Alveolar recruitment improves ventilation during thoracic surgery: a randomized controlled trial

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Editor’s key points
• Alveolar recruitment manoeuvres improve pulmonary function during anaesthesia by reducing atelectasis.
• An alveolar recruitment strategy was prospectively studied in patients undergoing open thoracotomy.
• A period of high pressure ventilation immediately before and after one-lung ventilation improved oxygenation and reduced alveolar dead space.

Background. This study was conducted to determine whether an alveolar recruitment strategy (ARS) applied during two-lung ventilation (TLV) just before starting one-lung ventilation (OLV) improves ventilatory efficiency.

Methods. Subjects were randomly allocated to two groups: (i) control group: ventilation with tidal volume (VT) of 8 or 6 ml kg\(^{-1}\) for TLV and OLV, respectively, and (ii) ARS group: same ventilatory pattern with ARS consisting of 10 consecutive breaths at a plateau pressure of 40 and 20 cm H\(_2\)O PEEP applied immediately before and after OLV. Volumetric capnography and arterial blood samples were recorded 5 min (baseline) and 20 min into TLV, at 20 and 40 min during OLV, and finally 10 min after re-establishing TLV.

Results. Twenty subjects were included in each group. In all subjects, the airway component of dead space remained constant during the study. Compared with baseline, the alveolar dead space ratio (\(V_{D_{alv}}/VT_{alv}\)) increased throughout the protocol in the control but decreased in the ARS group. Differences in \(V_{D_{alv}}/VT_{alv}\) between groups were significant (\(P<0.001\)). Except for baseline, all \(P_{aO_2}\) values in kPa (SD) were higher in the ARS than in the control group (\(P<0.001\)), respectively [70 (7) and 55 (9); 33 (9) and 24 (10); 33 (8) and 22 (10); 70 (7) and 55 (10)].

Conclusions. Recruitment of both lungs before instituting OLV not only decreased alveolar dead space but also improved arterial oxygenation and the efficiency of ventilation.

Keywords: lung, atelectasis; lung, gas exchange; surgery, thoracic; ventilation, dead space; ventilation, one-lung ventilation

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obtained from all the patients. Patients with ASA status II–III undergoing elective open thoracotomy in the lateral position were eligible for enrolment. Exclusion criteria were expected duration of OLV of <40 min, previous contralateral lobectomy, uncompensated cardiac disease, arrhythmias with haemodynamic repercussions, severe air trapping (residual volume >175%), tracheostomy, presence of large bullae, and intraoperative bleeding (>500 ml).

Anaesthesia, ventilation, and monitoring
Patients were monitored by ECG, pulse oximetry, nasopharyngeal temperature, and invasive arterial pressure using a Datex Ohmeda S/5 monitor (Datex-Ohmeda Division, Instrumentarium Corp., Helsinki, Finland).

Premedication with midazolam 5 mg i.v. was administered on arrival in the operating theatre. A paravertebral block at T6 level was performed by injecting 15 ml of bupivacaine 0.5% via a catheter. Anaesthesia was induced with propofol 1.5–2 mg kg⁻¹, fentanyl 5 µg kg⁻¹, and atracurium 0.5 mg kg⁻¹ i.v. Anaesthesia was maintained with a continuous infusion of propofol 6–10 mg kg⁻¹ h⁻¹ and supplemental fentanyl to keep arterial pressure and heart rate within 20% of the pre-induction values. The trachea was intubated with a left-sided double-lumen tube (DLT) (Broncho-part, Rush, Kernen, Germany: 39 F for male and 37 F for female patients); correct position was confirmed by fibreoptic bronchoscopy twice, with the patient supine and in the lateral position. Functional lung separation was confirmed by sequentially connecting the capnograph sensor with each lumen of the DLT while maintaining ventilation through the other lumen. During OLV, the lumen of the non-ventilated lung was left open to air.

After intubation, the lungs were ventilated with a Servo 1 ventilator (Maquet Critical Care, Solna, Sweden) using a constant flow, volume controlled mode with the following settings: tidal volume (VT) 8 ml kg⁻¹ predicted body weight, respiratory rate 12 bpm, inspiratory time of 33% including an end-inspiratory pause of 10%, PEEP of 8 cm H₂O, and FiO₂ = 1. During OLV, VT was reduced to 6 ml kg⁻¹ and respiratory rate increased to 15–18 bpm to maintain similar minute ventilation as during two-lung ventilation (TLV). PEEP was maintained at 8 cm H₂O. At the end of surgery, TLV was re-established using the above baseline settings.

