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Optimal duration of a sustained inflation recruitment maneuver in ARDS patients

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Abstract *Purpose:* To measure the dynamics of recruitment and the hemodynamic status during a sustained inflation recruitment maneuver (RM) in order to determine the optimal duration of RM in acute respiratory distress syndrome (ARDS) patients. *Methods:* This prospective study was conducted in a 12-bed intensive care unit (ICU) in a general hospital. A 40 cmH_2O sustained inflation RM maintained for 30 s was performed in 50 sedated ventilated patients within the first 24 h of meeting ARDS criteria. Invasive arterial pressures, heart rate, and SpO_2 were measured at 10-s intervals during the RM. The volume increase during the RM was measured by integration of the flow required to maintain the pressure at 40 cmH_2O, which provides an estimation of the volume recruited during the RM. Raw data were corrected for gas consumption and fitted with an exponential curve in order to determine an individual time constant for the volume increase. *Results:* The average volume increase and time constant were 210 ± 198 mL and 2.3 ± 1.3 s, respectively. Heart rate, diastolic arterial pressure, and SpO_2 did not change during or after the RM. Systolic and mean arterial pressures were maintained at 10 s, decreased significantly at 20 and 30 s during the RM, and recovered to the pre-RM value 30 s after the end of the RM (ANOVA, $p < 0.01$). *Conclusions:* In early-onset ARDS patients, most of the recruitment occurs during the first 10 s of a sustained inflation RM. However, hemodynamic impairment is significant after the tenth second of RM.

Keywords Mechanical ventilation · Recruitment maneuver · ARDS

Introduction

In acute respiratory distress syndrome (ARDS) patients, recruitment refers to the dynamic process of reopening previously collapsed lung units through an intentional transient increase in transpulmonary pressure [1]. The rationale for the use of recruitment maneuvers (RM) is to promote alveolar recruitment, leading to increased end-expiratory lung volume. An increase in end-expiratory lung volume may improve gas exchange, reduce the strain induced by ventilation [2], and prevent repetitive opening and closing of unstable lung units [3], all of which reduce ventilator-induced lung injury (VILI). Although a wide variety of RM have been described, it is uncertain which is the best method, and the optimal pressure, duration, and periodicity are unknown [4]. Because of viscoelastance and other time-dependent force-distributing phenomena, the tendency of a previously collapsed airway or alveoli to open is a function of both transpulmonary pressure and time [5]. Thus, the most commonly used RM in clinical studies is sustained application of continuous positive airway pressure (CPAP) of 30–50 cmH_2O for 30–40 s

(sustained inflation RM) [6–13]. In an animal model of ARDS, most of the recruitment occurs in the first seconds of sustained inflation RM [14]. Such information is missing in ARDS patients. The hypothesis of this study was that most of the recruitment occurs during the first seconds of sustained inflation RM in ARDS patients and that long-duration RM could compromise hemodynamic status. This prospective clinical study aimed to measure the dynamics of recruitment and the hemodynamic response during sustained inflation RM in order to determine the optimal duration of RM in ARDS patients.

Patients and methods

Patients

This prospective study was conducted from July 2007 to November 2008 in the 12-bed medical-surgical adult ICU of Font Pré Hospital in Toulon (France). The regional institutional review board (CPP of Nice) approved the protocol and informed consent was obtained from each patient's next of kin. Patients were included if they presented early-onset (≤24 h) ARDS as defined by the American-European consensus conference [15]. Inclusion criteria were a PaO_2/FiO_2 ratio measured by blood gas analysis of no greater than 200 mmHg after 30-min application of a 10 cmH_2O positive end-expiratory pressure (PEEP) with FiO_2 at least 50% [16]. Exclusion criteria were severe obesity (BMI > 35), pulmonary emphysema [4], severe chronic respiratory disease requiring long-term oxygen therapy or long-term mechanical ventilation, bronchopleural fistula, severe hypoxemia with PaO_2/FiO_2 ratio less than 60 mmHg, hemodynamic disorder requiring more than 1.4 μg/kg/min of epinephrine or norepinephrine, hypovolemia reflected by a variation in pulse arterial pressure (ΔPP) over 13% [17], increased intracranial pressure [18], pregnancy, and moribund status.

