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Lung recruitment maneuver improves right and left ventricular function in patients with acute respiratory distress syndrome

Alexis Lambour[1†], Yoann Zerbib[1,2†], Pablo Mercado[1,3], Loay Kontar[1], Bertrand De Cagny[1], Julien Maizel[1,2], Michel Slama[1,2] and Clément Brault[1,2*]

Abstract

Background Lung recruitment maneuvers (LRM) and high positive end-expiratory pressure (PEEP) may benefit some patients by reopening non- or poorly aerated alveoli. However, the effects of opening the lung with LRM on hemodynamics remain uncertain. This study aimed to evaluate the direct impact of LRM on cardiac function in patients with moderate-to-severe acute respiratory distress syndrome (ARDS).

Methods This post-hoc analysis included 34 patients with moderate-to-severe ARDS from two prospective cohort studies. The LRM consisted in a gradual increase in PEEP, starting from 25 cmH_2O ($PEEP_{pre}$, before the recruitment maneuver at PEEP 25 cmH_2O) until reaching 40 cmH_2O. After LRM, PEEP was decreased to 25 cmH_2O ($PEEP_{post}$, after the recruitment maneuver, also at PEEP 25 cmH_2O) followed by a decremental PEEP titration. We compared the size and function of the right ventricle (RV) and left ventricle (LV) between $PEEP_{pre}$ and $PEEP_{post}$.

Results The respiratory system compliance significantly increased from 21 ± 7 ml/cmH_2O at $PEEP_{pre}$ to 24 ± 7 ml/cmH_2O at $PEEP_{post}$ ($p < 0.001$), indicating effective lung recruitment. The RV end-diastolic diameter and the RV/LV ratio decreased after LRM (51 ± 11 vs. 41 ± 9 mm; $p < 0.001$, and 1.05 ± 0.21 vs. 0.90 ± 0.18; $p < 0.001$, respectively), suggesting reduced pulmonary vascular resistance. The RV free wall strain improved from -22 ± 10 to -25 ± 8% ($p = 0.040$). The cardiac index significantly increased from 2.1 ± 0.6 to 2.4 ± 0.7 L/min/m2 ($p < 0.001$) due to improved LV function, as demonstrated by a lower LV global longitudinal strain at $PEEP_{post}$ (-16 ± 4% vs. -19 ± 3%, $p = 0.002$).

Conclusions LRM may benefit both the lungs and the heart. The increase in transpulmonary pressure leads to an expansion in aerated lung volume, potentially reducing lung overdistension and collapse, thereby lowering RV afterload and improving RV systolic function.

Keywords Acute respiratory distress syndrome, Hemodynamic, Positive end-expiratory pressure, Lung recruitability, Respiratory mechanics

†Alexis Lambour and Yoann Zerbib contributed equally to this work.

*Correspondence:
Clément Brault
Brault.clement@chu-amiens.fr
[1]Medical Intensive Care Department, Amiens-Picardie University Hospital, 1 rue du Professeur Christian Cabrol, Amiens 80000, France
[2]MP3CV Laboratory, UR 7517, Jules Verne University of Picardie, Amiens, France
[3]Departamento de Paciente Crítico, Clínica Alemana de Santiago, Facultad de Medicina, Clínica Alemana - Universidad del Desarrollo, Santiago, Chile

Background

Acute respiratory distress syndrome (ARDS) is characterized by increased pulmonary vascular permeability, alveolar edema, and loss of aerated lung volume [1, 2]. Lung recruitment maneuvers (LRMs) and the application of high positive end-expiratory pressure (PEEP) may benefit certain patients by reopening the collapsed part of the lung and keeping it open. Although recent international guidelines advise against routine use of prolonged LRMs, the LUNG SAFE study reported that one-third of patients with severe ARDS received a recruitment maneuver [3].

During LRM, transpulmonary pressure – defined as the difference between airway pressure and pleural pressure – temporarily exceeds physiological limits, leading to the reaeration of collapsed alveoli. As a result, LRM can enhance oxygenation by increasing end-expiratory lung volume and reducing intrapulmonary shunt. Additionally, LRM may help prevent ventilator-induced lung injury by mitigating lung stress and strain during tidal inflation. Despite these potential benefits, recent meta-analyses have failed to demonstrate a survival benefit associated with LRM in ARDS [4–6]. Published in 2017, the ART trial even reported increased mortality among patients undergoing prolonged LRM combined with high PEEP [7]. These adverse outcomes might be partially attributed to the detrimental effects of LRM in terms of barotrauma and hemodynamic instability [8].

