**

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Introduction

Commonly used anaesthetic agents cause marked depression of both brainstem respiratory centres and respiratory muscles. Most patients under general anaesthesia, therefore, require ventilatory support to preserve arterial oxygenation and eliminate carbon dioxide. In addition to its depressing action on respiratory drive and mechanics, general anaesthesia also alters gas exchange [1], as seen, for example, in the increase in the alveolar–arterial oxygen tension gradient ($Aa-p_{O_2}$). Optimizing intraoperative ventilation requires appropriate understanding of the basic mechanisms responsible for gas-exchange impairment induced by anaesthesia.

Altered gas exchange can contribute to postoperative respiratory complications, which are among the most frequently occurring adverse events after surgery. Not surprisingly, it is currently considered that perioperative optimization of mechanical ventilation can improve outcome in patients [2–4].

In this review, the mechanisms responsible for hypoxaemia during general anaesthesia are described and recommendations for preventing or treating hypoxaemia are then provided in the light of these mechanisms. Finally, several features of mechanical ventilation that can help improve patient outcome are discussed (Table 1).

Pulmonary consequences of general anaesthesia and mechanical ventilation

Closing volume and functional residual capacity

The small airways lack cartilage and their patency relies on both the radial elastic traction exerted by the surrounding lung parenchyma and the negative pleural pressure [5]. Reducing the lung volume below a critical threshold, called the 'closing capacity or closing volume', results in reduced elastic recoil and less negative pleural pressure, thereby allowing closure of the small airway.

The gas volume remaining in the lung after a normal passive exhalation is called the resting lung volume or the functional residual capacity (FRC). It results from the balance between the inward elastic recoil of the lung and the outward force of the chest wall. Normally, FRC is larger than the closing volume, and airway closure does not occur.

Anaesthesia reduces functional residual capacity

Most patients undergo general anaesthesia while lying in the supine position, which itself causes an initial reduction in FRC [6]. Moreover, except for ketamine, all commonly used general anaesthetics produce some degree of diaphragmatic relaxation that further aggravates this reduction of FRC [1,7]. Therefore, under general anaesthesia, end-expiratory lung volume often approaches or even falls below the closing volume, leading to airway closure.

Intrapleural pressure is less negative in the basal regions than in the apex regions because the lung weight partly counterbalances the inward elastic recoil in these lower areas. Consequently, small airways and alveoli of dependent lung areas are narrower and more prone to collapse (Fig. 1).

Anaesthesia increases venous admixture

Airway closure reduces the regional alveolar ventilation. Because airway closure preferentially occurs in dependent

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Table 1 Summary and grading of recommendation

Preoxygenation		
Use of high F_IO_2 (0.8)	Reduces atelectasis	Grade B
CPAP 6 cmH_2O	Reduces atelectasis	Grade B
	Improves arterial oxygenation	Grade B
	Prolongs nonhypoxic apnoea time	Grade B
25° Head-up tilt	Reduces atelectasis	Grade B
	Improves arterial oxygenation	Grade B
	Prolongs nonhypoxic apnoea time	Grade B
Intraoperative management		
Use of PCV	Does not improve gas exchanges	Grade B
	Reduces peak airway pressure	Grade A
Reduce V_T to 5–8 $ml\,kg^{-1}$	Reduces alveolar inflammation	Grade B
	Reduces postoperative pulmonary dysfunction	Grade C
Use 5–10 cmH_2O PEEP	Reduces alveolar inflammation in association with low tidal volume ventilation	Grade B
	Improves arterial oxygenation in morbidly obese	Grade B
	Improves arterial oxygenation during one-lung ventilation	Grade B
	Prevents atelectasis relapse after a vital capacity manoeuvre	Grade B
Set F_IO_2 to 0.8	Reduces wound infection in major abdominal surgery	Grade A
	Does not reduce PONV	Grade B
	Protects cardiovascular system	ND

