**Influence of a Vital Capacity Maneuver on Pulmonary Gas Exchange After Cardiopulmonary Bypass**

Glenn S. Murphy, MD, Joseph W. Szokol, MD, Ronald D. Curran, MD, Timothy V. Votapka, MD, and Jeffery S. Vender, MD

**Objective:** To investigate the effect of a single, vital capacity breath (vital capacity maneuver [VCM]), administered at the end of cardiopulmonary bypass (CPB), on pulmonary gas exchange in patients undergoing coronary artery bypass graft surgery.

**Design:** Prospective, randomized, double-blind study.

**Setting:** University-affiliated hospital.

**Participants:** Forty patients scheduled for elective coronary artery bypass graft surgery and early tracheal extubation.

**Interventions:** Patients were randomized to 1 of 2 groups. VCM patients received a VCM at the conclusion of CPB. Control patients received no VCM.

**Measurements and Main Results:** Intrapulmonary shunt (Qs/QT), arterial oxygenation (Pao2), and alveolar-arterial oxygen gradients (P(A-a)O2) were measured after induction of anesthesia, CPB, intensive care unit (ICU) arrival, and extubation. The duration of postoperative intubation was recorded for each group. Qs/QT increased significantly 30 minutes after CPB in the control group (15.7 ± 1.8% to 27.4 ± 2.6%; p = 0.01). In the VCM group, a small decrease in Qs/QT occurred (16.1 ± 2.0% to 14.9 ± 2.0%). After ICU arrival and extubation, no significant difference in Qs/QT existed between the 2 groups. With the exception of a higher P(A-a)O2 in the control group at induction of anesthesia, no differences in Pao2 or P(A-a)O2 were present between the 2 groups at any measurement interval. Patients who received a VCM were extubated earlier than the control group (6.5 ± 2.1 hours vs 9.4 ± 4.2 hours; p = 0.01).

**Conclusion:** The use of a VCM prevented an increase in Qs/QT from occurring in the operating room. Although a VCM did not influence pulmonary gas exchange in the ICU, its application in the operating room appears to exert a beneficial effect on tracheal extubation times after cardiac surgery.

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**KEY WORDS:** vital capacity maneuver, cardiopulmonary bypass, intrapulmonary shunt, arterial oxygenation

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**METHODS**

After receiving institutional review board approval, informed consent was obtained from 40 patients scheduled for elective primary CAGB surgery. Previous studies have shown that Qs/QT doubles after CPB.9,10 Assuming that a VCM would eliminate an increase in shunt, a sample size of 40 would have an 80% power of determining such a difference with a 2-sided α of 0.05. Exclusion criteria included (1) pre-existing severe chronic pulmonary dysfunction (defined as pulmonary disease requiring home oxygen therapy or causing shortness of breath after 1 to 2 flights of stairs), (2) preoperative pulmonary edema, (3) ejection fraction < 40%, and (4) morbid obesity (a body mass index > 27). To avoid the influence of the time of intensive care unit (ICU) arrival (early afternoon vs early evening) on tracheal extubation, all patients enrolled in this study were the first cases of the day.

On the morning of surgery, patients were randomly assigned (sealed envelopes) to receive a VCM (VCM group) or no VCM (control group). The VCM was administered at the completion of CPB, immediately before resuming controlled ventilation of the lungs. The VCM was performed as previously described.4,5 The inflation of the lungs to an airway pressure of 40 cm H2O for 15 seconds, which corresponds to a volume close to vital capacity, has been shown to reduce Qs/QT in patients during general anesthesia. The VCM was administered by a clinician not involved in the management of the patient. Anesthesia providers stepped outside of the operating room when the VCM was applied (or not applied) to ensure blinding. All other participants in the study in the intraoperative and postoperative periods were blinded to group assignment. No re-expansion maneuver was used in control patients.

Patients were premedicated with midazolam, 2 to 4 mg, and fentanyl, 50 to 100 μg, in the holding area. A radial artery catheter and pulmonary artery catheter were placed under local anesthesia. A standardized
balanced anesthetic technique was used for each patient consisting of fentanyl, 15 to 18 μg/kg; midazolam, 4 to 6 mg; and supplemental isoflurane, 0.4% to 2.0%. Muscle relaxation was maintained with pancuronium. The lungs were mechanically ventilated with a tidal volume of 10 mL/kg and a fraction of inspired oxygen (FIO2) of 50% before and after CPB. Ventilatory rate was adjusted to maintain a PaCO2 of 32 to 36 mmHg. Positive end-expiratory pressure was not applied to any patient in the operating room or ICU. During CPB, the lungs were deflated and exposed to air. In the ICU, mechanical ventilation was standardized as previously described. After extubation, patients were placed on 50% high-flow oxygen masks.

