One lung ventilation: General principles

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INTRODUCTION

— In most patients requiring mechanical ventilation, both lungs are inflated and deflated together. One lung ventilation (OLV) refers to mechanical separation of the lungs to allow ventilation of only one lung. OLV is a standard approach to facilitate surgical exposure for thoracic surgeries, and may be used to isolate a pathologic

from a healthy lung to prevent soiling or to allow differential ventilation.

This topic will discuss the general principles and physiology of OLV, its management, and complications. Devices used for OLV, their placement, and comparative performance are reviewed separately. (See "Lung isolation techniques".)

INDICATIONS

— OLV is used either to improve exposure to the surgical field in thoracic surgery, or to anatomically isolate one lung from a pathologic process of the other lung [1].

Surgical exposure

— A non-ventilated, collapsed lung in the vicinity of the surgical field improves access to the thoracic cavity. OLV is often used during:

- Pulmonary resection, including pneumonectomy, lobectomy, and wedge resection (see "Anesthesia for open pulmonary resection", section on 'Airway control')

- VATS, including wedge resection, biopsy, and

pleurodesis (see "Anesthesia for video-assisted thoracoscopic surgery (VATS) for pulmonary resection", section on 'Airway management and surgical bronchoscopy')

- Mediastinal surgery (see "Anesthesia for patients with an anterior mediastinal mass", section on 'Airway management')

- Esophageal surgery (see "Anesthesia for esophagectomy and other esophageal surgery", section on 'Considerations for one lung ventilation')

- Thoracic vascular surgery (see "Anesthesia for descending thoracic aortic surgery", section on 'Induction and airway management')

- Thoracic spine surgery (see "Anesthesia for elective spine surgery in adults", section on 'Airway management')

- Minimally invasive cardiac valve surgery (see "Minimally invasive aortic and mitral valve surgery")

Lung isolation

— Lung isolation may be necessary to:

- Avoid cross contamination from one lung to the other in the following circumstances:
    - Pulmonary hemorrhage (see "Lung isolation techniques", section on 'Massive hemoptysis' and "Evaluation and management of life-threatening hemoptysis")
    - Purulent pulmonary secretions (see "Lung isolation techniques", section on 'Unilateral pulmonary infection')
    - During whole lung lavage for pulmonary alveolar proteinosis [2] (see "Treatment and prognosis of pulmonary alveolar proteinosis in adults", section on 'Whole lung lavage' and "Lung isolation techniques", section on 'Unilateral lung lavage')
- Decrease pressure or air flow on the side of pathology, as in bronchopleural fistula or unilateral cyst/bullae (see "Bronchopleural fistula in adults" and "Overview of pulmonary resection", section on 'Pulmonary blebs and bullae')

CONTRAINDICATIONS

— Contraindications to OLV include dependence on bilateral mechanical ventilation. Patients with severe pulmonary disease or prior pulmonary resection may not tolerate OLV; in these cases, selective lobar blockade may be an option. (See "Lung isolation techniques", section on 'Quality of lung deflation and reinflation'.)

Placement of a double-lumen endotracheal tube (DLT) may not be technically possible in a patient with an intraluminal airway mass restricting access to the tracheobronchial tree, and may be inadvisable if the intraluminal mass could be dislodged during placement, since this could cause complete airway obstruction.

PHYSIOLOGY

— The clinician using OLV needs to understand the changes in physiology caused by differential ventilation of the two lungs.

During normal ventilation, ventilation and perfusion are well matched anatomically, because dependent portions of the lungs receive both

greater blood flow (a result of gravity) and greater ventilation (from gravitational effects on lung compliance). The initiation of OLV stops all ventilation to one lung, which would create a 50 percent right-to-left shunt and relative hypoxemia if perfusion were unchanged. However, the actual shunt fraction is usually only 20 to 30 percent for the following reasons [3]:

- Surgical manipulation of the atelectatic lung obstructs vascular flow to the non-ventilated lung.

- Lateral positioning of the patient leads to a gravitational increase in perfusion to the dependent, ventilated lung. Most patients undergoing thoracic surgery are in the lateral decubitus position. The ventilated dependent lung is within the closed side of the chest and is exposed to the weight of the contralateral hemithorax contents. These factors result in an increase in lung elastance and the power delivered by the mechanical ventilator, even though tidal volume (TV) is reduced during OLV [4].

