Introduction

Mechanical ventilation is necessary during many surgical procedures, however a paradigm shift in ventilation has taken place in the past decades. There is convincing evidence that neuromuscular blockade and subsequent controlled mechanical ventilation applying intermittent positive pressure, also in patients with non-injured, healthy lungs, may impair the respiratory system, leading to postoperative pulmonary complications (PPCs), resulting in worse clinical outcome, prolonged hospitalization time and increased cost of hospital care. Multifactorial pathophysiology of ventilator induced lung injury (VILI) has been evaluated and a pulmonary protective ventilatory strategy [lung protective ventilation (LPV)], including the use of low tidal volumes [6 mL/kg, ideal body weight (IBW)], moderate or optimal levels of positive end-expiratory pressure (PEEP) and applying regular or targeted alveolar recruitment maneuvers (ARMs), has been developed. Recognizing the role of neuromuscular blockade during general anesthesia and even the importance of avoiding residual neuromuscular blockade in the early postoperative period regarding to postoperative respiratory impairment have become another, newer direction of research. Despite promising and convincing results of recent clinical trials, incidence of PPCs could not be reduced significantly and lung protective ventilation has remained to be a “hot topic” among researchers in the field of anesthesia and critical care. Maintaining spontaneous breathing during general anesthesia has some pathophysiological rationale worth to be dealt with, because it may be one of the options for further improvement. Physiology, advantages, disadvantages and potential role of spontaneous breathing during surgery as compared to intermittent positive pressure ventilation will be described in this article.

Keywords: Spontaneous breathing; lung protective ventilation; non-intubated thoracic surgery

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serious complications after cardiovascular events in the postoperative period (1,2).

Based on extensive research over the past two decades, a better understanding of the pathophysiology of ventilator induced lung injury (VILI) has been widely achieved and a pulmonary protective ventilatory strategy (lung protective ventilation, LPV), including the use of low tidal volumes [6 mL/kg, ideal body weight (IBW)], moderate or optimal levels of positive end-expiratory pressure (PEEP) and applying regular or targeted alveolar recruitment maneuvers (ARMs), has been developed (3-16). Additionally, advanced monitoring of respiratory mechanics, the use of compliance, plateau pressure, driving pressure or even transpulmonary pressure as target parameters, reducing lung strain and stress, accurate monitoring of gas exchange parameters and hemodynamics have become mandatory tools to optimize ventilatory settings and prevent VILI (17). Overall these results of recent trials in the field of protective ventilation have been very promising and convincing, and the role of this strategy has gained increasing importance during general anesthesia in routine anesthetic care.

Recognizing the role of neuromuscular blockade during general anesthesia and even the importance of avoiding residual neuromuscular blockade in the early postoperative period regarding to postoperative respiratory impairment have become another, newer direction of research. Results of a recent multicenter prospective observational study [“Post-anaesthesia pulmonary complications after use of muscle relaxants” (POPULAR) Study] indicated that the use of neuromuscular blocking agents (NMBAs) during general anesthesia is associated with an increased risk of PPCs. Additionally, neither monitoring neuromuscular transmission during anesthesia, nor the use of reversal agents could decrease this risk. The investigators of POPULAR Study recommended that anesthetists must balance the potential benefits of neuromuscular blockade against the risk of PPCs and suggested the superiority of the use of supraglottic devices and maintaining spontaneous breathing over the use of neuromuscular blockade, endotracheal intubation and subsequent controlled mechanical ventilation during minor surgical procedures (18). These results call attention that maintaining spontaneous breathing during general anesthesia may well be one of the options for further improvement. Moreover, this technique may be beneficial for surgical interventions at increased risk of PPCs, like thoracic surgeries. There is a growing experience-based evidence about the advantageous effects on respiration of non-intubated anesthesia in thoracoscopic and open thoracic surgery under spontaneous ventilation (19-25). However, one should be noted that neuromuscular blockade and controlled ventilation might be recommended during some procedures to meet surgical needs.