The CPB circuit consisted of a membrane oxygenator (Medtronic Cardiovascular, Brooklyn Park, MN), nonocclusive roller pumps, and an arterial filter. The oxygenator was primed with 2000 mL of crystalloid solution; 100 mL of mannitol 20%; sodium bicarbonate, 50 mEq; 50 mL of a 25% albumin solution; and bovine heparin, 10,000 units. Anticoagulation was induced with 300 units/kg of bovine heparin injected through a central catheter before aortic cannulation. Celite-activated coagulation times of >400 seconds were maintained during CPB. CPB was performed with flow rates of 2.4 to 2.8 L/min/m2 and mean blood pressures of 50 to 80 mmHg. During CPB, mild hypothermia of 33°C-35°C was used, and α-stat blood gas management was used on all patients. At the end of CPB, residual heparin was neutralized by protamine in a ratio of 0.75 mg of protamine for each 100 units of heparin administered.

Hemodynamic variables, PaO2, P(A-a)O2, and Qs/Qr, were obtained at 4 time intervals: 30 minutes after induction of anesthesia, 30 minutes after the termination of CPB, 30 minutes after ICU arrival, and 30 minutes after extubation. A pulmonary artery catheter (Abbott Critical Care Systems, Chicago, IL) was used to obtain pulmonary arterial hemodynamic variables, PaO2, P(A-a)O2, and Qs/Qr. In the control group, however, Qs/Qr returned to baseline values (14.4 ± 1.7%; p = 0.446). In the VCM group, Qs/Qr returned to baseline values (17.8 ± 1.7%; p = 0.585). Shunt increased in both groups after extubation, although this did not reach statistical significance (Table 4).

There were no differences in PaO2 between the control and VCM groups during any of the measurement periods. PaO2 significantly decreased in both groups after CPB and ICU arrival (p < 0.05, VCM and control) and was further reduced after extubation (p < 0.01, VCM and control). (A-a)O2 was higher in the control group than in the VCM group at baseline (131 ± 9 mmHg vs 107 ± 11 mmHg; p = 0.038). The 2 groups were similar in (A-a)O2 during the other measurement intervals. (A-a)O2 significantly increased from baseline values in both groups after CPB, ICU arrival, and extubation.

Every patient in this study was extubated within 24 hours of arrival in the ICU. Patients who received a VCM were extubated significantly earlier than patients in the control group (6.5 ± 2.1 hours vs 9.4 ± 4.2 hours; p = 0.01). There were no adverse clinical events associated with the application of the VCM.

### RESULTS

The main findings of this study were that the use of a VCM at the conclusion of CPB (1) prevented a significant

<table>
<thead>
<tr>
<th>Table 1. Demographic Data</th>
<th>Control (n = 20)</th>
<th>VCM (n = 20)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>60.2 ± 10.1</td>
<td>62.8 ± 9.9</td>
<td>NS</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>18/2</td>
<td>17/3</td>
<td>NS</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>172.9 ± 8.1</td>
<td>173.2 ± 11.4</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>87.3 ± 12.3</td>
<td>79.5 ± 15.7</td>
<td>NS</td>
</tr>
<tr>
<td>Concomitant cardiac medications</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-adrenergic blockers</td>
<td>16</td>
<td>15</td>
<td>NS</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>7</td>
<td>9</td>
<td>NS</td>
</tr>
<tr>
<td>Nitrates</td>
<td>6</td>
<td>9</td>
<td>NS</td>
</tr>
<tr>
<td>Associated diseases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>11</td>
<td>13</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes</td>
<td>4</td>
<td>4</td>
<td>NS</td>
</tr>
<tr>
<td>Prior myocardial infarction</td>
<td>5</td>
<td>6</td>
<td>NS</td>
</tr>
<tr>
<td>Renal insufficiency</td>
<td>1</td>
<td>1</td>
<td>NS</td>
</tr>
</tbody>
</table>

NOTE: Data are mean ± SD or number of patients. Abbreviations: VCM, vital capacity maneuver; NS, not significant.

