

A Study of the Physiologic Responses to a Lung Recruitment Maneuver in Acute Lung Injury and Acute Respiratory Distress Syndrome

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OBJECTIVE: To determine the magnitude, duration, and consistency of the effects of lung recruitment maneuvers (RMs) on oxygenation, lung mechanics, and comfort in patients with acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). **METHODS:** We conducted a prospective physiologic study at 3 tertiary-care hospitals. We enrolled 28 consecutive eligible patients with ARDS or ALI and a ratio of P_{aO_2} to fraction of inspired oxygen (P_{aO_2}/F_{IO_2}) ≤ 250 mm Hg while receiving $F_{IO_2} \geq 0.50$. We performed RMs twice daily for 3 days. The first RM was at 35 cm H₂O for 20 s. If initial response was equivocal, the clinician immediately administered another RM at a higher pressure (40 cm H₂O, then 45 cm H₂O) or for longer period (30 s, then 40 s), in a randomized order. Each patient had up to 6 sets of up to 3 RMs. **RESULTS:** Twenty-seven patients met the criteria for ARDS at baseline; 1 had ALI. There was no net effect on oxygenation or pulmonary mechanics following the first or subsequent RMs. The largest rise in P_{aO_2} was from 61 mm Hg to 71 mm Hg, and the largest decrease was 6 mm Hg following the first RM. Augmenting the inflation pressure or duration had no significant effect. These findings precluded analyses about predictors of response or consistency of response. Over the entire study of 122 RMs, 5 patients developed ventilator asynchrony, 3 appeared uncomfortable, 2 experienced transient hypotension, and 4 developed barotrauma that required intervention. **CONCLUSIONS:** These results do not support the addition of scheduled RMs to usual treatment for ALI or ARDS. *Key words:* recruitment maneuver, acute respiratory distress syndrome, acute lung injury, critical illness, respiration, mechanical ventilation. [Respir Care 2008;53(11):1441–1449. © 2008 Daedalus Enterprises]

Introduction

The therapeutic concept of lung-protective ventilation, which dates back to the 1960s,¹⁻³ has emerged in the past decade as the most important advance in the management of acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). The lung recruitment maneuver (RM) is one of many experimental lung-protective strategies.⁴

Often described as a “sigh” on the ventilator, an RM includes an inspiratory breath-hold. The goal is to open the lung by inflating it to total lung capacity.⁵

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Several mechanisms may underlie lung protection from RMs. First, recruiting collapsed alveoli may temporarily improve gas exchange and thus reduce the required fraction of inspired oxygen (F_{IO_2}). Second, with an open lung, a lower pressure can expand the lungs through the tidal range. Third, if applied with adequate positive end-expiratory pressure (PEEP), an RM may reduce repetitive opening and closing that causes shear stress that can damage terminal lung units.⁶ In summary, an RM that achieves an open lung may reduce the risk of oxygen toxicity, overdistention injury, and shear-stress injury.

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There are several approaches to RMs. One of the most feasible and popular approaches is to increase the static airway pressure to a fixed level (eg, 40 cm H₂O) for a fixed period (eg, 40 s). Amato et al used this approach in a randomized trial of a multifaceted lung-protective ventilation strategy with RMs following all ventilator disconnects.⁷ Their experimental open-lung strategy was associated with a large and statistically significant reduction in 28-day mortality (relative risk 0.53, 95% confidence interval 0.31–0.91), but the contribution of RMs to that survival benefit could not be independently evaluated. That work led to additional studies of RMs for patients with ALI.

Historically, RMs were found to safely improve arterial oxygenation in patients who develop atelectasis due to general anesthesia,⁸ and in neonates undergoing high-frequency oscillation for respiratory distress syndrome.⁹ Case series in patients with ALI or ARDS, in which the RM inflation pressure range was 30–60 cm H₂O and the duration 3 s to 3 min, have reported various improvements in gas exchange.^{10–16} Few adverse effects have been reported.

