Desaturazione e ipercapnia in OLV

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Oxyhemoglobin desaturation during OLV

Definition

SaO2 < 90% ??

It is important to consider the necessary oxygen saturation for each patient (relative hypoxemia rather than a specific oxygen saturation level (absolute hypoxemia))

Chronically depressed oxygen saturations in patients with significant reductions in diffusion lung capacity do not need to be improved upon intraoperatively

Oxygen delivery more important than actual saturation levels in terms of organ perfusion and tissue oxygenation

“satisfactory” SaO2 differs between patients and varies in an individual patient over time
Predictors of intraoperative hypoxemia during OLV

The individual response to OLV (efficiency of HPV) is hard to predict ....

complicated interaction between the patient’s genetic background (HPV response), underlying disease, anesthetic management, and surgical procedure

- Abnormally low arterial oxygen tension (PaO2) in blood gas analysis during the preoperative workup (PaO2 levels during spontaneous ventilation)

or during two-lung ventilation before OLV (a reliable indicator of abnormal lung function)

**Distribution of perfusion** between the two lungs
the less the perfusion of the nonventilated lung is, and the more the perfusion of the ventilated lung is,⇒ the higher the PaO2 is during OLV
Predictors of Hypoxemia during OLV

Side of Operation
right lung is larger than the left lung $\Rightarrow$ oxygenation during OLV may be better during left thoracotomy

Underlying Lung Function Abnormalities: basal spirometry
Fev1 ?? not always predictive
the less the forced expiratory volume was in 1 s, the better the oxygenation during OLV (paradoxical relation)

....... air trapping in the ventilated lung may generate auto–positive end-expiratory pressure (PEEP) during OLV, thus decreasing the likelihood of atelectasis in the ventilated lung and improving oxygenation
Also, air trapping in the nonventilated lung may delay the onset of desaturation
Predictors of hypoxemia during OLV

**position and the size of the tumor** (influence on Ventilation and perfusion, and consequently shunt)

Patients with large central tumors undergoing pneumonectomy or lobectomy will most probably have less perfusion to the operated non-ventilated lung (the lung is already partially preoperatively excluded ⇒ less intraoperative shunt like ) as compared with patients with peripheral masses.

large central tumors ⇒ much better oxygenation during OLV than those presenting for peripheral masses.

Sometimes..viceversa
gravity affects perfusion of the ventilated and nonventilated lung during OLV

in the supine position, gravity affects both lungs equally

in the lateral decubitus position gravity leads to a better perfusion of the lower, ventilated lung than of the upper, nonventilated lung

the semilateral... some perfusion of the NON ventilated lung remains (increased shunt)
Causes of hypoxemia during OLV

Tracheal-bronchial tube malposition

Adhesions, incomplete non-dependent lung collapse (partial ventilation maintains distension of extra-alveolar pulmonary vessels that oppose hypoxic pulmonary vasoconstriction in the non-ventilated lung)

Abnormal increase in intrathoracic pressure and PVR
Lung de-recruitment in the ventilated lung
Low mixed venous oxygen saturation secondary to low cardiac output

severe unbalance between oxygen consumption, and oxygen delivery
Excessive fluid administration
Where does (excess) fluid go in OLV?

due to HPV and pre-capillary vasoconstriction, the hypoxic non-dependent lung has a lower capillary pressure and less tendency to accumulated interstitial fluid

an excess of intravenous crystalloids (increased intracapillary hydrostatic pressure) goes across the endothelial capillary barrier and can rapidly cause desaturation of the pulmonary venous blood draining the dependent lung
Hypoxemia during OLV
Atelectasis in the ventilated dependent lung

compression caused by the weight of the mediastinum;
compression by abdominal contents, diaphragmatic muscle relaxation, diaphragm displacement

increased closure of small airways with old age, reduced elastic recoil, and the lateral decubitus rather than the erect position
occlusive secretions, blood

small tidal volumes, especially during high FIO2, may lead to more atelectasis and poor oxygenation

(very) prolonged OLV may cause fluid to trasudate into the dependent lung… this causes decrease in lung volumes and increases airway closure
Hypoxemia during OLV

Severe hypovolemia with reduction in CO and SvO2

Air trapping and high PEEP

Marked increase in CO $\Rightarrow$ Pa pressure, which increases perfusion of the non-ventilated lung $\Rightarrow$ increase in shunt fraction
hypoxemia in OLV

traditional teaching to prevent hypoxemia in OLV…

non-dependent lung compression to attenuate ventilation–perfusion mismatch by redirecting blood to the ventilated, dependent lung

However… compression may increase RV afterload, ⇒ reduce CO ⇒ reduce tissue oxygen delivery and lead to hypoxaemia
relationship between cardiac output and hypoxaemia

Various factors:

- arterial oxygen content \((CaO_2)\)
- capillary oxygen content \((CcvO_2)\)
  - shunt fraction \((Qs/Qt)\),
  - oxygen consumption \((VO_2)\),
  - cardiac output \((Qt)\)

Suboptimal cardiac output can result in increased peripheral O2 “extraction, and low venous oxygen content \((decreased \text{ PvO}_2)\).

