The open lung concept: effects on right ventricular afterload after cardiac surgery

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Background. The open lung concept (OLC) is a method of ventilation intended to maintain end-expiratory lung volume by increased airway pressure. Since this could increase right ventricular afterload, we studied the effect of this method on right ventricular afterload in patients after cardiac surgery.

Methods. We studied 24 stable patients after coronary artery surgery and/or valve surgery with cardiopulmonary bypass. Patients were randomly assigned to OLC or conventional mechanical ventilation (CMV). In the OLC group, recruitment manoeuvres were applied until $P_{aO2}/F_{IO2}$ was greater than 50 kPa (reflecting an open lung). This value was maintained by sufficient positive airway pressure. In the CMV group, volume-controlled ventilation was used with a PEEP of 5 cm H₂O. Cardiac index, right ventricular preload, contractility and afterload were measured with a pulmonary artery thermodilution catheter during the 3-h observation period. Blood gases were monitored continuously.

Results. To achieve $P_{aO2}/F_{IO2} > 50$ kPa, 5.3 (3) (mean, SD) recruitment attempts were performed with a peak pressure of 45.5 (2) cm H₂O. To keep the lung open, PEEP of 17.0 (3) cm H₂O was required. Compared with baseline, pulmonary vascular resistance and right ventricular ejection fraction did not change significantly during the observation period in either group.

Conclusion. No evidence was found that ventilation according to the OLC affects right ventricular afterload.

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Keywords: heart, right ventricular afterload; heart, right ventricular ejection fraction; lung, PEEP; lung, open lung concept; lung, recruitment manoeuvre

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The open lung concept (OLC) is a method of ventilation intended to reduce shear forces caused by repeated opening and closing of atelectatic lung.¹² This is done with a recruitment manoeuvre and application of sufficient positive end-expiratory pressure (PEEP) to counterbalance retractive forces, and with ventilation with the smallest possible pressure amplitude to prevent lung overdistention.³ However, this strategy increases intrathoracic pressure, which could increase right ventricular afterload and reduce safety.

Coronary bypass grafting (CABG) can be complicated by pulmonary dysfunction⁴ or by reduced right ventricular function.⁵ Patients with such complications could be vulnerable to increased right ventricular afterload. However, Dyhr and colleagues⁶ found that a lung recruitment manoeuvre followed by PEEP did not reduce cardiac output in patients after CABG who had been give a volume load. The increased right ventricular afterload could have been offset by the increased end-diastolic volume, since other effects of PEEP on cardiac output can be offset by preload augmentation.⁷–⁹

We set out to study the effect of ventilation according to the OLC on right ventricular afterload in patients ventilated after CABG and/or valve surgery.

Methods

The study was approved by the local human ethics research committee and informed written consent was given by each patient. We prospectively studied 24 stable patients who had...
underwent CABG and/or valve surgery with use of cardio-
pulmonary bypass (CPB). The patients we studied often need
prolonged ventilatory support after cardiac surgery because
of comorbidity and/or extensive surgery. We excluded
patients with severe airway obstruction (forced expired
volume in 1 s or vital capacity less than the predicted nor-
mal range, taken as 2 predicted value ±2 SD) or those who
required reoperation within the first 72 h (as blood loss
requiring reoperation could cause tamponade and affect
the circulation).

Preoperative risk factors were scored with the European
System for Cardiac Operative Risk Evaluation (Euroscore),
a scoring system used to predict mortality in cardiac surgery
patients, and expressed as a percentage.10

Anaesthesia was induced with midazolam 0.1 mg kg\(^{-1}\) i.v.
and sufentanil 2 \(\mu\)g kg\(^{-1}\). Muscle relaxation was with pancu-
ronium 0.1–0.2 mg kg\(^{-1}\) and was not reversed. Adminis-
tration of enoximone 0.5 mg kg\(^{-1}\) (effective for 3–6 h) was
used routinely to reduce myocardial stunning after CPB.

After induction of anaesthesia, a pulmonary artery catheter
(ACCO 774HF75 series; Edwards, Irvine, CA, USA) was
inserted through the right internal jugular vein. Anaesthesia
was maintained with midazolam 0.1 mg kg\(^{-1}\) and sufentanil
(1 \(\mu\)g kg\(^{-1}\)) as needed. None of the patients received corti-
costeroids.

