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

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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 (Q_S/Q_T), arterial oxygenation (PaO_2), and alveolar-arterial oxygen gradients $(P(A-a)O_2)$ were measured after induction of anesthesia, CPB, intensive care unit (ICU) arrival, and extubation. The duration of postoperative intubation was recorded for each group. Q_S/Q_T increased significantly 30 minutes after CPB in the control group ($15.7 \pm 1.8\%$ to $27.4 \pm 2.6\%$; $p = 0.01$). In the VCM group, a small decrease in Q_S/Q_T occurred ($16.1 \pm 2.0\%$ to $14.9 \pm 2.0\%$). After ICU arrival and extubation, no significant difference in Q_S/Q_T existed between the 2 groups. With the exception of a higher $P(A-a)O_2$ in the control group at induction of anesthesia, no differences in PaO_2 or $P(A-a)O_2$ 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 v 9.4 ± 4.2 hours; $p = 0.01$).

Conclusion: The use of a VCM prevented an increase in Q_S/Q_T 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

GAS EXCHANGE is frequently impaired after cardiopulmonary bypass (CPB).[1,2] An increase in intrapulmonary shunt (Q_S/Q_T) is a major component of pulmonary dysfunction after cardiac surgery, with shunts of 22% to 31% commonly observed in the early postoperative period.[1,2] The development of atelectasis has been identified as the primary cause of Q_S/Q_T in these patients, and these pathologic changes may contribute to prolonged requirements for mechanical ventilation after CPB.[3]

In patients undergoing general anesthesia, the use of a vital capacity or recruitment maneuver (inflation of the lungs to a peak airway pressure of 40 cm H_2O for 15 seconds) reduced Q_S/Q_T and atelectasis.[4-7] In a pig model, the performance of a vital capacity maneuver (VCM) immediately before the termination of CPB prevented an increase in Q_S/Q_T and atelectasis from occurring 1 hour post-CPB.[8] In cardiac surgical patients, a large positive-pressure breath is commonly employed at the end of CPB to expand the collapsed lungs. The influence of this practice on perioperative gas exchange has not been examined in humans.

The aim of this clinical study was to determine the effect of a VCM, administered at the conclusion of CPB, on Q_S/Q_T, alveolar-arterial oxygen gradients $(P(A-a)O_2)$, and arterial oxygenation (PaO_2) during the intraoperative and early postoperative periods. This study also assessed the impact of a VCM on extubation times in patients scheduled for elective coronary artery bypass graft (CABG) surgery and early tracheal extubation.

METHODS

After receiving institutional review board approval, informed consent was obtained from 40 patients scheduled for elective primary CABG surgery. Previous studies have shown that Q_S/Q_T 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 v 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 H_2O for 15 seconds, which corresponds to a volume close to vital capacity, has been shown to reduce Q_S/Q_T 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 (F_IO_2) of 50% before and after CPB. Ventilatory rate was adjusted to maintain a $PaCO_2$ 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/m^2 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, PaO_2, $P(A-a)O_2$, and Q_S/Q_T, 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 pressures, and cardiac output was calculated by the thermodilution technique. Heart rate was determined from the electrocardiogram. Tidal volume, minute volume, and peak airway pressures were recorded from the Drager anesthesia machine (North American Drager, Telford, PA). Arterial and mixed venous blood gases were collected at each of the 4 study intervals, and Q_S/Q_T and $P(A-a)O_2$ were calculated using standard formulae (see Appendix). All fluids (crystalloids, colloids, and blood products) administered to the patient until the time of extubation were recorded. The use of vasodilators and vasoconstrictors, which can influence the magnitude of Q_S/Q_T, was noted in each group.

On arrival in the ICU, patients were sedated with a continuous infusion of propofol, 25 to 75 μg/kg/min. The propofol infusion was discontinued during weaning from mechanical ventilation. Criteria for weaning from ventilatory support included an appropriate sensorium, hemodynamic stability (cardiac index, >2.2 L/min/m^2; mean arterial pressure, >60 mmHg; pulmonary artery diastolic pressure, <20 mmHg; and no significant arrhythmias), $PaO_2 > 60$ mmHg with an $F_IO_2 < 50\%$, minimal chest tube output, urine output > 0.5 mL/kg/h, and a temperature > 35.5° centigrade. After 15 minutes on continuous positive airway pressure support, patients were extubated if the oxygen saturation was >94%, end-tidal carbon dioxide was <50 mmHg, and a negative inspiratory force > -20 mmHg could be generated.

The data are presented as mean values ± SD. Differences in nominal values were analyzed using the chi-square test. The significance of a difference between 2 conditions was analyzed by Student's paired t-test. Comparisons of different time points versus baseline measures within 1 group were performed using analysis of variance for repeated measurements followed by Dunnett's post-test. Differences between groups at the same time point were analyzed using Student's t-test.

