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Use of a Vital Capacity Maneuver to Prevent Atelectasis after Cardiopulmonary Bypass

An Experimental Study

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Background: **Respiratory failure secondary to cardiopulmonary bypass (CPB) remains a major complication after cardiac surgery. The authors previously found that the increase in intrapulmonary shunt was well correlated with the amount of atelectasis. They tested the hypothesis that post-CPB atelectasis can be prevented by a vital capacity maneuver (VCM) performed before termination of the bypass.**

Methods: **Eighteen pigs received standard hypothermic CPB (no ventilation during bypass). The VCM was performed in two groups and consisted of inflating the lungs during 15 s to 40 cmH_2O at the end of the bypass. In one group, the inspired oxygen fraction (F_IO_2) was then increased to 1.0. In the second group, the F_IO_2 was left at 0.4. In the third group, no VCM was performed (control group). Ventilation–perfusion distribution was measured with the inert gas technique and atelectasis by computed tomographic scanning.**

Results: **Intrapulmonary shunt increased after bypass in the control group (from 4.9 ± 4% to 20.8 ± 11.7%; $P < 0.05$) and was also increased in the vital capacity group ventilated with 100% oxygen (from 2.2 ± 1.3% to 6.9 ± 2.9%; $P < 0.01$) but was unaffected in the vital capacity group ventilated with 40% oxygen. The control pigs showed extensive atelectasis (21.3 ± 15.8% of total lung area), which was significantly larger ($P < 0.01$) than the proportion of atelectasis found in the two vital capacity groups (5.7 ± 5.7% for the vital capacity group ventilated with 100% oxygen and 2.3 ± 2.1% for the vital capacity group ventilated with 40% oxygen.**

Conclusion: **In this pig model, postcardiopulmonary bypass atelectasis was effectively prevented by a VCM. (Key words: Gas exchange; lung; measurements techniques; multiple inert gases elimination.)**

IT is well known that cardiopulmonary bypass (CPB) impairs postoperative pulmonary gas exchange.[1,2] A common finding is a postoperative increase in the intrapulmonary shunt to 20–25%.[3,4] Hachenberg *et al.*[5] found a shunt of 26% in 11 patients with respiratory dysfunction in the early postoperative period, and this was well correlated with atelectasis on computed tomography scanning. Recently, we have shown in a pig model that CPB generates a larger amount of atelectasis (35% of the total lung area) than a control group without CPB (2%) and that atelectasis is well correlated with PaO_2 and shunt.[6]

Rothen *et al.*[7] have shown that inflating the lungs to vital capacity (peak airway pressure = 40 cmH_2O) for 15 s reexpanded virtually all atelectatic lung tissue during general anesthesia in patients with healthy lungs. They have also shown that the composition of inspiratory gas plays an important role in the recurrence of collapse after reexpansion.[8] With a moderate concentration of oxygen in nitrogen (inspired oxygen fraction [F_IO_2] = 0.4) atelectasis reappeared very slowly. In contrast, when 100% oxygen was used, atelectasis recurred within 5 min.

The purpose of this study was to estimate, in a pig model, if a vital capacity maneuver (VCM) performed

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before termination of CPB could prevent the occurrence of atelectasis as well as the increase in intrapulmonary shunt and hypoxemia commonly seen after cardiac surgery. The aim of the study was also to determine if the VCM had a more long-lasting effect when 40% oxygen in nitrogen was used instead of 100% oxygen.

Materials and Methods

Animals

After approval by the Animal Research Ethical Committee of Uppsala University, 18 pigs (mixed breed of Hampshire, Yorkshire, and Swedish landrace) weighing 27–40 kg were used in the study. The pigs were assigned to one of three groups. In the vital capacity/40% oxygen group (n = 6), the pigs were ventilated before and after bypass with 40% oxygen in nitrogen and a VCM was performed at the end of the CPB. In the vital capacity/100% oxygen group (n = 6), a VCM was done in the same way as in the first group but the pigs were ventilated with 100% oxygen after CPB. In the control group (n = 6), the pigs were ventilated with 40% oxygen before and after bypass, and no VCM was performed.

Vital Capacity Maneuver

The first four vital capacity pigs were also used to determine which peak airway pressure should be applied to obtain the same inflation of the lungs with an open thorax as achieved with a peak airway pressure of 40 cmH_2O with a closed chest before sternotomy. For this purpose, the lungs of these four pigs were inflated with 40% oxygen in nitrogen to a peak airway pressure of 40 cmH_2O while their thoraxes were still intact. This was thought to correspond to a maximum inflation, and the inflation volume was measured. At the end of CPB, when the thorax was open, the same volume was used to inflate the collapsed lung. A peak airway pressure of 37–42 cmH_2O was required in all four pigs. For the remaining pigs, the VCM was performed at the end of CPB by inflating the lungs to 40 cmH_2O and holding this pressure for 15 s. With this technique, the mean volume used for the hyperinflation was 45.1 ± 5.9 ml/kg; that is, nearly five times the tidal volume.

The similar airway pressure measured with inflation of a constant volume of gas in closed and open chest conditions, despite the absence of a chest wall elastance with an open chest, probably reflects the decrease in the functional residual capacity and respiratory compliance that might occur during CPB with the airway open to air.