During operation the lungs were ventilated with the follow-
ing settings: volume control mode, tidal volume
6–8 ml kg\(^{-1}\); PEEP 5 cm H\(_2\)O; I/E ratio 1:2; \(F_{\text{O}_2}\), 0.3–0.5; and
respiratory rate was adjusted to achieve a \(P_{\text{aCO}_2}\) between 4.5
and 6.5 kPa. These settings were called ‘conventional
mechanical ventilation’ (CMV). During CPB the lungs
were not ventilated. After CPB lungs were re-expanded by
manual inflation and ventilation was continued with the same
settings until randomization. After surgery the pericardium
was not closed. After sternum closure, the patients were
given fluids until left ventricular function did not increase
further. Left ventricular function was assessed by transeso-
ophageal echocardiogram by an experienced operator, who
assessed fractional area change on the transgastric mid-
papillary short-axis view. At optimal left ventricular func-
tion, right ventricular end-diastolic volume index (EDVI)
measured with the pulmonary artery catheter was defined
as optimal EDVI.

After surgery, patients were sedated with propofol
2–4 mg kg\(^{-1}\) h\(^{-1}\). An indwelling blood gas analyser probe
was inserted in a radial artery for continuous blood gas ana-
alyses (ParaTrend 7+; Philips, Boblingen, Germany). Fluid
management was guided by EDVI, aiming at optimal
EDVI as assessed during operation. Hypovolaemia was trea-
ted with a set plan using starch colloids. If a maximum daily
dose was reached, further fluid was given as pasteurized
plasma. If mean arterial pressure was less than 45 mm H\(_{\text{g}}\) and
hypovolaemia was excluded, an infusion of dobutamine
or phenylephrine i.v. was given.

Cardiovascular and respiratory measurements were made
every 30 min for 3 h. Measurements before randomization
were considered baseline measurements. Patients were
randomly assigned by envelope to the OLC group or the
CMV group. Randomization was not stratified for type of operation. The study group was ventilated according to
the OLC and in the CMV group ventilation was continued as
described above.

Ventilation according to the OLC was initiated by switch-
ing the ventilator to pressure control mode, PEEP 10 cm H\(_2\)O,
\(F_{\text{O}_2}\) 0.3–0.4, I/E ratio 1:1 and a pressure to obtain a tidal
volume of 4–6 ml kg\(^{-1}\), aiming at a \(P_{\text{aCO}_2}\) of 4.5–6.5 kPa. A
respiratory frequency of 40 b.p.m. was chosen to achieve
good carbon dioxide elimination with a low tidal volume.
A lung recruitment manoeuvre was applied by increasing
peak pressure to 40 cm H\(_2\)O for 40 s to increase the \(P_{\text{aO}_2}/
F_{\text{O}_2}\) ratio to a value greater than 50 kPa. If not, a recruit-
ment manoeuvre was repeated by increasing peak pressure 5
cm H\(_2\)O greater than before, up to a maximum peak pressure
of 60 cm H\(_2\)O until the \(P_{\text{aO}_2}/F_{\text{O}_2}\) ratio became greater than
50 kPa. If the \(P_{\text{aO}_2}/F_{\text{O}_2}\) ratio decreased slowly below 50 kPa
after recruitment, PEEP was increased by 2 cm H\(_2\)O and a
recruitment manoeuvre (beginning at 40 cm H\(_2\)O) was
repeated.

Cardiovascular measurements consisted of right atrial
pressure (RAP), mean pulmonary arterial pressure (\(P_{\text{amean}}\))
and pulmonary capillary wedge pressure (PCWP). A cardiac
output computer (Vigilence; Edwards) that was connected to
the pulmonary artery catheter and the monitor recorded heart
rate and calculated cardiac index (CI), EDVI and right ven-
tricular ejection fraction (REF). From these values, pulmon-
ary vascular resistance (PVR) was calculated.

After the 3-h study period, if temperature and cardio-
vascular measurements were satisfactory, sedation was stopped
and the patients were weaned from ventilation. Data on out-
come were not obtained in this study.

Statistics

To adjust for differences between patients, the changes from
baseline measurements were calculated and used to compare
the two groups.

Blood gas and cardiovascular measurements, as changes
from baseline, were compared using analysis of variance (\(\text{ANOVA}\))
for repeated measurements (PROC MIXED proce-
dures from SAS).11

Results are presented as mean (SD). The \(P\)-values given are
two-sided, and \(P<0.05\) was considered significant.

