Acute Permissive Hypercapnia during One Lung Ventilation: Impact on Right Ventricular Function during Lung Resection

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Abstract

Background: In a quasi-experimental design study, the haemodynamic and gas exchange effects of acute permissive hypercapnia and its effect on right ventricle during one-lung ventilation in 15 thoracotomy patients who had lung resection, were investigated.

Methods: Hypercapnia was induced by decreasing tidal volume until PaCO$_2$ increased to 60-70mmHg.

Results: Cardiac index (from 2.93 ±0.44 to 3.37±0.54l min$^{-1}$ m$^{-2}$, p-value <0.001), tricuspid annular plane systolic excursion (2.16±0.15 to 2.4±0.17cm, p-value <0.001) and right ventricular myocardial performance index (0.319 ±0.01 to 0.33±0.0, p-value <0.001) increased with hypercapnia. Systemic and pulmonary oxygenation improved.

Conclusion: Permissive hypercapnia as a component of OLV management is likely to be beneficial in selected patients. PaCO$_2$ levels up to 60-70mmHg are likely to be tolerated in the short-term and clearly beneficial in terms of lung injury attenuation.

Key Words: Permissive hypercapnia – One lung ventilation – Right ventricle.

Introduction

GALE and waters first described selective ventilation of one lung was in 1931 and quickly led to increasingly complex lung resection surgery, with the first published pneumonectomy for cancer in 1933 [1]. One-Lung Ventilation (OLV) in patients undergoing pulmonary resection is challenging and fraught of many complications. One of these complications is increased air way pressure of the dependent lung with potential risk of barotrauma [2]. During OLV, tidal volume (Vt) is frequently maintained at the same level as during two-lung ventilation without positive end-expiratory pressure, targeting normalization of partial carbon dioxide pressure (PaCO$_2$) [3]. This maintenance corresponds to high-volume ventilation with potentially deleterious effects, even for a period of <90min [4]. Normalizing of CO$_2$ at the expense of inducing undue lung stretch may not be appropriate.

In mechanically ventilated intensive care unit patients reduced Vt with an associated increased CO$_2$ (permissive hypercapnia) has become an accepted practice [5]. Michelet et al., [6] demonstrated that the protective ventilatory strategy based on the reduction of Vt is beneficial during OLV. Although the effect of permissive hypercapnia on hemodynamics and Right Ventricular (RV) function was previously reported in patients with ARDS [7], the effects of acute controlled hypercapnia on RV function during one-lung ventilation have not yet been investigated systematically.

The goal of this study is to detect the impact of acute permissive hypercapnia during one lung ventilation on right ventricular function during lung resection surgeries.

Patients and Methods

The present study included 15 patients scheduled to undergo pulmonary resection in Cairo University Hospital in 2012. The study was done after approval by local ethics committee and obtaining a written informed consent. The Clinical Trials. Gov Identifier. of the study is NCT025-19517.

Inclusion criteria:

Adult patients (>18yr.) scheduled to have elective primary pulmonary resection through thoracotomy with one lung ventilation.
Exclusion criteria:

Excluded from the study were patients with Systolic Pulmonary Arterial Pressure (SPAP) more than 50mmHg, patients with symptoms of intracerebral haemorrhage, those with pre-existing hypercapnia (PaCO₂ >45mmHg), Co-existing metabolic acidosis (pH <7.36, BE <-4), or patients with ischemic heart disease, patients with predicted postoperative forced expiratory volume in the first second (FEV₁) less than 800ml or less than 40% of the predicted in pneumonectomy patients. Also, patients in which TEE was contraindicated or failed to reveal necessary measurements were excluded from the study.

Anesthesia management:

Patients were monitored for temperature, invasive arterial blood pressure, 5-lead electrocardiogram, peripheral oxygen saturation, end-tidal carbon dioxide tension, hourly urinary output, and Central Venous Pressure (CVP). All patients underwent a comprehensive Transesophageal Echocardiography Examination (TEE) according to the American Society of Echocardiography/Society of Cardiovascular Anesthesiologists (ASE/SCA) guidelines [8] by anesthetists who are certified in adult transesophageal echocardiography by an international body.