RESULTS

The 2 groups were comparable with respect to age, height, weight, and cardiovascular medications (Table 1). There were

Table 1. Demographic Data

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

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

no significant differences between groups in surgical or anesthetic management (Table 2). Total doses (μg/kg) of fentanyl, pancuronium, and midazolam did not differ between groups. During the 4 measurement intervals, there were no significant differences between the VCM and control groups in heart rate, mean arterial pressure, pulmonary artery diastolic pressure, or cardiac index. Ventilatory parameters (minute volume, peak airway pressure, and oxygen saturation) were similar (Table 3).

At baseline, the control and VCM patients did not differ in Q_S/Q_T (15.7 ± 1.8% v 16.1 ± 2.0%; $p = 0.815$). Thirty minutes after termination of CPB, Q_S/Q_T markedly increased to 27.4 ± 2.6% in the control group ($p = 0.011$). In the VCM patients, a small decrease in Q_S/Q_T occurred (14.9 ± 2.0%; $p = 0.550$). Thirty minutes after ICU arrival, Q_S/Q_T remained at baseline levels in the VCM group (14.4 ± 1.7%; $p = 0.446$). In the control group, however, Q_S/Q_T 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 PaO_2 between the control and VCM groups during any of the measurement periods. PaO_2 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). $P(A-a)O_2$ was higher in the control group than the VCM group at baseline (131 ± 9 mmHg v 107 ± 11 mmHg; $p = 0.038$). The 2 groups were similar in $P(A-a)O_2$ during the other measurement intervals. $P(A-a)O_2$ 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 v 9.4 ± 4.2 hours; $p = 0.01$). There were no adverse clinical events associated with the application of the VCM.

DISCUSSION

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

Table 2. Intraoperative Data

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

increase in Q_S/Q_T from occurring in the operating room, (2) had no effect on PaO_2 or $P(A-a)O_2$ in the perioperative period, and (3) resulted in earlier tracheal extubation after CPB.

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[2] or mitral valve surgery,[9] an increase in Q_S/Q_T was the primary cause of worsening PaO_2 and $P(A-a)O_2$ in the perioperative period. Increases in Q_S/Q_T appear to contribute to prolonged requirements for mechanical ventilation after cardiac surgery.[3]

Lung management during CPB may have an influence on Q_S/Q_T and arterial oxygenation. Ventilation of the nonperfused lungs during CPB has been shown to increase Q_S/Q_T two hours after ICU arrival.[1] Studies examining the use of continuous positive airway pressure during CPB yielded conflicting results. Improvements in Q_S/Q_T and PaO_2,[16] transient improvements in $P(A-a)O_2$,[17] no change in Q_S/Q_T,[1,8] and worsening of Q_S/Q_T and PaO_2[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 neurosurgical or laparoscopic procedures, the use of a VCM nearly eliminated the increase in Q_S/Q_T that occurred after the induction of general anesthesia.[4,5] In a pig model of CPB, marked increases in Q_S/Q_T and decreases in PaO_2 were observed after CPB in the control animals. No abnormal changes in Q_S/Q_T or PaO_2 occurred in the pigs that received a VCM.[8]

In the control group in the present study, a significant increase in Q_S/Q_T was observed 30 minutes after the termination of CPB. A 2-fold increase in Q_S/Q_T at the conclusion of CPB is consistent with other published studies.[2,9,10] In the VCM group, no change in Q_S/Q_T from baseline was observed. The mechanism by which a VCM prevented an increase in Q_S/Q_T from occurring was not determined by this study. The development of atelectasis plays a primary role in the appearance of Q_S/Q_T 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), Q_S/Q_T remained close to baseline values in the VCM group. In the control group, however, improvements in Q_S/Q_T occurred so that no significant difference in shunt existed between the 2 groups. A reduction in Q_S/Q_T to presternotomy levels has been observed 4 hours after CABG surgery, which is attributed to the slow re-expansion of collapsed alveoli by positive-pressure ventilation.[2,9] Shunt again increased in both groups 30 minutes after extubation, although this did not reach statistical significance. No prior studies have measured Q_S/Q_T in cardiac patients shortly after extubation. A change from positive-pressure ventilation to spontaneous ventilation can result in increases in Q_S/Q_T 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 PaO_2 or $P(A-a)O_2$ at any of the measurement periods of the study. Although Q_S/Q_T was significantly higher in the control group 30 minutes after CPB (27.4 ± 2.6% v 14.9 ± 2.0%; $p = 0.01$), there was no difference in PaO_2 or $P(A-a)O_2$ between the 2 groups. These findings are consistent with the

