EDITORIAL

VENTILATION-PERFUSION MATCHING DURING ONE LUNG-VENTILATION IN THE LATERAL DECUBITUS POSITION

In the awake patient, breathing spontaneously in the lateral decubitus position (LDP), gravity results in a higher pleural pressure around the dependent lung which places the dependent lung on the steep part of the compliance (pressure-volume) curve. Also, gravity results in a higher pulmonary blood flow to the dependent lung than the blood flow to the nondependent lung. In the awake patient breathing spontaneously, and probably during natural sleep, the dependent lung receives more perfusion matched with more ventilation during the lateral decubitus position than the nondependent lung. Thus, adequate ventilation-perfusion matching is insured in both the dependent and the nondependent lungs of the awake patient during the LDP.

Induction of general anesthesia decreases the functional residual capacity (FRC) in both the dependent and non-dependent lungs with a consequent change of compliance; the dependent lung moves from the steep compliant position to the lower flat non compliant position of the compliance curve, while the nondependent lung moves from the upper flat non compliant position of the flow-volume curve down to the steep compliant portion. This will shift more ventilation away from the dependent to the nondependent lung. However, because of the gravity, blood flow to the dependent lung remains higher than the blood flow to the nondependent lung. Thus, following general anesthesia in the LDP, the dependent lung is more perfused than ventilated resulting in a shunt effect. In contrast, the non-dependent lung is more ventilated than perfused, resulting in a dead space effect. In order to maintain adequate oxygenation and carbon dioxide elimination, moderate hyperventilation with a high FiO2 is required. An alternative approach is to apply PEEP to the two lung, which can restore their FRC, and their original position on the pressure-volume curve, and optimize ventilation-perfusion matching during general anesthesia in the LDP.

During one-lung ventilation in the lateral decubitus position, the dependent lung is only ventilated. Also, the blood flows predominantly to the dependent lung. In contrast, the non-dependent lung is not ventilated, but is still perfused by about 40-50% of the cardiac output, creating a shunt effect. The degree of shunt is decreased by decreasing the blood flow to the non-dependent nonventilated lung by gravity and by lung collapse. However, the most important factor which decreases the degree of shunt from about 40-50% down to 20% is hypoxic pulmonary vasoconstriction (HPV) of the vessels of the nonventilated lung. HPV is obtunded in COPD patients, as well as in patients with respiratory bronchiolitis-associated interstitial lung disease (RB-ILD). Thus, transpulmonary shunting may persist in the non-ventilated lung, resulting in severe hypoxemia during OLV. In contrast, HPV remains very active in patients having left-to-right cardiovascular shunt such as patent ductus arteriosus and ventricular septal defect, which explains the high PaO2 during OLV of these patients.

Distribution of perfusion according to the lung disease is another factor which determines the degree of shunt during OLV. Patients with large central tumors undergoing pneumonectomy or lobectomy will most probably have less perfusion to the operated non-ventilated lung, as compared with patients with peripheral masses. Lung perfusion studies showed that perfusion is already impaired in patients with large central tumors, may be secondary to activation of HPV. That is why, patients undergoing pneumonectomy or lobectomy have a much better oxygenation during OLV than those presenting for peripheral masses.

Hypoxemia during OLV is not only related to the lung disease, but is also determined by several inter-related factors that determine the balance between oxygen consumption, and oxygen delivery:

Cardiac output x [(Hb x SaO2 x 1.34) + (PaO2 x 0.0031)]

Thus, hypoxemia during OLV is determined by both ventilation and perfusion, and hence may be attenuated by manipulation of ventilation and perfusion independently.

Protective ventilation strategy using a tidal volume of 5-6 mlKg-1 associated with PEEP versus ventilation by 10 mlkg-1 has been followed by a significantly lower inflammatory response. The inflammatory mediators may be also significantly lower in patients receiving inhalation sevoflurane than those who had intravenous propofol. It may be concluded that hypoxemia release of inflammatory mediators during one-lung ventilation may be attenuated by manipulation of ventilation and perfusion independently. From a ventilator perspective, protective ventilation strategy using a low tidal volume and positive end-expiratory pressure and avoidance of high tidal volume may attenuate the development of acute lung injury during one-lung ventilation. Hypoxemia during OLV may be also attenuated by adequate oxygen delivery, which can be achieved by maintenance of cardiac output, and avoidance of excessive hemodilution.