Anesthesia

Before the transport from the farm, the pigs were sedated with a neuroleptic (40 mg azaperone [Stresnil]; Janssen, Beerse, Belgium) given intramuscularly. Induction was done with 0.04 mg/kg atropine given intramuscularly, 6 mg/kg tiletamine/zolazepam (Zoletil; Reading Laboratories, Carros, France), and 2.2 mg/kg zylazine (Rompun; Bayer AG, Leverkusen, Germany). A cannula was inserted in an ear vein and 5 μg/kg fentanyl was injected. A tracheostomy was performed and a cuffed endotracheal tube (6-mm inner diameter) was inserted. Muscle relaxation was provided with 0.2 mg/kg pancuronium and artificial ventilation was instituted with a Servo ventilator in volume-cycled mode (Servo 900C; Siemens-Elema AB, Solna, Sweden). The tidal volume was 10 ml/kg and the frequency adjusted to maintain an end-tidal carbon dioxide tension between 5.2 and 5.6 KPa (39–42 mmHg) using a Capnomac Ultima analyzer (Datex, Helsinki, Finland). Thereafter the frequency was not changed. The inspiratory time was 25% and the inspiratory pause was 10% of the inspiratory cycle with a positive end-expiratory pressure of 4 cmH_2O. The ventilator settings were kept constant throughout the procedure. The F_IO_2 was 0.4, balance nitrogen. Anesthesia was maintained with a constant infusion of a hypnotic (clomethiazole at 400 mg/h), pancuronium (2 mg/h), and fentanyl (150 μg/h).

Catheterizations

A catheter (18 gauge) was inserted in the carotid artery *via* a cut-down for pressure measurements and blood sampling. A fiberoptic catheter (Pulsiocath 4F FT PV 2024; Pulsion Medical System, Munich, Germany) was inserted in the same artery and advanced into the aorta for lung water measurements. A Swan-Ganz thermodilution catheter was introduced in the external jugular vein *via* the same incision.

Cardiopulmonary Bypass

A median sternotomy was performed and the pericardium opened. Heparin sodium (porcine type; 400 IU/kg) was administered and the activated clotting time was kept above 400 s (Hemochron 400; International Technidyne Corp., Edison, NJ). A single 28-French venous return cannula was inserted through the right atrial appendage and a 16-French cannula was inserted into the ascending aorta. The extracorporeal circuit consisted of a membrane oxygenator (Univox Membrane Oxygenation Module, Bentley; Baxter, Irvine, CA), a cardiotomy reservoir with filter (BCR-2500, Bent-

ley; Baxter), and polyvinyl chloride tubing. Ringer's acetate solution (600 ml) and mannitol 15% (200 ml) were used to prime the circuit. Perfusion was conducted using a nonpulsatile pump (type PMO 10-220; Gambro, Lund, Sweden). After the beginning of the CPB, ventilation was stopped, the respirator disconnected, and the airway opened to the atmosphere. The aorta was clamped and cardioplegic solution (St. Thomas type I) with procaine (0.27 mg/l) was injected in the root of the aorta until cardiac arrest. A minimum of 15 ml/kg was always given and it was repeated every time cardiac activity was restarted (total, 19.2 ± 2.8 ml/kg). Hypothermia to 30°C was induced.

If the mean arterial pressure during CPB (49.2 ± 5.3 mmHg) decreased to less than 40 mmHg for a period longer than 5 min, a bolus of 50 μg epinephrine was given (this was only necessary in four pigs). The pump flow rate (56 ± 9.4 $ml \cdot kg^{-1} \cdot min^{-1}$) was limited by the venous return. To limit the hemodilution, no further crystalloid or allogenic blood were given.

A second dose (100 ml) of the cardioplegic solution was given 10 min before the aortic clamp was released (total duration of the cardiac ischemia, 45 min). Rewarming was then initiated. Cardiopulmonary bypass lasted 90 min. Fifteen minutes before termination of bypass, ventilation was reinstituted at one half the tidal volume. Just before termination of the bypass, the VCM was done for the two vital capacity groups. The lungs were then ventilated as before bypass, and the control group and vital capacity/40% O_2 group had their F_IO_2 maintained at 0.4 and the vital capacity/100% O_2 group had an F_IO_2 of 1.0. At the end of the bypass, all the pump prime was returned to the animals through the aortic cannula. The heparin effect was reversed with protamine (1 mg for each 100 IU used).

Measurements and Study Protocol

Baseline and Postbypass Measurements. A delay of 30 min was allowed after the surgical preparation before baseline measurements were made.

The infusion of inert gases was restarted after the end of the bypass; because of the time necessary to obtain a new steady state, the postbypass measurements of circulatory and ventilatory variables were not taken until 45 min after the bypass. During this waiting time, the CPB cannulas were removed and the chest closed.

Parameters measured included arterial and mixed venous blood gases (ABL 300 and OSM 3 Hemoximeter; Radiometer, Copenhagen, Denmark), heart rate, systemic and pulmonary arterial pressures (Tram series 7010 monitor; Marquette Electronics Inc., Milwaukee, WI), cardiac output measured by thermodilution, extravascular lung water and intrathoracic blood volume measured with the double-indicator dilution method, respiratory mechanics, and ventilation-perfusion relations.

Measurements of Thoracic Intra- and Extravascular Fluid Volumes. The same indicator bolus was used to determine cardiac output and lung fluid volumes and consisted of indocyanine green (an intravascular marker) mixed in 5 ml ice-cold 5% glucose (a thermal intra- and extravascular indicator). The bolus was injected in the right atrium. The dilution curves for dye and temperature were recorded simultaneously in the aorta with the thermistor-tipped fiberoptic catheter. A lung water computer (Pulsion COLD Z-021; Pulsion Medical Systems, München, Germany) determined the mean transit time for the thermal indicator and for the dye indicator and calculated cardiac output, total thermal volume, intrathoracic blood volume, and extravascular lung water. All measurements were made in triplicate, and the mean was calculated and used for statistical evaluation (for further details, see Hachenberg *et al.*[9])

Ventilatory Parameters. Compliance and resistance of the total respiratory system were measured using the technique of rapid airway occlusion during constant-flow inflation.[10] Resistance was calculated as the difference between peak airway pressure and the pressure after 2 s of end-inspiratory pause, divided by the flow.[11] Compliance was calculated as V_t divided by the end-inspiratory pressure minus the end-expiratory pressure. Pressure and flow were measured in the ventilator on the inspiratory side and fed into a computer for on-line signal processing (LabVIEW 3.1 software; C-O Sjöberg Engineering, National Instruments, Austin, TX). Gas compression in the ventilator tubings was corrected for when gas volume and flow were calculated. The mean value of two "inspiratory hold" maneuvers was used for statistical analysis.