- Hypoxic pulmonary vasoconstriction (HPV)

modulates the blood flow to hypoxic regions of the lungs. HPV reduces shunt flow through the nonventilated lung by 40 to 50 percent during OLV, moderating the degree of hypoxemia [5,6]. Alveolar hypoxia triggers the pulmonary vessels to constrict, directing blood away from poorly/nonventilated areas to better ventilated segments, thereby improving ventilation/perfusion matching. Clinically, HPV is proportional to the degree of local hypoxia, and is triggered primarily in response to an alveolar oxygen partial pressure (PaO_2) less than 100 mmHg [6]. The onset of HPV is two-phase with an initial rapid onset phase (minutes) and a delayed phase (hours). When oxygenation and ventilation is reestablished, the attenuation of HPV follows a reverse two-phase pattern and may not completely return to normal for several hours [5]. This is an important consideration in the case of bilateral sequential thoracic procedures, such as bilateral wedge resections for lung metastases, in which patients tend to desaturate more during the OLV of the second lung.

A number of anesthetic factors may influence the extent of HPV, some of which may be controlled by the management of the anesthetic.

HPV is potentiated (causing less shunt, improving oxygenation) by [6]:

- Metabolic and respiratory acidosis
- Hypercapnia
- Mild decreased mixed venous oxygenation
- Hyperthermia

HPV is decreased (causing increased shunt, worsening oxygenation) by:

- Metabolic and respiratory alkalosis
- Hypocapnia
- Hypothermia
- Increased left atrial pressure
- Administration of a volatile inhalation anesthetic at a dose >1 minimum alveolar concentration (MAC)
- Hemodilution

IMPROVING DEFLATION OF THE NONVENTILATED LUNG

— After placement of a lung isolation device to achieve OLV, expedited deflation of the nonventilated lung improves surgical access. One or more of the following maneuvers may help improve collapse of the nonventilated lung:

- Denitrogenate both lungs by ventilating with either 100 percent oxygen (O_2) or a nitrous oxide (N_2O)/O_2 mixture for several minutes prior to initiating OLV [7]. Replacing the poorly absorbed nitrogen in the nonventilated lung with more soluble O_2 or N_2O will facilitate collapse [8].

- Apply low suction (20 cmH_2O) until the nonventilated lung has collapsed well [9]. With a bronchial blocker, low suction is applied via the suction channel lumen. With a DLT, low suction is applied via a suction catheter placed into the endotracheal lumen that leads to the nonventilated lung. Low suction also prevents passive entrainment of air into the nonventilated lung [10].

- When a bronchial blocker is used, disconnect the anesthetic circuit and allow a prolonged expiration for 20 to 30 seconds, or until the end-tidal carbon dioxide ($ETCO_2$) falls to zero.

  Then begin OLV after inflating the cuff of the bronchial blocker under direct vision with the flexible intubating scope [8,11].

- Use pressure-controlled ventilation during OLV to avoid the peaks in airway pressure that may occur during volume-controlled ventilation (eg, due to surgical manipulation in the chest or patient coughing). This decreases the chance of inadvertent reinflation of the nonventilated lung due to gas being forced past the inflated cuff of the DLT or bronchial blocker device.

LUNG-PROTECTIVE VENTILATION STRATEGIES

— Thoracic surgery with OLV can result in a spectrum of lung injury ranging from mild lung injury to severe acute respiratory distress syndrome (ARDS) [12,13]. (See "Ventilator-induced lung injury".)