Changes in lung volume and intrathoracic (or pleural) pressure can significantly affect cardiac function because (i) the heart and lungs are confined within the chest cavity and (ii) they are connected through the pulmonary circulation [9]. Several studies have investigated heart-lung interactions during lung recruitment in ARDS patients, finding no significant hemodynamic alterations or only a transient reduction in cardiac output and/or systemic blood pressure [10–16]. However, most of these studies evaluated cardiac function before and after LRM, at different PEEP levels [10–14]. For instance, a previous study from our group assessed baseline cardiac function at clinical PEEP (i.e., at the PEEP set by the attending physician) while the effects of LRM were measured at the optimal PEEP [10]. Consequently, the direct impact of LRM on cardiac function remains unclear since LRM was followed by changes in the PEEP setting. In this study, we compared cardiac function before and after LRM, while applying the same PEEP level, in patients with moderate-to-severe ARDS.

Methods

Study population

This post-hoc analysis utilized data from two previously published prospective cohort studies conducted in a 16-bed medical intensive care unit (ICU) at Amiens University Hospital [10, 17]. These studies assessed the effects of the open lung strategy (LRM followed by decremental PEEP titration to determine the optimal PEEP) on hemodynamic and respiratory parameters in moderate-to-severe ARDS patients, according to the Berlin definition [1]. Exclusion criteria were severe hemodynamic instability (defined as an increase in vasoactive drug levels in the previous six hours), pneumothorax or pneumomediastinum, and very poor echogenicity (i.e., inability to delineate the endocardium in the apical four-chamber view). The studies involving human participants were reviewed and approved by the CPP Nord-Ouest II, Amiens, CEERNI 110. All patients or next of kin were informed about the study and consented to participate.

Ventilation strategy

Patients received sedatives (midazolam or propofol), analgesia (sufentanil) and neuromuscular blockade (cisatracurium or atracurium). We ensure the absence of spontaneous respiratory effort before the procedure. Patients were positioned in a semi-recumbent position and ventilated in volume-control mode using V500 (Dräger, Lubeck, Germany) or Servo I (Maquet, Solna, Sweden). The tidal volume was set to 6 mL per kg of predicted body weight, with plateau pressure maintained below 28–30 cmH_2O. The fraction of inspired oxygen (FiO2) was adjusted to achieve a peripheral oxygen saturation (SpO2) of 88–94%.

Recruitment maneuvers and PEEP Titration

Stepwise LRM was performed in pressure-control mode by applying a constant driving pressure of 15 cmH_2O. PEEP was initially set to 25 cmH_2O ($PEEP_{pre}$, at the start of the ascending branch of the LRM) and gradually increased to a maximum of 40 cmH_2O (in 5 cmH_2O increments every 2 min) to achieve full alveolar recruitment. Immediately following LRM, PEEP was reduced to 25 cmH_2O ($PEEP_{post}$, at the end of the descending branch), and we performed a decremental PEEP titration (See Supplementary Material, eFigure 1). Peripheral oxygen saturation, heart rate, and invasive arterial blood pressure were continuously monitored. The procedure was aborted in cases of hemodynamic instability or severe desaturation (see Supplementary Material, Appendix 1). Prior to LRM, a passive leg raising test was conducted to detect hypovolemia, and a fluid bolus was administered if preload responsiveness was detected.

Echocardiographic measurements

Comprehensive transthoracic echocardiography (TTE) was performed at $PEEP_{pre}$ and $PEEP_{post}$ (i.e. the same level of PEEP but before and after the LRM). All echocardiographic measurements were obtained using a Vivid S6 echocardiograph (GE Medical Systems, Milwaukee, WI,

USA) with a superimposed electrocardiogram and were subsequently analyzed using EchoPAC software version 203 (GE Vingmed Ultrasound AS, Horten, Norway). Following current guidelines, all measurements were taken at end-expiration and averaged over three consecutive cardiac cycles [18, 19].

Parasternal long-axis (PLAX), parasternal short-axis (PSAX), apical four-chamber (A4C), apical five-chamber (A5C), and subxiphoid (SX) views were recorded. The echocardiographic parameters assessed included left ventricular end-diastolic diameter (LVEDD), right ventricular end-diastolic diameter (RVEDD), left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), and aortic outflow tract area (obtained from PLAX). Left ventricular ejection function (LVEF) was measured from the A4C based on the modified biplane Simpson's method. The lateral mitral annulus plane systolic excursion (MAPSE) and tricuspid annulus plane systolic excursion (TAPSE) were obtained from the A4C view using M-mode. From the A5C view, we also measured the left ventricular outflow tract velocity-time integral (LVOT VTI) to estimate the stroke volume (SV), cardiac output (CO) and cardiac index (CI). Pulsed-Doppler echocardiography was used to measure peak early (E) and late diastolic (A) transmitral flow velocities. Using tissue Doppler imaging, we measured the early diastolic (E') and systolic (S) mitral and tricuspid annular velocities. The end-expiratory diameter of the inferior vena cava (IVC) was obtained in M-mode from the SX view. Moderate and severe right ventricular (RV) dilation were defined by an RVEDD/LVEDD ratio greater than 0.6 and 1.0, respectively [20]. The cardiac power index (expressed in W/m^2), reflecting cardiac pumping capability, was calculated as the product of the mean arterial pressure (MAP) and CI, divided by 451 [21] (See Supplementary Material, eFigure 2).