Results from randomized trials of RMs have been less encouraging. A randomized trial of RMs versus a sham procedure was conducted with 80 patients in the context of a larger study. RMs with pressures of 35–40 cm H₂O for 30 s had minimal effects on oxygenation and were associated with statistically significant decreases in blood pressure (mean 7 mm Hg, $P < .01$).¹⁷ A related trial of RMs with 30 patients with extrapulmonary ARDS used inflation pressure of 50 cm H₂O for 30 s and found no sustained oxygenation improvement.¹⁸

Our aim was to investigate the magnitude, duration, and consistency of effect of intermittent scheduled RMs on arterial oxygenation, F_{IO₂}, lung mechanics, heart rate, blood pressure, and patient comfort in patients with ARDS/ALI. We also measured the effects of increasing the RM inflation pressure and duration in patients whose oxygenation did not improve after the initial RM.

Methods

The study was conducted in the intensive care units at Hamilton Health Sciences and St Joseph's Healthcare in Hamilton, Ontario, and at Mt Sinai Hospital in Toronto, Ontario, Canada. These units care for general medical-surgical patients, trauma patients, neurosurgical patients,

and patients with burns. The research ethics board at each hospital approved the study. Each patient (or legal surrogate) provided informed consent for participation.

Patients

We included patients who were receiving mechanical ventilation and had ALI or ARDS ($P_{aO_2}/F_{IO_2} \leq 250$ mm Hg on $F_{IO_2} \geq 0.50$, regardless of the level of applied PEEP, and bilateral infiltrates on a frontal chest radiograph). We excluded patients whose mean arterial pressure was < 70 mm Hg, who had chronic interstitial lung disease, subcutaneous emphysema, pneumomediastinum, pneumothorax with a persistent air leak, previous pneumectomy, exacerbation of asthma or chronic obstructive pulmonary disease, cardiogenic pulmonary edema (based on a physician's assessment), whose anticipated duration of mechanical ventilation was < 72 h, or who had undergone lung biopsy or resection during the current hospital stay.

Randomization

We randomly allocated consecutive eligible patients to 2 groups, which corresponded to 2 methods of augmenting the RM pressure or duration if the first RM had an equivocal response. To conceal the blocked randomization scheme, we used a central telephone randomization system stratified by center.

Intervention

RMs were performed twice daily for 3 consecutive days, so each patient could receive up to 6 RMs. Research respiratory therapists (RTs) supervised many of the RMs. Clinical RTs conducted some of the study RMs without direct supervision from research personnel. For logistical reasons related to data collection, we generally did not initiate a study RM after 3 pm. We did not perform RMs after lung injury was resolved (in the judgment of the attending physician).

The response to RM was rated positive, negative, or equivocal based on the following criteria. A positive response was an absolute increase in arterial oxygen saturation measured via pulse oximetry (S_{pO_2}) of $\geq 3\%$ within 5 min of completing the RM. A negative response was an S_{pO_2} decrease of $\geq 3\%$ or systolic-blood-pressure decrease of ≥ 10 mm Hg. An equivocal response was neither positive nor negative.

We used pressure-control ventilation, delivered with either a Dura 2 (Dräger, Lubeck, Germany), a model 7200 (Puritan Bennett, Pleasanton, California), or (in 1 patient only) a model 840 (Puritan Bennett, Pleasanton, California) mechanical ventilator. Clinicians provided routine sedation at their discretion and did not administer additional

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analgesia, sedation, or neuromuscular blockade for the purpose of lung recruitment. We reasoned that any changes in the background level of sedation or paralysis following baseline assessments could confound the study results. If, however, the clinicians anticipated that a patient was insufficiently sedated for the procedure, additional background sedation or analgesia was administered and the baseline assessment was deferred until there was an apparent and consistent effect.

Prior to an RM the patient was suctioned and the F_{IO_2} was adjusted to achieve an S_{pO_2} of 90–92%, to maximize the detection of any P_{aO_2} improvement.

Beginning at end-expiration we delivered continuous positive airway pressure (with no pressure support) at 35 cm H_2O for 20 s, and then immediately restored the prior ventilation settings.

If the response to an RM after 5 min was equivocal, we repeated the RM immediately, with either a greater inflation pressure (40 cm H_2O) or a longer duration (30 s), as indicated by the randomization directives. If the response to the second RM was also equivocal, we conducted a third (and final) RM with either a greater inflation pressure (45 cm H_2O) or a longer inflation period (40 s).