Patients were orally intubated and mechanically ventilated using a Galileo Gold ventilator (Hamilton Medical AG, Rhäzüns, Switzerland) in adaptive support ventilation (ASV) mode [19]. Settings (minute volume and maximum inspiratory pressure) were adjusted to keep tidal volume (V_T) below 10 mL/kg of predicted body weight (PBW) with a plateau pressure below 30 cmH_2O [20, 21]. Patients were kept in a supine position with the head of the bed elevated to 30°. Sedation used a midazolam–fentanyl combination to reach a Ramsay score of 6, and patients were paralyzed for the purpose of the study with a single injection of cisatracurium. Electrocardiogram, intra-arterial blood pressure (radial or femoral artery), and pulse oximetry were monitored throughout the study.

Recruitment maneuver

The cuff of the endotracheal tube was transiently overinflated to 50 cmH_2O and, to ensure there were no air leaks, all equipment connections were verified. Absence of leak was confirmed when no changes were observed in airway pressure during a 10-s end-inspiratory pause. A single sustained inflation RM was performed using the previously described method [22, 23] (Fig. 1). In short, airway pressure was increased at a rate of 5 cmH_2O/s from 10 to 40 cmH_2O, which was sustained for 30 s (PV tool 2 Hamilton Medical AG, Rhäzüns, Switzerland). Afterwards, airway pressure decreased to 10 cmH_2O at a rate of 5 cmH_2O/s and basal ventilation resumed. To test the effect of starting pressure, 10 patients were studied starting the RM at a PEEP of 5 cmH_2O. The RM was immediately terminated if mean arterial pressure fell

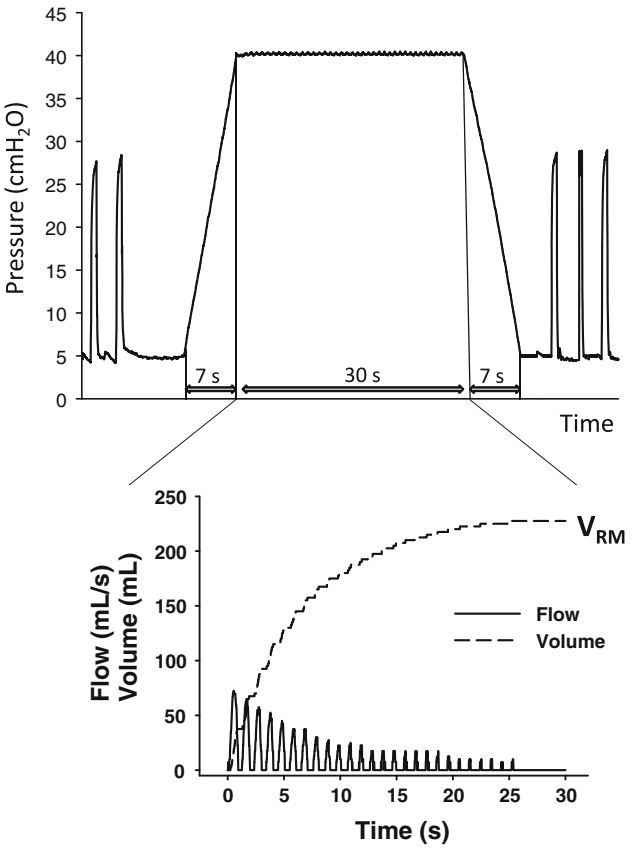

Fig. 1 Representation of the experimental protocol: airway pressure was increased from either 5 or 10 cmH_2O to 40 cmH_2O. RM used the sustained inflation method at 40 cmH_2O for 30 s (*upper panel*). If recruitment occurs, the total volume of the lung increases. As a consequence, airway pressure decreases. To maintain the airway pressure at 40 cmH_2O, the ventilator inflates the lung with spikes of flow (*solid line in lower panel*). Integration of the spikes of flow measured at the airway is used to calculate the volume increase during the RM (V_{RM}) (*dashed line in lower panel*) as an assessment of the volume recruited during the RM

below 55 mmHg, SpO_2 decreased to 85% or less [24, 25], or cardiac arrhythmia occurred. A chest X-ray was performed to detect extra-alveolar air within 24 h after RM in all patients.

Measurements

Airway pressure and flow were measured using the ventilator's proximal pneumotachograph (single-use flow sensor, PN 279331, Hamilton Medical, Bonaduz, Switzerland, linear between −120 and 120 L/min with a ±5% error of measure) inserted between the endotracheal tube and the Y-piece. The signal was acquired at 67 Hz and downloaded from the ventilator using specific acquisition software (Data logger, Hamilton Medical AG, Rhäzüns, Switzerland). Volume was obtained by integration of the flow signal. Static compliance (C_{STAT}) was measured by the least-squares fit method over the full respiratory cycle immediately before and after the RM [26]. Plateau pressure was measured using a 5-s end-inspiratory occlusion.