To minimize this risk, ventilation strategies adapted from approaches proven to be protective for patients with ARDS in the critical care setting are used during OLV. The goal of such protective ventilation is to minimize ventilator-induced lung trauma, inflammation, and injury due to alveolar over-distension and cyclic atelectasis, while maintaining adequate oxygenation. This protective ventilation strategy includes (see "Mechanical ventilation during anesthesia in adults", section on 'Lung protective ventilation during anesthesia') [14]:

- Low tidal volume (TV) ventilation: 4 to 6 mL/kg (adjusted from 6 to 8 mL/kg during two lung ventilation)
- Adjustment of respiratory rate to maintain end-tidal CO_2 ($ETCO_2$) and arterial carbon dioxide tension ($PaCO_2$) near the patient's baseline
- Positive end-expiratory pressure (PEEP): 5 to 10 cmH_2O if TV is low (0 to 5 cmH_2O in patients with chronic obstructive pulmonary disease [COPD])
- Limited airway pressures: plateau inspiratory

pressures <30 cmH_2O

- Minimum fraction of inspired oxygen (FiO_2): minimum level to maintain SpO_2 >90 percent

The incidence of clinically overt acute lung injury following lung resection generally ranges from 2 to 4 percent [15-17], though it is higher after larger resections, particularly pneumonectomy at 8 to 12 percent [16,18]. Mortality from acute lung injury is approximately 40 percent [6,19]. Measurement of inflammatory biomarkers (eg, cytokines) have been used to detect pulmonary injury and to predict development of postoperative multiorgan system dysfunction and mortality [12,20].

Low tidal volume

— To minimize lung injury caused by high TVs, the standard of care at the authors' institution is to use a TV of only 4 to 6 mL/kg predicted body weight during OLV (adjusted from 6 to 8 mL/kg during two lung ventilation). This strategy is based on proven benefits of low TV in critical care patients with previous lung injury, as well as some evidence of benefit in those without lung injury

[21,22]. (See "Overview of initiating invasive mechanical ventilation in adults in the intensive care unit", section on 'Tidal volume' and "Ventilator management strategies for adults with acute respiratory distress syndrome", section on 'Low tidal volume ventilation (LTVV): Initial settings'.)

In patients undergoing OLV, randomized and observational trials of ventilation with low TVs have noted decreased inflammatory markers, though not improved outcomes [23-26].

Permissive hypercapnia

— Use of low TVs without compensatory increases in respiratory rate leads to hypercapnia. This seems to be well-tolerated, and may be advantageous in OLV. Hypercapnia potentiates HPV [27] and causes a rightward shift of the oxyhemoglobin dissociation curve, enhancing oxygen delivery to tissue and thus potentially improving wound healing and reducing infectious complications [28]. However, benefits of hypercapnia must be balanced against the potential for increased intracranial pressure, pulmonary hypertension, myocardial depression, and decreased renal perfusion. (See "Permissive hypercapnia during mechanical ventilation in

adults".)

- Hypercapnia (to 64 mmHg) during OLV was well tolerated in one trial, though associated with a 42 percent increase in pulmonary vascular resistance [29].

- In two other series with higher levels of hypercapnia, patients tended to require inotropic support [30,31].

As a result of preexisting pulmonary disease in this patient population, a large P_aCO_2 to end tidal CO_2 gradient often exists during OLV, so regular arterial blood gas sampling is recommended.

PEEP level

— During OLV, patients being ventilated with low TVs should have positive end-expiratory pressure (PEEP) of 5 to 10 cmH_2O applied. However, excessive PEEP during OLV may shift perfusion away from the ventilated lung, resulting in increased shunt [32,33]. Also, the levels should be decreased or used with caution in patients with severe obstructive disease, in whom high levels of auto-PEEP make the impact of extrinsic PEEP

unpredictable.

PEEP is used during mechanical ventilation to prevent atelectasis and reduce injury from the mechanical stress of repetitive inflation/deflation cycles in the alveoli, which may lead to hypoxia and inflammation [34]. While there are no studies showing decreased lung injury associated with PEEP, equivalent or improved oxygenation and lower inflammatory markers resulted from use of PEEP (5 to 10 cmH_2O) with low TV (5 to 7 mL/kg) during OLV when compared with larger TV (9 to 15 mL/kg) without PEEP in several trials [23,24,35]. When lower TVs are used, PEEP is considered standard to prevent atelectasis; the need is illustrated by a small study in which 10 cmH_2O PEEP was required (in contrast to zero or 5 cmH_2O PEEP) to maintain baseline oxygenation and functional residual capacity during upper abdominal surgery [36]. (See "Mechanical ventilation during anesthesia in adults", section on 'Positive end-expiratory pressure' and "Overview of initiating invasive mechanical ventilation in adults in the intensive care unit", section on 'Positive end expiratory pressure'.)

