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 reduce shear forces caused by repeated opening and closing of atelectatic lung.¹ ² 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 given 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...
undergone CABG and/or valve surgery with use of cardiopulmonary 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 normal 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 suxamethonium 0.1–0.2 mg kg$^{-1}$ and was not reversed. Administration 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 (CCO 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 μg kg$^{-1}$) as needed. None of the patients received corticosteroids. During operation the lungs were ventilated with a set plan using starch colloids. If a maximum daily volume of 4–6 ml kg$^{-1}$, aiming at a $P_{aco2}$ 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 H2O for 40 s to increase the $P_{ao2}/F_{io2}$ ratio to a value greater than 50 kPa. If not, a recruitment manoeuvre was repeated by increasing peak pressure 5 cm H2O greater than before, up to a maximum peak pressure of 60 cm H2O until the $P_{ao2}/F_{io2}$ ratio became greater than 50 kPa. If the $P_{ao2}/F_{io2}$ ratio decreased slowly below 50 kPa after recruitment, PEEP was increased by 2 cm H2O and a recruitment manoeuvre (beginning at 40 cm H2O) was repeated.

Cardiovascular measurements consisted of right atrial pressure (RAP), mean pulmonary arterial pressure ($P_{a mean}$) 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 ventricular ejection fraction (REF). From these values, pulmonary vascular resistance (PVR) was calculated.

After the 3-h study period, if temperature and cardiovascular measurements were satisfactory, sedation was stopped and the patients were weaned from ventilation. Data on outcome 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 (ANOVA) for repeated measurements (PROC MIXED procedures from SAS).11

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 pneumothorax 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 $F_{IO2}$ to maintain $P_{aO2}$ greater than 10 kPa. In the OLC group, $5.3 (3)$ recruitment attempts were made with a mean peak pressure of $45.5 (2)$ cm H$_2$O to open the lung (Fig. 1). To keep the lung open, a total PEEP of $17.0 (2.7)$ cm H$_2$O 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 ($P_{A_{mean}}$–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 $P_{aCO2}$ 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).

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 mg 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, phleylephrine, 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\textsuperscript{14} 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.\textsuperscript{15–19} In these studies, however, greater PEEP was used without a recruitment manoeuvre, so that atelectasis would persist.\textsuperscript{20} Experimentally, atelectasis can increase right ventricular afterload, causing right ventricular failure in the longer term.\textsuperscript{21} Thus, a greater PEEP without recruitment may increase right ventricular afterload by affecting atelectatic lung regions or by overdistending healthy lung parts.\textsuperscript{22}

Table 2

<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>
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<td>84 (10)</td>
<td>82 (10)</td>
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<td>89 (16)</td>
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<td>79 (14)</td>
<td>75 (8)</td>
<td>81 (13)</td>
<td>86 (12)</td>
<td>83 (12)</td>
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<tr>
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<td>77 (10)</td>
<td>83 (13)</td>
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<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>
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<tr>
<td></td>
<td>CMV</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>
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<td>REF (%)</td>
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<td>29 (6)</td>
<td>34 (9)</td>
<td>31 (6)</td>
<td>37 (10)</td>
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<td>38 (12)</td>
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<td>151 (56)</td>
<td>147 (55)</td>
<td>141 (32)</td>
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<td>OLC</td>
<td>19 (2)</td>
<td>24 (3)</td>
<td>24 (3)</td>
<td>24 (5)</td>
<td>25 (4)</td>
<td>26 (5)</td>
<td>27 (8)</td>
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<td>21 (6)</td>
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<td>PCWP (mm Hg)</td>
<td>OLC</td>
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<tr>
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<td>CMV</td>
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<td>11 (3)</td>
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<td>11 (4)</td>
<td>10 (4)</td>
<td>11 (3)</td>
<td>0.8 (4)</td>
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<tr>
<td>EDVI (ml m\textsuperscript{-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>
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</tr>
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<td></td>
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<td>111 (26)</td>
<td>118 (32)</td>
<td>122 (30)</td>
<td>110 (21)</td>
<td>117 (27)</td>
<td>1.3 (8)</td>
</tr>
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</table>

HR=heart rate; MAP=mean arterial pressure; CI=cardiac index; REF=right ventricular ejection fraction; PVR=pulmonary vascular resistance; RAP=right atrial pressure; PA\textsubscript{mean}=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 H₂O PEEP did not increase right ventricular afterload, supporting our results. The open lung concept advocates recruitment manoeuvres followed by elevated PEEP levels and low driving pressures, resulting in low tidal volumes. 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.

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**References**