In the presence of a massive venous admixture in precapillary bed… hemoglobin saturation may not be complete in the end-capillary blood.

If significant intrapulmonary shunt: a great amount of desaturated venous blood will mix with oxygenated blood beyond the alveoli.
unrecognised factor for postoperative hypoxemia (suctioning and post.op edema)

severe hypoxemia and pulmonary edema in the non-ventilated caused by excessive suction via the bronchial blocker lumen with the cuff inflated

suction can result in excessive negative pressure within the lung resulting in excessive congestion of the lung and fluid “extravasation” (unilateral pulmonary edema)

Also, prolonged suctioning via the tracheal lumen during OLV whenever ventilation is interrupted for a prolonged period
Thoracic epidural and anesthesia
no inhibition of HPV, incidence of hypoxemia in OLV
comparable with general anesthesia alone
central and systemic hemodynamics maintained

Effects of thoracic epidural anesthesia on pulmonary venous admixture and oxygenation with isoflurane or propofol anesthesia during one lung ventilation

Reda S. Abdelrahman

The Effect of Thoracic Epidural Anesthesia on Pulmonary Shunt Fraction and Arterial Oxygenation During One-Lung Ventilation

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What “minimum” Hb-oxygen desaturation would be acceptable?

Systemic and pulmonary effects of hypoxemia .... individual susceptibility

impact on vital non pulmonary organ function may be variable.... but sometimes significant due to the increasing rate of co-morbid conditions in thoracic patients

Optimal SaO2 : stable saturation level above 92–94%.

Still acceptable : around 90%

Dangerous (?) : permanently <90%
What are the potential risks of hypoxemia? Is hypoxemia equally tolerated by different organ systems?

Different organ systems may have different degrees of tolerance to hypoxemia, some organs are more sensitive to the effects of hypoxia than are others.

The healthy brain and healthy systemic organs can generally tolerate hypoxemia better than diseased organs.

Even if systemic perfusion is well preserved, intra-organ perfusion dishomogeneities may be responsible for unpredictable injury from hypoxia.
Hypoxia does not affect all parts of the body equally

Of all the body organs, some parts of the brain (cortical pyramidal neurons, cerebellar Purkinje cells, hippocampal CA1 pyramidal neurons and subpopulations in the amygdala, striatum, thalamus and brainstem nuclei) and the ..... need more oxygen to function normally than do other parts, and are accordingly more sensitive to hypoxia.
Hypoxia does not affect all parts of the body equally

So increasingly severe hypoxia first causes malfunction, and then failure of those parts of the brain and eyes consuming more oxygen

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Survival Time (Minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrum, small pyramidal cells</td>
<td>8</td>
</tr>
<tr>
<td>Cerebellum, Purkinje’s cells</td>
<td>13</td>
</tr>
<tr>
<td>Medullary centers</td>
<td>20–30</td>
</tr>
<tr>
<td>Spinal cord</td>
<td>45–60</td>
</tr>
<tr>
<td>Sympathetic ganglia</td>
<td>60</td>
</tr>
<tr>
<td>Myenteric plexus</td>
<td>180</td>
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</tbody>
</table>

Brain activity under different SpO2

- **SpO₂ (%)**
  - 100: Normal brain function
  - 80: Increasing degrees of brain dysfunction (prefrontal, cerebellar, muscle paralysis)
  - 60: Total muscle paralysis results in apparent unconsciousness
  - 40: Unconsciousness and eventual death
  - 20: Hypoxia & Brain Function

(c) G.M. Woerlee
Risk of cerebral hypoperfusion during OLV even in the absence of severe Hb desaturation

Many factors leading to right ventricular and subsequently left ventricular dysfunction

an increase in right-sided filling pressures could increase cerebral venous blood pressure and volume and affect cerebral saturation (Stagnant hypoxia)

Given the lack of correlation between peripheral and cerebral saturation, ….increased cerebral desaturation with relatively low SaO2 would go undetected and put patients at further risk of non-pulmonary complications

Low cerebral saturation correlated with postoperative delirium and postoperative cognitive deficit
Thoracotomy is reported to be associated with a very high incidence of cerebral desaturation when one-lung ventilation (OLV) is instituted.