Table 3. Hemodynamic and Ventilatory Data

	Postinduction		CPB + 30 min		ICU + 30 min		Postextubation + 30 min	
	Control	VCM	Control	VCM	Control	VCM	Control	VCM
HR (beats/min)	71 ± 16	65 ± 14	84 ± 9	82 ± 11	83 ± 11	82 ± 9	89 ± 11	89 ± 12
MAP (mmHg)	84 ± 15	77 ± 11	75 ± 11	78 ± 11	79 ± 10	79 ± 12	76 ± 11	77 ± 10
PAD (mmHg)	14 ± 4	13 ± 3	12 ± 4	12 ± 4	13 ± 3	12 ± 3	12 ± 4	10 ± 3
CI (L/min/m²)	2.2 ± 0.3	2.3 ± 0.4	2.6 ± 0.5	2.6 ± 0.4	2.3 ± 0.5	2.4 ± 0.4	2.5 ± 0.3*	2.8 ± 0.5*
Minute volume (L)	6.7 ± 1.3	5.6 ± 1.5	6.4 ± 1.3	5.7 ± 1.3	6.8 ± 1.0	6.2 ± 1.2		
Paw (cm H_2O)	28.6 ± 3.5	27.6 ± 4.2	29.1 ± 4.9	27.4 ± 5.2	28.4 ± 4.2	26.6 ± 4.1		
Oxygen saturation (%)	98.6 ± 0.7	99 ± 0.7	98.3 ± 1.2	98.4 ± 1.1	98.4 ± 0.7	98.6 ± 1.2	97.8 ± 1.7	97.3 ± 2.0

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 v VCM).

Table 4. Gas Exchange Data

	Postinduction (Baseline)		CPB + 30 min		ICU Arrival + 30 min		Postextubation + 30 min	
	Control	VCM	Control	VCM	Control	VCM	Control	VCM
Shunt (%)	15.7 ± 1.8	16.1 ± 2.0	27.4 ± 2.6*§	14.9 ± 2.0§	17.8 ± 1.7	14.4 ± 1.7	21.4 ± 2.3	24.0 ± 2.1
PaO_2 (mmHg)	179 ± 14	208 ± 14	140 ± 18*	157 ± 14*	135 ± 11*	146 ± 11*	106 ± 7†	117 ± 8†
$P(A-a)O_2$ (mmHg)	131 ± 9‡	107 ± 11‡	167 ± 16*	148 ± 13†	164 ± 11*	159 ± 11†	195 ± 7†	188 ± 8†

NOTE. Data are mean $\pm$ SD.

Abbreviations: VCM, vital capacity maneuver; CPB, cardiopulmonary bypass; ICU, intensive care unit; PaO_2, arterial oxygen tension; $P(A-a)O_2$, alveolar-arterial PO_2 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).

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 Q_S/Q_T. This large reduction in Q_S/Q_T was accompanied by only minor improvements in PaO_2 or $P(A-a)O_2$.[4,5] Using the multiple inert gas elimination technique and computed tomography (CT) of the lungs, these studies showed that a VCM resulted in marked reductions in shunt and atelectasis. At the same time, the number of lung units with low ventilation-perfusion significantly increased. These findings suggest that a VCM may convert lung units with shunt into regions with low ventilation-perfusion, the net result being only small improvements in oxygenation.

It was hypothesized that one of the benefits of a VCM would be a reduction in the duration of postoperative intubation through improvements in Q_S/Q_T and oxygenation. Although the VCM patients were extubated significantly earlier, this was not due to improvements in pulmonary gas exchange. Many factors other than Q_S/Q_T and PaO_2 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.[21] In animal models, mechanical stretch of lung epithelial cells has been shown to result in sustained release of pulmonary surfactant for 30 minutes.[22] A VCM may have beneficial effects on the me-

chanical 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.[5] In a pig model, Magnusson et al[8] 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[16] 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 Q_S/Q_T 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 Q_S/Q_T has been observed,[23-25] and a VCM has been shown to significantly reduce atelectasis and Q_S/Q_T. It is likely that a VCM influenced Q_S/Q_T 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 Q_S/Q_T 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.

APPENDIX

Alveolar-arterial oxygen gradient:

$$P(A\text{-}a)O_2 = PAO_2 - PaO_2$$

$$PAO_2 = F_IO_2 \cdot (PB - PH_2O) - (PaCO_2/RQ)$$

$$PAO_2 = \text{alveolar } PO_2$$

$$PaO_2 = \text{arterial oxygen}$$

$$PaCO_2 = \text{arterial carbon dioxide}$$

$$F_IO_2 = \text{inspired oxygen concentration}$$

$$PB = \text{barometric pressure}$$

$$PH_2O = \text{water vapor pressure}$$

$$RQ = \text{respiratory quotient } (0.8)$$

Intrapulmonary shunt: $Q_S/Q_T = \dfrac{CcO_2 - CaO_2}{CcO_2 - CvO_2}$

$$CcO_2 = \text{end-capillary oxygen content}$$

$$CaO_2 = \text{arterial oxygen content}$$

$$C\bar{v}O_2 = \text{mixed venous oxygen content}$$

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