Intraoperative crystalloids were administered at a rate of 2–4 ml kg⁻¹ h⁻¹ and blood loss was initially replaced with colloids or with red blood cell concentrates if haemoglobin levels decreased below 80 g litre⁻¹. All patients were extubated in the operating theatre and admitted to a postoperative care unit for at least 24 h before transfer to the surgical ward.

Volumetric capnography and lung mechanics
Volumetric capnography was recorded continuously using the NICO capnograph connected to a laptop running the software DataColl (Respironics, Wallingford, CT, USA). Airway flow and CO₂-mainstream sensors were placed between the tracheal tube and the ‘Y’ piece of the ventilator circuit. Expired volume and CO₂ data were downloaded into a custom MatLab program (Mathworks, Natick, MA, USA) that constructed breath-by-breath volumetric capnograms for offline analysis. Analysis consisted of fitting to a Levenberg–Marquardt algorithm, defining a mathematical function from which all volumetric capnography-derived parameters were calculated.¹²

The VTCO₂,br is the amount of CO₂ eliminated during one breath obtained by integration of expired airway flow and PCO₂, P̅CO₂ is the partial pressure of CO₂ at the end of expiration. Airway dead space (VD₉₅) was calculated as the inflection point of phase II of the capnogram or the limit between VD₉₅ and the alveolar tidal volume (VT₉₅).¹²

Physiological dead space to tidal volume ratio (VD/VT) was calculated using the Bohr–Enghoff formula:¹³

\[
\frac{VD}{VT} = \frac{P_{aCO_2} - P_{ECO_2}}{P_{aCO_2}}
\]

where \(P_{ECO_2}\) is the mixed \(PCO_2\) of an expiration. Physiological dead space (VD₉₅) was then calculated by multiplying VD/VT and tidal volume. Alveolar dead space was obtained by subtracting VD₉₅ from VD₉₅ and then normalized by the alveolar tidal volume (VD₉₅/VT₉₅).

Dynamic compliance (Crs), airway resistance (R₉₅), peak inspiratory airway pressure (Pp), and mean airway pressure were determined by the NICO. Crs was automatically calculated as VT/(P₉₅ – PEEP). Plateau airway pressure (P₉₅) was obtained from the Servo. To minimize error and variability, the mean value of the last 20 breaths of each step was calculated for each variable.

Protocol and measurements
All measurements were performed in the lateral position. Subjects were randomly allocated to two groups using an Excel® random table (Microsoft, Redmont, WA, USA):

(i) Control group: ARS was not applied to the dependent lung at any point. After lung resection and the OLV period, the non-dependent lung was selectively re-expanded by a routine sustained inflation (vital capacity manoeuvre) using 40 cm H₂O of continuous positive airway pressure applied for 8 s.³ We used this controlled manoeuvre instead of the typical hand bagging to standardize non-dependent lung re-aeration in this group because otherwise neither VT nor airway pressures could have been controlled adequately.

(ii) ARS group: a lung recruitment manoeuvre (ARS₉₅) was performed on TLV 10 min after placing the patient in the lateral position and confirmation of appropriate DLT position. After the OLV period and just before re-establishing TLV, a second recruitment (ARS₂₉₅) of both lungs was performed to replace the traditional lung re-expansion manoeuvre by hand bagging.

ARS was performed as described previously.⁴–⁶ Briefly, the ventilator was switched to pressure-control ventilation,
adjusting the driving pressure ($P_{\text{plut}} - \text{PEEP}$) to obtain the same VT as during previous baseline ventilation in a volume-control mode. Pressure-control ventilation was then allowed to equilibrate for 3 min. Keeping the driving pressure constant, PEEP was increased from 8 to 10 cm H$_2$O and ventilation was maintained for 2 min to assess the haemodynamic response. If mean arterial pressure or heart rate changed by $\pm 20\%$ from baseline, ARS was not performed but 3 ml kg$^{-1}$ of colloid solution was infused. Once haemodynamic stability returned, ARS was tried again. During haemodynamic stability, PEEP was increased to 15 cm H$_2$O for 5 breaths and then to 20 cm H$_2$O. After reaching a PEEP of 20 cm H$_2$O, driving pressure was increased to 20 cm H$_2$O to reach a final $P_{\text{plut}}$ of 40 cm H$_2$O. These high recruitment pressures were maintained for 10 breaths at a respiratory rate of 12 bpm, which in total took <1 min. After completion of ARS, the ventilator was set back to baseline ventilation in volume-control mode at a PEEP of 8 cm H$_2$O.