Myocardial strain was assessed offline by 2D speckle tracking analysis with automated function imaging (AFI, GE Vingmed Ultrasound AS, Horten, Norway). Endocardial borders were manually traced at end-diastole, and the region of interest was adjusted to include the entire myocardium. Accurate tracking was confirmed through visual assessment. Images with inadequate tracking of the endocardium were excluded from analysis. Right ventricular free wall strain (RV FWS) was calculated as the average strain of the basal, mid, and apical segments from the A4C or SX view. Left ventricular global longitudinal strain (LV GLS) was measured from six segments of the A4C view (basal inferior septum, mid inferior septum, apicoseptal, apicolateral, mid anterolateral, and basal anterolateral) [18] (See Supplementary Material, eFigure 3).

Statistical analysis

Data are presented as numbers (percentages) or means ± standard deviations, unless otherwise specified. Continuous variables between $PEEP_{pre}$ and $PEEP_{post}$ were compared using a Student's paired t-test (for normally distributed data) or Wilcoxon matched-pairs signed-rank test (for non-normally distributed data). The Shapiro-Wilk test was used to assess the normality of the data. For categorical variables, the Chi-square test or Fisher's exact test was applied as appropriate. We performed subgroup analysis for comparing the absolute and relative change in echocardiographic parameters in COVID-19 and non-COVID-19 related ARDS. Correlations between relative changes in respiratory system compliance (Crs) and echocardiographic variables were analyzed using Spearman's rho correlation test.

Being a retrospective, physiological study, no formal sample size calculation was performed. Statistical analyses were conducted using GraphPad Prism software (version 9.0.0, GraphPad Software, San Diego CA, USA). Statistical significance was set at $p < 0.05$.

Results

Participants and baseline characteristics

Out of 50 patients enrolled in the original studies, 16 were excluded due to the absence of echocardiography examination at $PEEP_{pre}$ and/or $PEEP_{post}$ or due to poor echogenicity (See Supplementary Material, eFigure 4). Baseline characteristics (i.e. at clinical PEEP) of the 34 included patients are detailed in Table 1 and Supplementary Material eTable 1. Briefly, 12 patients had preexisting chronic cardiac disease, including 10 with ischemic cardiomyopathy (two had LVEF < 50%) and 4 with atrial fibrillation.

Lung recruitment tolerance

The LRM was successfully completed in 29 (85%) patients, reaching a maximal PEEP level of 40 cmH_2O. The remaining five patients did not complete LRM due to hemodynamic instability; however, no patient required an increase in catecholamine or fluid infusion after the procedure was interrupted. Systolic, diastolic, and mean blood pressures were similar between $PEEP_{pre}$ and $PEEP_{post}$. Heart rate was slightly higher at $PEEP_{post}$ (95 ± 20 vs. 99 ± 22 bpm; $p = 0.001$) (Table 2).

Respiratory mechanics and gas exchange

During LRM, Crs increased from 21 ± 7 ml/cmH_2O at $PEEP_{pre}$ to 24 ± 7 ml/cmH_2O at $PEEP_{post}$ ($p < 0.001$), indicating effective lung recruitment and a gain in reaerated tissue. The PaO2/FiO2 ratio also improved from 146 ± 79 mmHg at $PEEP_{pre}$ to 213 ± 106 mmHg at $PEEP_{post}$ ($p < 0.001$). One patient experienced a decrease in Crs,

Table 1 Baseline characteristics of patients

Parameters	All patients (*n* = 34)
Demographic data	
Age (years)	61 ± 13
Female sex	11 (31)
BMI (kg/m^2)	31.0 ± 6.7
SAPS II (pts)	48 ± 20
SARS-CoV-2 infection	16 (47)
Delay between orotracheal intubation and inclusion (days)	3 ± 5
Comorbidities	
Ischemic cardiomyopathy	10 (29)
LVEF < 50%	2 (6)
Atrial fibrillation	4 (12)
Hypertension	21 (62)
Diabetes mellitus	10 (29)
Ventilator settings	
FiO2 (%)	82 ± 20
Vt PBW (mL/kg)	6.0 ± 0.6
RR (breaths/min)	28 ± 3
PEEP (cmH_2O)	12 ± 4
Respiratory mechanics	
Ppeak (cmH_2O)	37 ± 7
Pplat (cmH_2O)	26 ± 6
DP (cmH_2O)	14 ± 5
Crs (mL/cmH_2O)	32 ± 10
Ers (cmH_2O/mL PBW)	2.3 ± 0.9
Gas exchange	
PaO2/FiO2	110 ± 54
PaCO2 (mmHg)	48 ± 11
pH	7.31 ± 0.09
Ventilatory ratio	2.13 ± 0.59

