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CLINICAL INVESTIGATIONS

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 maintain end-expiratory lung volume by increased airway pressure. Since this could increase right ventricular afterload, we studied the effect of this method on right ventricular afterload in patients after cardiac surgery.

Methods. We studied 24 stable patients after coronary artery surgery and/or valve surgery with cardiopulmonary bypass. Patients were randomly assigned to OLC or conventional mechanical ventilation (CMV). In the OLC group, recruitment manoeuvres were applied until Pa_{O_2}/FI_{O_2} was greater than 50 kPa (reflecting an open lung). This value was maintained by sufficient positive airway pressure. In the CMV group, volume-controlled ventilation was used with a PEEP of 5 cm H_2O. Cardiac index, right ventricular preload, contractility and afterload were measured with a pulmonary artery thermodilution catheter during the 3-h observation period. Blood gases were monitored continuously.

Results. To achieve $Pa_{O_2}/FI_{O_2} > 50$ kPa, 5.3 (3) (mean, SD) recruitment attempts were performed with a peak pressure of 45.5 (2) cm H_2O. To keep the lung open, PEEP of 17.0 (3) cm H_2O was required. Compared with baseline, pulmonary vascular resistance and right ventricular ejection fraction did not change significantly during the observation period in either group.

Conclusion. No evidence was found that ventilation according to the OLC affects right ventricular afterload.

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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 dysfunction[4] or by reduced right ventricular function.[5] Patients with such complications could be vulnerable to increased right ventricular afterload. However, Dyhr and colleagues[6] found that a lung recruitment manoeuvre followed by PEEP did not reduce cardiac output in patients after CABG who had been give 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 sufentanil 2 μg kg^{-1}. Muscle relaxation was with pancuronium 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 the following settings: volume control mode, tidal volume 6–8 ml kg^{-1}; PEEP 5 cm H_2O; I/E ratio 1:2; $F_{I_{O_2}}$ 0.3–0.5; and respiratory rate was adjusted to achieve a $P_{a_{CO_2}}$ between 4.5 and 6.5 kPa. These settings were called 'conventional mechanical ventilation' (CMV). During CPB the lungs were not ventilated. After CPB lungs were re-expanded by manual inflation and ventilation was continued with the same settings until randomization. After surgery the pericardium was not closed. After sternum closure, the patients were given fluids until left ventricular function did not increase further. Left ventricular function was assessed by transoesophageal echocardiogram by an experienced operator, who assessed fractional area change on the transgastric midpapillary short-axis view. At optimal left ventricular function, right ventricular end-diastolic volume index (EDVI) measured with the pulmonary artery catheter was defined as optimal EDVI.

After surgery, patients were sedated with propofol 2–4 mg kg^{-1} h^{-1}. An indwelling blood gas analyser probe was inserted in a radial artery for continuous blood gas analyses (ParaTrend 7+; Philips, Boblingen, Germany). Fluid management was guided by EDVI, aiming at optimal EDVI as assessed during operation. Hypovolaemia was treated with a set plan using starch colloids. If a maximum daily dose was reached, further fluid was given as pasteurized plasma. If mean arterial pressure was less than 45 mm Hg and hypovolaemia was excluded, an infusion of dobutamine or phenylephrine i.v. was given.

Cardiovascular and respiratory measurements were made every 30 min for 3 h. Measurements before randomization were considered baseline measurements. Patients were randomly assigned by envelope to the OLC group or the CMV group. Randomization was not stratified for type of operation. The study group was ventilated according to the OLC and in the CMV group ventilation was continued as described above.