Systolic, diastolic, and mean arterial blood pressure, heart rate (HR), and pulse oximetry were measured throughout the RM and recorded at five time points: T_0 (beginning of the RM), T_{10}, T_{20}, and T_{30} (10, 20, and 30 s, respectively, after the beginning of the RM), and T_{60} (60 s after the beginning of the RM, i.e., 30 s after the end of the RM).

Calculations

The volume increase during the RM (V_{RM}) was calculated by integration of the flow required to maintain the pressure at 40 cmH_2O assuming that in leak-free conditions, the additional volume needed to maintain the pressure is a recruited volume (Fig. 1) [22]. The volume increase during the RM was corrected for oxygen consumption [27].

To determine the dynamics of the individual volume increase during the RM, data were fitted with an exponential curve according to:

$$V(t) = V_{RM}\left(1 - e^{-t/\tau}\right)$$

where V_{RM} is the total volume increase, e is the base of natural logarithm, and τ is the time constant of the volume increase [28, 29]. The time to achieve 95% of V_{RM} was therefore calculated as $3 \times \tau$ and half of V_{RM} was obtained at $0.69 \times \tau$ [30]. Leaks were ruled out by visually checking the volume pattern over time during the 30-s sustained inflation assuming that a linear increase in volume without plateau indicated leaks, whereas in the absence of leaks the volume increase had an exponential shape with a plateau (Fig. 1). In addition, leaks were suspected when the data did not correctly fit the exponential function with a square Pearson coefficient of correlation of 0.95 or less. If leaks were suspected from visual or statistical analysis as defined before, data were rejected and not analyzed.

Statistical methods

Statistics were performed using SigmaStat (version 3.5, SPSS, Inc., Chicago, IL, USA). Data are reported as mean ± SD. Analysis of the dynamics of the volume increase used nonlinear regression (Sigma plot, version 11.0, SPSS, Inc., Chicago, IL, USA). A one-way analysis of variance for repeated measures (ANOVA) was used to analyze SpO_2, HR, and arterial pressures during the RM, followed by pairwise means comparison using Holm–Sidak post hoc tests. *T* test was used to compare results between patients with the RM initiated at a 5 cmH_2O PEEP and patients with the RM initiated at a 10 cmH_2O PEEP. Statistical significance was assumed for p value of 0.05 or less.

Results

Fifty-five patients were enrolled in the study. Five patients were excluded from analysis (two patients for an early termination of the RM because SpO_2 was 85% or less, and three patients for air leaks). In the same period, eight other patients with early-onset ARDS were screened but not included because of hemodynamic instability, lung emphysema, or inability to obtain informed consent [31]. Thus, 50 patients were analyzed, 10 patients with an initial PEEP of 5 cmH_2O and 40 patients with an initial PEEP of 10 cmH_2O. Baseline characteristics of the study population and outcomes are described in Table 1. Chest X-rays performed after RM revealed that extra-alveolar air was not found in any of the patients.

In the overall population, sustained inflation RM induced an average V_{RM} of 210 ± 198 mL. V_{RM} was higher with an initial PEEP of 5 cmH_2O as compared with 10 cmH_2O (390 ± 242 mL vs 178 ± 174 mL, for PEEP of 5 and 10 cmH_2O, respectively, $p = 0.008$). Figure 2 represents the individual volume increase during the RM. The average time constant of the volume increase was 2.3 ± 1.3 s. Half of V_{RM} was achieved after 1.6 ± 0.9 s and 95% of V_{RM} was achieved after 6.8 ± 4.0 s. More than 98% of V_{RM} was achieved at 10 s of the RM (T_{10}). Time constant of the volume increase was not significantly different with an initial PEEP of 5 cmH_2O as compared with 10 cmH_2O (2.6 ± 1.0 s vs 2.3 ± 1.3 s, for PEEP of 5 and 10 cmH_2O, respectively, $p = 0.55$). C_{STAT} increased from 30 ± 9 mL/cmH_2O before the RM to 33 ± 11 mL/cmH_2O immediately after the RM ($p < 0.001$).

