Lung recruitment maneuvers in acute respiratory distress syndrome

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Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) represent the spectrum of a syndrome characterized by increased permeability of the alveolar-capillary membrane. The syndrome leads to pulmonary edema rich in protein and consequent acute respiratory failure. This condition occurs after a genetically predisposed organism is exposed to a risk factor that triggers the development of increased-permeability pulmonary edema [1]. During ARDS, the volume of extravascular lung water rises. The high water content increases lung weight and promotes the collapse of peripheral airways and lung parenchyma mainly in the gravitationally dependent lung regions. This preferential collapse is explained by the progressively lower transpulmonary pressure along the ventrodorsal axis [1,2]. As a consequence, during the onset of ARDS, the pleural pressure gradient increases, respiratory effort and work of breathing increase, and gas exchange deteriorates [2,3]. Many ARDS patients must be intubated and coupled to a mechanical ventilator to maintain ventilation and oxygenation [4].

After initiation of mechanical ventilation, ARDS patients frequently require a high fraction of inspired oxygen (FiO2) to maintain acceptable arterial oxygenation and a high minute ventilation to maintain normal arterial CO2 levels [4]. In a study of 170 ARDS patients, Nuckton et al [5]...
found that dead-space fractions are elevated (0.58 ± 0.09) early in the course of ARDS. Dead-space fraction was higher among nonsurvivors than among survivors (0.63 ± 0.10 versus 0.54 ± 0.09) and was an independent risk factor for death [5].

The respiratory system of ARDS patients exhibits a decreased compliance and an increased resistance when compared with healthy humans. These differences are reflected in differences in the inflation limb characteristics of the pressure-volume (P–V) curve. In a healthy respiratory system, patent airways conduct gas to the lung parenchyma with normal elasticity, enclosed in a chest wall with normal elasticity. The resulting inflation P–V curve is essentially linear until an upper inflexion is observed near total lung capacity; this inflexion represents the changes in the elastic properties of the system that occur with overdistension (Fig. 1A). In contrast, ARDS patients usually have edema and collapse in the parenchyma and also in the airways at the beginning of inspiration associated with chest wall abnormalities. The resulting inflation limb of the P–V curve in these patients represents more complex phenomena, usually resulting in an overall curvilinear shape. This curvilinearity of the inflation P–V curve represents overlapping processes such as airway and alveolar recruitment, normal elastic expansion, deformation of chest wall, and nonelastic overextension of the respiratory system (Fig. 1A). The degree to which any of these pathologic phenomena exist varies in most patients according to the nature and extent of lung injury.

When a P–V loop is plotted during a slow inflation maneuver in ARDS patients, a poor compliance is initially observed as pressure and volume build from the origin, because many lung units are initially consolidated, liquid-filled, or collapsed, requiring relatively high opening pressures. Also, the small area of previously aerated parenchyma (ie, so-called “baby lung”) cannot accommodate too much air, resulting in a small volume gain. The resulting compliance (slope) of this first segment is particularly poor when massive airway collapse and flooding predominate and when airway collapse reaches the nondependent lung regions, because relatively few gas channels are patent. A rather abrupt upward inflection often marks a transition into the second segment of the P–V curve. In a patient with normal chest wall, this inflection usually occurs in the range of 8 to 18 cm H$_2$O, the range of airway opening pressures commonly found in non-dependent lung regions. This area of transition is commonly referred to as the lower inflection point or “Pflex” (a misleading term from a mathematical point of view). Beyond Pflex, a high-compliance segment occurs when pressure increments simultaneously redistribute luminal fluids, expand patent lung units, and recruit atelectatic units that achieve patency once their unique critical opening pressures are reached for the requisite time. Initially, these newly opened units are located in nondependent or intermediate lung regions; as inflation continues, the dependent zones become progressively involved. A third segment may then be entered in which almost all recruitable units are already aerated. This segment of the
curve displays the elastic properties of open units in various stages of distension. This third segment may be intermixed with the second or the fourth segment. In the fourth, final segment of the inflation curve, there is extensive overdistension of lung units, many of which are located in non-dependent regions (Fig. 1B), and the curve bends markedly to the right.

The deflation limb of the P–V curve displays a variable number of distinct subsections depending on the degree of lung injury and the maximum inspiratory pressure achieved in the respiratory system during the previous inspiration. In the first segment, the decrease in pressure represents the relaxation of overstretched units as volume descends from total lung

Fig. 1. (A) Inspiratory pressure-volume curve of the respiratory system of a normal healthy human and of an ARDS patient. (B) Inspiratory and expiratory pressure-volume curve of the respiratory system of an ARDS patient.
capacity. The second phase of the deflation curve primarily reflects the elastic-retraction properties of patent lung tissue. Relatively few units, only the most unstable, reach their critical closing pressures in this upper range. In the third phase of deflation the rate of closure progressively increases in tandem with deflation of open units. The curve bends downward. The narrower the distribution of closing pressures, the sharper is this downward shift. Depending on the size of the lung and on the heterogeneity of disease, this downward curvature may or may not match the range of pressures in Pflex. In the fourth and final phase, there is extensive airway (as opposed to alveolar) closure and gas trapping (Fig. 1B), with the curve bending leftwards.