Ventilation according to the OLC was initiated by switching the ventilator to pressure control mode, PEEP 10 cm H_2O, $F_{I_{O_2}}$ 0.3–0.4, I/E ratio 1:1 and a pressure to obtain a tidal volume of 4–6 ml kg^{-1}, aiming at a $P_{a_{CO_2}}$ 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 H_2O for 40 s to increase the $P_{a_{O_2}}/F_{I_{O_2}}$ ratio to a value greater than 50 kPa. If not, a recruitment manoeuvre was repeated by increasing peak pressure 5 cm H_2O greater than before, up to a maximum peak pressure of 60 cm H_2O until the $P_{a_{O_2}}/F_{I_{O_2}}$ ratio became greater than 50 kPa. If the $P_{a_{O_2}}/F_{I_{O_2}}$ ratio decreased slowly below 50 kPa after recruitment, PEEP was increased by 2 cm H_2O and a recruitment manoeuvre (beginning at 40 cm H_2O) was repeated.

Cardiovascular measurements consisted of right atrial pressure (RAP), mean pulmonary arterial pressure (PA_{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 are presented as mean (SD). The *P*-values given are two-sided, and $P<0.05$ was considered significant.

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.

Table 1 Characteristics of the groups. Data are mean (range) for age, or mean (SD) unless otherwise stated. *The Euroscore is expressed as median (first quartile)

Characteristic	OLC (n=10)	CMV (n=10)
Age (yr)	66.3 (54–78)	57.5 (29–77)
Weight (kg)	78.7 (17.6)	78.5 (10.2)
Male/female	6/4	9/1
CABG	6	4
Valve repairs or replacement	5	9
Euroscore (%)*	2.5 (2)	2.0 (0.9)
Pa_{O_2} (kPa)	10.5 (1.8)	10.7 (1.7)
Operation time (h)	5.49 (0.59)	5.41 (0.49)
Aortic clamp time (min)	76.2 (25.6)	102.0 (39.1)
Temperature at baseline (°C)	35.0 (0.48)	34.8 (0.56)
FEV_1 (%)	101 (13)	93 (12)
FVC (%)	102 (16)	93 (10)

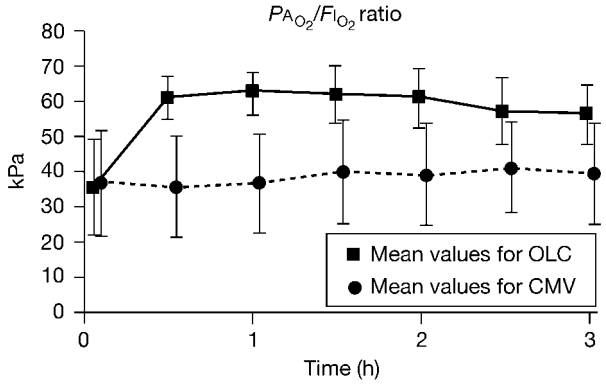

Fig 1 Changes in Pa_{O_2}/FI_{O_2} with time. Baseline values are given at t=0.

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 FI_{O_2} to maintain Pa_{O_2} 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_2O to open the lung (Fig. 1). To keep the lung open, a total PEEP of 17.0 (2.7) cm H_2O 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 (PA_{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 Pa_{CO_2} 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).

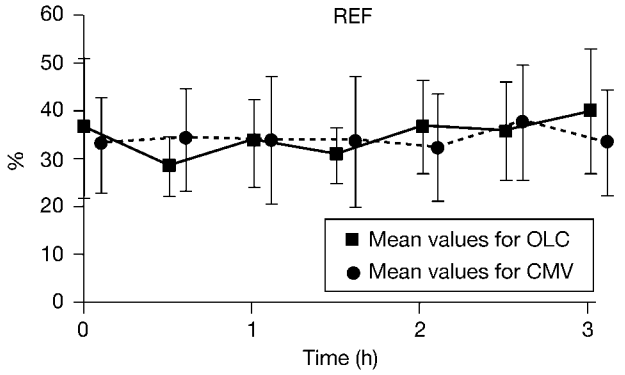

Fig 2 Changes in right ventricular ejection fraction (REF). Baseline values are given at t=0.