Results

We enrolled 24 patients. Four patients were excluded and the
data of 20 patients are presented. Three patients required
reoperation and were excluded: one in the CMV group
where reoperation occurred within 2 h, and two in the
OLC group where reoperation occurred at 3 and 7 h. One
other patient (OLC) was excluded because the continuous
blood gas analyser failed. No myocardial infarction or pneu-
mothorax occurred.
In the OLC group, the Euroscore was greater and more patients had surgery for CABG than in the CMV group. The groups were similar for other baseline characteristics (Table 1). In the CMV group, two patients required a greater FiO2 to maintain PaO2 greater than 10 kPa. In the OLC group, 5.3 (2) recruitment attempts were made with a mean peak pressure of 45.5 (2) cm H2O to open the lung (Fig. 1). To keep the lung open, a total PEEP of 17.0 (2.7) cm H2O had to be applied in the OLC group.

Compared with baseline REF (Fig. 2), CI and PVR changes did not differ between groups at any time (Table 2). In the OLC group, mean PA pressure and PCWP increased significantly during treatment compared with baseline (Table 2). The increase in PCWP, but not that in mean PA pressure, was significantly greater with OLC than with CMV (Table 2). Considering the decrease in pressure through the pulmonary circulation (PAmean–PCWP), the change from baseline to the treatment period was not different between the groups (Fig. 3).

The tidal volume in the OLC group was 4.5 (2) ml kg\(^{-1}\) and \(P_{A\text{CO}_2}\) remained in the target range (4.5–6.5 kPa). This resulted in peak inspiratory pressures that were comparable in the two groups despite the high PEEP values in the OLC group (Table 3).

Table 1  Characteristics of the groups. Data are mean (range) for age, or mean (SD) unless otherwise stated. *The Euroscore is expressed as median (first quartile)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>OLC (n=10)</th>
<th>CMV (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>66.3 (54–78)</td>
<td>57.5 (29–77)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>78.7 (17.6)</td>
<td>78.5 (10.2)</td>
</tr>
<tr>
<td>Male/female</td>
<td>6/4</td>
<td>9/1</td>
</tr>
<tr>
<td>CABG</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Valve repairs or replacement</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>Euroscore (%)*</td>
<td>2.5 (2)</td>
<td>2.0 (0.9)</td>
</tr>
<tr>
<td>(P_{A\text{CO}_2}) (kPa)</td>
<td>10.5 (1.8)</td>
<td>10.7 (1.7)</td>
</tr>
<tr>
<td>Operation time (h)</td>
<td>5.49 (0.59)</td>
<td>5.41 (0.49)</td>
</tr>
<tr>
<td>Aortic clamp time (min)</td>
<td>76.2 (25.6)</td>
<td>102.0 (39.1)</td>
</tr>
<tr>
<td>Temperature at baseline ((^\circ)C)</td>
<td>35.0 (0.48)</td>
<td>34.8 (0.56)</td>
</tr>
<tr>
<td>FEV1 (%)</td>
<td>101 (13)</td>
<td>93 (12)</td>
</tr>
<tr>
<td>FVC (%)</td>
<td>102 (16)</td>
<td>93 (10)</td>
</tr>
</tbody>
</table>

There was no significant difference in fluid balance (fluid administered minus fluid loss) between the groups during the 3-h study period (OLC, 230 (635) ml h\(^{-1}\); CMV, 11 (413) ml h\(^{-1}\)). In each group, five patients received phenylephrine (0.4 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)) and two patients received dobutamine.

**Discussion**

We found that greater PEEP after lung recruitment did not significantly affect PVR or REF in patients after cardiac surgery. Because baseline values were different, we analysed changes from baseline. These baseline differences probably occurred because of differences in the patients’ characteristics; the OLC group tended to have a greater predicted mortality (Euroscore) and more CABG operations. Factors that could affect the haemodynamic parameters, such as dobutamine, phenylephrine, propofol and enoximone, were used in both groups comparably. Because of the long operating times, the effect of enoximone would be almost over when the patients were studied.