Measurements of Ventilation–Perfusion Distribution. The multiple inert gas elimination technique is based on the steady-state elimination (obtained after a 40-min equilibration period) of six inert gases with different solubility in the blood (sulfur hexafluoride, ethane, cyclopropane, enflurane, diethylether, and acetone), as described by Wagner *et al.*[12-14] This mixture is dissolved in isotonic saline and infused at a constant rate (2-3 ml/min, depending on the minute ventilation) in a peripheral vein. After 40 min of infusion, under steady-state conditions, arterial and mixed venous blood was collected together with an expired gas sample and analyzed by gas chromatography (model 5890, series II, Hewlett-Packard, Little

Table 1. Ventilatory Parameters (Six Pigs in Each Group)

	Control		Vital Capacity/40% O_2		Vital Capacity/100% O_2	
	Baseline	Post-CPB	Baseline	Post-CPB	Baseline	Post-CPB
Tidal volume (ml/kg)	10.1 ± 0.6	10.3 ± 0.4	10.0 ± 0.2	10.0 ± 0.2	10.0 ± 0.2	10.0 ± 0.2
Minute volume ($ml \cdot kg^{-1} \cdot min^{-1}$)	237.3 ± 31.1	232.3 ± 34.7	238.0 ± 24.4	259.1 ± 31.8	242.2 ± 33.1	250.4 ± 35.2
Paw (cmH_2O)	20.4 ± 3.9	25.7 ± 4.3*	22.2 ± 3.7	28.7 ± 6.6†	21.6 ± 2.7	24.5 ± 4.9
$P_{ei}aw$ (cmH_2O)	16.8 ± 4.0	21.4 ± 3.3*	17.6 ± 3.0	21.9 ± 5.6†	17.2 ± 1.4	18.9 ± 2.9
Compliance (ml/cmH_2O)	26.7 ± 5.9	18.5 ± 4.9*	28.4 ± 6.0	22.9 ± 9.7	27.8 ± 3.1	25.1 ± 5.5
Resistance ($cmH_2O \cdot l^{-1} \cdot s^{-1}$)	16.9 ± 1.5	19.9 ± 6.5	14.6 ± 1.8	20.7 ± 9.0	15.1 ± 1.4	16.3 ± 5.9

CPB = cardiopulmonary bypass; Paw = peak airway pressure; $P_{ei}aw$ = end-inspiratory pressure.

* $P < 0.01$ *versus* baseline.

† $P < 0.05$ *versus* baseline.

Falls, DE). These data enable the construction of a virtually continuous distribution of blood flow or ventilation against ventilation-perfusion ratios (V_A/Q), with separation of shunt ($V_A/Q < 0.005$) from regions with low V_A/Q ratios ($0.005 < V_A/Q < 0.1$ = poorly ventilated lung units in relation to their perfusion) as well as the calculation of a normal V_A/Q region, units with high V_A/Q ratios ($10 < V_A/Q < 100$), and dead space ($V_A/Q > 100$). The dispersion of (V_A/Q) ratios is expressed as the logarithmic standard deviation of perfusion distribution. It describes the degree of V_A/Q mismatch.

Computed Tomography. At the end of the study, the pigs were moved to the computed tomography scan laboratory (Somaton Plus 4; Siemens, München, Germany). At approximately 1 h after the end of the bypass, an anteroposterior topogram covering the chest was obtained at end-expiration to define the limits of the lungs. One computed tomography scan was performed at end-expiration, 0-1 cm above the diaphragm (146 mA, 140 kV; slice thickness, 5 mm). The scans were analyzed for distribution of lung tissue density, and the total lung area was delineated manually. Nonaerated (atelectatic) lung tissues were defined as regions with attenuation values between −100 and +100 Hounsfield Units (HU),[15] and poorly aerated lung tissues were defined as regions presenting values between −500 and −100 HU. The extent of atelectasis and poorly aerated lung tissue was expressed as a percentage of the total lung area (excluding the mediastinum).

At the end of the experiment, the animals were killed

Table 2. Hemodynamic and Lung Water Parameters (Six Pigs in Each Group)

	Control		Vital Capacity/40% O_2		Vital Capacity/100% O_2	
	Baseline	Post-CPB	Baseline	Post-CPB	Baseline	Post-CPB
Heart rate (beats/min)	107.8 ± 11.1	119.8 ± 21.6	98.2 ± 9.1	112.5 ± 13.1	101.2 ± 20.4	111.3 ± 14.2
MAP (mmHg)	83.0 ± 7.6	59.3 ± 11.5*	75.7 ± 2.3	66.8 ± 6.1†	79.7 ± 4.1	68.8 ± 9.4‡
MPAP (mmHg)	16.2 ± 3.3	22.5 ± 3.7*	16.3 ± 2.5	24.7 ± 6.0†	17.3 ± 2.2	23.3 ± 2.3†
CVP (mmHg)	3.5 ± 1.8	4.8 ± 1.3	5.3 ± 0.8	8.8 ± 1.8‡§	5.3 ± 1.4	7.8 ± 1.0
PCWP (mmHg)	5.2 ± 1.7	5.7 ± 1.2	7.2 ± 1.3	9.2 ± 1.6§	7.2 ± 1.5	8.3 ± 1.0
CO (ml/min)	4.3 ± 0.7	3.8 ± 0.9	3.7 ± 0.6	3.0 ± 0.5	3.6 ± 0.3	3.0 ± 1.1
EVLWi (ml/kg)	5.1 ± 0.9	6.6 ± 5.0	4.7 ± 1.1	5.2 ± 1.0	4.6 ± 0.7	5.1 ± 1.1
ITBVi (ml/kg)	20.8 ± 2.3	17.8 ± 5.3	18.0 ± 1.3	15.5 ± 4.5	19.5 ± 2.3	15.3 ± 3.5†