An important finding in ARDS is that during deflation, varying degrees of lung volume can exist at the same pressure depending on the maximal inspiratory pressure achieved during the previous inflation (Fig. 2). This behavior is characterized as the multiple pressure-volume envelopes of the respiratory system in ARDS. Such behavior can be used to advantage during any lung recruitment trial or during positive end-expiratory pressure (PEEP) titration. The history of mechanical ventilation in the previous breaths strongly determines the working envelope at the current breath and the chances of promoting intratidal recruitment during mechanical ventilation.

![Pressure-volume curve](chart.png)

**Fig. 2.** The concept of pressure-volume envelopes: at the same airway pressure, the lung volumes during expiration are greater after previous inspirations to higher airway pressures. FRC, functional residual capacity; Pflex, lowest inflection point on the pressure-volume curve.
Thoracic radiographs and computerized tomography in acute respiratory distress syndrome

On conventional chest radiographs, patients who have ARDS demonstrate diffuse parenchymal infiltrates (Fig. 3A). Computerized tomography scans of the chest, however, have revealed that ARDS is in fact an inhomogeneous process; that is, lung densities are primarily located in dependent lung regions along a gravitational gradient (Fig. 3B). The CT-described morphology of ARDS varies with a number of factors such as cause (primary or secondary ARDS), mechanical ventilation parameters such as PEEP, tidal volume (VT) and recruitment maneuvers, and the patient’s body position (there is a redistribution of densities when the patient is shifted from the supine into the prone position) [6]. In addition, as unresolved ARDS progresses to a subacute phase, parenchymal fibrosis becomes a predominant feature, causing both distortion of the interstitial and bronchovascular markings on CT and a dramatic increase in subpleural cysts and bullae. It is important to recognize the subacute phase of ARDS because in that phase the efficacy of both PEEP and recruitment maneuvers is diminished, and the lungs may respond favorably to corticosteroid therapy [7].

Mechanical ventilation in acute respiratory distress syndrome

Evidence of lung injury induced by mechanical ventilation in experimental models of acute respiratory distress syndrome and the protective effects of positive end-expiratory pressure

High transpulmonary pressures associated with large VT ventilation causes ARDS-like lung injury in animals [8]. This effect is magnified as transpulmonary pressure increases. For example, as transpulmonary
pressure exceeds 35 to 40 cm H\textsubscript{2}O, microvascular permeability increases exponentially [9]. Dreyfuss et al [10,11] showed that high intermittent transpulmonary positive-pressure ventilation induces altered-permeability pulmonary edema that is accompanied by severe ultrastructural alterations. Furthermore, in animals, an ARDS-like injury is caused by the repetitive opening and closing of unstable lung units [12,13]. Hernandez et al [14], using an isolated, perfused rabbit lung model, demonstrated that an oleic acid–injured lung had a much greater capillary filtration coefficient after mechanical ventilation than did a normal lung or an injured lung without mechanical ventilation.

Several animal model studies have examined the effects of PEEP on acute-injured lungs. In 1974, Webb and Tierney [8] demonstrated that lung injury caused by high peak airway pressures and volumes could be reduced by the application of PEEP. Corbridge et al [15], using a hydrochloric acid-induced lung injury, found that the level of PEEP and VT influenced the magnitude of the initial lung injury. Using wet-lung-weight–to–dry-lung-weight–to–body ratios to assess injury, they found that dogs ventilated with a higher PEEP/lower VT (12.5 cm H\textsubscript{2}O, 15 mL/kg) exhibited less injury than those maintained at a lower PEEP/higher VT ventilation (3.2 cm H\textsubscript{2}O, 30 mL/kg). In both groups, peak alveolar pressure was maintained at 33 cm H\textsubscript{2}O [15].

In an ex vivo rat lung model, Muscedere et al [12] showed that in animals treated with PEEP set higher than P\textsubscript{flex}, lung compliance was better preserved than in control animals whose lungs were kept statically inflated with an airway pressure of 4 cm H\textsubscript{2}O. Two additional groups of animals were ventilated at either zero end-expiratory pressure (ZEEP) or PEEP set below the P\textsubscript{flex}. In both of these groups there was a large decrease in compliance and an increased incidence of histologic injury. The group ventilated with ZEEP had significantly greater injury to the bronchioles, whereas the group ventilated with a PEEP of 4 cm H\textsubscript{2}O (PEEP < P\textsubscript{flex}) had significantly greater alveolar duct injury [12].