To study the effects of ventilation on right ventricular performance, cardiac function is best considered in processes that affect right ventricular preload, contractility and afterload.\(^{12}\)

Efforts were made to maintain right ventricular preload constant. Fluid management was not based on wedge pressure, because this varied with intrathoracic pressure, but on the right ventricular EDVI. Compared with baseline, EDVI was comparable between the groups, suggesting comparable right ventricular preload. However, cardiac output depends on right as well as left ventricular preload. Since the ventricles share a common interventricular septum and are housed in a common pericardial sac, which limits their volume, right ventricular EDVI will affect left ventricle EDVI.\(^{12}\) In particular, increased PEEP can restrict left ventricular filling by leftward displacement of the interventricular septum.\(^{13}\) However, ventricular interdependence can be ruled out in our patients because the pericardial sac was not closed after surgery, and therefore could not limit ventricular volume.
Right ventricular afterload is difficult to assess, since the measures that could be used are not easy to obtain and do not only depend on afterload. Commonly used measures of afterload are PVR and REF.

PVR is often criticized because the calculation of resistance assumes that the vessels have rigid walls. Because the pulmonary vessels can collapse, their pressure–flow relationship is not linear, and a linear relationship is only likely if left atrial pressure is equal to or greater than pleural pressure (West zone 3). To assess pulmonary vascular resistance, Naeije suggests a pressure–flow diagram (Fig. 3). The pressure decrease across the pulmonary circulation is displayed on the vertical axis and CI on the horizontal axis. If changes in this plot are compared with baseline values, then pulmonary vasoconstriction or dilatation may be inferred.

