EJA

Eur J Anaesthesiol 2016; **33**:1–9

ORIGINAL ARTICLE

Lung recruitment improves right ventricular performance after cardiopulmonary bypass

A randomised controlled trial

Silvina Longo, Juan Siri, Cecilia Acosta, Alberto Palencia, Arturo Echegaray, Iván Chiotti, Andrés Parisi, Lila Ricci, Marcela Natal, Fernando Suarez-Sipmann and Gerardo Tusman

BACKGROUND Atelectasis after cardiopulmonary bypass (CPB) can affect right ventricular (RV) performance by increasing its outflow impedance.

OBJECTIVE The aim of this study was to determine whether a lung recruitment manoeuvre improves RV function by re-aerating the lung after CPB.

DESIGN Randomised controlled study.

SETTING Single-institution study, community hospital, Córdoba, Argentina.

PATIENTS Forty anaesthetised patients with New York Heart Association class I or II, preoperative left ventricular ejection fraction at least 50% and Euroscore 6 or less scheduled for cardiac surgery with CPB.

INTERVENTIONS Patients were assigned to receive either standard ventilation with 6 cmH_2O of positive end-expiratory pressure (PEEP; group C, $n = 20$) or standard ventilation with a recruitment manoeuvre and 10 cmH_2O of PEEP after surgery (group RM, $n = 20$). RV function, left ventricular cardiac index (CI) and lung aeration were assessed by transoesophageal echocardiography (TOE) before, at the end of surgery and 30 min after surgery.

MAIN OUTCOME MEASURES RV function parameters and atelectasis assessed by TOE.

RESULTS Haemodynamic data and atelectasis were similar between groups before surgery. At the end of surgery, CI had decreased from 2.9 ± 1.1 to 2.6 ± 0.9 l min^{-1} m^{-2} in group C ($P = 0.24$) and from 2.8 ± 1.0 to 2.6 ± 0.8 l min^{-1} m^{-2} in group RM ($P = 0.32$). TOE-derived RV function parameters confirmed a mild decrease in RV performance in 95% of patients, without significant differences between groups (multivariate Hotelling *t*-test $P = 0.16$). Atelectasis was present in 18 patients in group C and 19 patients in group RM ($P = 0.88$). After surgery, CI decreased further from 2.6 to 2.4 l min^{-1} m^{-2} in group C ($P = 0.17$) but increased from 2.6 to 3.7 l min^{-1} m^{-2} in group RM ($P < 0.001$). TOE-derived RV function parameters improved only in group RM (Hotelling *t*-test $P < 0.001$). Atelectasis was present in 100% of patients in group C but only in 10% of those in group RM ($P < 0.001$).

CONCLUSION Atelectasis after CPB impairs RV function but this can be resolved by lung recruitment using 10 cmH_2O of PEEP.

TRIAL REGISTRATION Protocol started on October 2014.

Published online xx month 2016

Introduction

Right ventricular (RV) failure during cardiac surgery is associated with difficult cardiopulmonary bypass (CPB) weaning and increased morbidity and mortality.[1–3] Several mechanisms have been proposed to explain the occurrence of RV dysfunction.[4–7] Among them, atelectasis induced by general anaesthesia and CPB can potentially affect RV function in the perioperative period.[8,9] Ranging from mild local hypoventilation to complete atelectasis, lung collapse increases RV outflow impedance by activation of the hypoxic pulmonary

From the Department of Anesthesia (SL), Department of Intensive Care (IC), Department of Cardiovascular Surgery, Hospital Privado Universitario de Córdoba, Córdoba (AP), Department of Anesthesia, Hospital Privado de Comunidad (CA, GT), Department of Mathematics Facultad de Ciencias Exactas, Universidad Nacional de Mar del Plata, Mar del Plata Argentina (LR, MN), Department of Surgical Sciences, Section of Anesthesia and Critical Care, Uppsala University Hospital; Hedenstierna Laboratory, Uppsala, Sweden (FS-S) and CIBERES. Madrid, Spain (FS-S)

Correspondence to Gerardo Tusman, MD, Department of Anesthesiology, Hospital Privado de Comunidad, Mar del Plata, Argentina.
Tel: +54 223 4990074; fax: +54 223 4990099; e-mail: gtusman@hotmail.com

DOI:10.1097/EJA.0000000000000559

vasoconstriction reflex and by the geometrical changes of pulmonary capillaries within atelectasis.[10,11] As a consequence, the post-CPB stunned RV could become dysfunctional when confronted with this resulting in increased impedance [12]. This possible mechanism of RV dysfunction might be more common than suspected because atelectasis affects almost all patients during cardiothoracic surgery.[13]

Preliminary data in an experimental animal model and in patients undergoing cardiovascular surgery have shown that a lung recruitment manoeuvre (RM), that is, a ventilatory intervention that restores lung aeration by means of a brief and controlled increase in airway pressure, decreases RV outflow impedance.[14,15] Even though these findings support the hypothesis that atelectasis play a role in RV dysfunction, a clear cause–effect relationship has never been established in a prospective controlled clinical study.

We hypothesised that atelectasis after CPB weaning contribute to RV function impairment by increasing its outflow impedance and that a recruitment manoeuvre is a simple and effective way to restore RV function. To verify this hypothesis, we studied patients with previously normal left ventricular (LV) function to avoid the bias introduced by the intraoperative use of inotropic support on RV contractility. RV function was assessed by means of transoesophageal echocardiography (TOE).

Methods

This was a randomised and controlled clinical trial performed at Hospital Privado de Córdoba, Argentina. We included patients (40 to 70 years of age) who signed informed consent and underwent scheduled cardiovascular surgery with CPB. Patients with New York Heart Association class I or II, preoperative LV ejection fraction $\geq$ 50% and Euroscore I $\leq$ 6 could enter the study. Patients in whom TOE was contraindicated and those who were haemodynamically unstable needing inotropic support or doses of noradrenaline more than 0.3 $\mu g\ kg^{-1}\ min^{-1}$ or nitroglycerine more than 3 $\mu g\ kg^{-1}\ min^{-1}$, were excluded.