Effects of several ventilatory settings with their associated grades of recommendation are given. Grade A recommendation is given in the presence of level 1 evidence, that is, data from large and powerful randomized controlled trials, meta-analyses or randomized controlled trials. Grade B is provided for level 2 evidence, meaning that data have come from low-powered randomized controlled trials or properly conducted nonrandomized and uncontrolled trials. Grade C is for levels 3 and 4 evidence, meaning that the data are from case–control studies, retrospective studies and observational epidemiologic studies. Grade D is formulated in the absence of scientific evidence but agreement among professionals. CPAP, continuous positive airway pressure; ND, no data; PCV, pressure-controlled ventilation; PEEP, positive-end expiratory pressure; PONV, postoperative nausea and vomiting.

lung areas–the ones that are best perfused for gravitational reasons–it reduces the ventilation-to-perfusion ratio (V_A/Q) in these lung regions.

An extreme example is seen in an intrapulmonary shunt in which V_A/Q is equal to zero. Such an intrapulmonary shunt frequently occurs shortly after the induction of general anaesthesia due to the development of atelectases. Indeed, anaesthesia-associated relaxation allows the diaphragm to move cephalad and compress dependent parts of the lung, leading to the so-called 'gravity dependent or compression atelectasis' [8,9]. Moreover, in low V_A/Q areas, gas uptake by pulmonary blood flow can exceed alveolar fresh gas inflow when high-inspired oxygen fractions (F_IO_2) are used, resulting in absorption atelectasis. High F_IO_2 indeed increases the alveolo-arterial oxygen gradient and subsequently the rate of oxygen absorption by pulmonary capillaries [8]. Eventually, both anaesthesia and positive pressure mechanical ventilation are thought to alter the alveolar-stabilizing function of the surfactant [10].

By increasing low V_A/Q lung areas and intrapulmonary shunt secondary to atelectasis, general anaesthesia raises

Fig. 1

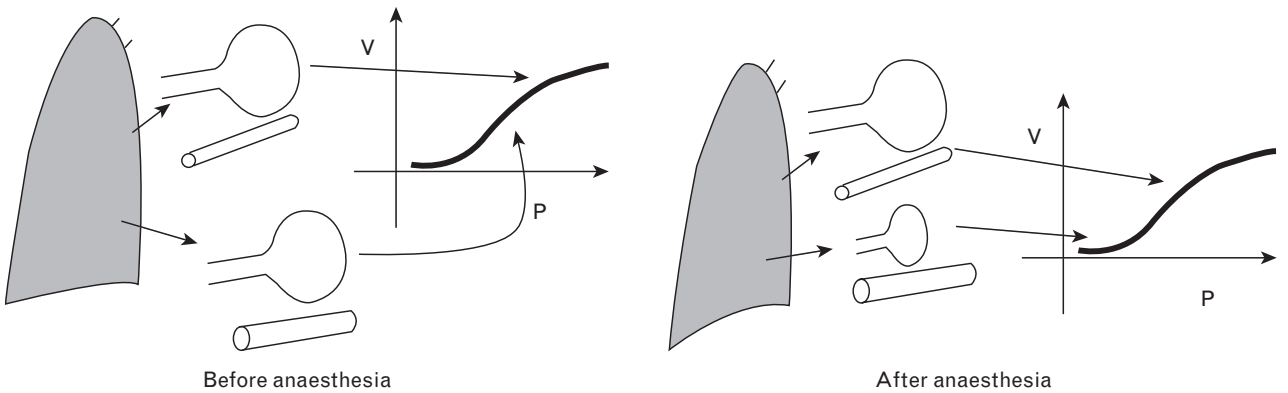


Illustration of regional alveolar sizes and ventilation/perfusion ratio (V_A/Q) changes associated with anaesthesia. Before anaesthesia apical alveoli are relatively greater than dependent alveoli and are located on the upper part of the pressure–volume curve. They are poorly ventilated owing to their low compliance and are poorly perfused for gravitational reasons. Alveoli from the base are more compliant, better ventilated and better perfused. After anaesthesia induction all alveoli move down the pressure–volume curve of the lung. Apical alveoli become less distended, more compliant and better ventilated. Dependent alveoli shrink, become less compliant, more prone to collapse and less ventilated while remaining well perfused. Consequently, anaesthesia is associated with V_A/Q mismatch and reduced lung volume mainly in basal areas, which become prone to airway closure and collapse. $P <$ pressure; V, volume.