More recently, Suh et al [13] demonstrated that repeated derecruitment can accentuate lung injury during mechanical ventilation. After lung injury induced by saline lavage, animals were ventilated at a VT of 10 mL/kg and randomly assigned to one of three groups. Each group was ventilated for 3 hours. The control group was ventilated with PEEP at P\textsubscript{flex} for 3 hours. The nonderecruitment group was ventilated at PEEP below P\textsubscript{flex} for the initial hour and then with PEEP at P\textsubscript{flex} for the remaining 2 hours. In the third group, derecruitment was repeatedly induced by 30-minute ventilation cycles consisting of 10 minutes at PEEP below P\textsubscript{flex} and 20 minutes with PEEP at P\textsubscript{flex}. Arterial oxygenation was profoundly affected by the ventilation pattern. In the control group, P\textsubscript{ao}2 remained greater than 500 mm Hg for 3 hours, whereas in the nonderecruitment group P\textsubscript{ao}2 increased to greater than 500 mm Hg once PEEP was increased to P\textsubscript{flex}. In contrast, the P\textsubscript{ao}2 of the derecruitment group progressively deteriorated to 220 plus or minus 130 mm Hg by 3 hours. Histologically, the derecruitment group
had more hyaline membrane formation than did controls and had a significantly higher mean bronchiolar injury score than did either controls or the nonderecruitment group. These findings suggest that ventilator lung injury had occurred in the derecruitment group [14].

Experimental evidence of protection from lung injury using an open-lung ventilation strategy

Rotta et al [16], using a repeated saline lavage model of lung injury, showed that an open-lung ventilatory strategy maintained PaO₂, lung volume, and protein concentration of bronchoalveolar lavage equal to those in healthy controls [16]. This strategy consisted of a peak inspiratory pressure of 33 cm H₂O, PEEP of 15 cm H₂O, inspiration: expiration ratio of 1:1, and a respiratory rate of 100. In contrast, the control group was ventilated at a peak inspiratory pressure of 26 cm H₂O, PEEP of 6 cm H₂O, inspiration: expiration ratio of 1:2, and respiratory rate of 30. Both the PaO₂ and lung volume were significantly decreased, whereas the protein concentration of bronchoalveolar lavage significantly increased compared with healthy controls [16]. The open-lung strategy provided similar degree of protection in a study performed by Hartog et al [17].

Evidence supporting the use of recruitment maneuvers in acute respiratory distress syndrome

Evidence of reduced ICU mortality and decreased inflammatory mediator activation using high positive end-expiratory pressure/low tidal volume ventilation and recruitment maneuvers, versus low positive end-expiratory pressure/high tidal volume ventilation strategies

Amato et al [18] demonstrated that a lung-protective ventilation strategy with high PEEP and a VT below 6 mL/kg, incorporating recruitment maneuvers with continuous positive airway pressure (CPAP) of 35 to 40 cm H₂O for 40 seconds, was associated with a 28-day survival rate of 62%. This rate contrasted with a survival rate of only 29% in patients managed with conventional ventilation [18]. A post hoc analysis of the study stratified the 53 patients into quartiles according to PEEP levels (<7 cm H₂O, 7–12 cm H₂O, 12–16 cm H₂O, and >16 cm H₂O) and analyzed the 28-day survival rate. The higher PEEP levels (>12 cm H₂O and especially >16 cm H₂O) correlated significantly with improved survival (Fig. 4) [19]. Ranieri et al [20] demonstrated that a higher PEEP/low VT ventilation strategy significantly decreases the levels of tumor necrosis factor-α, interleukin-8, and interleukin-6 in the bronchoalveolar lavage fluid and blood when compared with low PEEP/high VT ventilation strategy [20]. Recently, Takeuchi et al [21] used a lung-lavage animal model to demonstrate that higher PEEP levels (based on P–V curve analysis) are more effective in maintaining gas exchange and minimizing injury than PEEP titration targeted to maintain
adequate oxygenation at 50% oxygen concentration, even though the former generates higher plateau pressures [21].

The concept of pressure–volume envelopes of the respiratory system assessed by thoracic computerized tomography

The authors performed a tomographic study in ARDS patients ventilated with the same protective protocol used by Amato and colleagues [18] (PEEP = 2 cm H₂O above the Pflex and VT ≥6 mL/kg, after a recruiting maneuver with CPAP = 35–40 cm H₂O for 40 seconds). They observed that patients ventilated with this protocol still presented significant gravity-dependent lung densities. This finding explains the observation of a mean Pao₂/Fio₂ level of 198 plus or minus 16 mm Hg in patients in the protective ventilation arm of their initial clinical trial, indicating that the ARDS lungs were not fully recruited during this strategy [18]. Accordingly, the average mass of collapsed tissue observed in the CT images during the protective strategy corresponded to 20% to 30% of the total lung mass, producing shunt levels of similar magnitude.