Ethical approval for this study (N° HP 4–239) was provided by the Ethical Committee of Hospital Privado de Córdoba – CIEIS, Córdoba, Argentina (Chairperson Dr Adrián M. Kahn) on 23 October 2014. The study started at the end of October 2014.

Anaesthesia was induced with propofol 1 to 1.5 mg kg^{-1}, fentanyl 10 $\mu g\ kg^{-1}$ and vecuronium 0.08 mg kg^{-1} and was maintained with propofol and remifentanil. The lungs were ventilated in a constant flow mode using a tidal volume of 6 ml kg^{-1} of predicted body weight, respiratory rate of 15 breaths min^{-1}, positive end-expiratory pressure (PEEP) of 6 cmH_2O, inspiratory to expiratory ratio of 1 : 2, inspiratory pause of 10% and FIO_2 of 0.5 (Neumovent Graph, Tecme, Córdoba, Argentina). Static respiratory compliance was calculated as tidal volume divided by driving pressure (plateau pressure minus total PEEP).

The haemodynamic management aimed at maintaining mean systemic arterial pressures (MAPs) between 55 and 85 mmHg, heart rate (HR) between 50 and 90 bpm, central venous pressure (CVP) between 5 and 15 mmHg without TOE signs of hypovolaemia. Crystalloid solutions were infused at a baseline rate of 4 ml $kg^{-1}\ h^{-1}$ and boluses of 2 to 3 ml kg^{-1} were administered if MAP was less than 55 mmHg, CVP less than 5 mmHg and urine output less than 30 ml h^{-1}. Blood transfusion with packed red cells was given whenever haemoglobin concentration decreased to less than 7.5 g dl^{-1}. Doses of noradrenaline (0.01 to 0.3 $\mu g\ kg^{-1}\ min^{-1}$) or nitroglycerine (0.1 to 3 $\mu g\ kg^{-1}\ min^{-1}$) were allowed.

Mild hypothermia (32 to 34°C) and haemodilution were induced during CPB. Pump flow was maintained at at least 2.51 $min^{-1}\ m^{-2}$ and MAP between 55 and 75 mmHg. The lungs were not ventilated during CPB. Before CPB weaning, the lungs were reconnected to the breathing circuit starting baseline ventilation after five deep manual bag insufflations reaching approximately 30 cmH_2O of peak pressure. During CPB weaning, any electrolyte or pH imbalances and preload dependency, were promptly corrected and atrial–ventricular pacing was used as needed.

Prior to surgery, patients were randomised by the attending anaesthesiologist into one of two groups using a randomisation table. Patients in the control group (group C) were ventilated conventionally as described above, maintaining 6 cmH_2O of PEEP.

The remainder were allocated to the recruitment manoeuvre group (group RM). At the end of surgery, these patients were submitted to a lung recruitment manoeuvre.[16] Briefly, in a pressure-controlled ventilation mode with an initial constant driving pressure of 15 cmH_2O, PEEP was increased in steps of 5 cmH_2O, from 5 to 20, every five breaths (= 20 s at a respiratory rate of 15 bpm for each step). Finally, driving pressure was increased from 15 to 20 cmH_2O to reach the target recruitment pressure of 40 cmH_2O that was maintained during 10 breaths (40 s). Immediately thereafter, baseline ventilation was reinstituted but this time using 10 cmH_2O of PEEP to maintain lung aeration. The recruitment manoeuvre was immediately stopped if there were TOE signs of hypovolaemia, impairment in RV contraction or a change at least 15% of baseline MAP and HR.

Patients were studied in the operating room in the supine position and closed-chest condition at three different time points: before surgery, at the end of surgery (in the RM group before performing the manoeuvre) and 30 min after the end of surgery and after the lung recruitment had been

performed in the RM group. At each protocol step, haemodynamics, TOE parameters, atelectasis, arterial partial pressure of oxygen (PaO_2) and respiratory compliance were recorded.

A second anaesthesiologist, independent from the anaesthesia management and expert in TOE, performed TOE using a 5 MHz transoesophageal echoprobe (Sonosite Turbo, Bothell, Washington, USA). Hypovolaemia was ruled out by the absence of the papillary muscle kissing sign and a superior vena cava collapsibility index less than 20%.[17] Cardiac index (CI), pulmonary artery systolic pressure (PASP) and pulmonary vascular resistance (PVR) were determined using Doppler.[18–20] RV/LV end-diastolic diameter ratio was measured between basal and middle third segments in the 4-chambers view.[21] RV function was assessed by measuring the tricuspid annular plane systolic excursion (TAPSE) using the M mode, the tissue Doppler imaging-derived s' wave and the myocardial performance index (MPI).[19,20,22] We calculated RV ejection efficiency (RVEe), defined as TAPSE/PVR.[23] We performed RV TOE assessment in accordance with recent guidelines and recommendations.[24,25]

Lung images of the lower left lung were recorded at the mid-lower oesophageal position using the technique described by Tsubo *et al.*[26–28] Atelectasis by lung sonography was defined as the presence of a tissue-like pattern with dynamic air bronchograms associated with the presence of B lines.[29,30]

Statistical analysis

Our working hypothesis was that atelectasis after CPB impair RV function and that a recruitment manoeuvre reverses this. The statistical power to confirm this hypothesis was calculated assuming that atelectasis was present in 100% of the patients in group C and in only 50% of patients in group RM considering a β-power of 80% and an α-error of 5%.[31–33]

Patients' baseline characteristics were contrasted between RM and control groups, using Student *t*-tests (for normally distributed data) or Wilcoxon nonparametric tests (when normality assumption was rejected). For categorical variables, Pearson's χ^2 tests were used to compare both groups, except when expected frequencies were less than five: in these cases, a Fisher exact test was performed. For some relevant variables, means were contrasted between RM and control groups at the three conditions: before surgery, at the end of surgery and after surgery, using Student *t*-tests with Bonferroni's correction. Intragroup comparisons were also performed between adjacent conditions. Owing to the multivariate nature of these data, RM and control groups were contrasted using Hotelling *t*-tests for comparing two multivariate means in each set of parameters. Paired multivariate comparison was performed between moments in each set of parameters within each group. This test takes all intercorrelations into account.