Auto PEEP in obstructive disease — When positive end-expiratory pressure (PEEP) is added during OLV with higher TVs, or in patients with obstructive disease, the benefit is variable; this is most likely due to the contribution of dynamic hyperinflation, or auto-PEEP, in which incomplete expiration prior to the initiation of the next breath causes progressive air trapping. (See "Positive end-expiratory pressure (PEEP)", section on 'Auto (intrinsic) PEEP'.)

The majority of patients develop some level of auto-PEEP during OLV, at a level inversely correlated to the extent of obstructive pulmonary disease [37]. In patients with documented severe obstructive disease (low forced expiratory volume in one second [FEV_1]), or those in whom the expiratory flow is interrupted by the next breath, even low PEEP should be used judiciously to avoid dynamic hyperinflation.

- In one trial, patients received either 10 cmH_2O PEEP or no PEEP during OLV; PEEP improved oxygenation and lung compliance only in patients without obstructive disease [38].

- In a series of patients having OLV with 10 mL/kg TV, when PEEP 5 cmH_2O was added, oxygenation improved in 14 percent, was unchanged in 65 percent, and worsened in 21 percent [39]. The patients most likely to benefit from PEEP in this study had normal spirometry and low levels of intrinsic PEEP; the addition of extrinsic PEEP to those with obstructive disease likely resulted in excessive alveolar distension, increased pulmonary vascular resistance within the ventilated lung, increased shunting of blood flow to the non-ventilated lung, and worsened hypoxia.

Low airway pressures

— Use of pressure-controlled ventilation (PCV) rather than volume-controlled ventilation (VCV) during OLV has not been proven to decrease lung injury or improve oxygenation, but may be a reasonable choice to control high airway pressures and prevent barotrauma. (See "Mechanical ventilation during anesthesia in adults", section on 'Modes of intraoperative mechanical ventilation' and "Mechanical ventilation during anesthesia in adults", section on 'Plateau pressure'.)

- Two crossover studies during OLV demonstrated improved oxygenation and shunt fraction with PCV as compared with VCV, especially in patients with poor preoperative lung function [40,41]. This may be because PCV provides a more homogeneous distribution of tidal volume, thus improving oxygenation and dead space ventilation [42].

- However, two other trials were unable to demonstrate any improvement in oxygenation, although they did have lower airway pressures with PCV [43,44].

There are no prospective trials looking at outcome when ventilation is managed specifically to lower airway pressures. Two observational studies in patients undergoing OLV have associated ventilation at high airway pressures with the development of postoperative acute lung injury, but it is unclear whether this is simply an early marker for lung injury rather than a causative effect [15,45]. Pulmonary barotrauma is associated with plateau airway pressures greater than 35 cmH_2O, so maintaining plateau inspiratory pressures less

than 30 cmH_2O is a reasonable goal. (See "Diagnosis, management, and prevention of pulmonary barotrauma during invasive mechanical ventilation in adults".)

Oxygen concentration

— Patients should be ventilated with the minimum FiO_2 needed to maintain oxygen saturation above 90 percent for the following reasons (see "Mechanical ventilation during anesthesia in adults", section on 'Fraction of inspired oxygen'):

- A high FiO_2 allows absorption atelectasis in the ventilated lung, increasing shunt and worsening oxygenation [46].
- Hyperoxia produces toxic oxygen free radicals, though the threshold above which oxygen toxicity and acute lung injury occur is unknown [47]. Re-expansion of the surgical lung worsens this oxidative stress, resulting in increased vascular permeability and alveolar-capillary membrane edema. As a result, re-expansion using low FiO_2 may attenuate this damage [48].

- Potentially preventable hyperoxemia and substantial oxygen exposure are common during OLV [49]. Although use of 100 percent oxygen is often justified to minimize the risk of desaturation, the incidence of desaturation below 90 percent during OLV is only approximately 5 percent [3,50]. If desaturation occurs while lower oxygen concentrations are used, any increase in FiO_2 should be temporary while measures are taken to remedy the cause of the problem. (See 'Treatment of hypoxemia' below.)