The Puritan Bennett ventilators could deliver continuous positive airway pressure at ≥ 35 cm H_2O , but the Dura 2 could not, so with the Dura 2 we adapted the airway-pressure-release-ventilation (APRV) mode to achieve continuous positive airway pressure in 20 patients.¹⁹ For the RM the APRV mode was set as follows. The “high pressure” parameter was set to the desired inflation pressure, and the “low pressure” parameter was set to 35 cm H_2O , which was the maximum “low pressure” option. The “high time” parameter was set at 30 s, which was the longest inspiratory-time option. The “low time” parameter was set to 0.1 s, which was the shortest inspiratory-time option. We manually measured the inspiratory time. The Dura 2 reliably delivered the prescribed continuous positive airway pressure.

In pre-study assessments we connected a Dura 2 to a calibration analyzer (Timeter RT200, Allied Healthcare Products, St Louis, Missouri). Pressure-control mode that simulated the patient experience was followed by APRV at inflation pressure of 35 cm H_2O for 20-s, 30-s, and 40-s RMs. We also tested inflation pressures of 40 cm H_2O and 45 cm H_2O for 20 s each. The airway pressure always exceeded the desired pressure by 0.5–5 cm H_2O , and never fell below the desired pressure.

Safeguards

We continuously monitored blood pressure and S_{pO_2} during the RM. The RM was immediately terminated if mean arterial pressure fell below 65 mm Hg or 70% of the baseline pressure. Volume-depletion was considered and

an intravenous fluid bolus was administered if the supervising clinician deemed it clinically appropriate. We resumed RMs after the patient regained stability, and only with the approval of the supervising clinician. RMs were also terminated if S_{pO_2} decreased to $< 85\%$ or if the patient appeared uncomfortable. Patients who experienced hypotension (mean arterial pressure < 65 mm Hg), desaturation, or discomfort on 2 consecutive RMs received no further RMs.

If a study patient became unstable at any time during the 3-day period, further study was either discontinued or postponed until the patient’s condition stabilized.

Concurrent Respiratory Support

F_{IO_2} was increased if S_{pO_2} decreased to $< 85\%$ within 15 min of the RM, or to $< 88\%$ thereafter.

Use of inhaled nitric oxide was permitted during the study. Prone positioning was permitted only for refractory hypoxemia ($S_{pO_2} \leq 90\%$, $F_{IO_2} \geq 90\%$, and progressively increasing PEEP), during which further study was postponed. RM was not performed within 30 min of any change in mechanical ventilation. F_{IO_2} and PEEP were not reduced for 6 h following RM.

Measurements

We collected demographic data, including patient age, sex, height, weight, Acute Physiology and Chronic Health Evaluation II score at admission to the intensive care unit, and all risk factors that contributed to lung injury.

A designated study RT collected the following baseline data immediately after endotracheal suctioning and prior to each RM: P_{aO_2} , F_{IO_2} , arterial oxygen saturation (S_{aO_2}), S_{pO_2} , minute volume (\dot{V}_E), mean airway pressure, intrinsic PEEP, total PEEP, ratio of inspiratory time to expiratory time, plateau and mean airway pressures, ventilation mode, use of neuromuscular blockers, use of prone positioning, presence of subcutaneous emphysema/pneumothorax/other air leaks, and mean arterial blood pressure. The most recent chest radiograph from that day was considered the baseline chest radiograph. We also documented the Glasgow coma score and the amount of sedation, analgesia, and neuromuscular blocker administered.

The study RT prospectively recorded data immediately after the RMs and at 5 min, 20 min, 40 min, and 1, 2, 3, 4, 5, and 6 h after the RMs.

Analyses

Starting with the first set of RMs with each patient, we evaluated changes from baseline (ie, immediately prior to the RM set) to 5 min after the final RM in a set. We evaluated P_{aO_2} changes at constant F_{IO_2} and PEEP. We

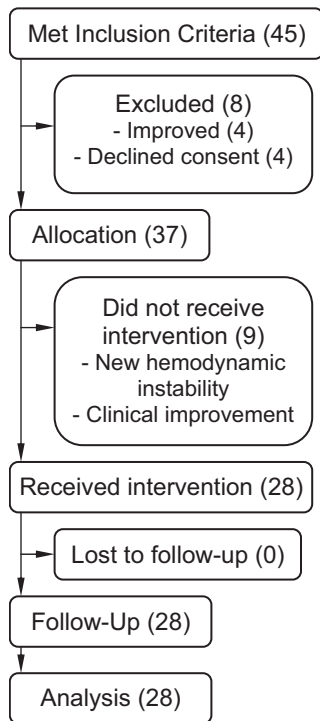


Fig. 1. Patient flow through the study.

