Repetitive vital capacity manoeuvres after cardiopulmonary bypass: effects on lung function in a pig model

L. MAGNUSSON, S. WICKY, H. TYDÉN AND G. HEDENSTIERNA

Summary
Respiratory failure following cardiopulmonary bypass (CPB) is a major complication after cardiac surgery. A vital capacity inflation of the lungs, performed before the end of CPB, may improve gas exchange, but the necessity to repeat it is unclear. Therefore, we studied 18 pigs undergoing hypothermic CPB. A vital capacity manoeuvre (VCM) was performed in two groups and consisted of inflating the lungs for 15 s to 40 cm H2O at the end of CPB. In one group, VCM was repeated every hour. The third group served as controls. Atelectasis was studied by CT scan. Intrapulmonary shunt increased after bypass in the controls and improved spontaneously 3 h later without returning to baseline values. From 3 to 6 h after CPB, there was no more improvement and more than 10% atelectasis remained at 6 h. In contrast, the two groups treated before termination of CPB with VCM showed only minor atelectasis and no abnormal changes in gas exchange directly after bypass or later. We conclude that the protective effect of VCM remained for 6 h after bypass, and there was no extra benefit on gas exchange by repeating the VCM. (Br. J. Anaesth. 1998; 80: 682–684)

Keywords: anaesthesia general; lung atelectasis; heart cardiopulmonary bypass; hypoxaemia; lung shunting; lung function; pig

Postoperative respiratory insufficiency is a major complication after cardiac surgery with cardiopulmonary bypass (CPB).1 In a recent study, using a pig model, we found that CPB caused more atelectasis (35% of the total lung area) than in a control group without CPB (2%).2 We also found that a vital capacity manoeuvre (VCM) (inflating the lungs to 40 cm H2O for 15 s before the end of CPB) prevented atelectasis and gas exchange impairment after CPB,3 but we only followed changes for 1 h after termination of bypass. In healthy patients during general anaesthesia, VCM has an effect limited in time, and atelectasis can recur.4 We hypothesized that atelectasis also recurs after CPB if VCM is applied at the end of bypass.

Therefore, in this study we assessed the duration of benefit of VCM performed just before termination of bypass and investigated if there was any advantage in repeating VCM.

Methods and results
After obtaining approval from the Animal Research Ethics Committee of Uppsala University, we studied 18 pigs subjected to CPB. In six pigs VCM was performed 5 min before the end of bypass (group VCM 1); in another six pigs, VCM was performed 5 min before termination of CPB and then repeated every hour (group VCM 6). The six remaining pigs were treated as controls and VCM was not performed (control group). VCM consisted of inflating the lungs to 40 cm H2O for 15 s. The same technique was applied to the two VCM groups with an opened chest at the end of bypass, and after bypass with a closed chest in group VCM 6.

After induction of general anaesthesia, tracheal intubation was performed and artificial ventilation instituted using a volume-cycled ventilator. Tidal volume was 10 ml kg−1 and ventilatory frequency was adjusted to maintain end-tidal carbon dioxide partial pressure at 5.2–5.6 kPa, with a positive end-expiratory pressure of 4 cm H2O. The ventilator settings were maintained constant throughout the procedure. The inspired oxygen fraction was 0.4 (balance nitrogen).

A median sternotomy was performed, and the pericardium and both pleura were opened. Percussion was conducted using a non-pulsatile pump primed with Ringer’s acetate solution 600 ml and 15% mannitol 200 ml. After the beginning of CPB, hypothermia to 30°C was induced, ventilation was stopped and the airway opened to atmosphere. The aorta was clamped and cardioplegic solution injected until cardiac arrest (total duration of cardiac ischaemia = 45 min). During CPB, chest drains were inserted in both pleura.

Before separation from bypass, the pigs were rewarmed for 40 min. Total duration of CPB was 90 min. At the end of bypass, all pump prime was returned to the animals via the aortic cannula. Fifteen minutes before termination of bypass, ventilation was reinstituted at half the tidal volume value. Just before termination of bypass, VCM was performed in the two VCM groups.

Measurements consisted of blood-gas tensions, standard haemodynamic variables, extravascular lung water and respiratory mechanics.2 Intrapulmonary
Table 1  Haemodynamic and respiratory variables (mean (SD)). CPB=Cardiopulmonary bypass, VCM=vital capacity manoeuvre, $P_{aw}=$end-inspiratory pressure, MPAP=mean pulmonary arterial pressure. *$P<0.05$, **$P<0.01$, ***$P<0.001$ compared with baseline; ††$P<0.05$, †††$P<0.01$, ††††$P<0.001$ compared with the control group; ‡‡‡$P<0.05$, ‡‡‡‡$P<0.01$ compared with the VCM 1 group.