venous admixture, that is, the amount of blood passing from the right to the left heart without complete equilibration with alveolar gas. Such an increase in venous admixture augments the PA–aO_2 gradient and favours hypoxaemia.

Anaesthesia increases dead space

General anaesthesia is also associated with an increased alveolar dead space. First, FRC reduction moves all lung areas down the pressure–volume curve of the lung. Consequently, under general anaesthesia, apex alveoli located on the linear part of this curve are best ventilated but less perfused for gravitational reasons. They, therefore, constitute areas of high V_A/Q ratio (Fig. 1). Moreover, increased alveolar pressure, and sometimes the reduced pulmonary artery pressure, further decreases the apical perfusion. These changes explain the enlargement of the alveolar dead space and the resulting increase of the gradient existing between arterial and end-tidal CO_2 (Pa-ETCO_2). Such an increase in alveolar dead space reduces the efficacy of alveolar ventilation in eliminating CO_2.

Positive pressure ventilation has haemodynamic consequences

Positive pressure ventilation profoundly alters heart filling and ejection regimen. Positive inspiratory pressure transitorily increases venous return to the left ventricle by driving blood out of the pulmonary capillaries, whereas the left ventricular afterload decreases due to increased systolic extracardiac pressure. At the same time, the inspiratory rise in intrathoracic pressure reduces venous return to the right ventricle by directly compressing the thoracic part of the vena cava and by increasing the right atrial pressure. Moreover, during inspiration, right ventricular afterload can increase because of the compression of pulmonary capillaries by the alveolar pressure. Together, these changes result in a brief increased left ventricular ejection and a reduced right ventricular output during inspiration [11]. The global effect of positive pressure ventilation is a reduction in the cardiac output proportional to the rise in the mean intrathoracic pressure. This reduction is further aggravated by hypovolaemia and by positive end-expiratory pressure (PEEP) [12].

Susceptibility factors to perioperative hypoxaemia

Although a small increase in F_IO_2 usually compensates for anaesthesia-induced gas-exchange impairment, some patients or surgical procedures are at increased risk of intraoperative hypoxaemia.

Patient-related factors

Because intraoperative hypoxaemia mainly results from a rupture of the safety margin existing between FRC and closing volume, infants [13], morbidly obese patients [14] and pregnant women [15] who have reduced FRC are particularly vulnerable. On the other hand, patients with chronic obstructive pulmonary disease, smokers [16] and the elderly [17] are characterized by an increased closing volume and are also more prone to alteration in intraoperative gas exchange.

Procedure-related factors

Intraoperatively, patient positioning associated with FRC reduction such as reverse Trendelenburg or forced lithotomy position can adversely affect gas exchanges [18]. The use of high-inspired oxygen without PEEP is also associated with progressive hypoxaemia by transforming low V_A/Q units into absorption atelectasis [19,20]. Eventually, anaesthesia duration is a risk factor for hypoxaemia probably because it is associated with progressive lung derecruitment [13].

Management of mechanical ventilation during anaesthesia

The preoxygenation

Unexpected difficult airway is one of the major fears of the anaesthetist during induction of general anaesthesia. Complete denitrogenation by preoxygenation with pure oxygen for 5 min before anaesthesia induction significantly prolongs the duration of nonhypoxic apnoea by providing an oxygen reserve equivalent to the FRC [21]. For this reason, pure oxygen was used during the induction phase as a safety measure in many institutions; however, in the context of anaesthesia-associated lowering in V_A/Q, high-inspired oxygen becomes a major determinant of absorption atelectasis [22].