evaluated changes in lung mechanics, as reflected by change in \dot{V}_E over that brief period, at constant driving pressure and sedation level. Compliance was not specifically measured. We repeated these assessments for the subsequent sets of RMs, from set 2 to 6, as applicable.

Next we evaluated changes in oxygenation and ventilation over the 6-hour period following each RM set.

Lack of response rendered irrelevant our planned regression modeling to evaluate potential predictors of response. We did use regression modeling to test for an interaction between RM responses and baseline PEEP.

Results

Over the 1-year period June 1999 through May 2000, 45 patients met the inclusion criteria and 28 (62%) completed the study (Fig. 1 and Table 1). Table 2 shows the numbers of RM sets conducted. We conducted a total of 122 RM sets. Reasons for not administering 46 scheduled RMs included clinical instability or death in 7 of 17 patients (41%), barotrauma in 5 (29%), and resolution of lung injury in 5 (29%).

Table 3 summarizes the baseline data (from before the first set of RMs). The severity of lung injury ranged from mild to very severe. The mean P_{aO_2}/F_{IO_2} was 118 ± 39 mm Hg, and mean PEEP was 10 ± 4 cm H_2O . The patients were heavily sedated; their mean abbreviated Glasgow coma score (ie, only the motor and eye components of the Glasgow coma scoring system) was 4.7 ± 3.3 .

Table 1. Subjects* (n = 28)

	Total	Allocated to Pressure-Augmented RMs	Allocated to Time-Augmented RMs
Female/male (n)	10/18	5/9	5/9
ARDS/ALI (n)	27/1	28/0	27/1
Age (mean \pm SD y)	57 \pm 19	59 \pm 19	55 \pm 19
APACHE-II Score at ICU admission (mean \pm SD)	23 \pm 8	25 \pm 7	22 \pm 10
ARDS risk factors per patient (mean \pm SD)	1.5 \pm 0.5	1.5 \pm 0.5	1.5 \pm 0.5
ARDS risk factors			
Severe sepsis (n)	15	8	7
Pneumonia (n)	8	1	7
Shock (n)	7	7	0
Gastric aspiration (n)	3	1	2
Burn injury (n)	3	0	3
Pulmonary contusion (n)	2	2	0
Acute pancreatitis (n)	2	1	1
Multiple transfusions (n)	1	0	1
Drug overdose (n)	1	1	0
Smoking history† (n)	9	2	7

* Data collected prior to the first set of recruitment maneuvers

† Unknown for 7 patients

RM = recruitment maneuver

ARDS = acute respiratory distress syndrome

ALI = acute lung injury

APACHE = Acute Physiology and Chronic Health Evaluation

Table 2. Recruitment Maneuver Implementation*

RM Sets	Patients Receiving RM Sets n (%)
1	28 (100)
2	26 (93)
3	21 (75)
4	20 (71)
5	16 (57)
6	11 (39)
Total	122 (100)

* 14 patients were allocated to pressure-augmented recruitment maneuvers, and 14 were allocated to time-augmented recruitment maneuvers.
NA = not applicable

Twelve patients (43%) received neuromuscular paralysis: 10 prior to baseline, one 4 h after RM, and one 5 h after RM. Eight patients (29%) received inhaled nitric oxide: 7 prior to RM, and one 4-h after RM. One patient was ventilated in the prone position throughout the study.