Anesthesia was induced with intravenous propofol (2mg/kg), fentanyl (2ug/kg), and atracurium (0.5mg/kg) to facilitate endotracheal intubation and was repeated intraoperatively as required to maintain muscle relaxation. When adequate relaxation was achieved, a left-sided Double-Lumen Endotracheal Tube (DLT) was inserted via direct laryngoscopy and Fibre-Optic Bronchoscopy (FOB) confirmed its correct placement.

After intubation, two-lung ventilation was initiated in the volume-controlled mode (25% inspiratory time, 10% pause) with constant inspiratory flow (DatexOhmeda (GE medical system, Milwaukee, Wisconsin, 53201 USA). Anesthesia was maintained using isoflurane (1-1.5 expired MAC) in 100% oxygen Initial tidal volume of 8ml/kg was used with 3cmH₂O of PEEP. Tidal volume was reduced to 6ml/kg after switching from two-lung to one-lung ventilation and the rate adjusted to maintain arterial PaCO₂ between 30 and 35 mmHg. After establishing OLV with normocapnia (OLV-N) for 15min., hemodynamic, respiratory variables and echocardiographic data were obtained (T₁). Hypercapnia was then induced by decreasing tidal volume to reach a PaCO₂ between 60 and 70 mmHg with a pH >7.1 (OLV- H) and was allowed to settle for another 15min (T₂) for data collection. Afterwards, T₃ was increased to restore PaCO₂ back to baseline values (OLV-N) and after another 15min hemodynamic, respiratory and echocardiography measurements were recorded (T₄). PaCO₂ was considered stable when the fluctuations within the 15min were less than 5%.

All patients had lung resection via a lateral thoracotomy through the 5th intercostal space. The nondependent, operative lung was collapsed when required by the surgeon.

TEE data:

1- Cardiac Output (CO): Cardiac output (ml/min) was calculated by transesophageal echo-Doppler as follows:

\[
\text{CO} = \text{Stroke volume} \times \text{heart rate}.
\]

Stroke Volume = Stroke Distance (VTI) X Cross Sectional Area (CSA).

Cardiac index = CO / Body surface area.

VTI was calculated by tracing the continuous wave Doppler (CW) spectral display of the flow across the aortic valve from TG long axis or deep transgastric long axis view while CSA of the aortic valve was estimated in the midesophageal short axis view of the aortic valve by planimetry of an equilateral triangle orifice observed in mid-systole [9].

2- RV systolic pressure. For quantitative assessment of RVSP, the peak Tricuspid Regurgitant (TR) jet velocity, obtained on continuous wave Doppler, was used to calculate peak pressure gradient between the RV and RA during systole using the modified Bernoulli’s equation, AP = 4V². RVSP was then calculated by adding measured CVP to the measured gradient [10].

3- Tricuspid Annular Plane Systolic Excursion (TAPSE): TAPSE measures the longitudinal systolic motion of the free edge of the tricuspid valve annulus in the mid-esophageal four-chamber (ME 4Ch) view, typically on the lateral annulus [11].

4- Right Ventricular Myocardial Performance Index (RVMPI), Tei index: The RVMPI has been described as a nongeometric index of global ventricular function. First, tricuspid diastolic inflow velocity profile was recorded from the ME 4Ch view with the Pulsed-Wave (PW) Doppler sample volume positioned at the tips of the tricuspid leaflets. Then the RV outflow velocity was recorded from the mid-esophageal ascending aorta short axis view with the PW Doppler sample volume positioned just below the pulmonary valve.
The tricuspid closure time (a-interval) was measured from the end of TV diastolic inflow Doppler display to the onset of the next inflow profile. The Right Ventricular Ejection Time (RVET) (b-interval) was measured from the start to the end of the RV outflow Doppler velocity profile. Mean values were obtained by averaging five beats [12] Fig. (1). The “a” interval equals the sum of Isovolumetric Contraction Time (ICT), Ejection Time (ET), and Isovolumetric Relaxation Time (IRT). The sum of ICT and IRT was obtained by subtracting “b” from “a”. The index of RV global function was calculated as