A small reduction in the inspired oxygen concentration from 100 to 80% during the induction of anaesthesia only modestly reduces the nonhypoxic apnoea time, whereas it efficiently prevents atelectasis [23].

Moreover, applying a 6 cmH_2O continuous positive airway pressure (CPAP) using the facemask during this preoxygenation phase allows compensation for anaesthesia-induced FRC reduction. It, therefore, increases the duration of nonhypoxic apnoea [24], prevents atelectasis formation and subsequently improves intraoperative oxygenation [25]. Use of CPAP was also shown to be effective in morbidly obese patients, who are especially prone to rapid arterial desaturation [26]. In these patients, preoxygenation in a 25° head-up tilt position improves the FRC and prolongs the duration of nonhypoxic apnoea [27].

To summarize, using 80% oxygen during preoxygenation allows a reduction in atelectasis. Compared with preoxygenation with 100% oxygen, the nonhypoxic apnoea time can probably be restored or even increased by adding CPAP or using the head-up tilt position during induction.

Ventilatory mode

Volume-controlled ventilation

Volume-controlled ventilation (VCV) is the most widely used ventilatory mode during anaesthesia. In VCV, the tidal volume (V_T) is usually delivered by constant flow insufflation. The parameters to be set are V_T, respiratory rate, inspiratory/expiratory (I/E) ratio and the extent of an optional end-inspiratory pause.

In addition to its effect on mean airway pressure, use of an inspiratory pause allows the two levels of inspiratory pressure to be measured. The peak airway pressure is measured at the end of the effective insufflation time, whereas the plateau pressure is measured at the end of the end-inspiratory pause. The peak airway pressure magnitude is a function of the inspiratory flow, the respiratory system compliance and the resistance. Inspiratory flow, in turn, depends on the tidal volume and the inspiratory time, the latter being a function of respiratory rate and the I/E ratio. The plateau pressure depends only on the tidal volume and the whole respiratory system compliance. With constant tidal volume and inspiratory time, peak and plateau airway pressures provide information on the resistance and compliance of the thoracopulmonary system. Indeed, any modification in the peak or the plateau airway pressure or both can be related to changes in total respiratory system resistance or compliance or both. An increased resistance of either the respiratory circuit or the patient's airway will result in an increase in the peak airway pressure without any change in the plateau pressure. On the contrary, a reduction in thoracopulmonary compliance increases the peak and plateau pressures to the same extent (Fig. 2). Pressure curve monitoring is, therefore, helpful for diagnosing tracheal tube obstruction or kinking, bronchospasm, reduced respiratory system compliance caused by retractors, peritoneal CO_2 insufflation, respiratory muscles activation or patient position.