BMI: body mass index, Crs: respiratory system compliance, DP: driving pressure, Ers: respiratory system elastance, FiO2: fraction of inspiratory oxygen, LVEF: left ventricular ejection function, PaO2: partial arterial pressure of oxygen, PaCO2: partial arterial pressure of carbon dioxide, Ppeak: peak pressure, Pplat: plateau pressure, PBW: predicted body weight, PEEP: positive end-expiratory pressure, RR: respiratory rate, SAPS: simplified acute physiology score, SARS-CoV-2: severe acute respiratory syndrome coronavirus 2, Vt: tidal volume. Data are quoted as the mean ± SD or n (%)

and two patients had a reduction in PaO2/FiO2 at $PEEP_{post}$ (Table 2).

The strongest observed correlation was between changes in Crs and RV FWS ($r = 0.51$, $p = 0.079$), suggesting a moderate positive relationship, though not statistically significant. Similarly, changes in the PaO2/FiO2 ratio showed a trend toward positive correlation with the cardiac output ($r = 0.48$, $p = 0.166$) and negative correlation with the RVEDD/LVEDD ratio (r =-0.46, $p = 0.118$), but these did not reach statistical significance (eTable 4).

Right ventricular size and function

The RVEDD decreased significantly from 51 ± 11 mm at $PEEP_{pre}$ to 41 ± 9 mm at $PEEP_{post}$ ($p < 0.001$). Similarly, the RVEDD/LVEDD ratio decreased from 1.05 to 0.90 ($p < 0.001$). There was a trend toward a lower prevalence of moderate and severe RV dilatation at $PEEP_{post}$ compared to $PEEP_{pre}$ (100 vs. 88%; $p = 0.114$, and 57 vs. 23%; $p = 0.100$, respectively).

The TAPSE increased from 15.8 ± 4.2 mm at $PEEP_{pre}$ to 18.7 ± 4.6 mm at $PEEP_{post}$ ($p < 0.001$). The prevalence of patients with systolic RV dysfunction (defined as TAPSE < 17 mm) was lower at $PEEP_{post}$ (37 vs. 69%; $p = 0.019$). Additionally, RV free wall strain improved at $PEEP_{post}$ (-22 ± 10 vs. -25 ± 8; $p = 0.040$) (Table 2; Fig. 1).

Left ventricular preload

Compared to $PEEP_{pre}$, both LVEDD and LVEDV decreased significantly at $PEEP_{post}$. The peak E-wave and A-wave velocities remained stable, and the E/A ratio was unaffected by LRM (1.2 ± 0.3 vs. 1.1 ± 0.4; p = 0.784). Similarly, the E/E' ratio did not change significantly, increasing from 9.7 ± 6.1 at $PEEP_{pre}$ to 10.0 ± 4.6 at $PEEP_{post}$ ($p = 0.294$), indicating no significant alteration in LV filling pressures (Table 2; Fig. 1).

Left ventricular contractility and output

Left ventricular systolic function improved after LRM, as we observed a significant increase in LVEF (55 ± 13 vs. 59 ± 12%; $p = 0.003$), MAPSE (9.9 ± 1.9 vs. 12.5 ± 2.3 mm; $p < 0.001$), and LV GLS (-16 ± 4 vs. -19 ± 3%; $p = 0.002$) at $PEEP_{post}$. The prevalence of patients with LV systolic dysfunction (defined as LVEF < 50%) was lower at $PEEP_{post}$ compared to $PEEP_{pre}$ (32 vs. 10%; $p = 0.053$).

The cardiac index increased from 2.1 ± 0.6 L/min at $PEEP_{pre}$ to 2.4 ± 0.7 L/min at $PEEP_{post}$, driven by a higher heart rate and stroke volume ($p < 0.001$). The cardiac index power also improved significantly from 0.36 ± 0.11 W/m² to 0.42 ± 0.16 W/m² ($p = 0.010$). Only two and four patients showed a decrease in cardiac index and cardiac power index at $PEEP_{post}$, respectively (Table 2; Fig. 1).

Subgroup analysis

The relative and absolute changes concerning ventricle sizes and functions were similar between non-COVID-19 and COVID-19 related ARDS (eTable 3).

Discussion

In this study, we evaluated the direct impact of LRM on cardiac function in ARDS patients. Left and right ventricle sizes and functions were assessed at the beginning and end of the LRM, with the same PEEP level applied. The main results are summarized as follows:

- Both RV and LV sizes and volumes decreased at $PEEP_{post}$, suggesting a reduction in preload, with no significant change in LV filling pressures.