CPB = cardiopulmonary bypass; MAP = mean arterial pressure; MPAP = mean pulmonary arterial pressure; CVP = central venous pressure; PCWP = pulmonary capillary wedge pressure; CO = cardiac output; EVLWi = extravascular lung water indexed for the weight; ITBVi = intrathoracic blood volume indexed for the weight.

* $P < 0.001$ *versus* baseline.

† $P < 0.01$ *versus* baseline.

‡ $P < 0.05$ *versus* baseline.

§ $P < 0.05$ *versus* control group.

Table 3. Ventilation–Perfusion Relationship, Gas Exchange, and Acid–Base Status Parameters (Six Pigs in Each Group)

	Control		Vital Capacity/40% O_2		Vital Capacity/100% O_2	
	Baseline	Post-CPB	Baseline	Post-CPB	Baseline	Post-CPB
Shunt (%)	4.9 ± 4.0	20.8 ± 11.7*	3.3 ± 2.1	5.0 ± 3.9§	2.2 ± 1.3	6.9 ± 2.9*§
Log SD_Q	0.60 ± 0.10	0.66 ± 0.12	0.61 ± 0.13	0.78 ± 0.13*	0.54 ± 0.05	0.60 ± 0.12
Pa_{O_2}/FI_{O_2} (mmHg)	433.2 ± 67.4	267.5 ± 100.5*	471.0 ± 67.6	465.8 ± 101.0§	497.3 ± 34.6	499.2 ± 50.7**
Pa_{CO_2} (mmHg)	43.5 ± 3.5	51.9 ± 8.0†	42.3 ± 3.4	42.0 ± 4.6§	39.7 ± 2.9	42.7 ± 2.9*¶
Atelectasis (%)		21.3 ± 15.8		2.3 ± 2.1§		5.7 ± 5.7¶
Poorly aerated (%)		31.8 ± 6.4		21.0 ± 7.6¶		30.1 ± 17.6
Base excess (mM)	5.8 ± 1.0	−4.9 ± 4.3†	6.5 ± 3.4	−5.6 ± 5.3‡	5.7 ± 2.7	−4.4 ± 2.9‡
*p*H	7.45 ± 0.05	7.24 ± 0.08‡	7.47 ± 0.05	7.29 ± 0.08‡	7.48 ± 0.06	7.31 ± 0.06‡

CPB = cardiopulmonary bypass; shunt = intrapulmonary shunt as measured with the inert gases technique; Log SD_Q = logarithmic standard deviation of perfusion distribution; Atelectasis, Poorly aerated lung tissues = measured with CT scanning and calculated in % of the total lung area.

* $P < 0.05$ *versus* baseline.

† $P < 0.01$ *versus* baseline.

‡ $P < 0.001$ *versus* baseline.

§ $P < 0.01$ *versus* control group.

¶ $P < 0.05$ *versus* control group.

** $P < 0.001$ *versus* control group.

with an intravenous injection of potassium chloride and the lungs were removed for inspection.

Statistics

Data in the text, tables, and figures are presented as means ± SD. Analysis of variance with the Bonferroni/Dunn *post hoc* test was used for baseline comparisons of normally distributed parameters, and the remaining parameters (mean arterial pressure, pulmonary capillary wedge pressure, and the hematocrit concentration) were compared using the Mann-Whitney U test. For the comparison of the three groups, analysis of variance for repeated measurements was used. Probability values < 0.05 were considered significant.

Results

Baseline

The pigs were of the same size in all groups (vital capacity/40% O_2 group: 33.2 kg ± 3.9; vital capacity/100% O_2 group: 30.2 kg ± 2.1; control group: 30.8 kg ± 2.4). There were no significant prebypass differences between the three groups in any measured variable (see tables 1–3).

After Cardiopulmonary Bypass

Ventilation and Respiratory Mechanics. Small increases in airway pressures were seen in all groups after CPB. These changes were only significant in the control and vital capacity/40% O_2 groups. In the control group, a significant increase was also seen in compliance (table 1).

Hemodynamics and Lung Fluids. There were no significant changes after CPB in heart rate or cardiac output compared with baseline (table 2). In all groups the mean systemic arterial pressure decreased and the mean pulmonary arterial pressure increased. The central venous pressure tended to increase in all groups, but this was only significant in the vital capacity/40% O_2 group.

No changes in the extravascular lung water were seen in any group. The intrathoracic blood volume tended to decrease, but this change was significant only in the group ventilated with 100% oxygen (table 2).

Ventilation–Perfusion Relations and Gas Exchange. In the vital capacity/40% O_2 group there were no changes after the CPB, compared with baseline, regarding intrapulmonary shunt, partial pressure of oxygen (PaO_2), or the partial pressure of carbon dioxide ($PaCO_2$). Shunt increased in the vital capacity/100% O_2 group and even more in the control group (fig. 1). The PaO_2 decreased and $PaCO_2$ increased in the control group (table 3 and fig. 2). Thus, in the control group, shunt and $PaCO_2$ were significantly higher while PaO_2 was significantly lower compared with the two vital capacity groups.