Table 1 Baseline characteristics of the study population and outcomes for the 50 patients included

Parameter	Value
Age (years)	62 ± 20
Sex (male/female)	32/18
SAPS II	52 ± 15
Body mass index (kg/m^2)	22 ± 8
Duration mechanical ventilation before inclusion (days)	0.3 ± 0.9
Tidal volume/PBW (mL/kg)	8.0 ± 1.2
Plateau pressure (cmH_2O)	24 ± 4
C_{STAT} (mL/cmH_2O)	30 ± 9
FiO_2 (%)	71 ± 20
pH	7.25 ± 0.10
PaO_2/FiO_2 (mmHg)	129 ± 37
$PaCO_2$ (mmHg)	45 ± 10
ARDS causes (*n*/%)	
Inhalation	21/42
Pneumonia	13/26
Septic shock	7/14
Near-drowning	6/12
Pulmonary contusion	2/4
Acute pancreatitis	1/2
Total duration of mechanical ventilation (days)	10 ± 10
Total duration of ICU stay (days)	11 ± 10
Mortality in ICU (*n*/%)	23/46

Values are mean ± SD
PBW, predicted body weight; C_{STAT}, static compliance

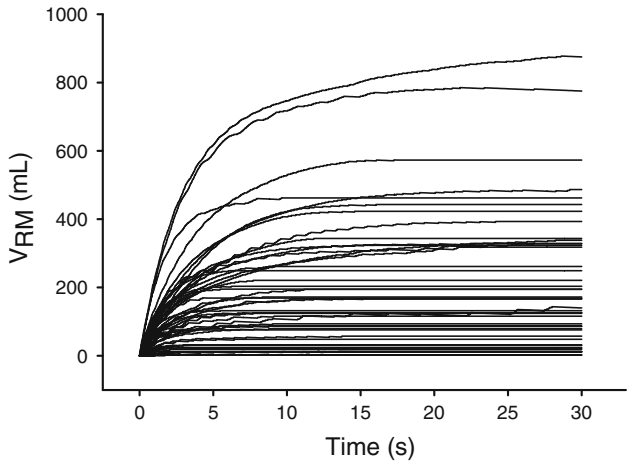

Fig. 2 Individual dynamics of volume increase during the RM. Each *thin line* represents a patient

HR, diastolic arterial pressure, and SpO_2 did not change during or after the RM (Fig. 3). Systolic and mean arterial pressures decreased significantly at T_{20} and T_{30} and recovered to the pre-RM value at T_{60} ($p < 0.01$) (Fig. 4). Figure 5 shows a representative case with the volume increase and the hemodynamic compromise.

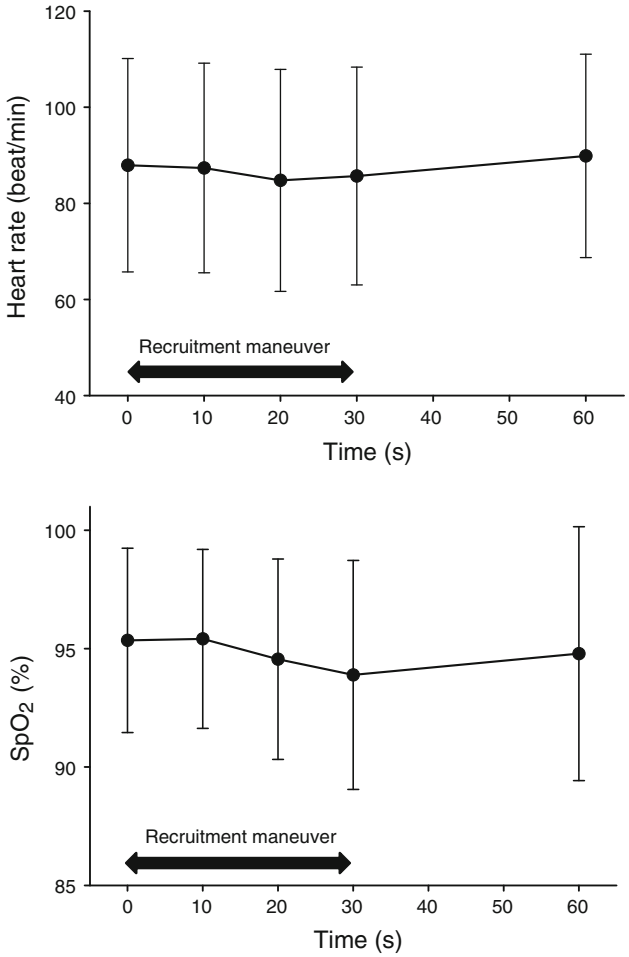

Fig. 3 HR (*upper panel*) and SpO_2 (*lower panel*) during (0–30 s) and after the RM (30–60 s). ANOVAs were not significant with $p = 0.76$ and $p = 0.53$ for HR and SpO_2, respectively

Discussion

This study revealed that most of the volume increase during a sustained inflation RM is achieved within 10 s, and arterial pressures decreases after 10 s. These results favor the use of a short duration for the sustained inflation RM.