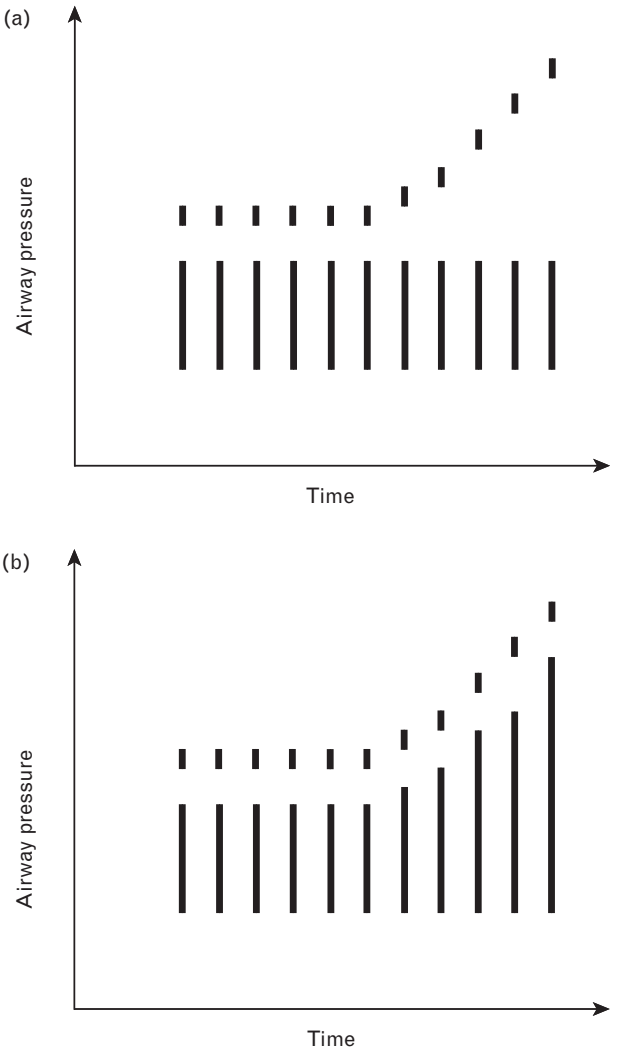
In VCV, any change in respiratory system compliance or resistance will result in modifications of airway pressures without affecting the delivery of the preset tidal volume, except when the 'high-pressure limit' is reached. VCV is, therefore, a safe ventilatory mode because minute ventilation is guaranteed independent of changes in airway compliance and resistance. This is particularly useful during anaesthesia in surgical procedures that affect respiratory system compliance, for example change in patient's position, peritoneal CO_2 insufflation or abdominal retractors.

Pressure-controlled ventilation

In PCV, the ventilator produces an inspiratory flow aimed at achieving and maintaining the preset pressure in the proximal airway [28]. This pressure progressively equilibrates with alveolar pressure, resulting in an exponentially decelerating inspiratory flow. The tidal volume becomes dependent on the preset pressure, the inspiratory time (T_I) and the respiratory system compliance and resistance.

Although more complicated than VCV, PCV provides some theoretical advantages. With the decelerating inspiratory flow, the bulk of the V_T is delivered early during inspiration and its residency time in the lung is longer. Moreover, for the same V_T and T_I, PCV results in a higher mean airway pressure [29] but a reduced peak airway pressure and should theoretically provide better arterial oxygenation [30]; however, despite these potential benefits, clinical studies failed to demonstrate gas exchange improvement during one-lung ventilation [31] in obese patients [32] and during laparoscopy [33]. PCV,

Fig. 2



Monitoring of peak (small bars) and plateau (large bars) pressure over time. (a) Isolated increase in peak airway pressure suggests an increase in airway resistance. (b) Both peak and plateau pressures increase to the same extent during reduced respiratory system compliance.

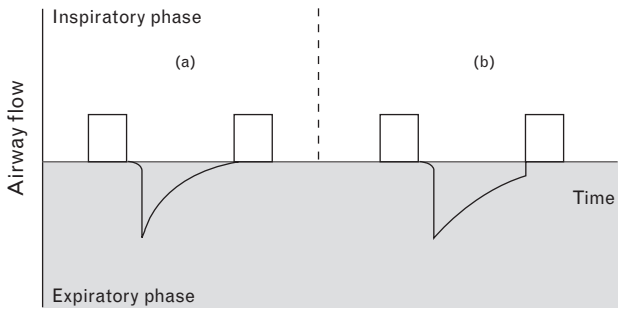
however, seems useful and well tolerated for ventilating children using supraglottic devices such as a laryngeal mask airway. Because of its association with reduced peak airway pressures, PCV helps to reduce air leak and gastric insufflation [34].

Unlike VCV, PCV does not guarantee minute ventilation because any change in respiratory system compliance or resistance will affect the tidal volume delivery. Moreover, PCV does not allow the determination of respiratory system resistance and compliance. Clinicians are, therefore, deprived of a helpful diagnostic tool in cases of sudden change in the thoracopulmonary system. Finally, when using PCV, the flow–time curve should be continuously monitored to adjust optimal inspiratory and expiratory times. The minimum T_I is the time required for inspiratory flow to reach zero, which ensures that inspiratory pressure reaches the alveolar level.