In one study of patients undergoing thoracic surgery, continuous titration of FiO_2 did not reduce oxygen exposure during OLV, but did reduce it after pulmonary vascular ligation [51].

Efficacy of lung protective strategies

— Protective ventilation, using a combination of smaller TVs, PEEP, lower airway pressures, and an FiO_2 less than 100 percent may improve clinical outcomes when compared with previously advocated strategies (see "Mechanical ventilation during anesthesia in adults", section on 'Lung

protective ventilation during anesthesia' and "Overview of initiating invasive mechanical ventilation in adults in the intensive care unit"). This has been demonstrated during OLV in the following studies:

- A randomized trial of 100 patients scheduled for elective lobectomy demonstrated that the incidence of pulmonary dysfunction (defined as PaO_2/FiO_2 less than 300 mmHg, lung infiltrates, or atelectasis) was 4 percent with protective OLV (TV 4 to 6 mL/kg, PEEP 5 cmH_2O, FiO_2 0.5, pressure-controlled ventilator settings), compared with 22 percent with conventional OLV ventilation (TV 10 mL/kg, no PEEP, FiO_2 1, volume-controlled ventilator settings) [52].

- In a randomized trial of 52 esophagectomy patients, a protective ventilation management strategy during OLV (TV 5 mL/kg with PEEP 5) resulted in lower inflammatory markers, higher pO_2/FiO_2 both during OLV and one hour postoperatively, and shorter postoperative mechanical ventilation duration

(115 versus 171 minutes), compared with a conventional ventilation strategy (TV 9 mL/kg with no PEEP) [23].

- An observational cohort study compared 1091 patients before and after implementation of a protective lung ventilation protocol during OLV, which included a smaller TV of 5.3 ± 1.1 versus 7.1 ± 1.2 mL/kg in the historical control group, as well as addition of PEEP and recruitment maneuvers, with limited maximal pressure ventilation [53]. Compared with the historical control group, a lower incidence of acute lung injury (from 3.7 to 0.9 percent), atelectasis (from 8.8 to 5 percent), admission to an intensive care unit (from 9.4 to 2.5 percent), and length of hospital stay (from 14.5 ± 3.3 to 11.8 ± 4.1 days) was noted after implementation of this lung protective ventilation strategy.

MANAGEMENT OF HYPOXEMIA

— The incidence of hypoxemia (oxygen saturation less than 90 percent) during OLV is approximately 5 percent [3,50]. This is most commonly due to shunting; if severe hypoxemia occurs acutely, it

should resolve with the resumption of bilateral ventilation. Because lung expansion may interrupt the surgical procedure, a series of other maneuvers are attempted first if the onset is more gradual (table 1).

Predicting hypoxemia during OLV

— Several factors predict increasing likelihood of hypoxemia during OLV [3,54-56]:

- Low pO_2 prior to OLV
- Left-sided ventilation (due to smaller left lung)
- Higher (normal) FEV_1 (see 'Auto PEEP in obstructive disease' above)
- Supine position as compared with lateral (see 'Physiology' above)

Treatment of hypoxemia

— We suggest the following approach to the management of hypoxemia during OLV (table 1):

- Initial treatment of hypoxemia is to increase the FiO_2 to 100 percent. If this is not effective in bringing the oxygen saturation above 90 percent, temporarily return to two lung

ventilation.

- The flexible intubating scope (FIS) should be used to check the lung isolation device and reposition as needed. Tube malposition (often caused by changes in patient position or surgical manipulation) was responsible for 60 percent of hypoxic episodes during OLV in a large series [50].

- Occlusion of major bronchi by secretions or blood may be diagnosed and treated with the FIS and suction.

- Cardiac output should be optimized. Many patients requiring OLV have poor cardio-respiratory reserve, and low cardiac output may result from hypovolemia, pump failure, or increases in pulmonary vascular resistance due to hypercapnia or auto-positive end-expiratory pressure (PEEP).