There were no "low VA/Q" regions either before or after CPB. The logarithmic standard deviation of perfusion

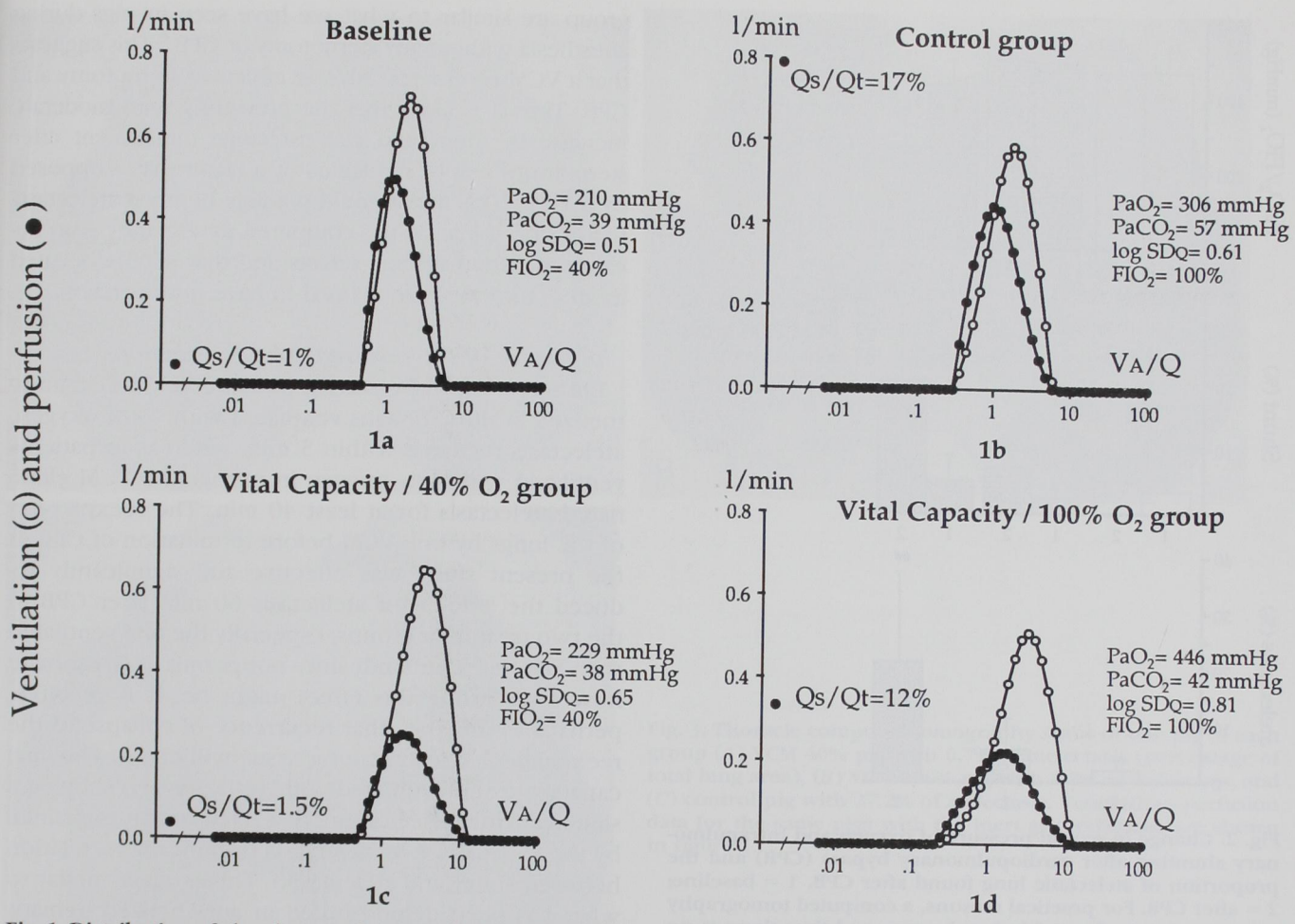

Fig. 1. Distribution of alveolar ventilation and blood flow against ventilation–perfusion (VA/Q) ratio. (*A*) Representative example at baseline. (*B*) Example of VA/Q after cardiopulmonary bypass (CPB) of the control group. (*C* and *D*) Representative examples of VA/Q after CPB in the two groups treated with a vital capacity maneuver (VCM) and ventilated thereafter with 40% oxygen (VCM 40% group) or 100% oxygen (VCM 100% group), respectively. Note the marked increase in the shunt (Qs/Qt) seen after CPB in the control group, which is less marked in the VCM group with 100% oxygen and the absence of changes found in the VCM group with 40% oxygen. The computed tomography scans of the same pigs are shown in figure 3.

distribution tended to increase in all groups, but this was significant only in the vital capacity/40% O_2 group.

After bypass, arterial *p*H decreased and base excess became more negative in all groups (table 3).

Atelectasis and Aeration of the Lung. Little or no atelectasis was seen in the vital capacity/40% O_2 group (mean, 2.3%; fig. 2A). Despite the fact that the proportion of atelectatic area (mean, 5.7%; fig. 2B) in the vital capacity/100% O_2 group was twice as large as in the vital capacity/40% O_2 group, this difference was not statistically significant. The control group showed large atelectasis that accounted for more than 20% of the total lung area (mean, 21.3%; fig. 3C), and the atelectasis was significantly larger than in the two vital capacity groups (table 3). There was also a significant correlation between the magnitude of shunt and the size of the atelectasis when the three groups were pooled (according to the equation, shunt = 6.8 + 0.41 × atelectasis, R = 0.52, $P < 0.05$; where shunt is expressed as a percentage of cardiac output and atelectasis as a percentage of the total lung area). Areas with poor aeration amounted to approximately 20–30% of the lung area but with large variation within the groups.

