LUNG RECRUITMENT

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INTRODUCTION

More than 30 years ago, it was shown that in normal lungs, low tidal volume ventilation during anesthesia led to progressive lung atelectasis and then hypoxemia.\textsuperscript{1,2} Several reports showed that collapse of the lungs appeared to be directly related to degree of hypoventilation, and that this could be prevented by large tidal volumes, even if they were delivered intermittently.\textsuperscript{3} Subsequently, procedures termed “recruitment maneuvers” began to be used, whereby sustained inflation pressures were used to recruit atelectatic regions of the lung and to improve oxygenation.\textsuperscript{4,5} Lately, recruitment maneuvers (RM) have been advocated to supplement protective ventilation strategies involving low lung volumes.\textsuperscript{6}

By lung recruitment, we mean reestablishing ventilation to collapsed or partially collapsed lung units. Lung recruitment is most beneficial in patients with ARDS or ALI. In these settings, hypoxemia is a result of a decreased functional residual capacity (FRC) related to areas of lung collapse and consolidation. Oxygenation is improved when FRC is increased. Several techniques currently are being evaluated to facilitate lung recruitment: sustained high inspiratory pressures (recruitment maneuvers) and optimal PEEP, prone positioning, and liquid ventilation.\textsuperscript{7}

PROBLEMS WITH ALVEOLAR COLLAPSE

Lungs with areas of alveolar collapse require increased airway pressures to ventilate owing to increased elastic recoil worsened by surfactant dysfunction.\textsuperscript{8} These patients are at risk for ventilator-induced lung injury as a result of alveolar overdistension and repetitive opening and closing of unstable lung units, which may activate the inflammatory cascade.\textsuperscript{9,10} These patients require high FiO\textsubscript{2} to prevent hypoxemia. All of these potential adverse effects decrease the probability of recovery and increase the likelihood of infection and more severe lung injury, potentially inducing a systemic inflammatory response.\textsuperscript{11,12}

High Fraction Of Inspired Oxygen

High FiO\textsubscript{2} increases the risk of developing oxygen toxicity. The safe limit of FiO\textsubscript{2} in critically ill patients is unclear. However, most would agree that FiO\textsubscript{2} greater than 0.8 should be avoided, and FiO\textsubscript{2} between 0.6 and 0.8 should be limited in duration, with the target FiO\textsubscript{2} in critically ill patients less than 0.5.\textsuperscript{13}
Induced Lung Injury

It has been shown clearly that high transpulmonary pressure associated with large tidal volume and the repetitive opening and closing of unstable lung units induces an injury in animals similar to ARDS and ALI. Numerous investigators have demonstrated a reduction in the adverse effects of high peak airway pressure on lung injury by the application of PEEP.

The mechanism by which PEEP attenuates the injury induced by mechanical ventilation is unclear. It has been speculated that PEEP reduces the shear stress associated with the repetitive opening and closing of unstable lung. The effect of shear stress across unstable lung units has been modeled by Mead et al. At a transpulmonary pressure of 30 cm H₂O, they calculated that the pressure exerted at the junction between a collapsed lung unit and the surrounding expanded lung units could exceed 150 cm H₂O. As the collapsed lung units open, shear stress decreases.

Inflammatory Mediator Activation

The use of ventilatory strategies that do not keep the lung recruited are associated with increased inflammatory mediator activation. Tremblay and colleagues in an ex vivo healthy and injured rat lung model, demonstrated that both pro-inflammatory and anti-inflammatory mediators are activated by ventilatory patterns associated with high peak alveolar pressure causing overdistension and the absence of PEEP above Pflex. Imai and colleagues and Takata and colleagues noted greater lung tumor necrosis factor alpha messenger RNA (TNF-alpha mRNA) expression with conventional ventilation at low PEEP (5 cm H₂O) compared with high-frequency oscillation at a mean airway pressure of 15 cm H₂O. More recently, Ranieri and colleagues showed that a “lung protective strategy” avoiding alveolar overdistension and cyclic collapse significantly decreased lavage TNF-alpha and IL-8 and IL-6 levels and systemic blood levels of these mediators compared to control ventilation.