Arterial blood gases, volumetric capnography, and ventilatory and haemodynamic data were recorded at the following study points (Fig. 1):

(i) TLV$_{\text{baseline}}$: 5 min after placing the patient in the lateral position during TLV but before performing the lung recruitment manoeuvre (ARS$_1$) in the treated group.
(ii) TLV$_{20}$: 20 min after placing the patient in the lateral position during TLV.
(iii) OLV$_{20}$: 20 min after OLV ventilation.
(iv) OLV$_{40}$: 40 min after OLV ventilation.
(v) TLV$_{\text{end}}$: once the non-dependent lung was re-expanded either by a manual vital capacity manoeuvre in the control or by ARS$_2$ in the treated group (Fig. 1), TLV was re-established and measurements were taken 10 min later.

After TLV$_{\text{baseline}}$ measurements, all subjects received a 3 ml kg$^{-1}$ of colloid infusion. The first four measurements were recorded before clamping any major pulmonary vessel of the non-dependent lung. If pulse oximetry decreased to <90% during OLV, surgery was temporarily interrupted to resume TLV (intermittent ventilation) until oxygen saturation recovered to at least 97%.

Blood samples were always processed within <5 min of extraction using an ABL 520 blood gas analyzer (Radiometer, Copenhagen, Denmark), and values were corrected for body temperature.

### Statistical analysis

The sample size for this study was determined for the primary endpoint, the change in VD/VT after the lung recruitment manoeuvre. Preliminary data were available from a study that tested the effects of this ventilatory strategy on the efficiency of ventilation (VD/VT) during OLV, thus an endpoint similar to that of the current study could be used to analyse statistical power. To detect a difference of 0.07 in VD/VT between OLV before ARS and OLV after ARS with a two-sided approximation, accepting an $\alpha$ error of 5% and a $\beta$ error of 10%, and anticipating a dropout rate of 30%, a total of 40 subjects was determined.

Quantitative data are expressed as mean (SD). Baseline parameters and procedural characteristics were compared between groups using Fisher’s exact test for categorical variables and the two-sample t-test for continuous variables. Between-group comparisons were done by repeated-measures analysis of variance. In all cases, $P<0.05$ was considered statistically significant. Statistical analysis was performed using SPSS version 15.0 (SPSS, Chicago, IL, USA).

### Results

A total of 40 subjects were enrolled, 20 in each group (Fig. 2). There were no differences between groups regarding characteristics or clinical data (Table 1). The duration of surgery was 152 min (92–215) in the control group and 162 min (89–185) in the ARS group. The timing between OLV$_{40}$ and TLV$_{\text{end}}$ was 66 min (12–123) in the control group and 63 min (10–117) in the ARS group.

Figure 3 shows variables derived from volumetric capnography. VD/VT remained unchanged in the control group but decreased significantly in the ARS group throughout the study. The VD/VT values were different between groups ($P<0.001$). In both groups, VD$_{\text{aw}}$/VT$_{\text{aw}}$ showed no significant changes. VD$_{\text{alv}}$/VT$_{\text{alv}}$ increased in the control group but decreased in the ARS group at TLV$_{20}$. Except for the baseline condition, all VD$_{\text{alv}}$/VT$_{\text{alv}}$ values were significantly lower in the ARS group than in the control group ($P<0.001$).

From TLV$_{\text{baseline}}$ to TLV$_{20}$, VTCO$_{2\text{br}}$ increased 10% in the ARS group and decreased 7% in the control group ($P=0.004$). When compared with baseline values, VTCO$_{2\text{br}}$ at TLV$_{\text{end}}$ increased 7% in the ARS group but decreased 25% in the control group ($P<0.001$) and these values were also different between groups ($P<0.001$).
At TLV20, $P_{a} - E_{CO2}$ increased 31% in the control group but decreased 29% in the ARS group when compared with baseline values (Table 2). $P_{a} - E_{CO2}$ values remained lower than baseline throughout the study in the ARS group but were higher in the control group ($P < 0.05$); these differences between groups reached significance at all points of the protocol ($P < 0.001$ in all except for TLV baseline).