Table 2 Respiratory, hemodynamic and echocardiographic parameters measured at the start ($PEEP_{pre}$) and the end ($PEEP_{post}$) of the lung recruitment maneuver, applying the same positive end-expiratory pressure level (i.e. 25 cmH_2O)

Parameters	$PEEP_{pre}$	$PEEP_{post}$	*P* value
ARDS characteristics			
Crs (mL/cmH_2O)	21 ± 7	24 ± 7	< 0.001
SpO2/FiO2	105 ± 28	114 ± 29	< 0.001
PaO2 (mmHg)	121 ± 80	179 ± 115	< 0.001
PaO2/FiO2	146 ± 79	213 ± 106	< 0.001
PaCO2 (mmHg)	52 ± 14	59 ± 19	0.015
pH	7.29 ± 0.08	7.24 ± 0.11	0.004
Ventilatory ratio	1.65 ± 0.47	2.21 ± 0.53	< 0.001
Hemodynamics			
SAP (mmHg)	113 ± 22	114 ± 27	0.843
DAP (mmHg)	58 ± 9	58 ± 10	0.661
MAP (mmHg)	75 ± 11	75 ± 12	0.747
PP (mmHg)	56 ± 18	56 ± 22	0.990
HR (beats per minute)	95 ± 20	99 ± 22	0.001
Conventional echocardiography			
LVOT VTI (cm)	15.5 ± 3.8	17.4 ± 3.5	< 0.001
LVEDV (mL)	104 ± 52	88 ± 32	0.019
LVESV (mL)	52 ± 32	39 ± 21	< 0.01
LVEF (%)	55 ± 13	59 ± 12	0.003
LVEF < 50%	9 (32)	3 (10)	0.053
SV (mL)	55 ± 15	62 ± 14	< 0.001
CO (L/min)	4.9 ± 1.5	5.7 ± 1.6	< 0.001
CI (L/min/m^2)	2.1 ± 0.6	2.4 ± 0.7	< 0.001
MAPSE (mm)	9.9 ± 1.9	12.5 ± 2.3	< 0.001
E-wave velocity (cm/s)	70 ± 17	71 ± 17	0.652
A-wave velocity (cm/s)	62 ± 22	65 ± 25	0.444
E'-wave velocity (cm/s)	8 ± 3	8 ± 3	0.490
S-wave velocity (cm/s)	11 ± 4	11 ± 5	0.973
E/A ratio	1.2 ± 0.3	1.1 ± 0.4	0.784
E/E' ratio	9.7 ± 6.1	10.0 ± 4.6	0.294
E-dt (ms)	199 ± 59	194 ± 55	0.648
LVEDD (mm)	49 ± 7	46 ± 8	0.016
RVEDD (mm)	51 ± 11	41 ± 9	< 0.001
RVEDD/LVEDD ratio	1.05 ± 0.21	0.90 ± 0.18	< 0.001
RVEDD/LVEDD ratio > 0.6	34 (100)	30 (88)	0.114
RVEDD/LVEDD ratio > 1.0	17 (57)	7 (23)	0.100
TAPSE (mm)	15.3 ± 4.2	18.7 ± 4.6	< 0.001
TAPSE < 17 mm	18 (69)	10 (37)	0.019
Tricuspid S-wave velocity (cm/s)	14 ± 5	14 ± 4	0.831
IVC (mm)	22 ± 5	20 ± 5	0.010
Speckle tracking			
RV free wall strain (%)	-22 ± 10	-25 ± 8	0.040
RV free wall strain > -20%	7 (44)	6 (38)	0.719
LV GLS (%)	-16 ± 4	-19 ± 3	0.002
LV GLS > -17%	10 (67)	8 (53)	0.710
Cardiac power index (W/m^2)	0.36 ± 0.11	0.42 ± 0.16	0.010

ARDS: acute respiratory distress syndrome, CI: cardiac index, CO: cardiac output, Crs: respiratory system compliance, DAP: diastolic arterial pressure, E-dt: E-wave deceleration time, E-wave: peak E-wave velocity, GLS: global longitudinal strain, HR: heart rate, IVC: inferior vena cava, LV: left ventricle, LVEDD: left ventricular end-diastolic diameter, LVEDV: left ventricular end-diastolic volume, LVEF: left ventricular ejection function, LVESV: left ventricular end-systolic volume, LVOT VTI: left ventricular outflow tract velocity-time integral, MAP: mean arterial pressure, MAPSE: mitral annulus plane systolic excursion, PP: pulse pressure, RV: right ventricle, RVEDD: right ventricular end-diastolic diameter, SAP: systolic arterial pressure, SpO2/FiO2: peripheral oxygen saturation to fraction of inspiratory oxygen, SV: stroke volume, TAPSE: tricuspid annulus plane systolic excursion. Data are quoted as the mean ± SD or n (%). Differences between 25INC and 25DEC, for normally distributed continuous variables, are tested using the Student's paired t-test, while non-normally distributed variables are tested using the Wilcoxon matched-pairs signed rank test. For categorical variables, we used Chi-square test or Fisher's exact test, as appropriate

- LRM improved RV function, evidence by a significant reduction in RV dilatation and increase in RV contractility.
- The observed improvements in RV and LV systolic function likely reflect beneficial changes in loading conditions, particularly a reduction in RV afterload.