\[ RVMPI = \frac{a-b}{b} = \frac{ICT + IRT}{ET} \]

\[ \text{Index} = \frac{(a-b) \cdot (ICT + IRT)}{b} = \frac{ICT + IRT}{ET} \]

\[(a - b) = ICT + IRT\]

Tricuspid inflow

ICT = (a - b) – IRT

ET

IRT = (c - d)

Fig. (1): Transthoracic echocardiography schema of Doppler intervals. Index [(a-b)/b] is calculated by measuring two intervals: (1) A is interval between cessation and onset of tricuspid inflow and (2) B is ejection time (ET) of right ventricular (RT) outflow [12].

Data collection:

At the 3 time points, the primary outcome to be measured was the change in cardiac index as the level of PaCO₂ increased from 35 to 60mmHg. Heart rate, systolic blood pressure, and Central Venous Pressure (CVP), arterial blood gas analysis, central venous oxygen saturation (ScVO₂), Peak Inspiratory Pressure (PIP), RVSP, TAPSE and RVMPI were also recorded.

Statistical analysis:

Power analysis was based on a previous study demonstrating a progressive increase in cardiac index by 15% as the level of PaCO₂ increased from 35 to 60mmHg [13]. This analysis revealed that at least 13 patients will provide a power more than 0.9 and error 0.05. Data were analysed using one-way repeated measures ANOVA, with post hoc Dunnet’s test for comparisons against baseline values. A p-value <0.05 was considered statistically significant. The software SPSS V 17.0 for windows (SPSS, Inc., Chicago, IL) was used for statistical analysis.

Results

Fifteen patients completed the study. All patients had an uneventful course during surgery and none of them suffered complications as a result of the study (Table 1).

Cardiac index significantly increased during the period of OLV-H compared to OLV-N [3.3 (0.44) vs. 2.9 (0.54) L/min/m², p<0.001] and the significant increase remained even after restoring normocapnia during OLV-N₁ [3.3 (0.49) vs. 2.9 (0.54) L/min/m², p<0.001]. Similarly, cardiac output increased during both OLV-H and OLV-N₁ compared to OLV-N [6.5 (1.3), 6.3 (1.3), and 5.6 (1.2) L/min respectively, p<0.001]. Both TAPSE and RVMPI significantly increased during OLV-H and OLV-N₁ compared to OLV-N. TAPSE increased from 2.16 (0.15) cm during OLV-N to 2.4 (0.17) cm during OLV-H, p<0.001 and during OLV-N₁ it was 2.35 (0.14) cm, p<0.001. In the same context RVMPI significantly increased at the same time points [OLV-H, 0.32 (0.01), p<0.001 and OLV-N₁, 0.33 (0.15), p<0.003] compared to the initial reading OLV-N [0.32 (0.01)] (Table 2).

Of the hemodynamic measurements, heart rate and systolic blood pressure significantly increased during OLV-H compared to OLV-N. Heart rate increased from 81 (6.4) bpm to 98 (8.1) bpm, p<0.001. Systolic blood pressure also increased from 123 (6.6) mmHg to 130 (6.0) mmHg, p=0.001. On returning to OLV-N₁ heart rate remained significantly high 94 (7.4) bpm, p<0.001 while systolic blood pressure readings were comparable. Other hemodynamic, arterial blood gas analysis and respiratory measurements are all shown in (Table 3).

Table (1): Demographic characteristics of study participants. Data are presented as mean ± SD, frequency as appropriate.

| Age (yr) | 53±9 |
| Weight (kg) | 77±15 |
| Height (cm) | 170.5±9.5 |
| Male/Female | 9/6 |
| BSA (m²) | 1.92±0.23 |

BSA: Body Surface Are.