Arterial oxygenation improved in the ARS group 28% at TLV20 ($P < 0.001$), but decreased 7% in the control group (Table 2). $P_{a}O_{2}$ values were significantly higher in the ARS group than in the control group throughout the study ($P < 0.001$) (Table 2). During OLV, one subject in the control group had arterial oxygen saturation decrease below 90%. In this subject, intermittent ventilation was performed twice within a 5 min period to achieve $S_{a}O_{2}$ of 97%. Thereafter, $S_{a}O_{2}$ remained > 90% throughout OLV. The lowest recorded value of $P_{a}O_{2}$ during OLV was 8.11 kPa in the control group and 15.7 kPa in the ARS group.

Lung mechanics and haemodynamic data are shown in Table 3. Dynamic respiratory compliance increased after the

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**Table 1** Subject characteristics. Data are presented using mean (SD) for continuous variables and n for categorical variables. FVC, forced vital capacity; FEV1, forced expiratory volume in 1 s

<table>
<thead>
<tr>
<th></th>
<th>ARS group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Age (yr) [mean (range)]</td>
<td>61 (24–78)</td>
<td>57 (21–78)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>71.4 (12.02)</td>
<td>68.8 (11.61)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>167.6 (7.80)</td>
<td>164.8 (9.13)</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>15 M/5 F</td>
<td>15 M/5 F</td>
</tr>
<tr>
<td>ASA (II/III)</td>
<td>8/12</td>
<td>9/11</td>
</tr>
<tr>
<td>Lobectomies/wedge resections</td>
<td>15/5</td>
<td>14/6</td>
</tr>
<tr>
<td>Preoperative FVC (% predicted)</td>
<td>82.2 (7.05)</td>
<td>90.3 (11.90)</td>
</tr>
<tr>
<td>Preoperative FEV1 (% predicted)</td>
<td>78.2 (21.01)</td>
<td>82.2 (20.23)</td>
</tr>
<tr>
<td>Preoperative FEV1/CVF (% predicted)</td>
<td>70.4 (12.75)</td>
<td>69.9 (10.96)</td>
</tr>
<tr>
<td>Preoperative $P_{a}O_{2}$ (mm Hg)</td>
<td>10.0 (1.15)</td>
<td>10.5 (0.79)</td>
</tr>
<tr>
<td>Preoperative $P_{a}CO_{2}$ (mm Hg)</td>
<td>5.1 (0.42)</td>
<td>5.0 (0.26)</td>
</tr>
</tbody>
</table>
The recruitment manoeuvre was well tolerated without any apparent pulmonary or haemodynamic complications. Arterial pressure was continuously monitored and closely

recruitment manoeuvre in the ARS group ($P=0.009$). However, it did not reach statistical significance between groups at any time point during the study.

Fig 3 Volumetric capnography-derived variables. VD/VT, physiological dead space to tidal volume; VDaw/VT, alveolar dead space to alveolar tidal volume; VDalv/VTalv, airway dead space to tidal volume and VTCO₂,br, expired volume of CO₂ per breath. *TLVbaseline against TLV₂₀, †TLV₂₀ vs OLV₂₀, §TLVbaseline vs TLVend, and ¶ when compared with the control group; all $P<0.05$.

### Table 2
Mean values (sd) of intraoperative blood gases. $P$O₂ arterial oxygen tension; $P$CO₂ arterial carbon dioxide tension; $E$CO₂ end-tidal $P$CO₂; $Pa − E$CO₂ arterial to end-tidal $P$CO₂ difference. TLVbaseline, 5 min after placing the patient in the lateral position during TLV; TLV₂₀, 20 min after placing the patient in the lateral position during TLV; OLV₂₀, after 20 min of OLV; OLV₄₀, after 40 min of OLV; TLVend, 10 min after TLV was re-established once the resection was finished. *TLVbaseline vs TLV₂₀, †TLV₂₀ vs OLV₂₀, §TLVbaseline vs TLVend, and ¶ when compared with the control group; all $P<0.05$