Discussion

Effect of the Vital Capacity Maneuver

Our results show that a VCM is helpful in preventing the impairment in gas exchange that was seen after CPB

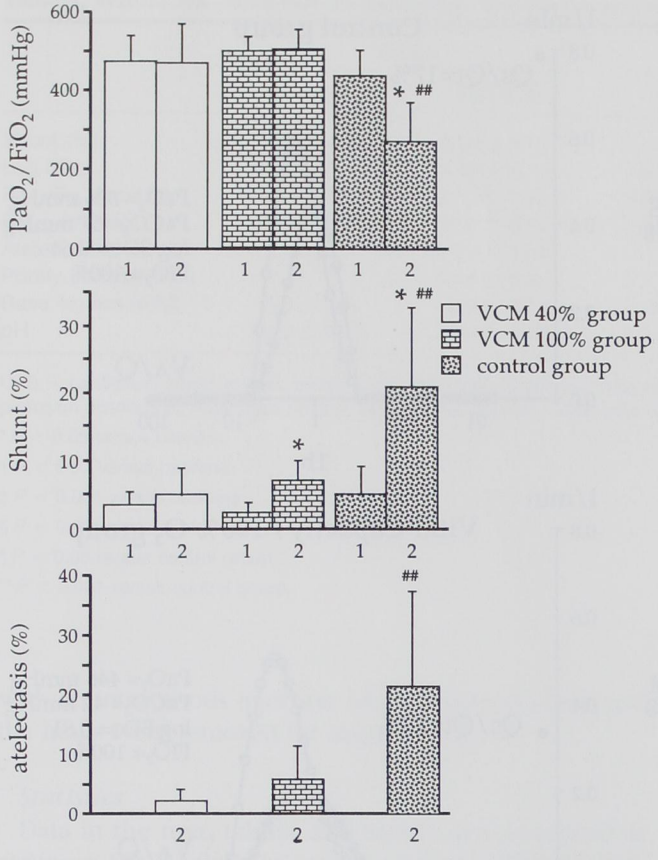

Fig. 2. Change in partial pressure of oxygen and intrapulmonary shunting after cardiopulmonary bypass (CPB) and the proportion of atelectatic lung found after CPB. 1 = baseline; 2 = after CPB. For practical reasons, a computed tomography scan was not performed before bypass, and thus there is no value for atelectasis at baseline. *$P < 0.05$ between two measurements in the same group. ##$P < 0.01$ between the control group and the other groups after CPB.

in pigs. Thus the control pigs showed extensive atelectasis after the CPB, accounting for more than 20% of the total lung area (fig. 3C). There was also a marked increase in the intrapulmonary shunt and a decrease in P_aO_2. In contrast, the two groups treated before termination of CPB with a VCM showed only minor or moderate atelectasis (table 3, figs. 3A, 3B). When a VCM was performed before termination of bypass and the lungs were ventilated throughout the procedure with 40% oxygen in nitrogen, no abnormal changes in the intrapulmonary shunt or in the PaO_2 could be detected after CPB. These results confirm our previous results that showed that atelectasis is one of the major causes of post-CPB gas exchange impairment.[6] Furthermore, the nearly complete absence of atelectasis and only minor shunt in the vital capacity/40% O_2 group are similar to what we have seen in pigs during anesthesia without any sternotomy or CPB.[6] This suggests that a VCM reverses all adverse effects of sternotomy and CPB. Thus it is likely that the previously seen moderate increase in shunt and gas exchange impairment after sternotomy[6] can be explained by a factor that is opposed by a VCM. This factor could possibly be microatelectasis that is not visible on the computed tomography scan because of partial volume effects and that may be located in areas that we have defined to have poor aeration.

Influence of the Inspired Oxygen Fraction

Rothen *et al.*[8] have shown that after a VCM, in anesthetized healthy humans ventilated with 100% oxygen, atelectasis recurred within 5 min, whereas in patients ventilated with 40% oxygen in nitrogen, a VCM eliminated atelectasis for at least 40 min. The reexpansion of the lungs by one VCM before termination of CPB in the present study was effective and significantly reduced the amount of atelectasis 60 min after CPB in the two treatment groups, especially the one ventilated with FiO_2 0.4. Our study does not permit us to estimate how long lasting this effect might be. It is possible, perhaps even likely, that recurrence of collapse of the reexpanded atelectatic lung tissue will occur. The vital capacity group ventilated with 100% oxygen showed a significant increase in shunt that is reasonably explained by the appearance of atelectasis (significant correlation between shunt and atelectasis). This is again similar to what has been demonstrated in anesthetized humans ventilated with 100% oxygen after a VCM, although there seems to be quantitative differences that may be related to species differences.