The respiratory data (not shown) taken just before the subsequent RMs for each patient were similar to the data

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Table 3. Baseline Data From Before the First Recruitment Maneuver*

	Total	Allocated to Pressure-Augmented RM	Allocated to Time-Augmented RM
P _{aO₂} /F _{IO₂} (mean ± SD mm Hg)	118 ± 39	117 ± 35	120 ± 44
F _{IO₂} (mean ± SD)	0.60 ± 0.19	0.59 ± 0.17	0.60 ± 0.20
P _{aO₂} (mean ± SD mm Hg)	65 ± 9	64 ± 9	66 ± 9
S _{aO₂} (mean ± SD %)	94 ± 3	93 ± 2	94 ± 3
S _{pO₂} (mean ± SD %)	92 ± 2	93 ± 2	91 ± 2
Set PEEP (mean ± SD cm H ₂ O)	10 ± 4	9 ± 4	11 ± 4
Total PEEP (mean ± SD cm H ₂ O)†	10 ± 4	10 ± 4	11 ± 5
Plateau pressure (mean ± SD cm H ₂ O)	33 ± 6	33 ± 8	33 ± 5
Mean airway pressure (mean ± SD cm H ₂ O)	20 ± 5	19 ± 5	21 ± 4
Minute ventilation (mean ± SD L/min)	12.9 ± 3.6	12.9 ± 3.8	12.9 ± 3.4
Total respiratory rate (mean ± SD breaths/min)	23 ± 7	21 ± 8	24 ± 6
Tidal volume (mean ± SD mL)	603 ± 186	649 ± 204	556 ± 160
Indexed tidal volume (mean ± SD mL/kg)	8.4 ± 3.0	8.7 ± 3.4	8.2 ± 2.6
Abbreviated Glasgow coma score‡	4.7 ± 3.3	3.9 ± 3.2	5.4 ± 3.3
Ventilator dyssynchrony (n, %)	1 (4)	0 (0)	1 (7)
Recent increase in analgesia (n, %)	2 (7)	1 (7)	1 (7)
Recent increase in sedation (n, %)	2 (7)	1 (7)	1 (7)
Paralyzed (n, %)	10 (36)	6 (43)	4 (29)
Prone positioning (n, %)	1 (4)	0 (0)	1 (7)
Inhaled nitric oxide (n, %)	7 (25)	5 (36)	2 (14)
Chest-wall restriction (n, %)	1 (4)	0 (0)	1 (7)

* All patients were ventilated with a pressure-control mode.

† Total PEEP was not measurable in 4 patients.

‡ The abbreviated Glasgow Coma Score includes only the motor and eye components. Data is missing for one patient.

RM = recruitment maneuver

F_{IO₂} = fraction of inspired oxygen

S_{aO₂} = arterial oxygen saturation

S_{pO₂} = arterial oxygen saturation measured via pulse oximetry

PEEP = positive end-expiratory pressure

in Table 3. The remaining data presented herein refer to the first set of RMs for each patient, unless otherwise specified.

Figure 2 shows the changes in oxygenation following the first RM, in each patient. Following the first RM, 4 (14%) of 28 patients had a positive response (increase oxygen saturation). The largest corresponding P_{aO₂} improvement was 10 mm Hg (from 61 mm Hg to 71 mm Hg). Sixteen of 28 patients (57%) had a negative response (decreased oxygenation). The largest P_{aO₂} decrease was 6 mm Hg (from 62 mm Hg to 56 mm Hg in one patient, and from 70 mm Hg to 64 mm Hg in another). Eight patients (29%) had an equivocal response. Augmenting the inflation pressure or duration gave no benefit.

Overall, the mean change in P_{aO₂} 5 min after the first set of RMs was 2.4 ± 5.0 mm Hg. Among the 7 patients who were receiving inhaled nitric oxide at baseline, the mean change in P_{aO₂} after the first RM set was 1.7 ± 4.8 mm Hg, which did not differ from those who were not receiving inhaled nitric oxide (*P* = .68). The results of subsequent sets of RMs were very similar; there was no apparent benefit (data not shown). Figure 3 summarizes the oxy-

genation responses to the RMs. We found no effect of baseline PEEP on any of the results reported above.

Changes in \dot{V}_E were similarly small. The mean change in \dot{V}_E at 5 min was 0.08 ± 0.79 L. In 20 (71%) patients \dot{V}_E did not change by > 0.5 L/min in either direction. In 5 patients (18%) \dot{V}_E increased, which indicates improved lung mechanics; the largest \dot{V}_E rise was from 11.6 L/min to 14.2 L/min. In 3 patients (11%) \dot{V}_E decreased, which indicates worse lung mechanics; the largest \dot{V}_E decrease was from 17.3 L/min to 15.9 L/min. The results of subsequent RMs were very similar; there was no appreciable improvement after RM.