A sevoflurane-based volatile anesthetic regimen has been also shown to provide protection against inflammatory mediators release during OLV, and better cognitive function following open heart surgery than intravenous proprofol-based anesthesia, suggesting preconditioning of cerebral hypoxemia by the inhalational anesthetic.

Despite optimizing ventilation by the protective ventilation strategy and optimizing perfusion by maintaining cardiac output and avoiding excessive hemodilution, serious hypoxemia during OLV can result from malpositioning of the lung isolation devices such as the double-lumen tubes and the bronchial blockers. The safety margin following left bronchial intubation is greater than following right bronchial intubation because the distance from the carina to the inlet of left upper lobe bronchus is longer than the distance to the inlet of the right upper lobe bronchus. Unfortunately, despite the safety margin, blocking of the inlet of the left upper lobe bronchus by down migration of the tube which may occur during positioning of the patient will result in severe hypoxemia because ventilation will be limited to the left lower lobe only, and hence hypoxemia will not be effectively counteracted by the HPV. Also, overinflation by limiting ventilation to one lobe can result in catastrophic barotrauma. Achieving one-lung ventilation by a tracheal tube and bronchial blocker may be also complicated by severe hypoxemia and unilateral pulmonary oedema whenever suction via the bronchial blocker lumen in practiced while the cuff of the blocker is inflated before thoracotomy. In this situation, suction can result in excessive negative pressure within the lung resulting in excessive congestion of the lung complicated by unilateral pulmonary edema and severe hypoxemia. Also, prolonged suctioning even via the tracheal lumen during OLV can result in hypoxemia whenever ventilation is interrupted for a prolonged period, because the oxygen store will be limited to FRC of one lung only.

The factors that influence distribution of ventilation and perfusion are also important postoperatively. In adults with unilateral lung disease; gas exchange is optimal when the good lung is dependent (down with the healthy lung). In contrast, gas exchange in infants is optimal when the healthy lung is nondependent, a finding opposite to that achieved in adults. Ventilation may be distributed differently in infants and adults. The more unstable rib cage in infants result in a lower FRC close to residual volume, making airway closure likely to occur in the dependent lung, even during tidal volume breathing, and thereby redistribute ventilation

to the non dependent lung. It is not known at which age the adult pattern appear. It is suggested that during the postoperative period, the child with unilateral lung disease should be nursed in both the lateral decubitus position, as well as the supine position to determine the position of optimal gas exchange.

In conclusion, hypoxemia during one-lung ventilation can be predicted preoperatively by severity of the lung pathology, by lung scan, and by the oxygen saturation during two-lung ventilation. Whenever hypoxemia develops following one-lung ventilation, the proper position of the intubation device should be ensured by fiberoptic bronchoscopy. Also, protective ventilation strategy using inhalation anesthetic in 100% oxygen can improve oxygenation and decrease inflammatory mediators. In addition, cardiac output should be optimized, and severe hemodilution avoided. Whenever hypoxemia persists, two-lung ventilation must be resumed, and PEEP to the dependent lung optimized after recruitment. Applying CPAP using 100% oxygen to the nondependent non-ventilated lung may also improve oxygenation significantly. However, the technique is not welcomed during thoracoscopy since it will distend the lung on the operative side, and may interfere with the surgical procedure. NO inhalation can be also used to improve perfusion of the ventilated lung during one-lung ventilation. As a last resort, pulmonary artery clamping on the operated side by the surgeon can improve oxygenation by discontinuing perfusion of the non-ventilated lung, and shifting the cardiac output to the ventilated lung with a consequent decrease of shunt.

Anis Baraka, MD, FRCA (Hon)
Emeritus Professor, Department of Anesthesiology
American University of Beirut
Emeritus Editor-in-Chief, Middle East Journal of Anesthesiology

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