In PCV, further lengthening of T_I increases the mean airway pressure and can improve the arterial oxygenation [30]. In VCV, prolonged T_I does not necessarily increase the mean airway pressure, which can be achieved by increasing the inspiratory pause time; however, the usefulness of such a manoeuvre has not been studied. Whatever the *I/E* ratio and the duration of the end-inspiratory pause, attention should be paid to keeping a sufficient expiratory time to allow end-expiratory flow to reach zero and avoid the development of intrinsic PEEP (Fig. 3) [35].

We consider that VCV remains, for safety reasons, the preferred ventilatory mode during anaesthesia; however, the clinician who is familiar with the setting of PCV can take advantage of this ventilatory mode in some particular situations. Development of new ventilators guaranteeing a preset tidal volume delivered by a decelerating inspiratory flow deserves further attention.

Fig. 3



Examples of flow–time curve monitoring. (a) The end-expiratory flow reaching the zero-flow line indicates the completion of expiration. (b) The expiratory flow is interrupted by the subsequent insufflation, suggesting the presence of an intrinsic positive end-expiratory pressure.

The tidal volume (V_T)

As discussed in the introduction, anaesthesia and muscle paralysis induce lung volume reduction that is responsible for gas-exchange impairment. Original studies suggested that a large V_T could prevent atelectasis and improve oxygenation during anaesthesia [19]. More recently, use of large V_T up to $20\,ml\,kg^{-1}$ was shown to be ineffective in improving gas exchange and preventing atelectasis in both normal individuals [36] and morbidly obese patients [37,38].

Moreover, growing evidence suggests that large V_T ventilation without PEEP increases alveolar inflammation and can adversely affect the lung [39,40]. In the intensive care setting, V_T above $6\,ml\,kg^{-1}$ was shown to be harmful for patients with acute respiratory distress syndrome (ARDS) [41] and to favour subsequent respiratory failure in patients who were free of respiratory disease at admission [42,43]. Regarding surgical patients, intraoperative ventilation with large V_T was identified as a risk factor for early postoperative respiratory failure after pneumonectomy [3]. Similarly, the use of reduced V_T ($5\,ml\,kg^{-1}$) with a 5 cmH_2O PEEP during one-lung ventilation for oesophagectomy resulted in both reduced systemic inflammatory response and improvement of early postoperative lung function [4].

Eventually, a large V_T has more adverse haemodynamic consequences. Under positive pressure ventilation, the cardiac output is reduced proportionally to tidal volume [44].

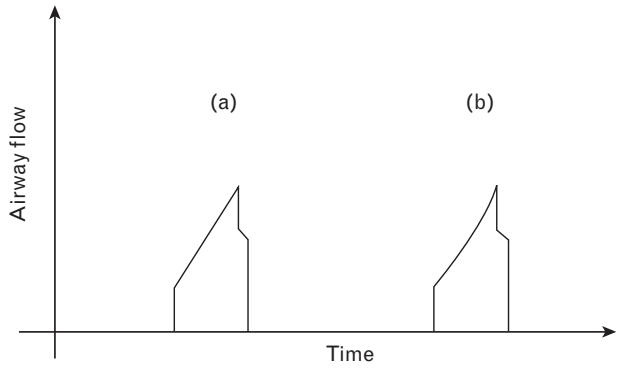
For all these reasons, we consider that V_T exceeding $10\,ml\,kg^{-1}$ of ideal body weight should be avoided because this exposes the patient to the risk of both low cardiac output and pulmonary overdistension. Further reduction to $6\,ml\,kg^{-1}$ is advised for patients susceptible to lung injury [45].

Positive end-expiratory pressure

By increasing both the expiratory and inspiratory alveolar diameters, PEEP allows us to compensate for anaesthesia-induced reduction in FRC. It can, therefore, prevent the end-expiratory lung volume from dropping below the closing capacity and subsequently prevent small airways from collapsing.