LRM improves RV systolic function by decreasing RV afterload

In mechanically ventilated ARDS patients, high PEEP can lead to hyperinflation of non-injured alveoli and compression of wall-embedded alveolar capillaries, resulting in increased pulmonary vascular resistance (PVR). In a seminal paper, Zapol et al. documented a three-fold elevation in PVR among ARDS patients [22]. Consequently, moderate-to-severe pulmonary hypertension (mean pulmonary artery pressure > 30 mmHg) and acute cor pulmonale (characterized by a dilated RV with echocardiographic systolic and diastolic dysfunction) are common in ARDS patients and may be associated with poor outcomes [22–26].

Our findings demonstrated that LRM improved RV systolic function, evidenced by a significant increase in TAPSE and RV free wall strain at $PEEP_{post}$. This enhancement in RV function likely reflects a reduction in RV afterload, the main determinant of which is PVR. Although we did not directly measure pulmonary artery pressure (or the tricuspid regurgitation velocity as a surrogate), the observed reduction in RVEDD and RVEDD/LVEDD ratio at $PEEP_{post}$ suggests a decrease in PVR. We hypothesize that mechanisms improving PVR are directly linked to the reopening of non- or poorly aerated alveoli. The initial drop in compliance at from baseline to $PEEP_{pre}$ may indicate overdistension in non-recruitable regions. However, the subsequent increase in compliance after LRM (from $PEEP_{pre}$ to $PEEP_{post}$) supports the presence of recruitable lung tissue, where opening previously