Basic principles of respiration

Physiologic respiration is a result of complex and precise interaction between the chest wall and the lungs. Contribution of respiratory muscles, elastic components of the chest wall and the lungs play a central role in generating a pressure gradient across the respiratory system (between the mouth and the external surface of the chest wall), resulting in an airflow during the airways to allow air to enter the alveolar space where gas exchange takes place. During mechanical ventilation, especially in the intraoperative settings, due to the use of anesthetics and analgesics or even NMBAs, respiratory drive and activity of the musculature may be significantly reduced, or in most cases completely extinguished. In this case the ventilator must generate a positive pressure to create airflow. Simplified, ventilation occurs when a pressure difference occurs across the respiratory system, regardless of its origin. This pressure difference (gradient) is determined by the following universal equation:

\[ P_{ao} + P_{mus} = P_{EEP} + (Ers \times V) + (Rrs \times Flow) \]

In this equation \( P_{ao} \) represents the pressure at the airway opening and \( P_{mus} \) is the pressure generated by respiratory muscles. PEEP is positive end-expiratory pressure, \( Ers \) is the elastance and \( Rrs \) is the resistance of the respiratory system, \( V \) stands for tidal volume, and \( Flow \) means the airflow (26).

It is evident that these main parameters—pressure gradient, elastance (or the inverse of elastance, namely compliance), volume, resistance and flow—determine ventilation, it follows that they should be monitored carefully and continuously during mechanical ventilation (27-29).

Respiratory physiology during spontaneous breathing

During physiological (unassisted) spontaneous inspiration movement of the chest wall and an increase in thoracic cavity and lung volumes due to active contraction of respiratory muscles decrease the already negative pleural pressure further and generate a pressure gradient termed transpulmonary pressure (\( P_{tp} \)) resulting in a “physiological negative pressure” ventilation. It is well known that regional
distribution of ventilation is heterogenous due to the elastic properties of the lungs and vertical gradient of pleural (and transpulmonary) pressure (30).

There are 2 groups of the muscles of the thoracic wall: those involved in inhalation and those responsible for forced exhalation. The principal muscle is the dome-shaped diaphragm whose contraction increases either the vertical dimension of the thorax by pushing downward the abdominal content, or the anterior-posterior dimension by an outward traction of the ribs. Contraction of the external intercostals elevates the lateral part of the ribs resulting in an increase of the transverse diameter of the chest. This excursion of the diaphragm is not homogenous, as well as ventilation and perfusion. Researches using fluoroscopic imaging proved that the diaphragm can be divided into three segments functionally: top (nondependent, anterior tendon plate), middle and dorsal (dependent, posterior). During spontaneous breathing (SB) the posterior part move more than the anterior, opposing alveolar compression, preventing ventilation/perfusion (V/Q) mismatch and resulting in improved ventilation of the dependent regions of the lungs. These advantages remain even in supine position (31,32).

During exhalation an opposite process takes place: the diaphragm and external intercostals relax, and due to the elastic elements of the lungs, the natural recoil of the lungs decreases the thoracic space, squeezing the air out of the lungs. This elastic recoil is sufficient during normal breathing thus expiration is a passive process. However, during forced expiration several other muscles (rectus abdominis and internal intercostal muscles) are recruited to increase the power and effectiveness of expiration.

Moreover, one should not forget that breathing patterns, respiratory rate and amplitude is variable during spontaneous ventilation to achieve metabolic requirements.

Advantages of SB during mechanical ventilation are summarized in Table 1.

It should be mentioned that there are also several disadvantages of SB during mechanical ventilation. Disadvantages include the possibility of uncontrolled inspiratory efforts that may worsen lung injury due to volutrauma or barotrauma; increased heterogeneity of ventilation leading to “occult pendelluft” (regionally elevated $P_L$ despite a safe mean value); regional dorsal atelectrauma due to cyclic opening and closing of small airways (33,34); patient-ventilator asynchrony resulting patient distress; increased alveolo-capillary pressure gradient leading to interstitial edema; impaired hemodynamics; difficulties in feasible measuring of respiratory mechanics parameters (e.g., driving pressure); impossibility of using NMBAs that may make endotracheal intubation and secured airway difficult. Respiratory depression effect of major analgesics may be also a problem that needs attention.