Figure 4 shows the F_{IO₂} changes over the 6 h following the first RM. In one patient F_{IO₂} was reduced from 1.0 to 0.9 at 1 h and further to 0.80 after 5 h. Five patients (18%) had F_{IO₂} increased. No patients had a PEEP decrease. One patient required a PEEP increase after 4 h. Subsequent sets of RMs had a similar lack of effect. The ventilator respiratory rates and driving pressures were not appreciably influenced by RM during the 6 h that followed (data not shown). The levels of sedation, analgesia, and paralysis were essentially constant during that time frame.

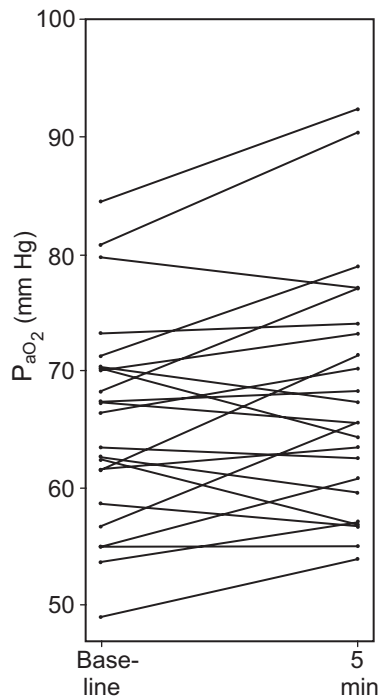


Fig. 2. Initial changes in P_{aO_2} during the first lung recruitment maneuvers. There were no significant changes in fraction of inspired oxygen or positive end-expiratory pressure in any patient 5-min after the lung recruitment maneuver.

Table 4 summarizes the adverse events associated with the 122 RMs. The average change in heart rate over 5 min was 1 ± 5.2 beats/min, and the average change in mean arterial blood pressure was 0.1 ± 8.0 mm Hg. Though those mean values did not change appreciably, individual fluctuations occurred. In 2 patients (7%) mean blood pressure fell 16 mm Hg (from 102 mm Hg to 86 mm Hg, and from 80 mm Hg to 64 mm Hg). The maximum increase in mean blood pressure was 23 mm Hg (from 70 mm Hg to 93 mm Hg). In the 5 min following RM the maximum decrease in mean arterial pressure was 18 mm Hg (from 82 mm Hg to 64 mm Hg), and the maximum increase was 35 mm Hg (from 77 mm Hg to 112 mm Hg). During RM, heart rate fell as much as 36 beats/min in 1 patient (from 126 beats/min to 90 beats/min) and rose by as much as 13 beats/min in another (from 106 beats/min to 119 beats/min).

Four patients (14%) developed barotrauma, all of which were clinically detected and confirmed on repeat chest radiography on the day of the RM. One 46-year-old male with septic shock who had bilateral chest tubes previously placed for pleural fluid drainage developed, during his first RM, a new air leak through one tube, and a new basal pneumothorax was noted on his next scheduled chest radiograph. A 20-year-old male with pulmonary contusion had a pre-existing, undocumented, and undrained pneu-

mothorax reported on computed tomogram that should have excluded him from the study. Within 6 h of his first RM there was clinical evidence of barotrauma, and bilateral chest tubes were inserted. A 79-year-old male with aspiration pneumonitis developed barotrauma after his second RM. The RM appeared uncomplicated at the time, but within hours he had bilateral chest tubes inserted for pneumothorax, pneumomediastinum, and pneumopericardium. Finally, a 45-year-old woman developed pneumothorax and subcutaneous emphysema following her fifth study RM, with no change in heart rate or blood pressure up to the time of chest tube insertion, 2 h after the RM. Because of progressing subcutaneous emphysema, a second chest tube was inserted.

Three patients (11%) experienced discomfort during RM, manifested primarily as coughing, but this was not associated with subsequent barotrauma. Moreover, none of the 5 instances of patient-ventilator asynchrony resulted in barotrauma. Two patients developed substantial hypotension (defined as a mean arterial pressure < 65 mm Hg), and no patients developed substantial desaturation during an RM (defined as an oxygen saturation of $< 85\%$).