In several well defined circumstances, including anaesthesia of morbidly obese patients [46], the use of high inspired oxygen concentration [20] and single-lung ventilation [47], 5–10 cmH_2O of PEEP is associated with reduced atelectasis and improved oxygenation. Excessive levels of PEEP should, however, be avoided during single-lung ventilation in the lateral decubitus position because the resulting increased alveolar pressure can drive pulmonary blood flow towards the nonventilated lung [48].

Fig. 4



Examples of pressure–time curve monitoring. (a) The linear increase in the airway pressure during inspiration indicates that the pulmonary compliance remains constant. (b) Upward inflexion of the pressure–time curve at the end of the inspiration suggests a reduction in the pulmonary compliance secondary to lung overdistension.

In addition to its positive effect on oxygenation, PEEP is thought to protect against ventilator-induced lung injury associated with low V_T ventilation. In this case, PEEP prevents repeated opening and closure of small airways [49] and helps avoid adverse consequences of progressive lung derecruitment.

Application of PEEP should be considered with extreme care in case of a larger V_T. PEEP also increases the end-inspiratory lung volume and can favour lung overdistension. Moreover, addition of PEEP to the airway pressure resulting from the V_T insufflation potentially leads to barotrauma. In VCV, careful monitoring of the pressure–time curve is useful in diagnosing overdistension and helps in adjusting ventilatory settings. Reduced compliance associated with overdistension results in a late upward inflexion of this curve during insufflation (Fig. 4) [50].

PEEP also reduces the venous return to the right heart and, consequently, cardiac output [12]. Moreover, levels of PEEP equal to or higher than 15 cmH_2O cause further reduction in the cardiac output by increasing the pulmonary vascular resistance [51]. Finally, because of interventricular interaction, right ventricular distension secondary to increased pulmonary vascular resistance reduces the left ventricular filling. PEEP should, therefore, be used with caution in patients with hypovolaemia and those with right ventricular dysfunction [12].

Another side-effect of PEEP during anaesthesia is the potential rise in the intracranial pressure. Although it is still the subject of controversy, small levels of PEEP, up to 6 cmH_2O, do not seem to significantly increase the intracranial pressure. PEEP could, however, be avoided when intracranial hypertension is a concern [52].

The current literature, therefore, suggests that most anaesthetized patients can benefit from a small level of PEEP because it improves gas exchanges and helps to prevent ventilator-induced pulmonary damages.

The respiratory rate

Because we recommend using V_T between 6 and 10 $ml\,kg^{-1}$ of ideal body weight, the respiratory rate becomes an important determinant of alveolar ventilation. It should, therefore, be set to target the desired $PaCO_2$ or the end-tidal carbon dioxide tension (ET-CO_2).

We should, however, keep in mind that increasing the respiratory rate also increases the dead space ventilation. As a consequence, the efficacy of increasing respiratory rate on alveolar ventilation is reduced as demonstrated during laparoscopy [53].

Moreover, when high respiratory rates are used, we recommend continuous monitoring of the flow–time curve. Indeed, a high respiratory rate reduces the expiratory time and potentially induces intrinsic PEEP (PEEPi). Like extrinsic PEEP, PEEPi can be responsible for dynamic pulmonary hyperinflation, volutrauma and haemodynamic consequences.

Optimal $PaCO_2$ during anaesthesia depends on both the type of surgery and the patient's preexisting conditions. For example, intracranial neurosurgery requires $PaCO_2$ between 30 and 35 mmHg when intracranial hypertension is suspected. Patients with chronic obstructive pulmonary diseases may benefit from permissive hypercapnia to avoid volutrauma and barotrauma. Furthermore, permissive hypoventilation results in moderate hypercapnia, which is associated with an increased tissue oxygen tension [54,55] in the splanchnic area where it might reduce the risk of infection and improve wound healing [56]. The underlying mechanism of this beneficial effect is an increased cardiac output secondary to hypercapnia [57].