<table>
<thead>
<tr>
<th></th>
<th>TLVbaseline</th>
<th>TLV₂₀</th>
<th>OLV₂₀</th>
<th>OLV₄₀</th>
<th>TLVend</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>pH</strong></td>
<td>7.40 (0.04)</td>
<td>7.40 (0.04)</td>
<td>7.39 (0.04)</td>
<td>7.39 (0.04)</td>
<td>7.40 (0.04)</td>
</tr>
<tr>
<td><strong>$P$O₂ (kPa)</strong></td>
<td>59.0 (9.7)</td>
<td>54.8 (9.2)*</td>
<td>24.3 (10.6)*</td>
<td>22.1 (10.5)*</td>
<td>54.9 (9.8)*</td>
</tr>
<tr>
<td><strong>$P$CO₂ (kPa)</strong></td>
<td>54.8 (7.2)</td>
<td>70.2 (7.3)*§</td>
<td>33.4 (9.2)*§</td>
<td>32.8 (7.7)*§</td>
<td>70.1 (8.6)*§</td>
</tr>
<tr>
<td><strong>$E$CO₂ (kPa)</strong></td>
<td>5.8 (0.61)</td>
<td>5.4 (0.7)*§</td>
<td>5.6 (0.7)</td>
<td>5.5 (0.6)</td>
<td>5.2 (0.6)*§</td>
</tr>
<tr>
<td><strong>Pa − $E$CO₂ (kPa)</strong></td>
<td>4.6 (0.4)</td>
<td>4.4 (0.5)</td>
<td>4.4 (0.4)</td>
<td>4.3 (0.4)</td>
<td>4.2 (0.5)*§</td>
</tr>
<tr>
<td><strong>$PaO₂$ (kPa)</strong></td>
<td>1.1 (0.4)</td>
<td>1.4 (0.4)*§</td>
<td>1.5 (0.4)</td>
<td>1.6 (0.4)</td>
<td>1.4 (0.4)*§</td>
</tr>
<tr>
<td><strong>$PaCO₂$ (kPa)</strong></td>
<td>1.3 (0.4)</td>
<td>0.9 (0.4)*§</td>
<td>1.0 (0.4)*§</td>
<td>1.0 (0.3)*§</td>
<td>0.8 (0.2)*§</td>
</tr>
</tbody>
</table>
checked, especially during the recruitment manoeuvre during which no clinically relevant haemodynamic alterations were encountered. No patient required additional colloid infusion during ARS.

Discussion
This randomized controlled study showed that bilateral lung recruitment just before starting OLV not only increased the efficiency of ventilation by decreasing the alveolar component of dead space but also improved arterial oxygenation. Such recruitment-induced improvement in lung physiology was sustained throughout the entire surgical procedure. The control group, however, treated with the same ventilator settings but without recruitment showed increased alveolar dead space values throughout the study.

Calculating dead space, defined as the wasted portion of ventilation, is the most common way to assess efficiency of ventilatory settings. The lower the dead space, the higher the ventilatory efficiency and vice versa. Previous studies have consistently shown an increase in $\text{VT}_{\text{CO}_2}$ in response to lung recruitment. Our results are in line with these studies and suggest an improvement in gas exchange, lung perfusion, or both with ARS, assuming that ventilation and metabolism did not usually change in the study population during the surgical procedure.

With lung recruitment, we observed a reduction in the alveolar but not in the airway component of dead space. Since we used Enghoff’s modification of Bohr’s formula (replacing alveolar $PcO_2$ by arterial $PaCO_2$), the reduction in $VD_{alv}/VT_{alv}$ in the ARS group can be explained unequivocally by decrements not only in the ‘real’ but also in the ‘apparent’ shunt-related $VD_{alv}$. This fictitious part of $VD_{alv}$ is created by the effect of shunt. Therefore, it seems plausible that the beneficial effect of lung recruitment on dead space is mainly attributed to the reversal of lung collapse and shunt. During OLV, the differences in dead space and gas exchange between the two groups are most likely related to the recruitment-induced reversal of lung collapse in the dependent lung.