205
Table (2): Echocardiographic parameters under normocapnic (OLV-N) and hypercapnic (OLV-H) one-lung ventilation. Data are presented.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>OLV-N</th>
<th>OLV-H</th>
<th>OLV-N1</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO (ml/min)</td>
<td>5625±1233</td>
<td>6507±1319</td>
<td>692±1325</td>
<td>Overall: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: &lt;0.001</td>
</tr>
<tr>
<td>CI (L/min/m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>2.93±0.44</td>
<td>3.37±0.54</td>
<td>3.33±0.49</td>
<td>Overall: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: &lt;0.001</td>
</tr>
<tr>
<td>RVSP (mmHg)</td>
<td>19.5±3.4</td>
<td>20.54</td>
<td>19.9±3.75</td>
<td>Overall: 0.039 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: 0.032 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: n.s.</td>
</tr>
<tr>
<td>TAPSE (cm)</td>
<td>2.16±0.15</td>
<td>2.4±0.17</td>
<td>2.35±0.14</td>
<td>Overall: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: &lt;0.001</td>
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<tr>
<td>RVMPI</td>
<td>0.319±0.01</td>
<td>0.33±0.01</td>
<td>0.325±0.015</td>
<td>Overall: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: 0.003</td>
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</table>


Table (3): Hemodynamic parameters and arterial blood gases under normocapnic and hypercapnic OLV.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>OLV-N</th>
<th>OLV-H</th>
<th>OLV-N1</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>81±6.4</td>
<td>98.62±8.1</td>
<td>93.9±7.4</td>
<td>Overall: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: &lt;0.001</td>
</tr>
<tr>
<td>SBP</td>
<td>123±6.6</td>
<td>130±6</td>
<td>127±7.1</td>
<td>Overall: 0.003 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: 0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: n.s.</td>
</tr>
<tr>
<td>CVP</td>
<td>10±1.4</td>
<td>10±1.1</td>
<td>10±0.9</td>
<td>Overall: n.s. T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: n.s. T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: n.s.</td>
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<tr>
<td>PaCO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>39±2.8</td>
<td>61.8±1.6</td>
<td>41±3.3</td>
<td>Overall: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: n.s.</td>
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<tr>
<td>PaO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>190±11.2</td>
<td>195±11.9</td>
<td>191±12.3</td>
<td>Overall: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: n.s.</td>
</tr>
<tr>
<td>PH</td>
<td>7.42±0.05</td>
<td>7.24±0.02</td>
<td>7.37±0.05</td>
<td>Overall: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: 0.001</td>
</tr>
<tr>
<td>HCO&lt;sub&gt;3&lt;/sub&gt;</td>
<td>23.5±2</td>
<td>22.8±2.09</td>
<td>22.9±1.89</td>
<td>Overall: 0.003 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: 0.004 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: 0.012</td>
</tr>
<tr>
<td>PIP</td>
<td>39±2.03</td>
<td>29.8±2.03</td>
<td>38.8±1.72</td>
<td>Overall: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: &lt;0.001 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: NS</td>
</tr>
<tr>
<td>ScVO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>80±2.37</td>
<td>81.6±2.61</td>
<td>80±2.13</td>
<td>Overall: 0.002 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;2&lt;/sub&gt;: 0.002 T&lt;sub&gt;1&lt;/sub&gt; vs T&lt;sub&gt;3&lt;/sub&gt;: NS</td>
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</table>

SBP: Systolic Blood Pressure in mmHg. CVP: Central Venous Pressure cm H<sub>2</sub>O. PIP: Peak Inspiratory Pressure mmHg. ScVO<sub>2</sub>: Central Venous Saturation %.