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 μg 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, phenylephrine, 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.

Table 2 Cardiovascular measurements at baseline and during the study. Data are mean (SD). †$P<0.05$ compared with zero; ‡$P<0.05$, OLC *vs* CMV

Variable	Group	Baseline	30 min	60 min	90 min	120 min	150 min	180 min	Mean change from baseline
HR (min^{-1})	OLC	82 (13)	84 (10)	82 (10)	84 (10)	82 (13)	89 (16)	89 (16)	2 (3)
	CMV	90 (10)	89 (6)	90 (10)	92 (6)	95 (10)	94 (13)	94 (13)	3 (3)
MAP (mm Hg)	OLC	78 (9)	81 (11)	79 (14)	75 (8)	81 (13)	86 (12)	83 (12)	2 (6)
	CMV	76 (13)	77 (10)	83 (13)	81 (10)	82 (13)	80 (10)	78 (10)	4 (6)
CI (litre min^{-1} m^{-2})	OLC	2.7 (0.5)	2.4 (0.4)	2.6 (0.4)	2.6 (0.5)	2.8 (0.5)	2.7 (0.5)	3.0 (0.6)	0.0 (0.3)
	CMV	3.2 (1.0)	3.2 (0.7)	3.2 (0.6)	3.3 (0.7)	3.4 (0.8)	3.6 (0.7)	3.6 (0.7)	0.3 (0.3)
REF (%)	OLC	37 (14)	29 (6)	34 (9)	31 (6)	37 (10)	36 (10)	41 (13)	−1.5 (7.9)
	CMV	33 (10)	35 (11)	34 (13)	34 (13)	33 (11)	38 (12)	34 (11)	1.1 (7.6)
PVR (dynes cm m^2)	OLC	149 (40)	167 (35)	160 (37)	154 (47)	151 (56)	147 (55)	141 (32)	2.0 (41)
	CMV	125 (87)	146 (82)	138 (68)	118 (47)	122 (51)	128 (51)	109 (47)	0.9 (41)
RAP (mm Hg)	OLC	10 (2)	14 (2)	14 (3)	14 (4)	15 (3)	15 (3)	17 (7)	5.1 (3)†
	CMV	9 (3)	9 (2)	10 (2)	10 (2)	10 (3)	10 (3)	10 (3)	0.6 (3)‡
PA_{mean} (mm Hg)	OLC	19 (2)	24 (3)	24 (3)	24 (5)	25 (4)	26 (5)	27 (8)	5.2 (4)†
	CMV	19 (5)	20 (5)	21 (6)	21 (5)	21 (5)	21 (4)	20 (3)	2.0 (4)
PCWP (mm Hg)	OLC	10 (2)	14 (3)	14 (3)	15 (2)	15 (5)	17 (6)	18 (8)	6.0 (4)†
	CMV	10 (3)	10 (2)	11 (3)	11 (3)	11 (4)	10 (4)	11 (3)	0.8 (4)‡
EDVI (ml m^{-2})	OLC	93 (21)	95 (18)	95 (15)	102 (17)	97 (12)	90 (15)	87 (19)	−1.5 (8)
	CMV	107 (22)	112 (21)	111 (26)	118 (32)	122 (30)	110 (21)	117 (27)	1.1 (8)

HR=heart rate; MAP=mean arterial pressure; CI=cardiac index; REF=right ventricular ejection fraction; PVR=pulmonary vascular resistance; RAP=right atrial pressure; PA_{mean}=mean pulmonary artery pressure; PCWP=pulmonary capillary wedge pressure; EDVI=right ventricular end diastolic volume index.