$VTCO_2$, indicates how much $CO_2$ is eliminated in one breath. It depends on the mass balance between metabolic production, lung perfusion, gas exchange area, and alveolar ventilation. Previous studies have consistently shown an increase in $VTCO_2$ in response to lung recruitment. Our results confirm these findings.
The significant differences in the variables $Pa - E_{CO_2}$ and $PaO_2$ support the above explanations. $Pa - E_{CO_2}$ is an index representing CO2 exchange through the alveolar-capillary membrane that is related to lung aeration. Strang and colleagues described a close correlation between $Pa - E_{CO_2}$ and the amount of atelectasis in animals with healthy lungs, while Tuomanen and colleagues found similar correlations between $Pa - E_{CO_2}$ and the amount of atelectasis in acutely injured lungs of pigs. Our study supports the idea that $Pa - E_{CO_2}$ can be used to indirectly assess the amount of atelectasis during mechanical ventilation. $Pa - E_{CO_2}$ values were lower in the ARS group than in the control group throughout the surgery, showing that lung recruitment optimizes the area of gas exchange by decreasing the amount of shunting through collapsed tissue. Our data are in agreement with clinical studies in thoracic and non-thoracic surgery, where $Pa - E_{CO_2}$ decreases once lung collapse is reversed by a lung recruitment manoeuvre.

Oxygenation is known to improve with lung recruitment due to reversion of atelectasis in the dependent lung. Our results coincide with these previous findings, suggesting that an ARS applied before OLV might prevent hypoxaemic episodes during OLV. Our data are also in agreement with findings that lung recruitment applied before OLV in patients with normal preoperative pulmonary function improved $PaO_2$ during OLV. However, differences between that study and our protocol must be highlighted: first, our study was carried out in patients with considerably poorer pulmonary function; and secondly, the highest airway pressures during our recruitment manoeuvre were maintained for <1 min rather than 12 min. Thus, it seems that ARS is effective in patients with both normal and poor preoperative functional testing and that a brief period of high airway pressures not exceeding 1 min is enough to reexpand atelectatic lung.

Although dynamic compliance increased after the recruitment manoeuvre in the ARS group, there was a lack of difference between groups. This can be attributed to the small degree of atelectasis. Moreover, the pre-existing difference in baseline values between groups could also have played a role. Our results are in line with those of Park and colleagues who did not find differences in lung compliances between groups. However, they are in disagreement with reports of differences between groups when different intraoperative ventilatory strategies were compared in obese and elderly patients.

Although we did not monitor cardiac output continuously, arterial pressure was monitored online and closely checked during the recruitment manoeuvre. We did not encounter any clinically relevant haemodynamic alterations during ARS. This could be explained by the optimization of haemodynamic status by administering 3 ml kg$^{-1}$ of colloids before ARS. Our results agree with those of Weingarten and colleagues who did not observe acute haemodynamic compromise during recruitment manoeuvres in elderly patients undergoing laparotomy.

**Potential clinical implications of findings**

Our study sheds new light on two important clinical issues in thoracic surgery. One is ventilator-induced lung injury, which is proportional to the stress on lung tissue determined by the size of VT and Ppl applied during OLV. Therefore, applying protective ventilatory strategies in the intraoperative period would seem reasonable. As lung recruitment decreases dead space, VT and Ppl can be reduced to a minimum because ventilation and thus the clearance of CO2 is more efficient. The other issue is episodes of hypoxaemia during OLV. Lung recruitment improved arterial oxygenation throughout thoracic surgery. Therefore, ARS could increase the margin of safety for hypoxaemia throughout the entire surgery. Moreover, ARS has additional clinical value as a rescue therapy in severely hypoxaemic patients as it can increase $PaO_2$ to a safer level instantaneously. Surprisingly, subjects in our control group did not show the changes in lung physiology that we anticipated. For instance, the incidence of hypoxaemia in the control group was in the lower range of what has been reported in the literature. Therefore, these potential benefits of ARS could be even more pronounced in critically ill patients. Further and larger studies in critically ill patients undergoing OLV are needed.

**Limitations**

The main limitation of this clinical study is that we did not provide direct evidence for the presence of atelectasis by imaging or shunt measurements using a pulmonary artery catheter. Imaging would have been technically difficult to use while the invasive pulmonary artery catheter could not be clinically justified in our patients. Thus, we were confined to using $PaO_2$, $Pa - E_{CO_2}$, and alveolar dead space as surrogate for atelectasis to test the effect of lung recruitment on atelectasis and shunt.

In conclusion, recruitment of both lungs before instituting OLV not only made ventilation more efficient but also improved arterial oxygenation during the entire surgical procedure.

**Declaration of interest**

F.S.-S. performs consultant services for Maquet Critical Care.

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**References**