Discussion

This randomized trial was unique in the conduct and evaluation of *scheduled* RMs in addition to usual care for patients with ALI and ARDS. On average, RM had no significant benefits for gas exchange or pulmonary mechanics. Even the greatest improvements in oxygenation and pulmonary mechanics in any individual patient were small, and there was no evidence that more aggressive RMs had any benefit. In patients with an initial equivocal response to the RM, increasing the inflation pressure to 45 cm H₂O or the duration from 20 s to 40 s did not achieve noteworthy responses. The lack of response to RM performed as a routine intervention in usual care precluded subsequent attempts to assess potential predictors of response or consistency. Though benefit was absent, 14% of patients developed barotrauma, and 7% developed substantial hypotension during RMs. Approximately 50% of RMs were associated with a decrease in arterial saturation, by at least 3%.

The failure of RMs to improve oxygenation in this study contrasts with several reported series.^{14,20-24} However, we performed regularly scheduled RMs in stable patients across the spectrum of ALI, whereas other investigators used RM for patients with deteriorating gas exchange, where greater effects may be expected.^{12,25} Other contrasting case series may have been subject to selective reporting of patients with positive findings or selective publication of positive reports. At any rate, many of the reports of improved oxygenation with RM found that this response was very transient.^{11,12,23,25,26}

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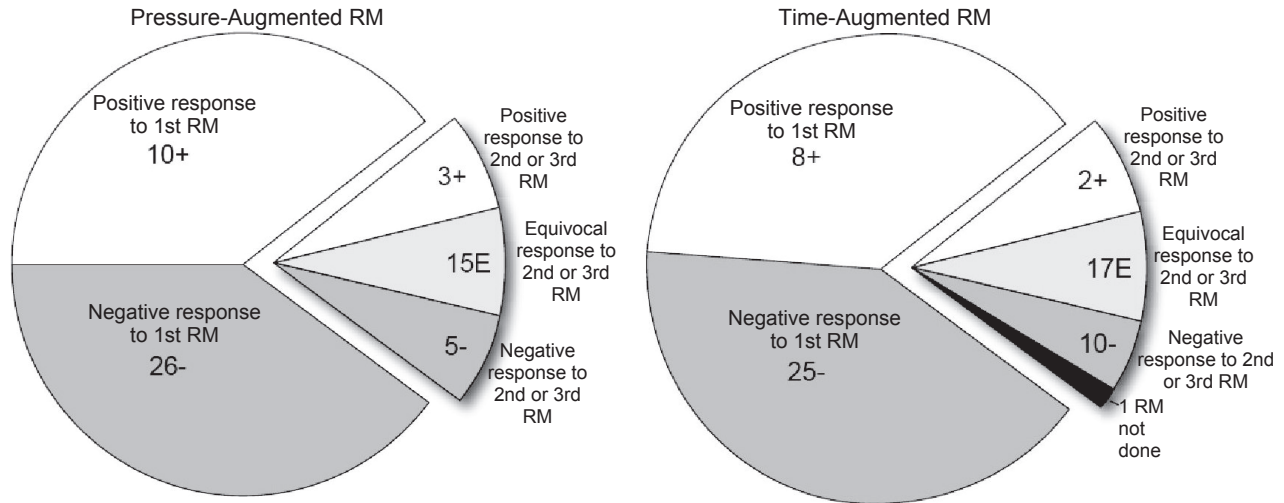


Fig. 3. Oxygenation response to 122 sets of lung recruitment maneuvers (RMs). A plus symbol denotes a positive response, defined as an absolute increase in arterial oxygen saturation measured via pulse oximetry (S_{pO_2}) of $\geq 3\%$. A minus symbol denotes a negative response, defined as S_{pO_2} decrease of $\geq 3\%$ or a systolic-blood-pressure decrease of ≥ 10 mm Hg. “E” denotes an equivocal response (neither positive nor negative).