Avarenics: VCM, vital capacity maneuver; NS, not significant.
Impaired pulmonary gas exchange is a major complication after cardiac surgery. Proposed mechanisms include surgical trauma, leukocyte activation,11 accumulation of extravascular lung water,12,13 microaggregation in the pulmonary capillaries,14 oxygen toxicity,15 and atelectasis. In patients undergoing CABG surgery or mitral valve surgery,2 an increase in Qs/QT was the primary cause of worsening PaO2 and P(A-a)O2 in the perioperative period. Increases in Qs/QT appear to contribute to prolonged requirements for mechanical ventilation after cardiac surgery.3

Lung management during CPB may have an influence on Qs/QT and arterial oxygenation. Ventilation of the nonperfused lungs during CPB has been shown to increase Qs/QT two hours after ICU arrival.1 Studies examining the use of continuous positive airway pressure during CPB yielded conflicting results. Improvements in Qs/QT and PaO2,16 transient improvements in P(A-a)O2,17 no change in Qs/QT,1,8 and worsening of Qs/QT and PaO2,16 have been shown after the application of continuous positive airway pressure during CPB. The influence of a VCM on gas exchange has not been previously studied in humans undergoing cardiac surgery. In patients undergoing elective neurological or laparoscopic procedures, the use of a VCM nearly eliminated the increase in Qs/QT that occurred after the induction of general anesthesia.4,5

In a pig model of CPB, marked increases in Qs/QT and decreases in PaO2 were observed after CPB in the control animals. No abnormal changes in Qs/QT or PaO2 occurred in the pigs that received a VCM.8

In the control group in the present study, a significant increase in Qs/QT was observed 30 minutes after the termination of CPB. A 2-fold increase in Qs/QT at the conclusion of CPB is consistent with other published studies.2,9,10 In the VCM group, no change in Qs/QT from baseline was observed. The mechanism by which a VCM prevented an increase in Qs/QT from occurring was not determined by this study. The development of atelectasis plays a primary role in the appearance of Qs/QT after CPB,19 and the application of a VCM has been shown to eliminate atelectasis and shunt.4,5

Thirty minutes after ICU arrival (approximately 2 hours after CPB), Qs/QT remained close to baseline values in the VCM group. In the control group, however, improvements in Qs/QT occurred so that no significant difference in shunt existed between the 2 groups. A reduction in Qs/QT to pre sternotomy levels has been observed 4 hours after CABG surgery, which is attributed to the slow re-expansion of collapsed alveoli by positive-pressure ventilation.2,20 Shunt again increased in both groups 30 minutes after extubation, although this did not reach statistical significance. No prior studies have measured Qs/QT in cardiac patients shortly after extubation. A change from positive-pressure ventilation to spontaneous ventilation can result in increases in Qs/QT in patients on the 1st postoperative day after cardiac surgery.2,20

The authors did not observe a beneficial effect of a VCM on either PaO2 or P(A-a)O2 at any of the measurement periods of the study. Although Qs/QT was significantly higher in the control group 30 minutes after CPB (27.4 ± 2.6% vs 14.9 ± 2.0%; p = 0.01), there was no difference in PaO2 or P(A-a)O2 between the 2 groups. These findings are consistent with the

### Table 2. Intraoperative Data

<table>
<thead>
<tr>
<th>Anesthetic management</th>
<th>Control (n = 20)</th>
<th>VCM (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total fentanyl (µg/kg)</td>
<td>15.4 ± 0.8</td>
<td>15.8 ± 0.4</td>
</tr>
<tr>
<td>Total pancuronium (mg)</td>
<td>19.4</td>
<td>20.0</td>
</tr>
<tr>
<td>Vasodilator use</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Vasoconstrictor use</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>Inotropic use</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Units of PRBC administered</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Total crystalloid</td>
<td>2942 ± 152</td>
<td>2730 ± 173</td>
</tr>
</tbody>
</table>

**Surgical management**

| Saphenous vein grafts | 2.5 ± 0.7 | 2.4 ± 0.9 |
| Internal mammary artery | 20 | 20 |
| Duration of CPB | 123.2 ± 4.7 | 124.0 ± 7.5 |

**NOTE.** Data are mean ± SD or number of patients. No statistically significant differences in any of these variables were found.

Abbreviations: VCM, vital capacity maneuver; PRBC, packed red blood cells; CPB, cardiopulmonary bypass.