The present study found a short time constant to describe the volume increase during an RM. This result is in line with experimental and clinical studies. In an animal model of acute lung injury, the time constant of aeration during inflation measured by dynamic CT scan was 0.5 s [29]. Using in situ microscopy to measure recruitment in individual alveoli as well as macroscopic visualization of recruitment at the whole lung level in a rat model of ARDS, Albert et al. [14] reported that most of the recruitment occurs during the first 2 s of RM. In patients with healthy lungs, the dynamics of re-expansion of atelectasis after anesthesia was evaluated using CT

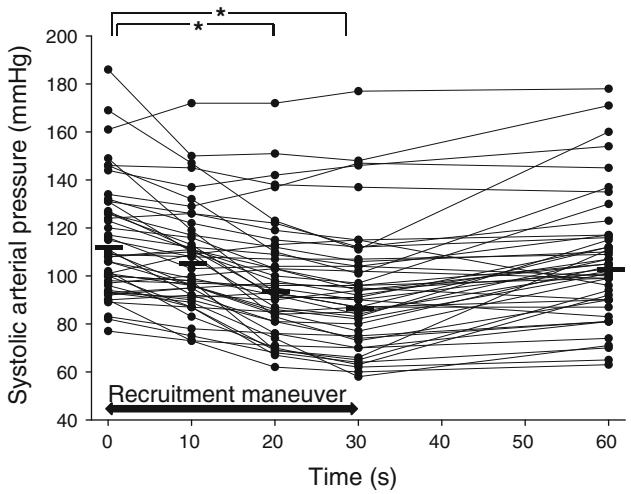

Fig. 4 Individual systolic arterial pressures during (0–30 s) and after the RM (30–60 s). Each *thin line* represents a patient. ANOVA was significant with $p < 0.01$. $*p < 0.05$ for pairwise means comparisons with T_0. *Thick lines* are mean values

scan measurements and revealed a mean time constant of 2.6 s which is very close to the 2.3 s found in the present study in ARDS patients [28]. In acute lung injury and ARDS patients, it has been shown that extending the duration of sustained inflation RM from 20 to 30 s and 40 s produces no benefit in terms of oxygenation [24]. These results imply that more than 98% of the recruitment is achieved at 10 s. Interestingly, the dynamics of lung recruitment was not influenced by the initial level of PEEP setting. Starting RM with lower PEEP resulted in a larger volume recruited, which suggests that the higher inspiratory pressure associated with PEEP 10 cmH_2O efficiently recruited part of the lung.

The most frequently observed side effect of sustained inflation RM is transient hypotension [3]. Despite a careful fluid management prior to RM to maintain pulse pressure variation below 13% [32], systolic and mean arterial pressures decreased progressively throughout the RM and became significant at 20 and 30 s with a rapid recovery of the basal condition 30 s after the end of the RM. Overall systolic and mean arterial pressures decreased by a median value of 16 [8–28] mmHg and 8 [2–13] mmHg, respectively, from the beginning to the end of the RM. Such an impairment may have clinical consequences, especially as arterial pressure underestimates the true effect of the RM on cardiac output [10]. An animal study has shown an almost immediate peripheral vasoconstriction in response to the RM, which preserved the arterial pressure much better than cardiac output [33]. A 10-s sustained inflation RM would have limited the decrease in systolic and mean arterial pressures. Studies comparing hemodynamic parameters before and after the RM reported no hemodynamic compromise, probably because of this transient effect [6, 13, 34]. In animal models, hemodynamic compromise was constant but differed according to the model used (pneumonia being worse than oleic acid injury or VILI) and the RM performed (40-s sustained inflation RM being worse than incremental PEEP) [32]. Grasso et al. [8] recorded hemodynamic parameters during a 40 cmH_2O/40 s sustained inflation RM in 22 ARDS patients and observed a substantial reduction of mean arterial pressure and cardiac output in oxygenation non-responders (<50% increase in PaO_2/FiO_2 ratio after the RM) and in patients with a low chest wall compliance. In the present study, the hemodynamic impairment was not correlated with the volume increase. The hemodynamic impairment was delayed relative to the anatomical recruitment. This favors the