The inspired oxygen fraction (F_IO_2)

Intraoperative F_IO_2 frequently needs to be increased in order to compensate for anaesthesia-induced gas-exchange impairment; however, use of excessive inspired oxygen concentrations is potentially deleterious for the lung. First, oxygen is toxic for the tracheobronchial tree and the lung. In healthy animals, pure oxygen produces tracheobronchial irritation after a couple of hours [58]. Symptoms of severe respiratory failure may develop as soon as 24 h after administration of 100% oxygen and more prolonged exposure can lead to irreversible pulmonary fibrosis [59]. Second, as detailed above, high inspired oxygen fractions favour absorption atelectasis. Finally, high F_IO_2 increases lung susceptibility to ventilation with excessive V_T or airway pressures [60].

Apart from these deleterious consequences, intraoperative use of supplemental oxygen has been associated with several advantages. During major abdominal surgery, the use of high-inspired oxygen fractions (0.8 versus 0.35) results in reduced incidences of wound infections [2,61] and postoperative complications [62]. Supplemental oxygen increases PaO_2 as well as tissue and wound oxygen tension. Increased tissue PaO_2, in turn, favours collagen synthesis in the wound and prevents infection, possibly through the formation of free radicals [63]. Intraoperative administration of high F_IO_2 should be part of a multimodal approach to improve postoperative outcome and reduce the length of hospital stay in patients undergoing major abdominal surgery. Whether benefits of intraoperative high F_IO_2 can be extended to other types of surgeries deserves further study. Data from initial studies of intraoperative oxygen supplementation also suggested a reduced incidence of postoperative nausea and vomiting (PONV) in the group of patients given an F_IO_2 of 0.8 as compared with those given an F_IO_2 of 0.35 [64]. Other studies carried out in patients undergoing abdominal surgery as well as other surgical procedures, however, did not confirm these early results [65–67]. A recent meta-analysis dedicated to supplemental oxygen and PONV concluded that there was no evidence of any beneficial effect [68]. Therefore, there is currently no reason to use high F_IO_2 in order to prevent PONV, irrespective of the type of surgery. Increasing inspired oxygen fraction to 100% was finally shown to reduce mean cardiac index, heart rate and stroke volume index in both healthy volunteers and patients under propofol or sevoflurane anaesthesia. The mean arterial blood pressure was unaffected by F_IO_2, whereas systemic vascular resistance increased with increase in inspired oxygen [69]. The physiological consequences of these haemodynamic changes remain unclear as no outcome study has ever been performed. It can, however, be hypothesized that high oxygen tension could provide some degree of perioperative cardiovascular protection similar to the haemodynamic effects of β-blockers [70].

The choice of F_IO_2 should, therefore, take into account the benefits of supplemental oxygen and their potential risks. To summarize, levels of F_IO_2 in excess of 0.8 expose the patient to the risk of oxygen toxicity and atelectasis. Therefore, prolonged administration of F_IO_2 over 0.8 should not be routinely recommended unless necessary to correct intraoperative hypoxaemia. For major abdominal surgery, robust data suggest a reduction in postoperative complications with a F_IO_2 of 0.8h; however, these high F_IO_2 levels should be avoided in combination with large V_T and high airway pressures to reduce the risk of ventilator-induced lung injury.

Conclusion

General anaesthesia and mechanical ventilation *per se* induce impairment of gas exchanges. Appropriate management of the patient from the preoxygenation phase is worthwhile both to avoid hypoxaemia and to prevent ventilator-induced lung injuries. During anaesthesia, continuous monitoring of airway pressure–time and flow–time curves is useful for diagnosing adverse consequences of mechanical ventilation such as lung overdistension or intrinsic PEEP.

Moreover, recent data suggest that some ventilatory settings including use of small V_T, low level of PEEP and high-inspired oxygen fraction can positively affect patient outcome.

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