Data are presented as *n* (%) for proportions and mean ± SD for continuous variables. A p value less than 0.05 was considered statistically significant. All calculations were performed using the R statistical package (R Core Team, 2015, Foundation for Statistical Computing, Vienna, Austria).

Results

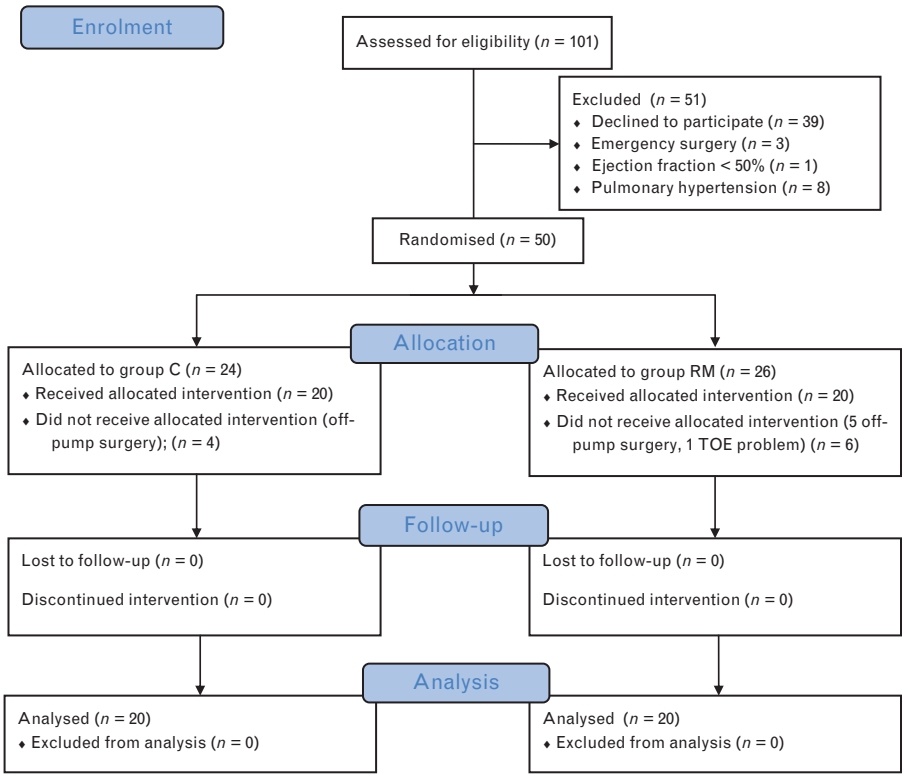
Between the study approval in October 2014 and July 2015, we screened 101 cardiac surgery patients, of whom 40 patients (39.6%) were enrolled. The flow chart of screened and enrolled patients is presented in Fig. 1. Patients' clinical and surgical characteristics were well balanced between groups (Table 1).

Table 2 describes the incidence of atelectasis during the protocol. Both groups had a similar presence of atelectasis before surgery and at the end of surgery. However, after surgery, atelectasis was observed in 100% patients in group C but only in 10% of the patients in group RM ($P < 0.001$). Figure 2 and Video 1 (supplemental material, http://links.lww.com/EJA/A107) show lung ultrasound images of representative patients of both groups.

Figure 3 illustrates the changes in static respiratory compliance and arterial oxygenation during the protocol. Compliance was similar at all protocol steps in group C but increased by 27% after surgery in group RM ($P < 0.001$). Comparing before surgery with the end of surgery, PaO_2 significantly decreased in both, group C (−21%, $P = 0.03$) and group RM (−19%, $P = 0.03$). Comparing the end of surgery with 30 min after surgery, PaO_2 increased after surgery by 8.8% in group C ($P = 0.21$) and by 29% in group RM after lung recruitment ($P = 0.01$). Although the improvement in PaO_2 after surgery appeared to be higher in group RM than in group C, no significant differences between groups were found ($P = 0.07$).

The recruitment manoeuvre was well tolerated in all patients in the treated group. MAP did not decrease more than 15% from baseline and myocardial contraction directly observed by TOE remained unchanged. Table 3 presents the comparison of haemodynamic parameters between group C and group RM before, at the end of surgery and 30 min after surgery. Table 4 shows the changes within group C and within group RM during the study. In both groups, the absolute values of HR, MAP and CVP remained without important changes during the protocol steps. PASP and PVR significantly increased at the end of surgery when compared with baseline measurements in both groups. However, after surgery, PASP and PVR decreased only in group RM ($P < 0.001$ for both, intra and inter-group comparisons). We observed no intra or inter-group differences in CI before and at the end of surgery (Fig. 4). After surgery, CI did not change in group C but increased by 42% in group RM ($P < 0.001$ for both intra and inter-group comparisons).

Fig. 1



Study flow chart. 'Off pump' surgery, surgeons decided in the operating room to perform the procedure without cardiopulmonary bypass; TOE problem, impossible to place the probe in the correct oesophageal position. Group C, control group; group RM, respiratory manoeuvre group; TOE, transoesophageal echocardiography.

The RV TOE-derived parameters were similar between groups before surgery (Table 3). Almost all patients (95%) presented abnormal RV function according to TOE analysis at the end of surgery. In group C, multivariate analysis showed that TAPSE, s' and MPI worsened during the study; however, in group RM, these parameters improved significantly after surgery (Table 4). The inter-group comparison confirmed that TAPSE, s' and MPI were different at this last protocol step ($P < 0.001$, Table 3).