The control group in our previous study was ventilated with 100% oxygen after bypass and showed atelectasis accounting for 32% of the lung area,[6] whereas in this study the control group was ventilated with 40% oxygen after bypass and showed atelectasis accounting for 21% of the lung area. Thus the FiO_2 may have an effect on the atelectasis formation, as shown during routine anesthesia.[8]

Previous Studies

Many efforts have been made to prevent postbypass lung function impairment. In particular, lung management during CPB has been studied for this purpose. Continuous positive airway pressure with varying levels of airway pressure has given different results. Some studies have shown a decrease in lung function with no difference compared with lungs that were left to collapse during CPB.[16-19] Oth-

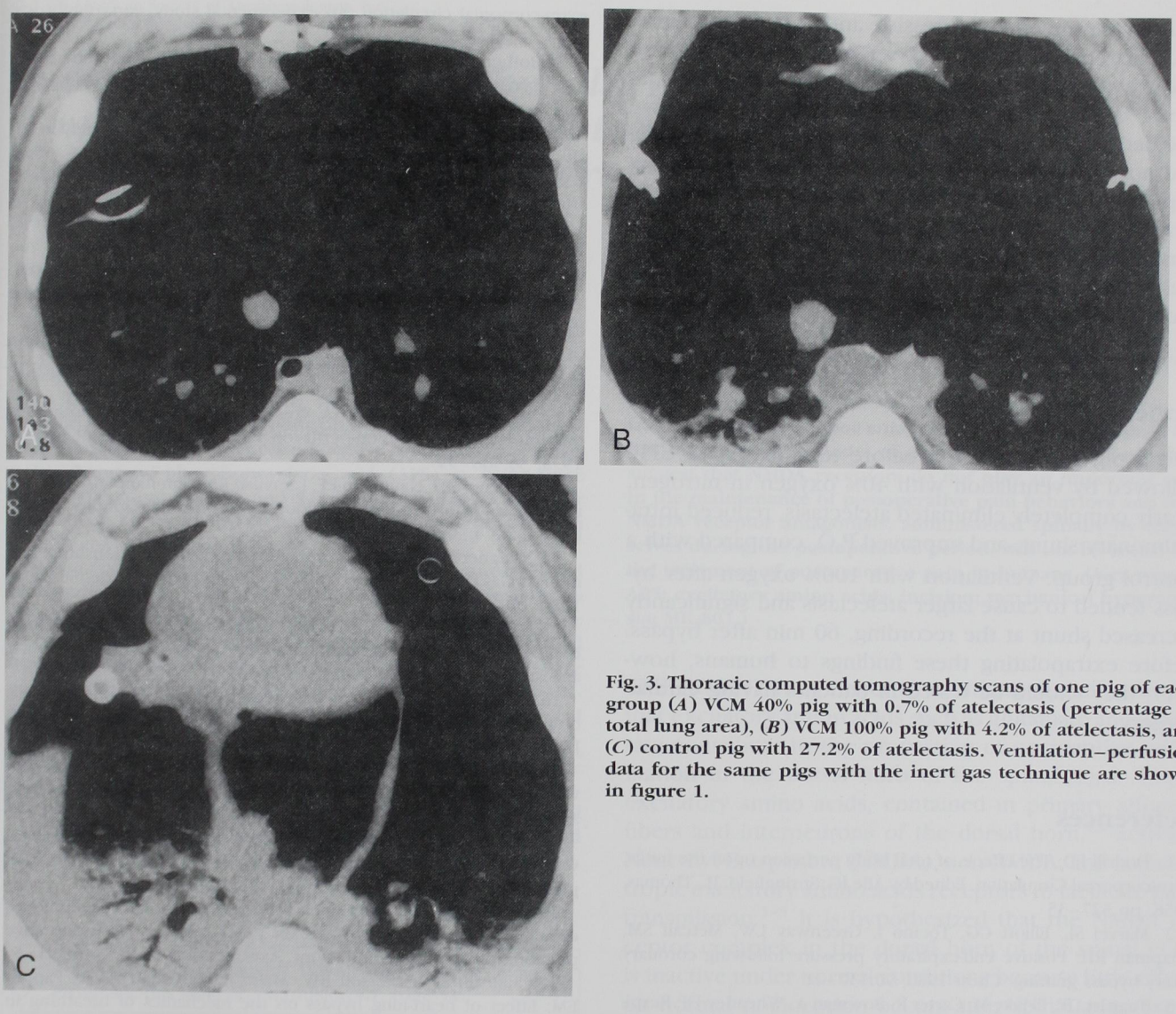

Fig. 3. Thoracic computed tomography scans of one pig of each group (*A*) VCM 40% pig with 0.7% of atelectasis (percentage of total lung area), (*B*) VCM 100% pig with 4.2% of atelectasis, and (*C*) control pig with 27.2% of atelectasis. Ventilation–perfusion data for the same pigs with the inert gas technique are shown in figure 1.

ers have shown better lung function after CPB when continuous positive airway pressure was used,[20–22] but the improvement had disappeared 5 h after CPB.[20,22] Only Boldt *et al.*[21] concluded that moderate continuous positive airway pressure seemed to optimize postbypass pulmonary function. Maintaining standard ventilation has also been studied with varying results: no difference with the preoperative values,[16] decrease in the lung function identical to the decrease found if the lungs were left to collapse,[17,18] or even with worse results when compared with no ventilation.[19,23]

These conflicting results may be due to varying study protocols, the use of different species, different CPB techniques, different gas mixtures (air, pure oxygen, helium), different respiratory rates when the lungs were ventilated, and different PEEP levels. The length of bypass has also varied and there may have been different fluid balance regimens. Finally, the methods for assessing pulmonary function have also varied among studies (PaO_2, shunt, $P_{(A-a)}O_2$, extravascular lung water, compliance) as have the postoperative period until measurements were made.[16–23]

Fluid Balance and Lung Tissue Edema

We used a large amount of mannitol (approximately 1 g/kg) in the priming fluid of the heart-lung machine

to increase the oncotic pressure and to force the diuresis. This could explain why we observed no increase in extravascular lung water after CPB. Similarly, to counteract hemodilution and avoid blood transfusion, we decided to limit as much as possible the priming volume and not to add Ringer's solution during CPB. This may explain why the blood flow during CPB was low and why the pigs were acidotic at the end of CPB. Acidosis may have some deleterious effect on post-CPB myocardial function, but we found no difference in the cardiac output between the groups.