- If hypoxemia persists, management of ventilation should be reassessed. We suggest attempting, in the following order:

    - Recruitment maneuvers before, during, and after OLV are effective in improving

oxygenation, lowering shunt, and decreasing dead space; maneuvers include short periods of higher airway pressures and tidal volumes that re-expand atelectatic lung tissue [57-63]. However, recruitment maneuvers can cause lung injury in animal models, so their use must be weighed against the risks of atelectasis [64,65]. (See "Mechanical ventilation during anesthesia in adults", section on 'Recruitment maneuvers'.)

- Increasing PEEP up to 10 cmH_2O to the ventilated lung can help minimize atelectasis and improve oxygenation, especially when low tidal volumes are used [60-62,66]. However, PEEP should be used with caution in patients with obstructive disease since they may have high levels of intrinsic PEEP [67,68]. (See 'PEEP level' above and 'Auto PEEP in obstructive disease' above.)

- Continuous positive airway pressure (CPAP) 5 to 10 cmH_2O to the

nonventilated lung reduces shunt fraction by providing oxygen to the circulation of the nonventilated lung [69]. This is used only in response to hypoxemia, as any flow to the operative lung will cause partial inflation, potentially worsening surgical exposure.

- Insufflation of a low flow of oxygen (3 L/min) via a non-occluding suction catheter in the lumen of the DLT to the non-ventilated lung at the start of OLV has been shown to decrease the incidence of subsequent hypoxemia. It is unclear if this effect is primarily due to absorption of the insufflated oxygen or due to prevention of the passive entrainment of room air into the non-ventilated lung [70].

- High frequency jet ventilation to the nonventilated lung has been used as an alternative to CPAP to improve oxygenation and decrease shunt [71]. A bronchial blocker or the FIS may be used to insufflate oxygen/CPAP into

only the nonoperative lobe(s) of the operative lung to treat hypoxemia [34].

- The nonventilated lung may be intermittently reinflated and ventilated, although this often requires interruption of the surgical procedure.

- Patients who do not tolerate collapse of an entire lung may tolerate collapse of the operative lobe. This can be achieved with a bronchial blocker placed in the bronchus of this lobe [72,73]. (See "Lung isolation techniques", section on 'Quality of lung deflation and reinflation'.)

- The surgeon can decrease shunt by mechanically restricting pulmonary blood flow to the operative lung [74].

Inhaled nitric oxide (NO) as a pulmonary vasodilator has been proposed to increase perfusion of the ventilated lung and thus minimize shunt, but it was ineffective in improving oxygenation in clinical trials [75-77]. The use of nebulized prostacyclin or its derivative iloprost to the

ventilated lung seems to be useful for treatment of hypoxemia during OLV, particularly during TIVA [78].

ANESTHETIC CHOICE

— There is insufficient evidence that choice of anesthetic type (inhalation versus intravenous) affects oxygenation during OLV, or incidence of lung injury; therefore, the decision to use inhalation or intravenous anesthetics should be based on standard considerations unrelated to OLV [3]. Evidence of the impact of adding thoracic epidural to general anesthesia is contradictory in terms of effects on oxygenation and shunt during OLV, and should not impact the decision to use an epidural [79-83].

Intravenous versus inhalation anesthetics

— We typically select a technique based on the potent volatile inhalation anesthetic sevoflurane as the primary agent for maintenance of anesthesia [1], and we often administer supplemental intravenous agents such as opioids, ketamine, and/or a neuromuscular blocking agent (NMBA).

The bronchodilatory and anti-inflammatory effects of potent volatile agents may be advantageous in some patients [12]. However, small studies have noted that arterial blood concentrations of sevoflurane decrease by 26 percent 10 minutes after initiating OLV, without changes in end-tidal concentration [84,85]. Similar results were noted for desflurane, with a 29 percent decrease after 10 minutes [84]. Such reductions in arterial anesthetic concentration are likely due to ventilation-perfusion mismatch in the atelectatic nonventilated lung, as demonstrated by elevation of alveolar-to-arterial oxygen pressure gradients [84,85]. Thus, in patients who exhibit signs of inadequate anesthetic depth after initiation of OLV (eg, increased heart rate and/or blood pressure), it may be prudent to transiently increase the inspired concentration of inhalation agent. Blood concentrations of sevoflurane slowly increase back toward baseline during OLV lasting ≥40 minutes if inspired concentration is maintained at a stable level [84,85]; however, blood concentrations of desflurane remain reduced [84]. (See "Accidental awareness during general anesthesia", section on 'Risk factors'.)