**Table 1** Advantages of spontaneous breathing during mechanical ventilation

<table>
<thead>
<tr>
<th>Advantage</th>
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<tr>
<td>Intact respiratory muscle tone</td>
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<td>Restored diaphragmatic function</td>
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<tr>
<td>Improvement of dorsal ventilation</td>
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<td>Prevent ventral redistribution of ventilation</td>
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<td>Improved V/Q matching</td>
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<td>Improved gas exchange</td>
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<tr>
<td>Maintenance of distal airway patency</td>
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<tr>
<td>Prevent atelectasis of the lungs</td>
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<td>Improved FRC</td>
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<tr>
<td>Restoration of mucociliary clearance</td>
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<tr>
<td>Prevent PPCs</td>
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<tr>
<td>Improved hemodynamics</td>
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<tr>
<td>Avoiding the use of NMBAs</td>
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<tr>
<td>Decreased sedation</td>
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<td>Reduced recovery time after operation</td>
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V/Q, ventilation/perfusion ratio; FRC, functional residual capacity; PPCs, postoperative pulmonary complications; NMBAs, neuromuscular blocking agents.

**Respiratory physiology changes during positive pressure ventilation**

Positive pressure ventilation modes can be divided into two groups: invasive or non-invasive assisted spontaneous ventilation [e.g., pressure support ventilation (PSV)], and controlled ventilation [e.g., volume-controlled ventilation (VCV) or pressure-controlled ventilation (PCV) modes]. It is common to both modalities that a positive inspiration pressure is generated by a ventilator, but during assisted spontaneous ventilation the work of breathing is shared by the respiratory muscles and the ventilator, while during controlled modes muscles remain passive and all respiratory work is carried out by the machine. During assisted spontaneous ventilation alveolar pressure (Palv) decreases below PEEP for only a proportion of the inspiratory time,
while Pao and Pmus are positive. In controlled ventilation Pao and Palv are always positive, while Pmus = 0 cmH2O (26).

Beyond these major differences from physiological breathing, that is, mechanical ventilators pressurize the respiratory system, and a heterogenous redistribution of P1 occurs during positive pressure ventilation (30). This heterogenous redistribution of P1 in combination with inappropriate ventilatory settings might be responsible for both mechanical (barotrauma, volutrauma) and biological injury of the lungs (damage of the extracellular matrix due to cyclic opening and closing of the little airways and increased inflammatory response) leading to VILI and PPCs.

On the other hand, a typical redistribution of ventilation occurs during positive pressure ventilation, especially when neuromuscular blockade is also introduced. During controlled mandatory ventilation (CMV), main extent of ventilation is being shifted to the nondependent and less perfused anterior regions of the lung leading to V/Q mismatch and extent atelectasis in the dependent lung regions (31). These observed differences are based on the altered excursion of the diaphragm. Movement of the posterior, dependent part of the diaphragm decreased significantly but rather at anterior, nondependent part during controlled ventilation even when low tidal volumes were applied (35-37). These differences could only be more, or less equalized when tidal volumes were increased, but also remain regardless of whether PCV or PSV modes are used, however some authors suggested the superiority of PSV over either CMV or SB (32,35,37-39). Additionally, when NMBAs are used, redistribution of diaphragmatic excursion and the concomitant ventilatory impairments become much more striking.

Maintaining spontaneous breathing during thoracic surgery: NITS, a new approach

Thoracic surgery is considered high risk for PPCs. This risk has a dual origin: several surgery related risk factors and patient related risk factors are in the background. Patients scheduled for thoracic surgery commonly have long standing medical history of pulmonary disease [e.g., chronic obstructive pulmonary disease (COPD), restrictive disorders, tumors, etc.], most of them are smoking and have impaired respiratory mechanics and gas exchange. Other proportion of patients have an acute pulmonary or intrathoracic morbidity (e.g., pulmonary abscess, thoracic empyema, etc.). In one word: thoracic surgery is a high-risk intervention in a high-risk patient, that makes a challenge for the anesthetist.