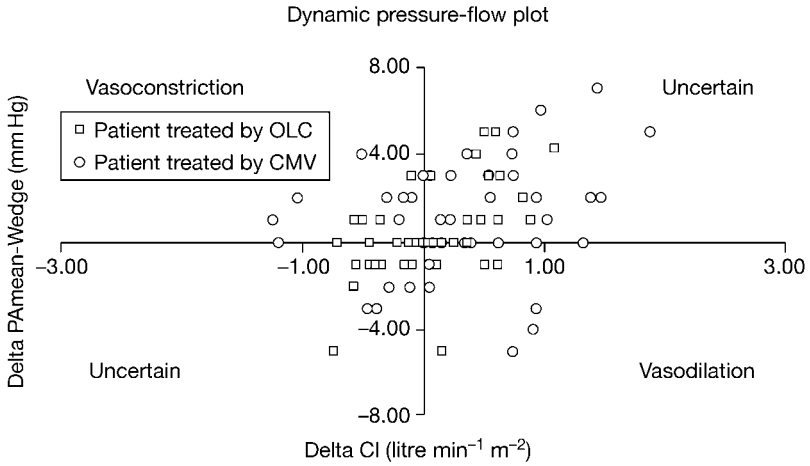

Fig 3 Dynamic pressure–flow diagram indicating changes in pulmonary vascular resistance. Baseline values of CI and the pressure decrease through the pulmonary circulation are placed at the intersection of the axes. Measurements at 60, 90, 120, 150 and 180 min are plotted as differences from baseline for each patient.

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

Table 3 Respiratory values during the study. Data are mean (SD)

Parameter	Group	Baseline	30 min	60 min	90 min	120 min	150 min	180 min
Pa_{CO_2} (kPa)	OLC	4.5 (0.3)	4.8 (0.5)	4.7 (0.4)	4.8 (0.5)	4.7 (0.5)	4.9 (0.8)	4.9 (0.8)
	CMV	5.1 (0.7)	4.8 (0.5)	5.0 (0.5)	5.0 (0.5)	5.0 (0.3)	5.0 (0.3)	5.0 (0.3)
P_{peak} (cm H_2O)	OLC	21.8 (3)	25.3 (5)	25.2 (5)	25.3 (4)	24.5 (4)	25.6 (4)	24.4 (4)
	CMV	21.9 (4)	22.1 (3)	22.0 (4)	22.5 (4)	21.7 (5)	21.9 (4)	21.0 (4)
dP (cm H_2O)	OLC	17 (3)	8 (2)	9 (3)	10 (4)	9 (3)	10 (4)	9 (2)
	CMV	17 (4)	17 (3)	17 (4)	17 (4)	17 (4)	17 (4)	16 (4)
$PEEP_{tot}$ (cm H_2O)	OLC	4.9 (0.4)	18.1 (3.7)	18.6 (2.7)	16.6 (2.0)	16.7 (2.6)	16.0 (2.6)	15.4 (1.7)
	CMV	4.9 (0.3)	5.1 (0.3)	5.2 (0.6)	5.2 (0.6)	5.2 (0.6)	5.0 (0.0)	5.0 (0.0)
Respiratory rate (b.p.m.)	OLC	12 (1)	41 (13)	45 (16)	45 (15)	43 (14)	45 (14)	43 (11)
	CMV	11 (0.7)	11 (0.7)	11 (0.8)	11 (0.8)	11 (0.8)	11 (0.8)	11 (0.7)
MV (litre min^{-1})	OLC	7.6 (1)	14 (5)	14 (4)	15 (4)	15 (4)	14 (3)	14 (2)
	CMV	8 (0)	7 (2)	8 (0)	7 (1)	7 (1)	7 (1)	7 (3)

P_{peak}=peak pressure; dP=driving pressure ($P_{peak}-PEEP_{tot}$); $PEEP_{tot}$=external PEEP+intrinsic PEEP; MV=minute ventilation.

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 H_2O PEEP did not increase right ventricular afterload,[23] supporting our results. The open lung concept advocates recruitment manoeuvres followed by elevated PEEP levels and low driving pressures, resulting in low tidal volumes.[3]

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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