Selection of a total intravenous anesthetic (TIVA) technique is a reasonable alternative to inhalation-based anesthesia. In a 2013 Cochrane review of 20 trials with 850 patients, use of a TIVA technique during OLV did not affect outcomes, compared with an inhalation anesthetic technique [86]. In most trials, there were clinically insignificant differences in oxygenation or shunt fraction [87-91], or in markers of inflammation [92-95]. One randomized trial in 180 patients noted lower mortality and complications with a sevoflurane-based inhalation technique compared with a propofol-based TIVA technique [96]. However, another randomized trial in 460 patients noted that use of a desflurane-based anesthetic was not associated with reduced complications compared with propofol-based TIVA [97]. In a 2017 meta-analysis of patients with COPD, use of dexmedetomidine as the primary or adjunct agent during OLV resulted in improved oxygenation and reduced intrapulmonary shunt compared with placebo (14 trials; 625 patients) [98].

General versus combined thoracic epidural/general anesthesia

— Trials of oxygenation during OLV under general anesthesia (intravenous and inhalation) with and without thoracic epidural anesthesia have reported contradictory results [79-82]. Given the low quality of evidence, and the lack of consistent results, the decision to use an epidural should be based on standard considerations.

RE-EXPANDING THE NONVENTILATED LUNG

— Except in the case of pneumonectomy, re-expansion of the nonventilated lung is necessary to reinflate all atelectatic areas. This is accomplished with a sustained inflation at low levels of positive airway pressure (eg, 20 to 30 cmH_2O for 10 to 15 seconds). Higher pressures (>30 cmH_2O) are avoided due to risk of exacerbation of acute lung injury or creation of new air leaks after pulmonary resection. (See 'Low airway pressures' above.)

Such gently sustained inflations may be repeated for optimal recruitment of lung tissue. If pulmonary resection was performed, sustained inflations also allow detection of bronchial air leaks. When feasible, expansion is accomplished with direct

observation of the lung in an open chest, or on the monitor during video-assisted thoracoscopic surgery (VATS). This ensures gradual but complete recruitment of residual lung tissue with application of the minimum sustained positive pressure that is necessary.

Subsequently, two lung ventilation is managed with a protective ventilation strategy and the minimum FiO_2 that maintains oxygen saturation >93 percent. (See 'Efficacy of lung protective strategies' above and 'Oxygen concentration' above.)

ONE LUNG VENTILATION IN COVID-19 PATIENTS

— Placement of a lung isolation device to achieve OLV is an aerosolizing procedure that typically involves intubation, bronchoscopy, deflating the operative lung, and airway suctioning. Establishing OLV places participating anesthesia personnel at high risk for infection if the patient has novel coronavirus disease 2019 (COVID-19) [99-102].

Key principles for management of tracheostomy, bronchoscopic evaluation, or other pulmonary or tracheobronchial surgery in a COVID-19 patient

include [99-102]:

- Rigorous adherence to the use of personal protective equipment (PPE). (See "COVID-19: Perioperative risk assessment and anesthetic considerations, including airway management and infection control", section on 'PPE during airway management or aerosol generating procedures'.)
- Use of disposable anesthetic equipment when feasible.
- Use of deep neuromuscular blockade to avoid coughing.
- Airway manipulation and bronchoscopy should by performed during apnea.
    - Place the lung isolation device, then immediately confirm its position with flexible intubating scope (FIS)
    - Ensure absence of airflow during apnea (by pausing ventilation and gas flow)
    - Maintain apnea until the lung isolation device is properly positioned and the

anesthesia circuit is reattached

    - Before resuming ventilation, ensure closure of the anesthesia circuit by confirming that all ports are closed and that endotracheal cuffs are adequately inflated

- During subsequent bronchoscopy, insert the FIS through the diaphragm of the bronchoscopy adapter to maintain a closed circuit.