<table>
<thead>
<tr>
<th></th>
<th>Control group</th>
<th>VCM 1 group</th>
<th>VCM 6 group</th>
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<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>6 h after CPB</td>
<td>Baseline</td>
</tr>
<tr>
<td>$P_{aw}$ (cm H₂O)</td>
<td>16.0 (1.0)</td>
<td>19.7 (1.4)**</td>
<td>15.7 (1.4)</td>
</tr>
<tr>
<td>Compliance (ml cm H₂O⁻¹)</td>
<td>26.2 (4.4)</td>
<td>16.3 (3.3)**</td>
<td>24.4 (3.8)</td>
</tr>
<tr>
<td>MPAP (mm Hg)</td>
<td>17.3 (2.7)</td>
<td>24.7 (1.6)**</td>
<td>17.8 (0.8)</td>
</tr>
<tr>
<td>Shunt (%)</td>
<td>4.2 (1.6)</td>
<td>8.8 (5.5)</td>
<td>4.3 (3.1)</td>
</tr>
<tr>
<td>$P_{aw}/P_{aw_{0}}$ (mm Hg)</td>
<td>476 (55)</td>
<td>349 (105)*</td>
<td>484 (98)</td>
</tr>
<tr>
<td>Atelectasis (%)</td>
<td>11.8 (7.3)</td>
<td>1.8 (2.2)†**</td>
<td>1.8 (2.2)†**</td>
</tr>
<tr>
<td>Poorly aerated lung (%)</td>
<td>36.1 (13.5)</td>
<td>17.0 (6.4)††</td>
<td>17.0 (6.4)††</td>
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shunt was calculated using standard formulae. A delay of 30 min was allowed after the surgical preparation before baseline measurements were made. Because of the time necessary to remove the CPB cannulae and close the chest, the first measurements after bypass were not obtained until 20–30 min after separation from bypass. The same measurements were repeated at 3 and 6 h after CPB. After the last series of measurement, the pigs were moved to the CT scan laboratory. One CT scan was performed at end-expiration, 0–1 cm above the diaphragm. Non-aerated lung tissue (atelectasis) was defined as regions with attenuation values between −100 and +100 Hounsfield Units (HU) and poorly aerated lung tissue as regions with values between −500 and −100 HU. The extent of atelectasis and poorly aerated lung tissue was expressed as a percentage of total lung area.

Power analysis indicated that six pigs were needed in each group to detect a 50% decrease in atelectasis by repeated VCM compared with a single manoeuvre, with known SD values in the atelectatic area and reproducibility of the method. Data are presented as mean (SD). Analysis of variance (ANOVA) with Bonferroni–Dunn as the post hoc test was used for baseline comparisons. The effect of bypass and different VCM exposures were calculated by ANOVA for repeated measurements. $P<0.05$ was considered significant.

The pigs were similar in weight (controls 31.0 (3.9) kg; group VCM 1 31.8 (3.9) kg; and group VCM 6 29.5 (3.2) kg). After bypass, end-inspiratory airway pressures ($P_{aw}$) decreased significantly in all groups but was more pronounced in the control group. Three hours after bypass, $P_{aw}$ was significantly in group VCM 6 than in the two other groups. Six hours after CPB, $P_{aw}$ had returned to pre-bypass values only in group VCM 6. Compliance decreased in the control group after bypass and was significantly less than in the two VCM groups. This difference persisted at 3 and 6 h after bypass. Compliance also decreased significantly in group VCM 1 at 3 and 6 h after bypass, and was significantly less than in group VCM 6. There were no significant changes in haemodynamic variables, except for mean pulmonary arterial pressure (table 1).

Intrapulmonary shunt increased markedly (from 4 to 17%; $P<0.05$) (fig. 1) and $P_{aw}/P_{aw_{0}}$ decreased (from 476 to 265 mm Hg; $P<0.01$) after bypass in the control group, and not in the two VCM groups. Three hours after bypass, intrapulmonary shunt decreased and $P_{aw}/P_{aw_{0}}$ increased in the control group and remained stable thereafter, but the difference compared with the two VCM groups was still significant (table 1, fig. 1). There was no difference in shunt between the two VCM groups.

Atelectasis accounted for more than 10% and poorly aerated lung tissue for 36% of the total lung area in the control group, which was significantly greater ($P<0.01$) than in the two VCM groups (table 1). There was no difference between the two VCM groups in the amount of atelectasis and poorly aerated lung.

Comment

We have shown previously in a pig model that VCM performed just before termination of CPB is effective in preventing atelectasis and gas exchange impairment after bypass. In this study, the control group demonstrated spontaneous improvement in gas exchange between measurements made directly after termination of bypass and 3 h later (fig. 1). This may indicate that conventional ventilation with a low level of PEEP (5 cm H₂O) slowly reopens partially collapsed lung tissue. However, from 3 to 6 h after the end of bypass there was no more improvement and more than 10% atelectasis remained at 6 h. Thus alveolar recruitment appears to be a slow and limited process with conventional ventilation with PEEP. In contrast, the two groups treated before termination of CPB with VCM showed only minor atelectasis and no abnormal changes in gas exchange directly after bypass or later. Thus the beneficial effect of VCM was still present 6 h after the end of bypass, with less atelectasis and gas exchange impairment compared with the control group.
group. This effect was significant despite the spontaneous improvement in the control group. There was no effect on atelectasis or gas exchange from repeating the VCM every hour but repeated lung inflation improved respiratory compliance.

Although previous workers have investigated the effect of various lung management procedures during CPB, few have followed this effect for some hours. Some found a beneficial effect of CPAP just after CPB but this difference was not evident at 4–8 h after the end of bypass. Boldt and colleagues found that lung water returned to normal 5 h after CPB in all patients except those with no CPAP or CPAP 15 cm H$_2$O, but shunt and $P_aO_2$ did not return to pre-bypass values in any group. In contrast with these results, our study showed that VCM performed before the end of bypass was effective in preventing postoperative impairment in gas exchange.

We conclude that spontaneous but limited improvement in gas exchange was seen in the control group, that the protective effect of VCM remained at least for 6 h and that there was no extra benefit on gas exchange by repeating the VCM every hour after CPB. However, before extrapolating these findings to cardiac surgical patients, they should be tested in human controlled studies, with particular attention to the potential harmful effects of VCM.

Acknowledgements

This study was supported by grants from the Swedish Medical Research Council (No.5315) and the Swedish Heart-Lung Fund.

References