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Lung Recruitment for Ventilation - Does it Work and Is it Safe?

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Ventilation strategies for infants, children, and adults with lung injury are active areas of translational and clinical research. The concept of the “Open Lung” continues to be developed. A large experimental literature demonstrates clearly that severe lung injury results if a lung is ventilated from collapse with each ventilator cycle. The lung will not collapse unless surfactant function is deficient and/or no positive end expiratory pressure (PEEP) is used. However, the distribution of injury/pneumonia or whatever the primary abnormality may be is seldom uniform across the lung. The open lung is a lung that is inflated as uniformly as possible with reversal of any atelectasis and with aeration of fluid filled alveoli. Consolidated lung volumes will not open despite the application of pressures that will severely overdistend the more normal lung (1). Thus, opening an injured lung is a relative goal - to open the regions that can be opened. The other way to cause lung for injury is overdistention, and a large experimental literature demonstrates that overstretch of a lung will cause stress-strain injuries to the airspace epithelium and vascular endothelium.

The lungs of adults or children with the acute respiratory distress syndrome (ARDS) can be opened by evaluating volume recruitment above the inflection point of a quasi-static pressure volume curve - a difficult maneuver to perform. Another approach is to empirically measure the oxygenation responses to progressive increases in mean airway pressures. There is no absolute volume that defines the lung as open because much of the lung may not be recruitable (1). In adult medicine, the approach of trying to open the lung and then to ventilate using relative low tidal volumes of about 6 ml/kg is becoming established. With newer approaches to ventilating the injured lung with lower tidal volumes and higher mean airway pressures, the lung may be ventilated at a volume above the normal functional residual capacity for the normal lung.

The practical difficulties are defining when the lung is open in an individual patient by measurements of pressure-volume curves, imaging or oxygenation responses and then determining how best to keep it open. One approach is to use recruitment maneuvers that increase mean airway pressures for variable periods, a variant of sigh cycles with ventilators. Despite the theoretical attractiveness of such maneuvers and their ability to recruit lung volume and increase oxygenation in some circumstances, two very large randomized controlled trials for ARDS and a meta-analysis of recruitment maneuvers for adults with lung injury showed no long-term benefits (2-4).

But, respiratory treatment strategies that do not work consistently in adults with lung injury can work in infants. The prime example is the use of surfactant to treat RDS in preterm infants,

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and pneumonia or meconium aspiration in term infants (5). Surfactant has not been effective for ARDS in adults. The open lung concept is certainly relevant for the newborn. If the lung is atelectatic or fluid filled, gas exchange is inadequate. The major effects of surfactant are to open the lung by increasing FRC and total lung capacity (5). Similarly, inhaled nitric oxide is ineffective unless the lung is open so that the gas can reach the distal airspaces. Clinicians have tried to gauge the status of lung inflation by using chest films, especially for infants on high frequency oscillation. Empirically, neonatologists have increased PEEP or continuous positive airway pressure delivered non-invasively to improve oxygenation in infants with RDS. However, the use of lung recruitment procedures has seldom been reported in infants and children.

Several years ago, de Jaegere et al (6) demonstrated that the lungs of ventilated preterm infants with RDS could be recruited by using the improvement in oxygenation to define when the lung was open. They used a high frequency oscillator and a protocol to increase mean airway pressure from a mean value of about 7 cmH₂O pressure by 2 cmH₂O pressure every 2-3 min until the FiO₂ was ≤ 0.25 or there was no further improvement in oxygenation. A poor oxygenation response was presumably because there was right to left shunt or non-recruitable lung. The surprising finding was that the mean airway pressure required to decrease the FiO₂ from 0.7 to 0.24 was 20.5 cmH₂O. The maneuver was performed at a median age of 3h in infants with RDS and birth weights of 1.3 ± 0.5 kg. The average time to perform this maneuver was about 20 min. They then decreased mean airway pressure until oxygenation decreased, which defined the closing pressure of the lung, which was about 12 cmH₂O pressure. They then “reopened” the lung by increasing the mean airway pressure to its opening pressure and then lowered the pressure to 2 cm above the closing pressure for subsequent ventilation.

Following surfactant treatment, the recruitment maneuver was then repeated, with a mean airway pressure of about 15 cmH₂O to open the lung (6). The closing pressure was about 7 cmH₂O after surfactant treatment. The total period required to assess these pressure and oxygenation responses must have been about an hour of continuous intensive adjustments of ventilation. In the original report, the blood pressures and heart rates did not change (6). However, the concept has been well developed in *The Journal* in recent years that blood pressures and heart rates do not define cardiovascular performance, particularly in preterm infants (7). In this issue of *The Journal*, de Waal et al (8) repeated the same lung recruitment maneuvers in preterm infants with RDS on high frequency oscillation and evaluated cardiovascular status. They found a small 17% decrease in right ventricular output, but no consistent changes in superior vena cava flow or ductal shunting when pressures were increased to the opening pressure. The effects of overdistention of the adult lung on cardiac output are well documented in animal models and patients. The gas volume of the fully recruited preterm lung is small (30-50 ml/kg) relative to the adult lung and the chest wall is very compliant. The increased mean airway pressures did not influence cardiovascular function in these preterms with RDS.

This recruitment maneuver works in the sense that lung oxygenation is improved and an “optimal” pressure for oscillation can be determined. There were no acute safety concerns as cardiovascular status was not compromised and no air leaks occurred. The information provided by these physiologic measurements in infants is valuable to the field. However, from my perspective, such a recruitment maneuver is not something the average neonatologist should take home and try. First, these are demanding and time consuming assessments. The mean airway pressures used to define the open lung are high, and in my experience with ventilation of preterm animals they can cause lung blebs and pneumothorax. Although not evaluated in this clinical experience, lung stretch may transduce inflammatory signals (9). Such recruitment maneuvers should not be necessary when the infant is treated with surfactant once RDS requiring a high FiO₂ is recognized. A surfactant treatment mitigates the need to open

the surfactant deficient lung using high pressures. However, the other extreme of clinical practice of using a PEEP of 5 for all infants with RDS also is not responding to optimization of oxygenation and lung recruitment.

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