We observed a decrease in cerebral oxygen saturation in all patients, whether they were undergoing open procedures or video-assisted thoracotomy.

In many cases pulse oximetry indicated that arterial oxygen saturation remained within clinically acceptable levels.
possible causes: a reduction in cerebral oxygen delivery that could be due to a reduction either in arterial oxygen content or in cerebral blood flow that might also be related to a decrease in cardiac output.

there is the potential of central venous pressure increases during OLV, which could result in increased back pressure for the cerebral venous circulation.

Given the recommended implementation of lower inspired concentrations of oxygen during thoracic surgery,… this recommendation needs to be weighed against the evidence of cerebral desaturation also with a FIO2 of 1.0 during OLV.
monitoring cerebral desaturation in patients at risk of severe hypoxemia during OLV?

cerebral intraoperative episodes of oxygen desaturation has been shown to be associated with postoperative Neurocognitive dysfunction.

Standard pulse oximetry is insufficient to detect a reduction in arterial oxygen content, and oxygen delivery (cardiac output) and an increase in metabolic consumption.

monitoring cerebral oxygenation (rSO2) using near infrared spectroscopy in patients who required OLV for thoracic surgery?

detecting cerebral desaturation (rSO2 monitoring) might be useful and allow for early intervention during OLV.
Intraoperative hypoxaemia and clinical outcome:
an increased risk of complications such as myocardial dysfunction, atrial fibrillation, renal failure, and pulmonary hypertension.

Hypoxemia may increase renal vascular resistance, leading to renal hypoperfusion and a decrease in glomerular filtration rate.

Intraoperative hypoxemia can lead to long-term cognitive deficits and structural neurologic damage.

If need for FiO2 = 1 to compensate for severe desaturation
⇒ high risk of absorption atelectasis in the ventilated lung.
Hypoxemia and myocardial function

acute hypoxic exposure is generally associated with increased heart rate, decreased stroke volume, increased indices of LV systolic function, maintained indices of RV systolic function, ....and altered diastolic filling patterns of both ventricles

hypoxia-induced sympathetic nervous system activation preserves LV contractility… but induces an abnormal LV filling pattern with decreased early filling and greater contribution of atrial contraction

hypoxemia can increase pulmonary artery pressure (↑ PVR through hypoxic pulmonary vasoconstriction)
Hypoxia, PVR and RV function

Development of hypoxic compartments in the dependent lung increases its PVR (HPV), thereby decreasing dependent and increasing non-dependent lung blood flow. The acutely hypoxic right ventricle remains able to preserve the coupling of its contractility to increased afterload (transiently).

However, it then limits its pump function because of decreased oxygen availability, right-ventricular dysfunction possible in right coronary artery disease and in COPD patients.
Hypoxia-induced lung injury

although alveolar epithelial cells can adapt to hypoxia, and exposure to low oxygen concentrations may be protective… Hypoxic alveolar cells develop alterations in cellular function that can impact clinical outcomes

Hypoxia appears to disturb the cell cytoskeleton in alveolar epithelial cells, induces release of inflammatory mediators and increase permeability of the epithelial barrier.

Epitelial cells may undergo apoptosis at an accelerated rate under hypoxic stress

Reduction in surfactant production

reduced LUNG EDEMA CLEARANCE

During acute hypoxemic respiratory failure, alveoli flood with edema, thus impairing the transfer of oxygen from the airspaces into the pulmonary circulation.
Is hypoxia-induced OLV always preventable?

On an individual basis.....complex physiology, diversity in the underlying conditions, and still inconclusive studies make it difficult to define which combination of tidal volume, respiratory rate, and PEEP will decrease the likelihood of desaturation during OLV

How to perfectly modulate Rv function/pulmonary circulation.... and completely prevent-eliminate atelectasis in the ventilated lung...and preserve its perfusion... remains a matter of debate and study
Deliberate permissive Hypoxia in OLV?

Prolonged OLV with high FiO2 harmful for lung epithelial cells. Potentially dangerous levels of FiO2 may be required in severe lung disease to maintain SaO2 >90%.

PERMISSIVE HYPOXEMIA \(\Rightarrow\) Valuable strategy to reduce high inhaled FiO2?