The gold standard ventilatory mode for thoracic surgery was considered invasive mechanical one lung ventilation (OLV) for decades. OLV under general anesthesia was required in most open thoracic procedures, especially in video-assisted thoroscopic surgery (VATS). OLV can be achieved by using a double-lumen endotracheal tube, or some types of bronchial blockers. The use of these airway devices provides adequate conditions for isolation either the right or the left lung and for surgery as well. Additionally, OLV had some pathophysiological rationale: gas exchange impairment (progressive hypoxia, hypercapnia and hypoxic pulmonary vasoconstriction) due to the operated collapsed lung during surgical pneumothorax with maintained SB was well known and was considered intolerable (40,41).

In the last decades, the widespread use of combined regional (epidural, local and plane blockades) and general anesthesia techniques along with technical development of ventilatory equipment, and also the improvement of the minimal invasive thoracic surgery have allowed to perform thoracic surgery on awake or only minimally (conscious) sedated patients in SB (41). Moreover, thank to extensive research, nowadays surgical pneumothorax can be considered a safe technique that allows maintenance of SB during thoracic surgery procedures. The technique is named non-intubated thoracoscopic surgery (NITS) or non-intubated VATS (NIVATS), while VATS performed under general anesthesia is commonly termed GAVATS in literature. NITS can be performed with or without laryngeal mask airway insertion as well.

NITS enables the maintenance of SB throughout the surgical procedure offering several advantages (including prevention of baro-, volu and atelectrauma, ventral redistribution of ventilation and attenuation of inflammatory response) as compared to intermittent positive pressure mechanical ventilation (IPPV) (42). Regarding to the common patient population scheduled for thoracic surgery, SB may protect against the harmful effects of IPPV as well, so the risk of VILI and consequently the development of PPCs may be reduced resulting improved outcome, shorter in-hospital stay and reduced health care costs. Either surgical or anesthetic techniques of NITS/NIVATS is well described, but there are some cornerstones to mention. First, adequate regional anesthesia (thoracic epidural, intercostal nerve or paravertebral blockade) supplemented with or without serratus plane blockade is essential, and infiltration of vagal nerve with local anesthetics—for prevention of coughing and bradycarryrhythmia during
the procedure—is suggested. According to some authors thoracic epidural anesthesia from T₁ to T₈ alone may be sufficient in most cases (42-45). Once surgical pneumothorax is performed and the nondependent lung is collapsed, patient may become dyspneic or tachypneic, signs of respiratory distress and panic can occur, therefore most of the NITS cases are performed under sedation. The most popular option is propofol sedation by the target-controlled infusion (TCI) guided by depth of anesthesia monitoring reached the surgical sedation level either (42). In all cases, incremental titration of opioid analgesics can also be used. All authors in the field of NITS agree, that moderate hypoxia and hypercapnia resulting mild, non-significant respiratory acidosis is common during non-intubated awake thoracic surgery. These changes resolve within some minutes to hours after successful operation (19,22,23,24,42). Postoperative recovery is also fast: patients are allowed to drink clear fluids 1 hour after the operation, breathing exercises and mobilization can be started as soon as possible, practically already in the post-anesthesia care unit (42). Further advantages of NITS as compared to conventional GAVATS are the decreasing occurrence of postoperative nausea and vomiting (PONV), the less frequently required nursing care and the reduced in-hospital length of stay (19). The main disadvantage is that in case of intraoperative deterioration, endotracheal intubation and conversion to conventional OLV can be difficult. Moreover, NITS requires practice, skills and excellent interdisciplinary cooperation between the anesthetist and the surgeon as well.

Conclusions

Despite promising and convincing results of recent clinical trials, lung protective ventilation has remained to be a “hot topic” among researchers in the field of anesthesia and critical care. Despite the well-evaluated pathophysiology of VILI and efforts have been made in the past decades to eliminate these pathophysiological factors, incidence of PPCs could not be reduced significantly. Neither low tidal volume ventilation, nor the use of moderate levels of PEEP and regular use of ARMs alone or in combination could have solved this worldwide healthcare problem: LPV concept seems to be a search for “The Holy Grail”. The reason for this may be that mechanical ventilatory support applying intermittent positive pressure, regardless to the mode of ventilation (controlled, assisted or intelligent dual-controlled mode), is non-physiological, to say the least.

Individualization of ventilatory settings and maintaining physiological spontaneous breathing during mechanical ventilation may provide the opportunity for further improvement.

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None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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