RV efficiency assessed by RVEe decreased in both groups at the end of surgery without reaching statistical significance when compared with before surgery. After surgery, RVEe remained low in group C ($P = 0.05$) but increased by 107% in group RM ($P < 0.001$). There were

Table 1 Characteristics of the patients

	Group C ($n = 20$)	Group RM ($n = 20$)
Age, years	62 ± 16	63 ± 10
Men, *n* (%)	8 (45)	14 (65)
BMI, kg m^{-2}	28 ± 10	32 ± 6
EF, %	56 ± 6	57 ± 6
Hypertension, *n* (%)	11 (65)	14 (70)
Smoking history, *n* (%)	15 (75)	9 (45)
Diabetes mellitus, *n* (%)	3 (15)	7 (35)
Myocardial infarction, *n* (%)	0 (0)	1 (5)
Stroke, *n* (%)	1 (10)	3 (15)
Euroscore	4.7 ± 2.8	4.4 ± 3.1
Type of surgery, *n* (%)		
Valvular repair/replacement	7 (35)	11 (55)
Coronary artery bypass graft	6 (30)	8 (40)
Valvular + coronary surgery	2 (10)	0 (0)
Other	4 (20)	1 (0,5)
CPB time, min	104 ± 54	112 ± 32
Aortic cross-clamp, min	80 ± 46	86 ± 27
Total i.v. fluid, ml	3062 ± 838	3112 ± 516
Noradrenaline, *n* (%)	20 (100)	20 (100)
Dose, μg kg^{-1} min^{-1}	0.045 ± 0.055	0.043 ± 0.092
Nitroglycerine, *n*	0	0

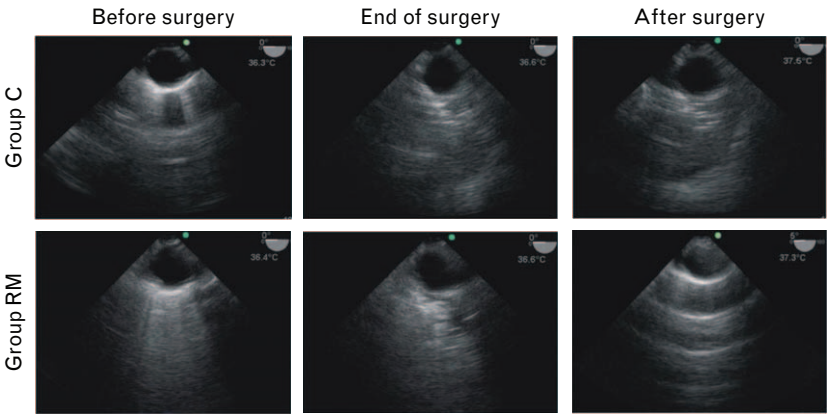
Data are presented as mean ± SD or number (%). Group C, control group; group RM, respiratory manoeuvre group. All comparisons non-significant.

Table 2 Atelectasis diagnosed by ultrasound images during the study

Protocol step	Group C ($n = 20$)	Group RM ($n = 20$)	P^a
Before surgery	NA = 3	NA = 6	
	A = 17	A = 14	0.22
End of surgery	NA = 2	NA = 1	0.88
	A = 18	A = 19	
30 min after surgery	NA = 0	NA = 18	<0.001
	A = 20	A = 2	

A, presence of atelectasis; Group C, control group; group RM, respiratory manoeuvre group; NA, no atelectasis. [a]Fisher exact test.

Fig. 2



Lung ultrasound images in a representative patient from each group. The group C patient presented a consolidation pattern in all protocol steps. The group RM patient presented consolidation before and at the end of surgery and a normal lung image 30 min after surgery. Group C, control group; group RM, respiratory manoeuvre group.

significant differences between groups at this last protocol step (Fig. 4).

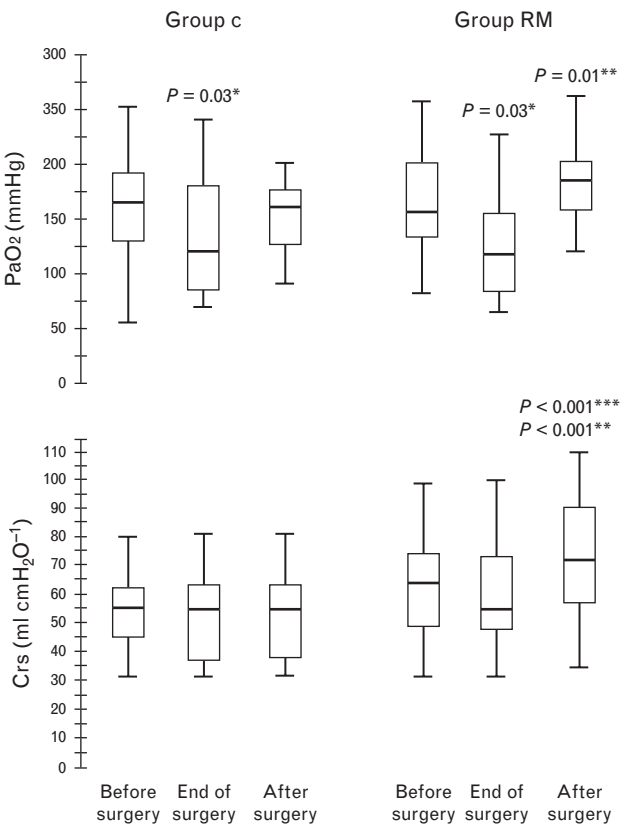
The RV/LV ratio increased in both groups at the end of surgery (intra-group comparison of $P < 0.001$; Fig. 4). However, the RV/LV ratio decreased after surgery only in group RM ($P < 0.001$ for both intra and inter-group comparisons). Figure 5 and Video 2 (supplemental material, http://links.lww.com/EJA/A108) include RV/LV images of representative patients of both groups during the protocol.