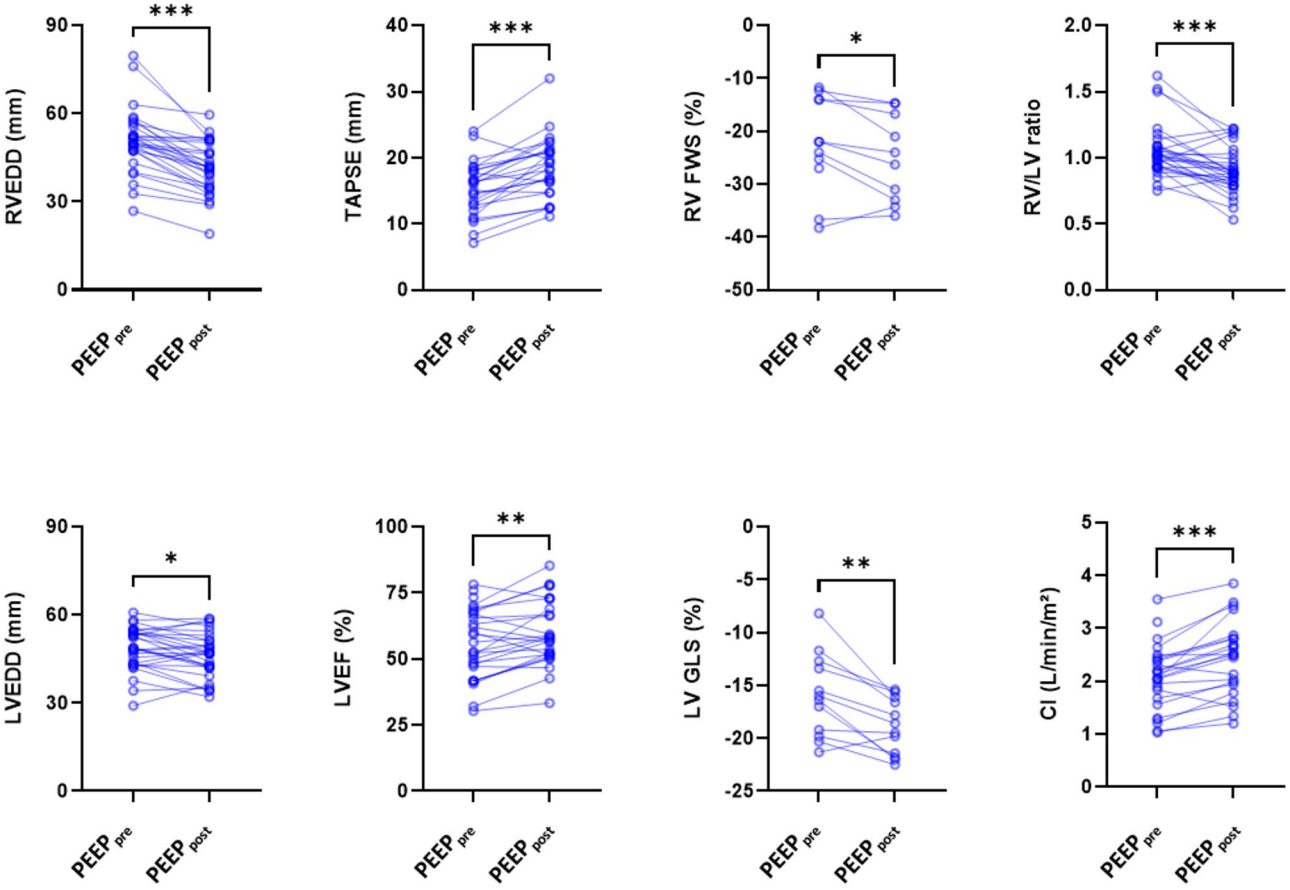

Fig. 1 Effect of lung recruitment maneuver on individual values of selected echocardiographic parameters. CI: cardiac index, LV: left ventricle, LVEDD: left ventricular end-diastolic diameter, LVEF: left ventricular ejection function, LV GLS: left ventricular global systolic strain, RV: right ventricle, RVEDD: right ventricle end-diastolic diameter, RV FWS: right ventricle free wall strain, SV: stroke volume, TAPSE: tricuspid annular plane systolic excursion. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$. Differences between $PEEP_{pre}$ and $PEEP_{post}$, for normally distributed continuous variables, are tested using the Student's paired t-test, while non-normally distributed variables are tested using the Wilcoxon matched-pairs signed rank test

collapsed alveoli improved overall respiratory mechanics. Borges et al. demonstrated that increasing alveolar pressure up to 60 cmH_2O (the same level we reached in our protocol) reduced lung collapse to less than 5% of the total lung mass [16]. Notably, they observed lower alveolar collapse and overdistension following LRM when equivalent pressures were applied during inflation. Similarly, Hansen et al. found that in pigs with lobar atelectasis, a selective LRM affected lung was hemodynamically well tolerated, whereas a "global" LRM led to circulatory side effects, likely due to hyperinflation of the healthy lung [27]. These findings support our hypothesis that efficient alveolar recruitment decreases RV afterload by reducing PVR. Recently, Borlino et al. showed that the PVR response to PEEP depends on the potential for lung recruitment – assessed by the recruitment-to-inflation (R/I) ratio. Patients with low recruitability (R/I ratio < 0.5) experienced a significant increase in PVR and RV dilatation in response to high PEEP, whereas those with high recruitability (R/I ratio ≥ 0.5) showed no significant change in PVR [28].

The relationship between PVR and lung volume follows a U-shaped curve, with PVR being lowest at functional residual capacity (FRC). Deviations from FRC due to collapse or overdistension increase PVR through compression of extra-alveolar and alveolar vessels, respectively [29]. By increasing of the number of open alveoli, LRM restores FRC, thereby reducing PVR. The RV afterload is influenced not only by PVR but also by vascular waterfall phenomena and changes in downstream pressure, as described by Permutt and colleagues. Recruitment-induced shifts in West zone physiology (i.e., transitions toward West zone 3) may reduce RV afterload by restoring perfusion to previously non-aerated alveoli [30–32]. Finally, LRM promotes ventilation-perfusion matching, improving gas exchange and reducing hypoxic pulmonary vasoconstriction [33].

LRM improves cardiac output through increased LV function

Due to ventricular interdependence, the effects of LRM on RV size and function influence the LV. Indeed, both ventricles shared a common pericardial sac and interventricular septum, and any change in RV size has an effect on LV function [34]. After LRM, the marked reduction in RV size suggests a decrease in RV preload, and despite the previously mentioned improvement in RV afterload and function, we showed a decline LV preload because the decrease in LVEDV at $PEEP_{post}$.

Despite this decrease in LV preload, we observed a significant increase in cardiac output at $PEEP_{post}$, driven by a greater stroke volume and, to a lesser extent, a higher heart rate. The increase in stroke volume was associated with improved LV systolic function, as indicated by significant increases in LVEF, MAPSE, and LV GLS at $PEEP_{post}$. Several mechanisms may contribute to this enhanced cardiac contractility, including reduced myocardial oxygen demand because the decrease in RV afterload and ventricular dilation. LRM may also improve coronary perfusion by lowering epicardial surface pressure [35]. Additionally, the increase in stoke volume might be related to the worsening of hypercapnia and hypercapnic acidosis found in our patients post-LRM. Although hypercapnia is typically considered harmful due to increased PVR, in certain conditions, it may have protective effects by improving coronary perfusion and inducing a hyperdynamic state [33, 36–40]. We hypothesize that, in our patients, this beneficial effects of mild hypercapnia outweighed its potentially harmful consequences.