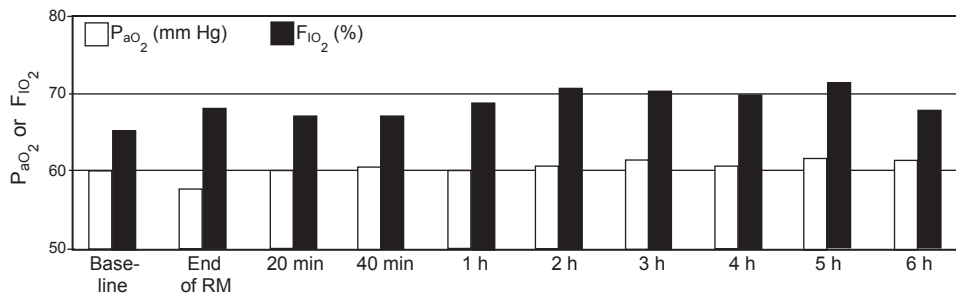


Fig. 4. Mean changes in oxygenation and fraction of inspired oxygen ($F_{I_{O_2}}$) at baseline (ie, just prior to) and over the 6 h after the lung recruitment maneuver (RM).

Table 4. Adverse Events Associated With 106 Recruitment Maneuvers

Type of Adverse Event	Number of Adverse Events	Number of Patients
Patient-ventilator dyssynchrony	5	5
Barotrauma on same-day chest radiograph	4	4
Patient discomfort	4	3
Mean arterial pressure decreased to < 65 mm Hg or $< 70\%$ of baseline	2	2

Our findings in the present study are consistent with 2 other randomized trials. Oczenski and colleagues found a significant short-term oxygenation improvement in patients with early ARDS who underwent an RM with inflating pressure of 50 cm H₂O, but that resolved within 30 min.¹⁸ In the ARDS Network trial the failure of RM may have been attributable to the high baseline PEEP ($13.8 \pm$

3 cm H₂O), which was even higher than our mean PEEP (10.1 ± 4 cm H₂O).¹⁷

Unique features of the present study include the randomized design, our focus on scheduled rather than ad hoc RMs, and our use of increased inflation pressure or duration if initial RM response was equivocal. The strengths of our study include minimal confounding effects due to different ventilator modes or setting changes. Patients were ventilated with a pressure-control mode, and the ventilator settings were not substantially changed during the 6 h following RM. There were no changes in $F_{I_{O_2}}$ or PEEP that might mask improvements in gas exchange due to RM. Differences in sedation and paralysis during the 6-h period after RM were largely avoided. Trained study RTs ensured standardization among the patients and sites in this multicenter randomized trial. We frequently collected data on ventilation parameters and gas exchange, and assessed for both improvement and adverse effects.

Limitations

We did not evaluate RMs in the setting of acute or subacute deterioration of gas exchange, so we cannot comment on the effect of RMs under those circumstances. Similarly, our findings do not apply to RMs after ventilator-disconnect or derecruitment caused by suctioning.²⁵ The aim of this trial was to test whether routine application of RMs in standard practice affects oxygenation, lung mechanics, or comfort in patients with ALI or ARDS. In addition, this trial preceded publication of the ARDS Network trial that found that mechanical ventilation with a target tidal volume of 6 mL/kg and airway pressure ≤ 30 cm H₂O saves lives. Our mean tidal volume at baseline was 8.45 ± 2.95 mL/kg, and mean plateau airway pressure was 33.2 ± 6.1 cm H₂O, and it is plausible that RMs may be more effective in the setting of lower tidal volume.

It is conceivable that the inflation pressure or duration were on average too low to open the lungs and achieve important benefit. More recent studies used much higher RM pressure and PEEP,⁸ and some case series have reported improved oxygenation after RM if PEEP was adjusted to avoid derecruitment.^{10,27,28} Indeed, in the present study the average baseline plateau airway pressure was relatively high, such that high recruiting pressure might be anticipated to have greater effect on oxygenation. However, the rate of negative responses and the lack of response to augmenting the inflating pressure do not support that notion. Logically, adverse effects must increase with pressure and duration; over 14% of our patients suffered barotrauma.

Finally, our sample size of 28 patients is relatively small, and arguably underpowered to detect an important effect. However, the total lack of RM benefit in our patients makes it unlikely that we have missed major benefits.

Conclusions

The findings from this randomized trial provide no support for the addition of routinely scheduled RMs (with pressure up to 45 cm H₂O or duration of 40 s) to the current management of critically ill patients with ALI or ARDS.

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