### Table 3. Hemodynamic and Ventilatory Data

<table>
<thead>
<tr>
<th>Postinduction</th>
<th>Control</th>
<th>VCM</th>
<th>Control</th>
<th>VCM</th>
<th>Control</th>
<th>VCM</th>
<th>Control</th>
<th>VCM</th>
<th>Postextubation + 30 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>71 ± 16</td>
<td>65 ± 14</td>
<td>84 ± 9</td>
<td>82 ± 11</td>
<td>83 ± 11</td>
<td>82 ± 9</td>
<td>89 ± 11</td>
<td>89 ± 12</td>
<td></td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>84 ± 15</td>
<td>77 ± 11</td>
<td>75 ± 11</td>
<td>78 ± 11</td>
<td>79 ± 10</td>
<td>79 ± 12</td>
<td>76 ± 11</td>
<td>77 ± 10</td>
<td></td>
</tr>
<tr>
<td>PAD (mmHg)</td>
<td>14 ± 4</td>
<td>13 ± 3</td>
<td>12 ± 4</td>
<td>12 ± 4</td>
<td>13 ± 3</td>
<td>12 ± 3</td>
<td>12 ± 4</td>
<td>10 ± 3</td>
<td></td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>2.2 ± 0.3</td>
<td>2.3 ± 0.4</td>
<td>2.6 ± 0.5</td>
<td>2.6 ± 0.4</td>
<td>2.3 ± 0.5</td>
<td>2.4 ± 0.4</td>
<td>2.5 ± 0.3*</td>
<td>2.8 ± 0.5*</td>
<td></td>
</tr>
<tr>
<td>Minute volume (L)</td>
<td>6.7 ± 1.3</td>
<td>5.6 ± 1.5</td>
<td>6.4 ± 1.3</td>
<td>5.7 ± 1.3</td>
<td>6.8 ± 1.0</td>
<td>6.2 ± 1.2</td>
<td>6.8 ± 1.0</td>
<td>6.2 ± 1.2</td>
<td></td>
</tr>
<tr>
<td>Paw (cm H₂O)</td>
<td>28.6 ± 3.5</td>
<td>27.6 ± 4.2</td>
<td>29.1 ± 4.9</td>
<td>27.4 ± 5.2</td>
<td>28.4 ± 4.2</td>
<td>26.6 ± 4.1</td>
<td>28.4 ± 4.2</td>
<td>26.6 ± 4.1</td>
<td></td>
</tr>
<tr>
<td>Oxygen saturation (%)</td>
<td>98.6 ± 0.7</td>
<td>99.0 ± 0.7</td>
<td>98.3 ± 1.2</td>
<td>98.4 ± 1.1</td>
<td>98.4 ± 0.7</td>
<td>98.6 ± 1.2</td>
<td>97.8 ± 1.7</td>
<td>97.3 ± 2.0</td>
<td></td>
</tr>
</tbody>
</table>

**NOTE.** Data are mean ± SD.

Abbreviations: VCM, vital capacity maneuver; CPB, cardiopulmonary bypass; ICU, intensive care unit; HR, heart rate; MAP, mean arterial pressure; PAD, pulmonary artery diastolic pressure; CI, cardiac index; Paw, peak airway pressure.

* p < 0.05 (control vs VCM).
results of 2 previous VCM studies. In patients undergoing neurosurgical or elective general surgical procedures, the use of a VCM resulted in a 3-fold to 7-fold reduction in Qs/Qt. This large reduction in Qs/Qt was accompanied by only minor improvements in PaO2 and P(A-a)O2. Although the VCM patients were extubated significantly earlier, this was not due to improvements in pulmonary gas exchange. Many factors other than Qs/Qt and PaO2 influence the decision on when to extubate cardiac surgical patients. The ICU nurses, who were blinded to group assignment, determined when the patients were ready to be weaned from ventilatory support and extubated. There were no differences between the control and VCM groups in any of the criteria the nurses used to guide weaning and extubation, however. Chest tube output was <50 mL/h in all patients. Hemodynamics were similar in the 2 groups in the ICU. Other than 2 patients in each group on renal dose dopamine, no other inotropic support was used. Core temperature was maintained at >35.5°C and urine output was >0.5 mL/kg/h in all subjects. During the study period, crystalloid and blood product requirements were similar.

The reasons why a VCM resulted in earlier extubation times were not established by this study. One possible explanation involves the release of surfactant; a single large breath may lead to the release of surfactant, which has been shown to improve alveolar stability. In animal models, mechanical stretch of lung epithelial cells has been shown to result in sustained release of pulmonary surfactant for 30 minutes. A VCM may have beneficial effects on the mechanical properties of the lung. In patients receiving general anesthesia, improvements in compliance of the respiratory system persisted for at least 40 minutes after the application of a VCM. In a pig model, Magnusson et al concluded that a VCM prevented a decrease in pulmonary compliance from occurring after CPB. Accumulation of extravascular lung water occurs after separation from CPB, which may contribute to postoperative pulmonary dysfunction. Boldt et al showed that the use of continuous positive airway pressure during CPB had a significant influence on the amount of extravascular lung water after CABG surgery. A VCM may prevent an increase in extravascular lung water from occurring. In the present study, however, lung water parameters were not measured.