Aiming at a level of oxygen delivery that is adequate to avoid tissue hypoxia may minimize the detrimental effects of the often toxic (↑RL) ventilatory support required to maintain normal arterial oxygenation.

goal-oriented OPTIMIZATION of cardiac output and, if necessary, hemoglobin concentration, to compensate for hypoxemia and maintain a normal value of oxygen delivery.
Deliberate permissive Hypoxia in OLV

Strategy focused primarily on the adequacy of tissue oxygenation rather than arterial oxygenation. The targeted SaO2 may be decreased as long as global DO2 can be maintained (careful balance between the target SaO2 and the ventilatory toxicity required to achieve a higher SaO2).

Organs function more likely to be determined by the amount of oxygen that actually reaches the tissues rather than the amount that circulates “free” in the blood.

Active manipulation of cardiac output and hemoglobin concentration (if necessary) to compensate for hypoxemia and maintain a normal (but not supranormal) value of tissue DO2 (preload optimization, O2 content, inotropic agents).
Deliberate permissive hypoxemia and Hb level

The adoption of a restrictive transfusion policy does not support the maintenance of permissive hypoxemia. A higher transfusion threshold is likely to be more appropriate.

Low Hb levels are associated with an increased oxygen extraction and an increased Hb-desaturation in the venous pool (inhibition of HPV by "very" low PVo2).

A sufficient amount of RBC is necessary to remove NO and adenosine, important factors that inhibit HPV.

Cardiac output augmentation improves the balance between organ DO2 and organ oxygen consumption, despite the presence of hypoxemia. Tissue hypoxia may not occur during hypoxemia if cardiac output is adequately increased.
↑ CO or ↑ SaO2 ??

A patient with an SaO2 of 80% and a Hb of 10 g/dL would still have normal DO2 if cardiac index is increased to 4.5 L/min/m2 (calculated DO2 is 480 mL/min/m2, normal value is 400–650).

On the contrary, another patient who has a cardiac index of 3 L/min/m2 and a Hb of 10 g/dL may suffer from tissue hypoxia despite having an SaO2 of 90% (calculated DO2 is 360 mL/min/m2).

Increasing the cardiac index from 3 L/min/m2 to 4.5 L/min/m2 would increase tissue DO2 much more than raising SaO2 from 80% to 90%.

Improving oxygen supply to hypoxic tissues through increase in cardiac output increases mixed venous oxygen saturation (↑ PvO2 if constant tissue oxygen extraction).

(↑SvO2 improves “saturation capacity” of capillary bed and ↑ SaO2)
Treatment of Hypoxemia in OLV

Hypoxemia during OLV: $\text{SpO}_2 < 90\%$

- **Increase FiO$_2$ to 100%**

**Life threatening ($\text{SpO}_2 < 90\%$)**
- and/or occurrence of arrhythmia and/or ST changes

**Stop surgery**
- Resume bipulmonary ventilation

**Treatable cause**
- **Fiberoptic bronchoscopy**
  - DLT position
  - Secretions/blood
- **Hemodynamic**
  - Low blood pressure
  - Too deep level of anesthesia
  - Blood loss
  - Right ventricular dysfunction

**Optimize ventilation**
- **Nonventilated lung**
  - Manual re-expansion (O$_2$ 100%)
  - CPAP (O$_2$ 100%)
- **Ventilated lung**
  - PEEP
  - Recruitment maneuver
  - iPEEP evaluation

**Non-life threatening ($\text{SpO}_2 > 90\%$)**
- Continue OLV

**Improve oxygenation**

**Optimize perfusion**
- Decrease shunt
  - IV Nitric oxide
- Surgical lung compression
- Pulmonary artery clamp

**Improve ventilated lung perfusion**
- Pressure-controlled ventilation
- Inhaled nitric oxide, PGI$_2$
Hypercapnia in OLV

Unpredictable consequence of Reduction in tidal volumes and ventilatory pressures, which allow to minimise mechanical stress and secondary volu- or barotrauma

However…if protective ventilation beneficial…. low tidal volume ventilation has the potential to worsen oxygenation, either due to lung de-recruitment with inadequate PEEP or due to pulmonary blood flow diversion with excessive PEEP

Low tidal volume ventilation increases dead-space… and CO2 elimination is therefore consistently impaired
Protective lung ventilation and hypercapnia

Once… in patients undergoing lung volume reduction surgery for advanced emphysema, permissive hypercapnea was used electively as part of a barotrauma avoidance strategy.