- Rather than clamping one lumen of a double-lumen tube (DLT) and then opening the corresponding bronchoscope port to facilitate lung deflation, consider maintaining a closed port after clamping. An alternative is to place a second viral filtration efficiency (VFE) filter on the end of the open DLT lumen.

- Minimize periods that the patient's respiratory system is open to the atmosphere.

- Avoid or limit the use of suctioning of airway secretions.

- Avoid or minimize coughing during tracheal

extubation. (See "COVID-19: Perioperative risk assessment and anesthetic considerations, including airway management and infection control".)

Further details regarding anesthetic care of COVID-19 patients are available in a separate topic. (See "COVID-19: Perioperative risk assessment and anesthetic considerations, including airway management and infection control".)

SUMMARY AND RECOMMENDATIONS

- **Indications for OLV –** Ventilation of a single lung while allowing the other to collapse is known as one lung ventilation (OLV). Indications for OLV include (see 'Indications' above):
    - Surgical exposure during thoracic surgery
    - Isolation of one lung from the other for the treatment of pulmonary pathology
- **Contraindications for OLV –** Contraindications include dependence on

bilateral mechanical ventilation, and intraluminal airway masses that restrict access to the tracheobronchial tree. (See 'Contraindications' above.)

- **Physiology of OLV –** Physiologic changes caused by differential ventilation of the two lungs include ventilation/perfusion mismatch and hypoxic pulmonary vasoconstriction. (See 'Physiology' above.)

- **Deflating the nonventilated lung –** After placement of a lung isolation device, expedited deflation of the nonventilated lung to improve surgical access can be achieved by one or more maneuvers. (See 'Improving deflation of the nonventilated lung' above.)

- **Lung-protective ventilation strategies –** During OLV, protective ventilation minimizes lung injury while maintaining oxygenation. We suggest using the following combination of ventilation strategies to the ventilated lung (**Grade 2C**) (see 'Lung-protective ventilation strategies' above):

    - Low tidal volume (TV) ventilation (4 to

6 mL/kg)

- Respiratory rate adjusted to maintain end-tidal CO_2 ($ETCO_2$) and arterial carbon dioxide tension ($PaCO_2$) near the patient's baseline
- Positive end-expiratory pressure (PEEP; 5 to 10 cmH_2O), except in patients with severe obstructive disease
- Limited airway pressures (plateau inspiratory pressures <30 cmH_2O), or pressure-controlled ventilation
- FiO_2 below 1 (as needed to maintain oxygen saturation >90 percent)

- **Management of hypoxemia during OLV –** We initially increase oxygen to 100 percent. If this is not effective, we temporarily return to two lung ventilation to bring the oxygen saturation above 90 percent, and then perform the following maneuvers (table 1) (see 'Treatment of hypoxemia' above):
    - Check lung isolation device position

using the flexible intubating scope (FIS), and reposition as indicated

- Suction blood or secretions from major bronchi
- Optimize cardiac output
- Perform recruitment maneuvers on the ventilated lung
- Add 5 cmH_2O PEEP to ventilated side (if not already applied); use with caution in patients with obstructive pulmonary disease
- Add continuous positive airway pressure (CPAP) 5 to 10 cmH_2O to the nonventilated lung
- The nonventilated lung may be intermittently reinflated and ventilated
- The surgeon may consider clamping flow to the operative lung

- **Anesthetic choices –** We typically employ an anesthetic technique based on the potent volatile inhalation anesthetic sevoflurane as

the primary agent for maintenance of anesthesia. Anesthetic choices (eg, inhalation versus intravenous agents, intraoperative use of supplemental epidural agents versus none) do not affect oxygenation during OLV; thus a total intravenous anesthetic (TIVA) technique is also reasonable. (See 'Anesthetic choice' above.)

- **Re-expanding the nonventilated lung –** Re-expansion is necessary to reinflate all atelectatic areas of the nonventilated lung and detect bronchial air leaks. This is accomplished with a sustained inflation at low levels of positive airway pressure (eg, 20 to 30 cmH_2O for 10 to 15 seconds), which may be repeated for optimal recruitment of lung tissue. (See 'Re-expanding the nonventilated lung' above.)

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