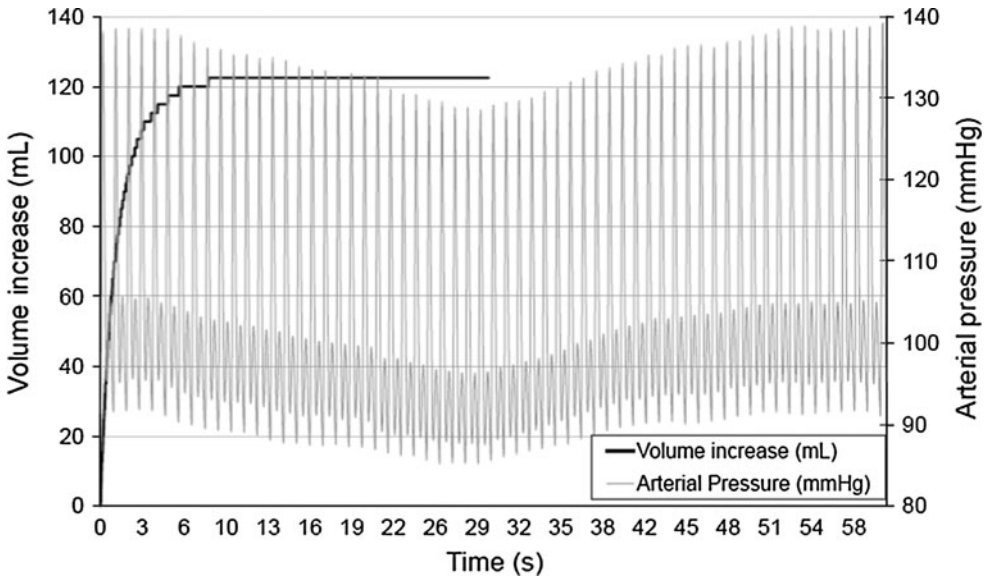

Fig. 5 Representative case of a patient showing the volume increase (V_{RM}) and the invasive arterial pressure during and after the RM

hypothesis of a decrease in venous return to explain the hemodynamic impairment. RM immediately decreases the right heart preload. However, it takes a few seconds for the blood to reach the left ventricle, which explains the delay in arterial pressure decrease. Interestingly, arterial pressure decrease was not associated with heart rate increase during the RM. We can speculate that the RM can be considered as a Valsalva maneuver. It may have stimulated the vagal nerve, which prevents an increased heart rate.

In this study, the full maneuver lasted 42 and 44 s (with an initial PEEP at 10 and 5 cmH_2O, respectively) with only 30 s at 40 cmH_2O of pressure. This progressive inflation was chosen because sudden changes in airway pressure can expose non-collapsed lung units to transient higher stress, potentially worsening lung damage [35]. Some recruitment may have occurred during the inflation phase, which would mean that V_{RM} and the dynamics of recruitment are underestimated. Thus, the dynamics of recruitment may be different if a rapid increase of pressure is used.

The main question arising in this study is the physiological meaning of V_{RM}. We assume that V_{RM} is mainly due to recruitment of previously collapsed alveoli instead of overdistension of aerated alveoli or airways. This is supported by the significant increase in C_{STAT} after the RM. Moreover, it is difficult to conceive that overdistension of previously inflated alveoli occurs at constant pressure. Interestingly, the mean V_{RM} found in this study is very similar to the recruited volume measured by the difference in end-expiratory lung volume before and after a sustained inflation RM reported by Grasso et al. [8] and Constantin et al. [36]. However, the physiological meaning of V_{RM} should be confirmed by local imaging.

The clinical implication of this study is to use a 10-s-duration sustained inflation RM in early-onset ARDS in order to achieve a plateau in the volume recruited and to prevent hemodynamic impairment.

In conclusion, this study provides direct evidence that most of the recruitment occurs early during a sustained inflation RM in ARDS patients which confirms the experimental animal study data [14]. However, hemodynamic impairment is a progressive phenomenon throughout the sustained inflation RM. These results could influence the design of optimal sustained inflation RM in ARDS patients. A 10-s sustained inflation RM may be recommended to achieve a plateau in the volume recruited and to prevent hemodynamic compromise.

Conflict of interest JMA was supported by Hamilton Medical in presenting the results of this study at international conferences. MW is an employee of Hamilton Medical and as the head of medical research was involved in the initial discussions regarding the design of the study and assisted in writing the manuscript. He was not involved in collecting and analyzing the data.

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