Nowadays..permissive hypercapnia considered a routine component of a PLV strategy for OLV (?)

Protective ventilation is not synonymous with low tidal volume ventilation, but includes all of: routine “appropriate” PEEP, lower FiO2, frequent recruitment … and particularly lower ventilator pressures through the acceptance of permissive hypercapnia in some circumstances.
Elevated CO2 level may be beneficial in OLV?

Moderate hypercapnia with lower tidal volumes and only marginal rate compensation potentiates the HPV response and is therefore unlikely to adversely affect oxygenation.

Hypercapnia causes coronary vasodilatation, which may be of further benefit during the period of hypoxemia.

**Systemic circulation and myocardial function:**

An increase in CO, SV, HR, SBP, DBP, and MAP has been observed with a slight reduction in SVR.

The direct effect of hypercapnia on the heart and vascular smooth muscle is to reduce contractility. However, these direct effects are opposed by a neurohumeral effect, thus resulting in an increase in sympathomimetic output.

This leads to an increase in HR, systemic vasodilatation, and decrease in left ventricular afterload, which results in an increase in CO.
Hypercapnic acidosis and oxygenation

HCA can improve tissue oxygenation:

Potentiation of HPV response

rightward shift of the oxyhemoglobin dissociation curve which decreases the affinity of hemoglobin for oxygen and facilitates oxygen release to the tissues (the Bohr effect)

HCA causes vasodilatation in microvessels, promoting oxygen delivery and tissue perfusion

owing to the combined effects of increased sympathetic tone, and enhancement of venous return by the lower mean intrathoracic pressure, and reduction in SVR…

hypercapnia usually improves CO

Hypercapnic acidosis may have beneficial effects during prolonged hypoxia by preserving pulmonary capillary integrity
Possible benefit of hypercapnic acidosis in ventilation associated lung injury

Review

**Bench-to-bedside review: Permissive hypercapnia**
Donall O’ Croinin¹, Martina Ni Chonghaile², Brendan Higgins³ and John G Laffey⁴

Effects of hypercapnia and hypercapnic acidosis on attenuation of ventilator-associated lung injury

N. M. ISMAIEL ¹, ², D. HENZLER ¹, ²

attenuation of pulmonary inflammation, reduction of apoptosis in alveolar epithelial cells, improvement in sepsis-induced ALI and protective effects on other organ systems,
Potential benefit of hypercapnic acidosis on the epithelial cells

**Hypercapnic acidosis** was demonstrated to attenuate the increases in lung permeability seen following free radical, ischaemia/reperfusion, and ventilator-induced ALI.

**Hypercapnic acidosis** appears to attenuate the production of higher oxides of nitric oxide, such as nitrite and nitrate, following both ventilator-induced and endotoxin-induced ALI.

**Hypercapnic acidosis** attenuates pulmonary apoptosis following pulmonary ischaemia/reperfusion (Laffey et al: Therapeutic hypercapnia reduces pulmonary and systemic injury following in vivo lung reperfusion. *Am J Respir Crit Care Med* 2000, 162:2287-2294)
in a cohort analysis by Licker et al. permissive hypercapnia has been shown to reduce the incidence of postoperative ALI

Conclusions: Implementing an intraoperative PLV protocol in patients undergoing lung cancer resection was associated with improved postoperative respiratory outcomes as evidenced by significantly reduced incidences of acute lung injury and atelectasis along with reduced utilization of intensive care unit resources.
potentially **therapeutic effects** of hypercapnic acidosis in different models of lung injury

Hypercapnia reduces the production of superoxide and other free radical compounds in these cells.

Therapeutic hypercapnia protects against pulmonary inflammation and apoptosis, preserves lung mechanics.

Hypercapnic acidosis is therapeutic for lung injury; buffering the acidosis worsens lung injury.

Hypercapnic acidosis improves the outcome of ventilator-induced lung injury.

Hypercapnic acidosis attenuates lung injury by reducing inflammation via inhibition of NF-κB activity.

Hypercapnic acidosis attenuates lung injury by reducing the production of reactive nitrogen species and neutrophil infiltration.