REF is inversely related to right ventricular afterload. In the present study, REF did not differ between groups, suggesting that the OLC treatment did not affect afterload. Our findings contrast with previous clinical studies, in which afterload was greater when PEEP was greater. In these studies, however, greater PEEP was used without a recruitment manoeuvre, so that atelectasis would persist. Experimentally, atelectasis can increase right ventricular afterload, causing right ventricular failure in the longer term. Thus, a greater PEEP without recruitment may increase right ventricular afterload by affecting atelectatic lung regions or by overdistending healthy lung parts.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Baseline</th>
<th>30 min</th>
<th>60 min</th>
<th>90 min</th>
<th>120 min</th>
<th>150 min</th>
<th>180 min</th>
<th>Mean change from baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (min⁻¹)</td>
<td>OLC</td>
<td>82 (13)</td>
<td>84 (10)</td>
<td>82 (10)</td>
<td>84 (10)</td>
<td>82 (13)</td>
<td>89 (16)</td>
<td>89 (16)</td>
<td>2 (3)</td>
</tr>
<tr>
<td></td>
<td>CMV</td>
<td>90 (10)</td>
<td>89 (6)</td>
<td>90 (10)</td>
<td>92 (6)</td>
<td>95 (10)</td>
<td>94 (13)</td>
<td>94 (13)</td>
<td>3 (3)</td>
</tr>
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<td>MAP (mm Hg)</td>
<td>OLC</td>
<td>78 (9)</td>
<td>81 (11)</td>
<td>79 (14)</td>
<td>75 (8)</td>
<td>81 (13)</td>
<td>86 (12)</td>
<td>83 (12)</td>
<td>2 (6)</td>
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<tr>
<td></td>
<td>CMV</td>
<td>76 (13)</td>
<td>77 (10)</td>
<td>83 (13)</td>
<td>81 (10)</td>
<td>82 (13)</td>
<td>80 (10)</td>
<td>78 (10)</td>
<td>4 (6)</td>
</tr>
<tr>
<td>CI (litre min⁻¹ m⁻²)</td>
<td>OLC</td>
<td>3.2 (1.0)</td>
<td>3.2 (0.7)</td>
<td>3.2 (0.6)</td>
<td>3.3 (0.7)</td>
<td>3.4 (0.8)</td>
<td>3.6 (0.7)</td>
<td>3.6 (0.7)</td>
<td>0.3 (0.3)</td>
</tr>
<tr>
<td></td>
<td>CMV</td>
<td>3.2 (0.5)</td>
<td>2.4 (0.4)</td>
<td>2.6 (0.4)</td>
<td>2.6 (0.5)</td>
<td>2.8 (0.5)</td>
<td>2.7 (0.5)</td>
<td>3.0 (0.6)</td>
<td>0.0 (0.3)</td>
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<tr>
<td>REF (%)</td>
<td>OLC</td>
<td>37 (14)</td>
<td>29 (6)</td>
<td>34 (9)</td>
<td>31 (6)</td>
<td>37 (10)</td>
<td>36 (10)</td>
<td>41 (13)</td>
<td>-1.5 (7.9)</td>
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<tr>
<td></td>
<td>CMV</td>
<td>33 (10)</td>
<td>35 (11)</td>
<td>34 (13)</td>
<td>34 (13)</td>
<td>33 (11)</td>
<td>38 (12)</td>
<td>34 (11)</td>
<td>1.1 (7.6)</td>
</tr>
<tr>
<td>PVR (dynes cm⁻²)</td>
<td>OLC</td>
<td>149 (40)</td>
<td>167 (35)</td>
<td>160 (37)</td>
<td>154 (47)</td>
<td>151 (56)</td>
<td>147 (55)</td>
<td>141 (32)</td>
<td>2.0 (41)</td>
</tr>
<tr>
<td></td>
<td>CMV</td>
<td>125 (87)</td>
<td>146 (82)</td>
<td>138 (68)</td>
<td>118 (47)</td>
<td>122 (51)</td>
<td>128 (51)</td>
<td>109 (47)</td>
<td>0.9 (41)</td>
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<tr>
<td>RAP (mm Hg)</td>
<td>OLC</td>
<td>10 (2)</td>
<td>14 (2)</td>
<td>14 (3)</td>
<td>14 (4)</td>
<td>15 (3)</td>
<td>15 (3)</td>
<td>17 (7)</td>
<td>5.1 (3)</td>
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<tr>
<td></td>
<td>CMV</td>
<td>9 (3)</td>
<td>9 (2)</td>
<td>10 (2)</td>
<td>10 (2)</td>
<td>10 (3)</td>
<td>10 (3)</td>
<td>10 (3)</td>
<td>0.6 (3)</td>
</tr>
<tr>
<td>PAmean (mm Hg)</td>
<td>OLC</td>
<td>19 (2)</td>
<td>24 (3)</td>
<td>24 (3)</td>
<td>24 (5)</td>
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<tr>
<td></td>
<td>CMV</td>
<td>19 (5)</td>
<td>20 (5)</td>
<td>21 (6)</td>
<td>21 (5)</td>
<td>21 (5)</td>
<td>21 (4)</td>
<td>20 (3)</td>
<td>2.0 (4)</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>OLC</td>
<td>10 (2)</td>
<td>14 (3)</td>
<td>14 (3)</td>
<td>15 (2)</td>
<td>15 (5)</td>
<td>17 (6)</td>
<td>18 (8)</td>
<td>6.0 (4)</td>
</tr>
<tr>
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<td>CMV</td>
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<td>11 (3)</td>
<td>11 (3)</td>
<td>11 (4)</td>
<td>10 (4)</td>
<td>11 (3)</td>
<td>0.8 (4)</td>
</tr>
<tr>
<td>EDVI (ml m⁻²)</td>
<td>OLC</td>
<td>93 (21)</td>
<td>95 (18)</td>
<td>95 (15)</td>
<td>102 (17)</td>
<td>97 (12)</td>
<td>90 (15)</td>
<td>87 (19)</td>
<td>-1.5 (8)</td>
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<td>CMV</td>
<td>107 (22)</td>
<td>112 (21)</td>
<td>111 (26)</td>
<td>118 (32)</td>
<td>122 (30)</td>
<td>110 (21)</td>
<td>117 (27)</td>
<td>1.1 (8)</td>
</tr>
</tbody>
</table>

HR=heart rate; MAP=mean arterial pressure; CI=cardiac index; REF=right ventricular ejection fraction; PVR=pulmonary vascular resistance; RAP=right atrial pressure; PAmean=mean pulmonary artery pressure; PCWP=pulmonary capillary wedge pressure; EDVI=right ventricular end diastolic volume index.

Fig 3 Dynamic pressure–flow diagram indicating changes in pulmonary vascular resistance. Baseline values of CI and the pressure decrease through the pulmonary circulation are placed at the intersection of the axes. Measurements at 60, 90, 120, 150 and 180 min are plotted as differences from baseline for each patient.
Recruitment manoeuvres could re-expand atelectatic lung, and, if combined with low tidal volumes, this would reduce the increment in the right ventricular afterload during ventilation with greater PEEP. In healthy, non-intubated volunteers without atelectasis, 12.5 cm H\(_2\)O PEEP did not increase right ventricular afterload, supporting our results. The open lung concept advocates recruitment manoeuvres followed by low tidal volumes.\(^3\) We found no evidence of increased right ventricular afterload during ventilation according to the OLC in patients after cardiac surgery. The question remains whether these results can be generalized to patients with an intact pericardium, and this question warrants further investigation.