Discussion

In this study, we found that RV function was affected during cardiac surgery with CPB in 95% of our patients with normal preoperative cardiac function. This mild RV dysfunction persisted for at least 30 min after surgery when ventilating partially collapsed lungs with standard protective ventilation but resolved after lung aeration was restored by a lung recruitment manoeuvre followed by PEEP adjustment. These findings suggest that atelectasis after CPB is a common but reversible contributing mechanism impairing RV function by means of an increase in outflow impedance.

This mechanism of RV dysfunction may have relevant clinical implications. First, as demonstrated by many previous studies, lung collapse was present in all patients after CPB and 95% had TOE signs of mild RV dysfunction. Second, the aeration of the lungs in response to the selected ventilatory strategy has a direct impact modulating RV performance and can thus affect RV/LV cardiac output. Third, acknowledging that this mild RV dysfunction is commonly subclinical and adequately managed with inotropes and vasoactive drugs, its resolution by a lung recruitment manoeuvre is a relatively simple intervention that may be more efficient as it acts direct on its

Fig. 3



Box plot of arterial oxygenation and respiratory compliance during the protocol. Intragroup comparison: before vs. end of surgery (*) and end vs. after surgery (**); Bonferroni's correction was applied. Intergroup comparison after surgery (***). Crs, static respiratory compliance; group C, control group; group RM, respiratory manoeuvre group; PaO_2, arterial partial pressure of oxygen.

Table 3 Haemodynamics and transoesophageal echocardiography data during the study

Parameter		Before surgery				End of surgery				After surgery			
		Group C	Group RM	P^a	P^b	Group C	Group RM	P^a	P^b	Group C	Group RM	P^a	P^b
Haemodynamics	HR bpm	60 ± 11	66 ± 14	0.16	0.24	74 ± 10	72 ± 12	0.53	0.46	73 ± 10	77 ± 13	0.30	<0.001
	MAP mmHg	74 ± 11	80 ± 16	0.16		72 ± 12	76 ± 12	0.29		76 ± 9	78 ± 10	0.58	
	PASP mmHg	23 ± 4	22 ± 5	0.63		27 ± 5	29 ± 7	0.37		31 ± 8	22 ± 4	<0.001	
	PVR WU	0.92 ± 0.4	0.83 ± 0.3	0.37		1.03 ± 0.3	1.11 ± 0.4	0.48		1.12 ± 0.3	0.69 ± 0.3	<0.001	
	CVP mmHg	12 ± 3	12 ± 3	0.30		13 ± 5	15 ± 4	0.20		13 ± 5	14 ± 3	0.85	
RV systolic function	TAPSE mm	18 ± 2	18 ± 2	0.48	0.74	14 ± 2	14 ± 2	0.68	0.16	13 ± 2	18 ± 2	<0.001	<0.001
	s' cm s^{-1}	11.0 ± 2	10.8 ± 3	0.74		9.8 ± 2	9.4 ± 2	0.62		8.6 ± 1	12.5 ± 4	<0.001	
	MPI	0.66 ± 0.2	0.61 ± 0.2	0.41		0.78 ± 0.2	1.03 ± 0.4	0.03		0.92 ± 0.2	0.54 ± 0.2	<0.001	

Comparison between control and RM groups before, at the end of surgery and 30 min after surgery. Group C, control group; group RM, respiratory manoeuvre group; CVP, central venous pressure; HR, heart rate; MAP, mean arterial pressure; MPI, myocardial performance index; PASP, pulmonary artery systolic pressure; PVR, pulmonary vascular resistance; TAPSE, tricuspid annular plane systolic excursion; s', tissue Doppler imaging-derived s′ wave; TOE, transoesophageal echocardiography; WU, Wood units. [a] Student's *t*-test. [b] Hotelling test for multivariate means.

causal factor. Finally, it is not unreasonable to consider that susceptible patients, such as those with previous pulmonary diseases, pulmonary hypertension, RV infarction or congenital cardiac disease may be at an increased risk of developing more severe RV failure by this mechanism. In these patients, the decision to treat RV failure by means of a recruitment manoeuvre must be balanced against the potential haemodynamic side-effects induced by the brief application of high intrathoracic pressures.

Accurate assessment of RV function is difficult because of the RV's complex structure and physiology but TOE is emerging as a useful well recognised and accepted clinical method.[18,19] TOE-derived parameters such as TAPSE, s' and MPI are relatively easy, reliable and reproducible measurements to evaluate RV function. They overcome the limitations of other measurements such as those dependent on the endocardial border definition.[34,35] They correlate well with RV ejection fraction and have been shown to be good predictors of perioperative mortality in many clinical conditions. However, these indices are less well validated in the intraoperative period, are load and Doppler beam alignment-dependent and can be affected by changes in the pattern of RV contractility caused by pericardial opening.[25,36–38] We tried to minimise these TOE limitations by analysing a combination of TOE parameters and views as suggested by recent guidelines[18,24] as well as adding newer TOE views to improve Doppler beam alignment.[25]

In addition, absolute PVR values estimated by TOE have many drawbacks that must also be recognised, such as the absence of proper validation in artificially ventilated patients.[20] For this reason, we measured the efficiency of RV ejection as described by Lopez-Candales *et al.*[23] RVEe is a good parameter describing global RV impedance, correcting TAPSE for changes in both transpulmonary pressure (tricuspid regurgitation velocity) and flow (RV outflow tract velocity/time integral ratio) loads.

For a proper interpretation of the changes in RV function during the protocol steps of this study, it is important to put them in the context of TOE images of atelectasis[26–28] and TOE load-independent measurements of global cardiac function, such as preload assessment,[17] the RV/LV ratio[21,39] and left heart CI.[18] We found that the sequence of atelectasis, increase in RV dimension and decrease in CI in the context of a normal preload condition was related to impairment of RV function (Figs. 4 and 5 and Table 3). On the contrary, after normalising lung aeration by lung recruitment, the RV size decreased and the impaired CI and RV performance improved. These improvements were not observed in the control group after surgery.