ALVEOLAR RECRUITMENT

In an effort to reduce the amount of lung collapse, high-pressure recruitment maneuvers have been used to provide the maximal amount of open lung. Although it has been suggested that PEEP can recruit alveoli, recent work suggests that very high pressures are needed to open collapsed lung units. In a porcine model of ARDS, Sjostrand and colleagues observed that pressure amplitudes of 40 cm H₂O with peak alveolar pressures of 55 cm H₂O were required to open collapsed lung regions. When opened, these lung units may be stabilized with lower pressures. A study in normal human subjects reported that 40 cm H₂O was needed to completely expand atelectatic regions induced by 20 mins of general anesthesia. Gattinoni and colleagues showed that pressures up to 46 cm H₂O resulted in lung recruitment in patients with ARDS. Therefore, it must be understood that PEEP holds open unstable lung units but of itself does not open them—sustained high pressures do that. Recruitment maneuvers are intended to establish initial alveolar patency that then can be maintained at lower tidal
pressures and PEEP levels than would otherwise be required. They are based on the principle that acutely injured lung units can be held open at pressures well below those needed to open them. The American-European consensus conference on ARDS proposed periodic use of recruiting maneuvers to prevent atelectasis when small tidal volume and/or low PEEP levels are used. On the basis of these recommendations, several studies have investigated the physiologic effects of recruiting maneuvers in patients with ARDS.

The approach to lung recruitment that has received the most interest has been that of sustained high pressure RMs. Greaves and colleagues indicate that 30 cm H₂O transpulmonary pressure is needed to recruit healthy lungs that are atelectasis. Peak alveolar pressure of 40 cm H₂O held for 7 to 15 seconds was required by Rothen and colleagues to recruit lungs of previously healthy individuals undergoing 20 minutes of general anesthesia. In a porcine model of ARDS, Sjostrand and colleagues required peak airway pressures of 55 cm H₂O to open collapsed lung. In patients with ARDS, Gattinoni and colleagues needed 46 cm H₂O peak airway pressure to recruit collapsed lung. However, Amato et al applied 35 to 40 cm H₂O CPAP for 30 to 40 seconds prior to establishing a lung protective ventilatory strategy and whenever mechanical ventilation was disrupted.

Lapinsky and colleagues demonstrated that patients could tolerate multiple RMs without significant hemodynamic compromise or development of barotrauma. They noted that if PEEP was not appropriately set post-RM the oxygenation benefit of the RM was rapidly lost. The authors varied the RM pressure applied between 30 to 45 cm H₂O based on the peak pressure obtained while ventilating at a tidal volume of 12 mL/Kg. In most patients, the benefit of the RM was sustained for 4 hrs. It can easily be argued based on animal data that if PEEP had been set higher and the ventilator circuit was not disconnected allowing derecruitment, the oxygenation benefit in all patients could have been maintained.

The highest recruiting pressure documented in humans was applied by Medoff and colleagues, who reported on the use of a peak pressure of 60 cm H₂O in a 32-year-old woman with severe ARDS secondary to streptococcal sepsis. Recruitment was preformed by PC ventilation, PEEP 40 cm H₂O with 20 cm H₂O PC, I:E of 1:1, and rate 10/min maintained for 2 minutes. Prior to this RM, recruitments at peak pressures of 50 and 55 cm H₂O were tried but had limited results. No hemodynamic compromise was reported, but it is difficult to ascertain if the maneuvers produced barotrauma because bilateral chest tubes were placed to drain effusions before the RMs. A PEEP of 25 cm H₂O was required to keep the recruited lung open. Lower PEEP levels could not sustain the improved oxygenation. It is interesting to note that the Pflex was estimated at about 18 cm H₂O.

SIDE EFFECTS OF RECRUITMENT MANEUVERS
The application of a high sustained airway pressure offers potential problems. Hemodynamic compromise and the development of barotrauma are always concerns. Clearly, in patients with pre-existing pulmonary cystic or bullous lung disease or preexisting airleaks, the benefits and potential risks of RMs must be weighed carefully. The groups reporting the use of RMs in either patients or animals have not reported the development of barotrauma.\textsuperscript{20,21,33,34} It must be assumed, however, that as RMs become more common, examples of barotrauma will be reported. Hemodynamic compromise is always a potential problem during positive pressure ventilation. RMs should be delayed until patients are hemodynamically stable. During any RM, arterial pressure, pulse rate and rhythm, and oxygen saturation measured by pulse oximetry should be monitored carefully, and the recruitment maneuver aborted if compromise develops.\textsuperscript{7}

**SUCCESSFUL RECRUITMENT**

The earlier in the course of ARDS and ALI, the greater the likelihood that lung is recruitable. As ARDS progresses and the lung becomes more fibrotic, RMs are less effective in improving PaO\textsubscript{2}, and the risk for barotrauma increases. The cause of ARDS also affects the likelihood of successful recruitment. Lung in secondary ARDS (e.g., sepsis, trauma) may be more recruitable than the lung in primary (e.g., pneumonia) ARDS.\textsuperscript{35} Little work comparing the response to RMs is available in these different groups however. It is recommended to perform RMs early in the course of ARDS and ALI regardless of the cause of ARDS.\textsuperscript{7}

**References:**