Conclusions

In a pig model, a VCM before termination of CPB, followed by ventilation with 40% oxygen in nitrogen, nearly completely eliminated atelectasis, reduced intrapulmonary shunt, and improved P_aO_2 compared with a control group. Ventilation with 100% oxygen after bypass tended to cause larger atelectasis and significantly increased shunt at the recording, 60 min after bypass. Before extrapolating these findings to humans, however, they should be tested in patients having cardiac surgery.

References

1. Dodrill FD: The effects of total body perfusion upon the lungs, Extracorporeal Circulation. Edited by Alle JG. Springfield, IL, Thomas, 1958, pp 327–35

2. Marvel SL, Elliott CG, Tocino I, Greenway LW, Metcalf SM, Chapman RH: Positive end-expiratory pressure following coronary artery bypass grafting. Chest 1986; 90:537–41

3. Taggart DP, El-Fiky M, Carter R, Bowman A, Wheatley DJ: Respiratory dysfunction after uncomplicated cardiopulmonary bypass. Ann Thorac Surg 1993; 56:1123–8

4. Lindberg H, Svennevig JL, Lilleaasen P, Vatne K: Pulsatile vs. non-pulsatile flow during cardiopulmonary bypass. A comparison of early postoperative changes. Scand J Thorac Cardiovasc Surg 1984; 18:195–201

5. Hachenberg T, Brüssel T, Roos N, Lenzen H, Möllhoff T, Gockel B, Konertz W, Wendt M: Gas exchange impairment and pulmonary densities after cardiac surgery. Acta Anaesthesiol Scand 1992; 36:800–5

6. Magnusson L, Zemgulis V, Wicky S, Tydén H, Thelin S, Hedenstierna G: Atelectasis is a major cause of hypoxemia and shunt after extra-corporeal circulation. An experimental study. ANESTHESIOLOGY 1997; 87:1153–63

7. Rothen HU, Sporre B, Engberg G, Wegenius G, Hedenstierna G: Re-expansion of atelectasis during general anaesthesia: A computed tomography study. Br J Anaesth 1993; 71:788–95

8. Rothen HU, Sporre B, Engberg G, Wegenius G, Högman M, Hedenstierna G: Influence of gas composition on recurrence of atelectasis after a reexpansion maneuver during general anesthesia. ANESTHESIOLOGY 1995; 82:832–42

9. Hachenberg T, Tenling A, Rothen HU, Nyström SO, Tyden H, Hedenstierna G: Thoracic intravascular and extravascular fluid volumes in cardiac surgical patients. ANESTHESIOLOGY 1993; 79:976–84

10. Bates JHT, Milic-Emili J: The flow interruption technique for measuring respiratory resistance. J Crit Care 1991; 6:227–38

11. Kochi T, Okubo S, Zin WA, Milic-Emili J: Flow and volume dependence of pulmonary mechanics in anesthetized cats. J Appl Physiol 1988; 64:441–50

12. Wagner PD, Saltzman HA, West JB: Measurement of continuous distributions of ventilation-perfusion ratios: Theory. J Appl Physiol 1974; 36:588–99

13. Wagner PD, Naumann PF, Laravuso RB: Simultaneous measurement of eight foreign gases in blood by gas chromatography. J Appl Physiol 1974; 36:600–5

14. Wagner PD, West JB: Ventilation-perfusion relationships, Pulmonary Gas Exchange. Edited by West JB. New York, Academic Press, 1980, pp 219–62

15. Lundquist H, Hedenstierna G, Strandberg A, Tokics L, Brismar B: CT-assessment of dependent lung densities in man during general anaesthesia. Acta Radiol 1995; 36:626–32

16. Edmunds Jr LH, Austen WG: Effect of cardiopulmonary bypass on pulmonary volume-pressure relationships and vascular resistance. J Appl Physiol 1966; 21:209–16

17. Ellis EL, Brown A, Osborn JJ, Gerbode F: Effect of altered ventilation patterns on compliance during cardiopulmonary bypass. Anesth Analg 1969; 48:947–52

18. Weedn RJ, Coalson JJ, Greenfield LJ: Effects of oxygen and ventilation on pulmonary mechanics and ultrastructure during cardiopulmonary bypass. Am J Surg 1970; 120:584–90

19. Stanley TH, Liu WS, Gentry S: Effects of ventilatory techniques during cardiopulmonary bypass on post-bypass and postoperative pulmonary compliance and shunt. ANESTHESIOLOGY 1977; 46:391–5

20. Sullivan SF, Patterson RW, Malm JR, Bowman FO Jr, Papper EM: Effect of heart-lung bypass on the mechanics of breathing in man. J Thorac Cardiovasc Surg 1966; 51:205–12

21. Boldt J, King D, Scheld HH, Hempelmann G: Lung management during cardiopulmonary bypass: Influence on extravascular lung water. J Cardiothorac Anesth 1990; 4:73–9

22. Berry CB, Butler PJ, Myles PS: Lung management during cardiopulmonary bypass: Is continuous positive airways pressure beneficial? Br J Anaesth 1993; 71:864–8

23. Svennevig JL, Lindberg H, Geiran O, Semb BK, Abdelnor M, Ottesen S, Vatne K: Should the lungs be ventilated during cardiopulmonary bypass? Clinical, hemodynamic, and metabolic changes in patients undergoing elective coronary artery surgery. Ann Thorac Surg 1984; 37:295–300