The study confirmed the high incidence of atelectasis described after cardiac surgery by other authors.[31–33] Factors such as anaesthesia, pleurotomy, intermittent apnoea, lack of ventilation during CPB and cardiogenic/noncardiogenic pulmonary oedema contribute to the development of atelectasis.[31–33] It is important to emphasise that PEEP alone or lung recruitment not followed by an individualised PEEP adjustment (i.e. a decremental PEEP titration) has a less efficient effect on atelectasis resolution. It is the combination of a recruitment manoeuvre with a postrecruitment PEEP adjustment that keeps the lungs free from collapse during noncardiac and cardiac operations.[16,40,41]

However, higher randomly set levels of PEEP can potentially compress nondependent pulmonary capillaries with a consequent increase in RV impedance, especially in the presence of persistent atelectasis. We decided to use 10 cmH_2O of PEEP after recruitment aiming at minimising nondependent pulmonary capillary compression while maintaining most of the lungs well aerated.[42] We cannot exclude the possibility that better results in RV function could have been obtained with an individualised postrecruitment manoeuvre PEEP adjustment, which may also explain the persistence of visible atelectasis in 10% of the patients in group RM.

The current study is in agreement with our preliminary series of pilot cardiac surgery patients with poor

Table 4 The changes of haemodynamic parameters within control and recruitment manoeuvre groups during the study

Parameter		Group C				Group RM			
		Before vs. end of surgery		End vs. after surgery		Before vs. end of surgery		End vs. after surgery	
		Paired *P* value	Multivariate paired *P* value	Paired *P* value	Multivariate paired *P* value	Paired *P* value	Multivariate paired *P* value	Paired *P* value	Multivariate paired *P* value
Haemodynamics	HR	0.01	<0.001	0.62	0.01	0.12	<0.001	0.12	<0.001
	MAP	0.48		0.06		0.20		0.54	
	PASP	0.01		0.05		<0.001		<0.001	
	PVR	0.29		0.19		0.003		<0.001	
	CVP	0.01		0.46		0.01		0.27	
RV systolic function	TAPSE	<0.001	<0.001	0.28	0.02	<0.001	<0.001	<0.001	<0.001
	s'	0.03		0.02		0.01		<0.001	
	MPI	0.05		0.04		<0.001		<0.001	

Univariate and multivariate paired comparison between protocol steps within control and RM groups. Group C, control group; group RM, recruitment manoeuvre group; CVP, central venous pressure; HR, heart rate; MAP, mean arterial pressure; MP, myocardial performance index; PASP, pulmonary artery systolic pressure; PVR, pulmonary vascular resistance; TAPSE, tricuspid annular plane systolic excursion; s', tissue Doppler imaging-derived s' wave.

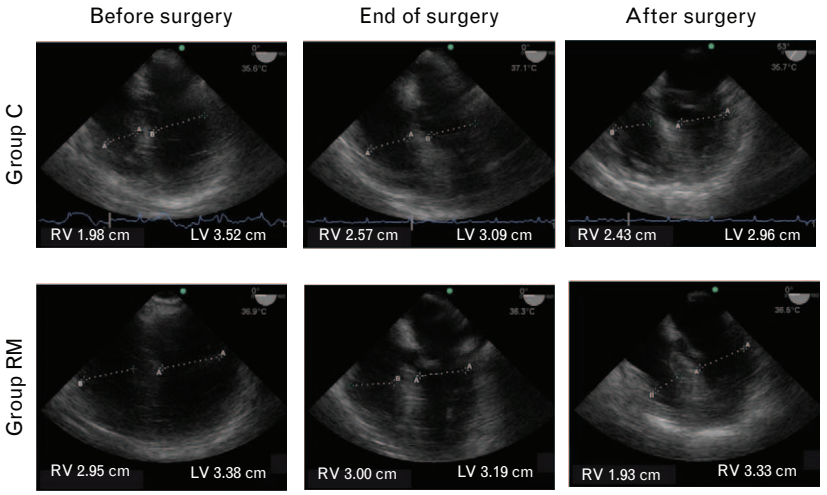
Fig. 4

Box plot of cardiac index, right ventricle efficiency and ventricular dimension during the protocol. Intra-group comparisons: before vs. end of surgery (*) and end vs. after surgery (**); Bonferroni's correction was applied. Intergroup comparisons after surgery (***). CI, cardiac index; group C, control group; group RM, respiratory manoeuvre group; RVEe, right ventricle ejection efficiency; RV/LV, right/left ventricular end-diastolic diameter ratio.

ventricular function (Ejection Fraction < 40%) evaluated by a pulmonary artery catheter.[14] We observed a significant increase in CI (from 3.0 ± 0.8 to 3.5 ± 0.6 l min^{-1} m^{-2}) and a decrease in PVR (from 274 ± 97 to 177 ± 62 dyne s cm^{-5}) in patients ventilated with 10 cmH_2O of PEEP immediately after a recruitment manoeuvre. However, in contrast with these findings, Reis Miranda *et al.*[43,44] did not find any changes in RV outflow impedance using 14 ± 4 or 17 ± 3 cmH_2O of PEEP after a recruitment manoeuvre when compared with 5 cmH_2O of PEEP without lung recruitment. These differences may be related to the populations studied, the effects of enoximone and phenylephrine used in Reis Miranda *et al.*'s studies on pulmonary circulation and/or the potential compression of pulmonary capillaries by the use of unnecessarily high PEEP levels after lung recruitment.[43,44]

There are some limitations to our study. We were unable to perform a standard and complete transthoracic lung ultrasound analysis[29] because of the intraoperative nature of our study and the presence of sterile surgical fields at some protocol steps. Thus, the TOE analysis could have

Fig. 5



Measurement of RV/LV ratio in representative patients from each group. RV and LV diameters were measured at the end of diastole between basal and mean thirds using the ME 4-chambers view. Group C, control group; group RM, respiratory manoeuvre group; LV, left ventricle; RV, right ventricle.

underestimated the presence of atelectasis as only the lower left lobe was analysed[26–28] and anaesthesia and CPB-induced lung collapse affects both lungs in a ventral–dorsal direction.[9,31] However, it is known that the weight of the heart makes the lower left lobe more prone to atelectasis than the rest of the lung regions as described in healthy volunteers, anaesthetised children and in the Acute Respiratory Distress Syndrome patients.[45,46] This means that the presence of significant atelectasis in the lower right lobe is very unlikely when the lower left lobe appears normally aerated after a lung recruitment manoeuvre. Even though TOE cannot quantify the aeration of the whole lung and has never been validated for this purpose, the method described by Tsubo *et al.*[26–28], which we used can easily detect or rule out the presence of atelectasis, as shown in Fig. 2 and Video 1, http://links.lww.com/EJA/A107.