Discussion

Permissive hypercapnia (PaCO<sub>2</sub> 60-70mmHg and pH >7.1) resulted in significant increase in both cardiac output and cardiac index in all study participants. Hypercapnia was tolerable in all cases with no need for inotropic support. In a study conducted by Zollinger A. et al.,[14] 24 patients had volume reduction surgery for advanced emphysema with permissive hypercapnia as part of barotrauma prevention strategy. Their mean PaCO<sub>2</sub> value was 56mmHg with a peak of 86mmHg, resulting in a mean pH value of 7.29. Apart from that more than 50% of the patients required inotropic support, hypercapnia was well tolerated. Decreased size of pulmonary vascular bed secondary to advanced diffuse pulmonary emphysema, the consequences of lung resection and the resulting increase in pulmonary artery pressure and right ventricular failure, and the higher PaCO<sub>2</sub> levels (up to 86mmHg) may be all held responsible for the need of inotropic support in their patients compared to ours. Similarly, Moriaski et al.,[15] described higher PaCO<sub>2</sub> levels in a series of 10 patients with severe emphysema managed with elective hypoventilation and hypercapnia for barotrauma avoidance. PaCO<sub>2</sub> values rose to a peak of 70-135mmHg, resulting in pH values as low as 7.03 (despite bicarbonate administration). Hypercapnia was not as well tolerated at these levels.

Hypercapnic acidosis has a direct suppressive effect on cardiac contractility, but it can also lead to a net increase in cardiac output through several mechanisms, as demonstrated in both animal and human studies[16,17]. First, sympathetically mediated release of catecholamines due to neuroadrenal stimulation results in an increase in end-systolic volume and venous return as described by Brofman et al.,[17]. In addition to an increase in heart rate, Nakahata et al.,[18] demonstrated that HCA induced cerebral and coronary vasodilation, an action mediated by ATP-sensitive K<sup>+</sup> channels. In the same context, Kitakaze et al.,[19] showed that acidosis had protective effects against myocardial ischemia-reperfusion injury.

Right ventricular and pulmonary artery systolic pressures increased in all cases but remained within normal values. Weber et al.,[20] and Thoren et al.,[21] in their clinical studies demonstrated that HCA increased the mean pulmonary arterial pressure in patients with ARDS. Similarly, Mekontso and colleagues[7] showed a lower right ventricular stroke index in patients with severe ARDS who were ventilated with higher positive end-expiratory
pertension

Not only RV but also LV systolic performance may have been recommended by American Society of Echocardiography (ASE) guidelines as part of routine echocardiographic evaluation [23].

In the current study, TAPSE values significantly increased with hypercapnia. TAPSE has been shown to correlate strongly with RV ejection fraction [22]. TAPSE is simple and highly reproducible, and has been recommended by American Society of Echocardiography (ASE) guidelines as part of routine echocardiographic evaluation [23].

TAPSE which is the longitudinal excursion of the lateral tricuspid annulus towards the RV apex in the mid esophageal four-chamber view demonstrated prognostic value in PAH [24]. TAPSE is used as a correlate of RV systolic function, since longitudinal displacement of RV base accounts for the greater proportion of total RV volume change. A reasonable correlation between TAPSE and RVEF assessed by radionuclide angiography was found [25,26].

Although simple, widely available, and feasible also in patients with poorly visualized RV endocardial borders, TAPSE is one dimensional and angle-dependent. Regional RV myocardial abnormalities are also neglected when using TAPSE as a single measure [27]. Moreover, its load dependency needs to be considered in case of significant tricuspid regurgitation, when RV base active excursion may inaccurately reflect overall RV contractile function. Not only RV but also LV systolic performance may influence TAPSE value due to ventricular interdependence [28].

Right ventricular myocardial performance index (RVMPI, Tei Index) values showed statistically significant change after mild degree of hypercapnia in our study but this change was within normal range values. RVMPI appears to be relatively independent of preload, afterload, and heart rate and has been useful in assessing patients with congenital heart disease and pulmonary hypertension [29]. In TTE literature, RVMPI is a predictor of adverse outcome in patients with primary pulmonary hypertension [12]. Some authors found a negative correlation between the RV MPI and RV ejection fraction, [30] while others failed to do so [31].