Limitations of the study

Our study has several important limitations. First, we included patients with ARDS from various causes, including COVID-19, which may differ in lung mechanics and recrutability. However, our findings, in line with previous studies, indicate that the cardiac and lung response to high PEEP are similar between patients with COVID-19 related ARDS and those with ARDS from other etiologies (see eTable 3) [41, 42]. Second, our findings may not be generalizable to other LRM procedures. We used a stepwise LRM approach, allowing a gradual increase in intrathoracic pressure with good hemodynamic tolerance [43]. Additionally, this method permits to continue tidal ventilation during LRM, limiting the risk of severe hypoxemia and/or hypercapnia [44]. In contrast, sustained inflation, another LRM technique commonly used in ICUs and operative rooms, interrupts tidal ventilation might trigger the pulmonary vasoconstriction reflex [45]. Third, the tolerance of the LRM may be influenced by other factors such as volemic status, lung morphology, and respiratory mechanics [46, 47]. For instance, chest wall compliance might affect the cardiac response to LRM, as it determines the level of pressure transmitted to the lung. Grasso et al. demonstrated that patients who responded to LRM (defined by changes in PaO2/FiO2 ratio) had lower chest wall compliance than nonresponders [13]. When available, these factors are provided in Supplementary Material eTable 2. Fourth, in 5 (15%) of our patients, the LRM was aborted before reaching the maximum PEEP level of 40 cmH_2O. While a lower PEEP level may be sufficient to restore lung volume in moderate ARDS, we may not have achieved full lung recruitment [48, 49]. Fifth, important echocardiographic parameters are missing, such as the maximal tricuspid regurgitation velocity, making it difficult to definitively assess the impact of LRM on RV afterload. This limitation may be attributed to difficulties in acquiring high-quality images at high PEEP levels and the short duration of ventilation during at $PEEP_{pre}$ and $PEEP_{post}$ (2 min each). Additionally, our study relied exclusively on echocardiographic assessment, which limited our ability to fully characterize heart-lung interactions. Due to its retrospective design, we were unable to incorporate invasive monitoring techniques—such as esophageal pressure measurements to evaluate pleural and transpulmonary pressure dynamics, or the use of a Swan-Ganz catheter to assess pulmonary vascular resistance. Moreover, echocardiographic parameters were recorded only at end-expiration in accordance with current guidelines, preventing assessment of the effects of tidal volume and the associated increase in transpulmonary pressure on cardiac function. Lastly, we evaluated only the immediate effects of LRM, and future studies are needed to investigate its long-term cardiac impact.

Conclusions

Although LRM might induce hemodynamic compromise during the procedure, it may lead to improved cardiac function afterward. For this purpose, we evaluated the direct impact of LRM by measuring echocardiographic parameters before and after LRM, at the same PEEP level. The temporary increase in transpulmonary pressure results in increased aerated lung volume, thereby reducing RV afterload by lowering PVR. Both RV and LV systolic function improved at the end of the procedure. Improvements in RV and LV systolic parameters likely reflect favorable changes in loading conditions—specifically, decreased RV afterload and potentially reduced LV transmural wall stress—rather than changes in intrinsic myocardial contractility. These effects may not persist over time and must be weighed against the potential risks of hemodynamic compromise. Thus, LRM should be used cautiously,

and ideally tailored to individual recruitability and hemodynamic tolerance.

Abbreviations

A4C	Apical four-chamber
A5C	Apical five-chamber
ARDS	Acute respiratory distress syndrome
CI	Cardiac index
CO	Cardiac output
Crs	Respiratory system compliance
FiO2	Fraction of inspired oxygen
FRC	Functional residual capacity
ICU	Intensive care unit
IVC	Inferior vena cava
LRM	Lung recruitment maneuver
LVEDD	Left ventricular end-diastolic diameter
LVEDV	Left ventricular end-diastolic volume
LVEF	Left ventricular ejection function
LVESVL	Left ventricular end-systolic volume
LV GLS	Left ventricular global longitudinal strain
LVOT VTI	Left ventricular outflow tract velocity-time integral
MAP	Mean arterial pressure
MAPSE	Mitral annulus plane systolic excursion
PEEP	Positive end-expiratory pressure
PLAX	Parasternal long-axis
PSAX	Parasternal short-axis
PVR	Pulmonary vascular resistance
RV	Right ventricular
RVEDD	Right ventricular end-diastolic diameter
RV FWS	Right ventricular free wall strain
SpO2	Peripheral oxygen saturation
SV	Stroke volume
Sx	Subxiphoid
TAPSE	Tricuspid annulus plane systolic excursion
TTE	Transthoracic echocardiography

Supplementary Information

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Supplementary Material 1

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Not applicable.

Author contributions
CB, AL, and MS contributed to the conception and design of the parent study. CB, AL, YZ, PM, LK, BDC and JM were responsible for the recruitment and clinical care of the patients. CB, AL, YZ and MS participated on the analysis and interpretation of the data. CB and AL drafted the manuscript. All the authors reviewed and approved the final version of the manuscript.

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Data availability
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study was reviewed and approved by the French research ethics committee (Comité de Protection des Personnes Nord-Ouest II, Amiens, CEERNI 110) and conducted in accordance with the Declaration of Helsinki. All patients or next of kin were informed about the study and consented to participate.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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