Another shortcoming was that we did not directly measure pulmonary pressure and PVR by a Pulmonary Artery catheter as its use is not justified in patients with normal heart function. Our preliminary data in patients with poor cardiac function, in whom we did use a Pulmonary Artery catheter, support the results of this study: an increase in PVR after CPB and the corresponding decrease after lung recruitment.[14]

We did not perform a TOE analysis in the late postoperative period and, therefore, we do not know how lung collapse or lung recruitment would have affected RV function in the later postoperative period. The main objective of this study was to describe this pathophysiological mechanism of RV dysfunction and the possibility to reverse it by a lung recruitment manoeuvre. Further studies should test the long-term effects of a recruitment manoeuvre on RV performance and on patients' outcome after surgery.

Conclusion

We found that lung collapse caused mild RV dysfunction in patients undergoing cardiac surgery with CPB. Reversal of atelectasis using recruitment manoeuvres and post-recruitment PEEP adjustment confirmed the mechanical nature of this RV dysfunction and constitutes a simple and fast solution for this condition.

Acknowledgements relating to this article

Assistance with the study: we thank Dr Alejandro Contreras for his fruitful comments and valuable academic contribution and Dr Sergio Gonorazky for his technical assistance.

Financial support: local hospital resources.

Conflicts of interest: none.

Presentation: none.

References

1 Davila-Roman VG, Waggoner AD, Hopkins WE, *et al.* Right ventricular dysfunction in low output syndrome after cardiac operations: assessment by transesophageal echocardiography. *Ann Thorac Surg* 1995; **60**:1081–1086.
2 Maslow AD, Regan MM, Panzica P, *et al.* Precardiopulmonary bypass right ventricular function is associated with poor outcome after coronary artery bypass grafting in patients with severe left ventricular systolic dysfunction. *Anesth Analg* 2002; **95**:1507–1518.
3 Denault AY, Pearl RG, Michler RE, *et al.* Tezosentan and right ventricular failure in patients with pulmonary hypertension undergoing cardiac surgery: the TACTICS trial. *J Cardiothorac Vasc Anesth* 2013; **27**:1212–1217.
4 Vlahakes GJ. Right ventricular failure following cardiac surgery. *Coron Artery Dis* 2005; **16**:27–30.
5 Mehta SR, Eikelboom JW, Natarajan MK, *et al.* Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. *J Am Coll Cardiol* 2001; **37**:37–43.
6 Schultz JM, Karamlou T, Swanson J, *et al.* Hypothermic low-flow cardiopulmonary bypass impairs pulmonary and right ventricle function more than circulatory arrest. *Ann Thorac Surg* 2006; **81**:474–480.