The RVMPI has a number of limitations, such as pseudonormalization of the index, the partial preload dependence, [32] the infeasibility or imprecision of the determination of the index in patients with atrial fibrillation, frequent supraventricular and ventricular extrasystoles.

In the present study, we used ScvO2 as a surrogate to Svo2 as an index of tissue oxygenation. Trends but not individual values of central venous oxygen saturation agree with mixed venous oxygen saturation during varying hemodynamic conditions. There was significant increase in ScvO2 values with hypercapnia. Balanos et al., [33] stated that moderate hypercapnia potentiates the HPV response and is therefore unlikely to adversely affect oxygenation, however, the same may not hold true for extreme CO2 elevations [15].

There was significant increase in PaO2 during normcapnic and hypercapnic one lung ventilation in this study. The beneficial effects of hypercapnic acidosis in increasing arterial and tissue oxygenation is evident from multiple in vivo studies and has been demonstrated in healthy humans [16,34]. HCA can improve tissue oxygenation by several mechanisms. First, a shift of the oxyhemoglobin dissociation curve to the right which facilitates oxygen release to the tissues (the Bohr effect) [35]. Second, HCA causes microvascular vasodilatation promoting oxygen delivery and tissue perfusion. However, high concentrations of PCO2 (101mmHg) will surpass the beneficial vasodilatory effects of HCA and result in vasoconstriction as demonstrated by Komori M. et al., [36]. Third, HCA improves ventilation-perfusion (V/Q) matching by potentiating hypoxic pulmonary vasoconstriction [33,37]. Fourth, as cardiac output is one of the major determinants of peripheral oxygen delivery, one can expect that a CO2-mediated increase in cardiac output augments peripheral oxygen delivery [38].

Limitation of the study:

The relatively short duration spent since the stabilization of ventilation at any given point till the switch the next phase might have had an impact on the readings obtained during the study. Though this was dictated by the rapidly changing intraoperative environment, we still believe this impact was uniform along all study phases.

Conclusion:

Permissive hypercapnia as a component of OLV management is likely to be beneficial in selected patients. PaCO2 levels up to 60-70mmHg are likely to be tolerated in the short-term and clearly beneficial in terms of lung injury attenuation.
References


من ن电话ية الة الواحدة لمرضى الاستناد الرئوي له العديد من المضاعفات، منها زيادة الضغط في طريق الهواء للثة السفلي والخاطر المتصلة من الصدمة التائية عن الضغط، اوعمل نفس الحجم أتى تهوية الة الواحدة أو الرئتين كان يستخدم الخاطر على نفس المستوى الطبيعي لضبط الجنسين أتى أوكسيد الكربونس بدون ضغط الحم أخر الزفير. وذلك حتى لو كان الوقت لتنديد تسعين دقيقة، قد يؤدي إلى زيادة كمية الرئة.

وعلى الرغم من أن تأثير الزيادة المسموح بها لثاني أوكسيد الكربون على ديناميكا الدم والبطين الكبير كان قد نذكر سابقا في مرضى متلازمة الضائقة التنفسية الحادة؛ لكن تأثير ذلك على وظائف البطين الكبير أثناء تهوية الة الواحدة لم يتم التحقيق فيه بشكل منهجي حتى الآن.

وقد أوضحت الدراسة الحالية أنه في وجود احتياطي معقول للقلب والأوعية الدموية وخاصة البطين الكبير، فإن الدرجات القليلة من ارتفاع الضغط الجنسين لثاني أوكسيد الكربون ما بين 20-70 مم زئيقي ودرجة حوضية لا تقل عن 75% قد مصير زيادة في الناتج القلبي مؤذن.

وقد أوضحت الدراسة أن الزيادة المسموحة في الضغط الجنسين لثاني أوكسيد الكربون تكون ممتصة وفعالة كجزء من تهوية الة الواحدة، فيما يتعلق بتجنب إصابة الرئة.