**Acknowledgement**

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**References**

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**Table 3** Respiration values during the study. Data are mean (SD)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>Baseline</th>
<th>30 min</th>
<th>60 min</th>
<th>90 min</th>
<th>120 min</th>
<th>150 min</th>
<th>180 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>( P_{\text{aw}} ) (kPa)</td>
<td>OLC</td>
<td>4.5 (0.3)</td>
<td>4.8 (0.5)</td>
<td>4.7 (0.4)</td>
<td>4.8 (0.5)</td>
<td>4.7 (0.5)</td>
<td>4.9 (0.8)</td>
<td>4.9 (0.8)</td>
</tr>
<tr>
<td>CMV</td>
<td>5.1 (0.7)</td>
<td>4.8 (0.5)</td>
<td>5.0 (0.5)</td>
<td>5.0 (0.5)</td>
<td>5.0 (0.3)</td>
<td>5.0 (0.3)</td>
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<td></td>
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<tr>
<td>( P_{\text{peak}} ) (cm H(_2)O)</td>
<td>OLC</td>
<td>21.8 (3)</td>
<td>25.3 (5)</td>
<td>25.2 (5)</td>
<td>25.3 (4)</td>
<td>24.5 (4)</td>
<td>25.6 (4)</td>
<td>24.4 (4)</td>
</tr>
<tr>
<td>CMV</td>
<td>21.9 (4)</td>
<td>22.1 (3)</td>
<td>22.0 (4)</td>
<td>22.4 (5)</td>
<td>21.7 (5)</td>
<td>21.9 (4)</td>
<td>21.0 (4)</td>
<td></td>
</tr>
<tr>
<td>( P_{\text{PEEPtot}} ) (cm H(_2)O)</td>
<td>OLC</td>
<td>12 (1)</td>
<td>14 (3)</td>
<td>14 (4)</td>
<td>14 (5)</td>
<td>15 (4)</td>
<td>14 (4)</td>
<td>14 (3)</td>
</tr>
<tr>
<td>CMV</td>
<td>7.6 (1)</td>
<td>14 (5)</td>
<td>14 (4)</td>
<td>14 (5)</td>
<td>14 (4)</td>
<td>14 (3)</td>
<td>14 (2)</td>
<td></td>
</tr>
<tr>
<td>Respiratory rate (b.p.m.)</td>
<td>OLC</td>
<td>11 (0.7)</td>
<td>11 (0.7)</td>
<td>11 (0.8)</td>
<td>11 (0.8)</td>
<td>11 (0.8)</td>
<td>11 (0.8)</td>
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<td>CMV</td>
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<td>7 (2)</td>
<td>8 (0)</td>
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<td>7 (3)</td>
<td></td>
</tr>
<tr>
<td>MV (litre min(^{-1}))</td>
<td>OLC</td>
<td>4.9 (0.3)</td>
<td>5.1 (0.3)</td>
<td>5.2 (0.6)</td>
<td>5.2 (0.6)</td>
<td>5.2 (0.6)</td>
<td>5.0 (0.0)</td>
<td>5.0 (0.0)</td>
</tr>
<tr>
<td>CMV</td>
<td>17 (3)</td>
<td>17 (3)</td>
<td>17 (4)</td>
<td>17 (4)</td>
<td>18 (4)</td>
<td>17 (4)</td>
<td>16 (4)</td>
<td></td>
</tr>
<tr>
<td>( \Delta P ) (cm H(_2)O)</td>
<td>OLC</td>
<td>8 (0)</td>
<td>7 (2)</td>
<td>8 (0)</td>
<td>7 (1)</td>
<td>7 (1)</td>
<td>7 (1)</td>
<td>7 (3)</td>
</tr>
</tbody>
</table>

\( P_{\text{peak}} \)= peak pressure; \( \Delta P = P_{\text{peak}} \) – \( P_{\text{PEEPtot}} \); \( P_{\text{PEEPtot}} = \) external PEEP + intrinsic PEEP; MV = minute ventilation.