7 Haddad F, Couture P, Tousignant C, *et al.* The right ventricle in cardiac surgery, a perioperative perspective: II. Pathophysiology, clinical importance, and management. *Anesth Analg* 2009; **108**:422–433.
8 Hedenstierna G, McCarthy GS. Airway closure and closing pressure during mechanical ventilation. *Acta Anaesthesiol Scand* 1980; **24**:299–304.
9 Brismar B, Hedenstierna G, Lundquist H, *et al.* Pulmonary densities during anaesthesia with muscular relaxation: a proposal of atelectasis. *Anesthesiology* 1985; **62**:422–428.
10 Moudgil R, Michelakis ED, Archer SL. Hypoxic pulmonary vasoconstriction. *J Appl Physiol* 2005; **98**:390–403.
11 Simmons DH, Linde LM, Miller JH, *et al.* Relation between lung volume and pulmonary vascular resistance. *Circ Res* 1961; **9**:465–471.
12 Braunwald E, Kloner RA. The stunned myocardium: prolonged, postischemic ventricular dysfunction. *Circulation* 1982; **66**:1146–1149.
13 Magnusson L, Zemgulis V, Wicky S, *et al.* Atelectasis is a major cause of hypoxemia and shunt after cardiopulmonary bypass. An experimental study. *Anesthesiology* 1997; **87**:1153–1163.
14 Tusman G, Bohm SH, Suarez Sipmann F, *et al.* Alveolar recruitment decreases pulmonary vascular resistance after cardiopulmonary by-pass (Abstract). *Anesthesiology* 2006; **105**:A229.
15 Santos Oviedo A, Gomez Peñalver E, Borges JB, *et al.* Open lung ventilation improves conditions for right ventricle performance by decreasing pulmonary vascular wave reflections in an experimental model of ARDS. *Intensive Care Med Exp* 2014; **2**:P70.
16 Tusman G, Bohm SH, Suarez-Sipmann F. Alveolar recruitment during mechanical ventilation: where are we in 2013? *Trends Anaesth Crit Care* 2013; **3**:238–245.
17 Vieillard-Baron A, Chergui K, Rabiller A, *et al.* Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. *Intensive Care Med* 2004; **30**:1734–1739.
18 Hahn RT, Abraham T, Adams MS, *et al.* Guidelines for performing a comprehensive TEE examination: recommendations from the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. *J Am Soc Echocardiogr* 2013; **26**:921–964.
19 Rudski LG, Lai WW, Afilalo J, *et al.* Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography. *J Am Soc Echocardiogr* 2010; **23**:685–713.
20 Abbas AE, Fortuin FD, Schiller NB, *et al.* A simple method for noninvasive estimation of pulmonary vascular resistance. *J Am Coll Cardiol* 2003; **41**:1021–1027.
21 Fremont B, Pacouret G, Jacobi D, *et al.* Prognostic value of echocardiographic right/left ventricular end-diastolic diameter ratio in patients with acute pulmonary embolism: results from a monocenter registry of 1,416 patients. *Chest* 2008; **133**:358–362.
22 Murphy GS, Marymont JH, Szokoi JW, *et al.* Correlation of the myocardial performance index with conventional echocardiographic indices of systolic and diastolic function: a study in cardiac surgical patients. *Echocardiography* 2007; **24**:26–33.
23 Lopez-Candales A, Lopez FR, Trivedi S, *et al.* Right ventricular ejection efficiency: a new echocardiographic measure of mechanical performance in chronic pulmonary hypertension. *Echocardiography* 2014; **31**:516–523.
24 Bartels K, Karhausen J, Sullivan BL, *et al.* Update on perioperative right heart assessment using transesophageal echocardiography. *Semin Cardiothorac Vasc Anesth* 2014; **18**:341–351.
25 Kasper J, Bolliger D, Skarvan K, *et al.* Additional cross-sectional trans-esophageal echocardiography views improve perioperative right heart assessment. *Anesthesiology* 2012; **117**:726–734.
26 Tsubo T, Sakai I, Suzuki A, *et al.* Density detection in dependent left lung region using transesophageal echocardiography. *Anesthesiology* 2001; **94**:793–798.
27 Tsubo T, Yatsu Y, Suzuky A, *et al.* Daily changes of the area of density in the dependent lung region: evaluation using transesophageal echocardiography. *Intensive Care Med* 2001; **27**:1881–1886.
28 Tsubo T, Patsy Y, Tanabe T, *et al.* Evaluation of density area in dorsal lung region during prone position using transesophageal echocardiography. *Crit Care Med* 2004; **32**:83–87.
29 Volpicelli G, Elbarbary M, Blaivas M, *et al.* Conference Reports and Expert Panel: international evidence-based recommendations for point-of-care lung ultrasound. *Intensive Care Med* 2012; **38**:577–591.
30 Acosta C, Maidana GA, Jacoviti D, *et al.* Accuracy of transthoracic lung ultrasound for diagnosing anesthesia induced atelectasis in children. *Anesthesiology* 2014; **120**:1370–1379.
31 Tenling A, Hachenberg T, Tyden H, *et al.* Atelectasis and gas exchange after cardiac surgery. *Anesthesiology* 1998; **89**:371–378.
32 Ng CS, Wan S, Yim AP, *et al.* Pulmonary dysfunction after cardiac surgery. *Chest* 2002; **121**:1269–1277.
33 Asimakopoulos G, Smith PL, Ratnatunga CP, *et al.* Lung injury and acute respiratory distress syndrome alter cardiopulmonary bypass. *Ann Thorac Surg* 1999; **68**:1107–1115.
34 Ryan T, Petrovic O, Dillon JC, *et al.* An echocardiographic index for separation of right ventricular volume and pressure overload. *J Am Coll Cardiol* 1985; **5**:918–927.
35 Ashes C, Roscoe A. Transesophageal echocardiography in thoracic anesthesia: pulmonary hypertension and right ventricular function. *Curr Opin Anesthesiol* 2015; **28**:38–44.
36 Guazzi M, Bandera F, Pelissero G, *et al.* Tricuspid annular plane systolic excursion and pulmonary arterial systolic pressure relationship in heart failure: an index of right ventricular contractile function and prognosis. *Am J Physiol Heart Circ Physiol* 2013; **305**:H1373–H1381.
37 Unsworth B, Casula RP, Yadav H, *et al.* Contrasting effect of different cardiothoracic operations on echocardiographic right ventricular long axis velocities, and implications for interpretation of postoperative values. *Int J Cardiol* 2013; **165**:151–160.
38 Tamborini G, Muratori M, Brusoni D, *et al.* Is right ventricular systolic function reduced after cardiac surgery? A two-dimensional echocardiographic study. *Eur J Echocardiogr* 2009; **10**:630–634.
39 Lang RM, Badano LP, Mor-Avi V, *et al.* Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr* 2015; **28**:1–39.
40 Reis Miranda D, Struijs A, Koetsier P, *et al.* Open lung ventilation improves functional residual capacity after extubation in cardiac surgery. *Crit Care Med* 2005; **33**:2253–2258.
41 Dyhr T, Nygard E, Laursen N, *et al.* Both lung recruitment manoeuvre and PEEP are needed to increase oxygenation and lung volume after cardiac surgery. *Acta Anaesthesiol Scand* 2004; **48**:187–197.
42 Huemer G, Kolev N, Kurz A, *et al.* Influence of positive end-expiratory pressure on right and left ventricular performance assessed by doppler two-dimensional echocardiography. *Chest* 1994; **106**:67–73.
43 Reis Miranda D, Gommers D, Struijs A, *et al.* The open-lung: effects on right ventricular afterload after cardiac surgery. *Br J Anaesth* 2004; **93**:327–332.
44 Reis Miranda D, Klompe L, Mekel J, *et al.* Open lung ventilation does not increase right ventricular outflow impedance: an echo-Doppler study. *Crit Care Med* 2006; **34**:2555–2560.
45 Malbouisson LM, Busch CJ, Puybasset L, *et al.* Role of the heart in the loss of aeration characterizing lower lobes in acute respiratory distress syndrome. *Am J Respir Crit Care Med* 2000; **161**:2005–2012.
46 Tusman G, Böhm SH, Tempra A, *et al.* Effects of recruitment maneuver on atelectasis in anesthetized children. *Anesthesiology* 2003; **98**:14–22.