The present study has some potential limitations. The mechanism by which a VCM reduced Qs/Qt was not determined. In subjects under general anesthesia, a strong correlation between the amount of atelectatic lung tissue (measured by CT scanning) and the magnitude of Qs/Qt has been observed. A VCM has been shown to significantly reduce atelectasis and Qs/Qt. It is likely that a VCM influenced Qs/Qt after CPB through a reduction in atelectasis, as was reported in previous animal models. The present study provided no radiologic evidence to support this hypothesis, however. Transporting patients to the radiology suite for CT scanning was not possible during the intraoperative and early postoperative measurement periods. As discussed previously, the authors were unable to ascertain the reasons why a VCM performed in the operating room resulted in shorter postoperative intubation times. Improvements in pulmonary compliance and extravascular lung water may have occurred but were not measured in this study. Future studies to further define the effects of a VCM on respiratory mechanics and lung water are needed.

In conclusion, this study has shown that the application of a VCM at the end of CPB prevents an increase in Qs/Qt from occurring in the operating room. Although a VCM had no influence on pulmonary gas exchange in the early postoperative period, its use appears to have a beneficial effect on the duration of intubation after CABG surgery.

### Table 4. Gas Exchange Data

<table>
<thead>
<tr>
<th>Time Period</th>
<th>Control</th>
<th>VCM</th>
<th>Control</th>
<th>VCM</th>
<th>Control</th>
<th>VCM</th>
<th>Control</th>
<th>VCM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Shunt (%)</td>
<td></td>
<td>Qt (%)</td>
<td></td>
<td>Qt (%)</td>
<td></td>
<td>Qt (%)</td>
<td></td>
</tr>
<tr>
<td>Postinduction (Baseline)</td>
<td>15.7 ± 1.8</td>
<td>16.1 ± 2.0</td>
<td>27.4 ± 2.6*</td>
<td>14.9 ± 2.0*</td>
<td>17.8 ± 1.7</td>
<td>14.4 ± 1.7</td>
<td>21.4 ± 2.3</td>
<td>24.0 ± 2.1</td>
</tr>
<tr>
<td>CPB + 30 min</td>
<td>179 ± 14</td>
<td>208 ± 14</td>
<td>140 ± 18*</td>
<td>157 ± 14*</td>
<td>135 ± 11*</td>
<td>146 ± 11*</td>
<td>106 ± 7†</td>
<td>117 ± 8†</td>
</tr>
<tr>
<td>ICU Arrival + 30 min</td>
<td>131 ± 9†</td>
<td>107 ± 11‡</td>
<td>167 ± 16*</td>
<td>148 ± 13†</td>
<td>164 ± 11*</td>
<td>159 ± 11†</td>
<td>195 ± 7†</td>
<td>188 ± 8†</td>
</tr>
<tr>
<td>Postextubation + 30 min</td>
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<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**NOTE.** Data are mean ± SD.

Abbreviations: VCM, vital capacity maneuver; CPB, cardiopulmonary bypass; ICU, intensive care unit; PaO2, arterial oxygen tension; P(A-a)O2, alveolar-arterial PO2 difference.

* p < 0.05 (compared with baseline).
† p < 0.01 (compared with baseline).
‡ p < 0.05 (control v VCM).
§ p < 0.01 (control v VCM).
APPENDIX

Alveolar-arterial oxygen gradient:

\[
\text{P}(A-a)O_2 = \text{PAO}_2 - \text{PaO}_2
\]

\[
\text{PAO}_2 = F_iO_2 \cdot (PB - PH_2O) - (\text{PaCO}_2/RQ)
\]

\[
\text{PaO}_2 = \text{arterial oxygen}
\]

\[
\text{PaCO}_2 = \text{arterial carbon dioxide}
\]

\[
F_iO_2 = \text{inspired oxygen concentration}
\]

\[
PB = \text{barometric pressure}
\]

\[
PH_2O = \text{water vapor pressure}
\]

\[
RQ = \text{respiratory quotient (0.8)}
\]

REFERENCES