Hypercapnic acidosis attenuates injury in early and prolonged septic lung injury.
Potentially **harmful consequences** of permissive Hypercapnia during OLV

The potentiating effect of hypercapnia on the hypoxic response of pulmonary vasculature may cause a marked increase in pulmonary vascular resistance

an enhanced HPV not only affects the non-dependent extraalveolar vessels..... but also the “non-ventilated” areas of dependent lung

The “net” effect on PVR of dependent lung will determine both RV afterload and increase in Pap

hypercapnic acidosis (deliberate hypoventilation) may attenuate the component of injury that is due to lung stretch..... but not that due to repeated collapse and re-expansion of non-ventilated lung (protective only in the dependent ventilated lung)
Permissive (??) hypercapnia in OLV

Permissive hypercapnia should probably be used with caution in patients with heart disease and is contraindicated in those with elevated intracranial pressure (cerebral vasodilation yielding increased intracranial pressure).

The increased discharge of the sympathetic nervous system has a pro-arrhythmic effect.

An increase in cardiac output (hypercabic acidosis-induced) not paralleled with an increase in alveolar ventilation may increase the shunt fraction.
Permissive Hypercapnia Impairs Pulmonary Gas Exchange in the Acute Respiratory Distress Syndrome

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amount as with hypercapnia. Permissive hypercapnia increased $Q$ by an average of 1.4 L · min$^{-1}$ · m$^2$, decreased arterial oxygen tension from 109 ± 10 mm Hg to 92 ± 11 mm Hg ($p < 0.05$), markedly increased true shunt ($Q_s/Q_T$), from 32 ± 6% to 48 ± 5% ($p < 0.0001$), and had no effect on the dispersion of $V_A/Q$. On reinstatement of baseline $V_T$ with maintenance of a high $Q$, $Q_s/Q_T$ remained increased, to 38 ± 6% ($p < 0.05$), and $P_{aO_2}$ remained decreased, to 93 ± 4 mm Hg ($p < 0.05$). These results agreed with effects of changes in $V_T$ and $Q$ predicted by the mathematical lung model of the MIGET. We conclude that permissive hypercapnia increases pulmonary shunt, and that deterioration in gas exchange is explained by the combined effects of increased $Q$ and decreased alveolar ventilation.
Potentially harmful consequences of permissive Hypercapnia during OLV

Deliberate hypoventilation with “deliberate” hypercapnia (improper PEEP, no recruitment, etc) may decrease lung compliance… and then

high Vt OLV needed to increase lung compliance and increase dependent blood flow

Figure 4 Increased PVR at extremes of lung volumes. This figure
potentially **harmful effects** of hypercapnic acidosis in different models of lung injury

Hypercapnic acidosis worsens lung injury by increasing the production of NO and inflammation and causes severe hypotension.

Hypercapnic acidosis injures alveolar epithelial cells by increasing the production of NO.

Hypercapnic acidosis reduced plasma membrane wound repair in alveolar epithelial cells.

Hypercapnia impairs alveolar epithelial cell function by causing the endocytosis of Na+/K+ ATPase.

Hypercapnic acidosis worsens ALI by exacerbating pulmonary bacterial infection via its immunosuppressive properties.
Permissive hypercapnia in OLV

At which level CO2 accumulation may induce excessive sympathetic stimulation, cardiac rhythm disturbances and/or cardiac dysfunction?

Individual tolerance, duration of acidosis, depth of anesthesia, neuroendocrine “stress”, buffering manoeuvres….

Assuming a reasonable cardiovascular reserve, and in particular right ventricular function, PaCO2 levels up to 70 mmHg are well tolerated in the short term and clearly beneficial in terms of lung injury avoidance and attenuation

Higher levels (?) should be avoided due to the risk of hemodynamic negative effects
CO2 management in Severe pulmonary disease

In cases of severe lung disease with respiratory acidosis, variable pulmonary hypertension or right ventricular dysfunction, increase in cardiac output (CO2-mediated) no longer sustains pulmonary vasoconstriction

pH, per se, and not CO2 seems to be responsible for the impaired RV function

“protective” low-tidal volume – high rate ventilation should be abandoned.....in favor of higher tidal volume ventilation (or even change from OLV to TLV) at a lower respiratory rate to maximize CO2 elimination

acceptable balance between ⇒ potential risk of ALI vs imminent risk of hemodynamic dysfunction (excessive sympathetic stimulation, cardiac rhythm disturbances and/or cardiac collapse)
Conclusion

Mild hypoxemia and hypercarbia are well tolerated in OLV, provided they are not determined by severe atelectasis, low cardiac output, or tube malposition.

Protective lung ventilation is not simply low tidal ventilation.

Increasing FiO2 in the ventilated or adding 100% oxygen to the non-dependent lung, by apparently “solving” an immediate desaturation episode, may instead mask an underlying incautious OLV conduction.
Merry Christmas and happy new year