Nevertheless, those densities disappeared with a more prolonged, stepwise recruiting maneuver achieving plateau pressures as high as 60 cm H₂O, followed by slightly higher PEEP levels [22]. The authors also found that the lungs showed diminished dependent alveolar densities on the
deflation limb on the P–V curve (ie, after recruitment maneuvers) than when equivalent pressures were applied during the inflation limb. This finding is in accordance with the concept of hysteresis and the multiple P–V envelopes (see Fig. 2). In ARDS lungs, this particular behavior is mainly explained by recruitment during inspiration, followed by derecruitment during expiration, provided that the distribution of closing pressures is shifted to the left in comparison to opening pressures (ie, average closing pressures are lower than average opening pressures, as shown in Fig. 5) [23,24].

The importance of maintaining lung recruitment with adequate positive end-expiratory pressure levels: achieving homogeneous distribution of tidal ventilation

Conceptually, protective ventilation for the ARDS lung should avoid injury from overdistension and from repeated opening and closing of alveoli. Tidal recruitment is defined as a recruitment that occurs at the level of tidal ventilation, as illustrated in Figs. 6 and 7. After a recruiting

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Fig. 5. The thoracic tomography aspects of ARDS lungs at different levels of the inspiratory and expiratory pressure-volume curves. (A) CT scan at zero end-expiratory pressure (ZEEP) showing gravitational dependent lung opacities. (B) CT scan at L-PFlex showing a more open lung but still with lung opacities in the gravitational-dependent regions. (C) A nearly fully recruited ARDS lung at the end of inspiration. (D) CT scan at the same airway pressure (L-PFlex) as in C during expiration. The lungs are more aerated during expiration than during inspiration at the same pressure. Note the opening of the airways at the L-Pflex level.
maneuver, it makes sense to maintain a PEEP level adequate to maintain patent alveoli and to keep tidal recruitment to a minimum. Insufficient PEEP levels after a recruitment maneuver are usually associated with a period of tidal recruitment followed by progressive and sticky collapse after a few minutes, depending on the plateau pressures employed. The situation can regress to that before the recruiting maneuver very quickly. On the contrary, if carefully pursued, the avoidance of cyclic collapse and recruitment stabilizes the dependent lung, and the open-lung status can be
maintained in the long run. At the same time, avoiding tidal recruitment may favor more dependent regional ventilation, because dependent airways are now patent, decreasing the cyclic overdistension of the upper lung.

The failure to observe this simple principle may render ineffective any attempt to recruit the lung. In fact, because the recruitment maneuver certainly causes short-term overdistension, the use of insufficient PEEP afterwards imposes a double risk: neither the risks of short-term overdistension nor the risks of long-term collapse are avoided with repeated reopening.

Fig. 7. (A) A PEEP level of 25 cm H₂O can keep the dependent ARDS lung region opened in this patient after the recruitment maneuver but not before. (B) A PEEP level of 20 cm H₂O after the recruitment maneuver can keep the lungs of this ARDS patient opened with a minimal tidal recruitment observed during the tidal ventilation with a pressure control of 15 cm H₂O.
Performing recruitment maneuvers in acute respiratory distress syndrome: techniques and possible results

Grasso et al [25] studied 22 ARDS patients who had been ventilated with the National Institutes of Health ARDS-Network lung-protective strategy [26] and applied recruitment maneuvers with CPAP 40 cm H2O for 40 seconds. Patients were classified as responders or nonresponders according to the occurrence of a 50% increase in PaO2/Fio2 after recruitment maneuver. Two minutes after the recruitment maneuver, they observed a 20% plus or minus 3% increase in PaO2/Fio2 in the nonresponder group and a 175% plus or minus 23% increase in the responder group. Responders were characterized as having a higher lung and chest wall compliance, were ventilated for a shorter period of time, and had less hemodynamic impairment than nonresponders [25].

In a randomized, clinical trial on ARDS, Amato et al [18] used 40-cm H2O CPAP for 40 seconds, with PEEP set 2 cm H2O above the Pflex. Afterwards they observed a sustained increase in PaO2/Fio2, a progressive increase in the respiratory system compliance, and an improved 28-day survival rate compared with controls [18]. The results show that this moderate recruiting maneuver, with maximum airway pressures at 40 cm H2O, has proven clinical efficacy, although the results may be suboptimal.

Intermittent sighs

Pelosi et al [27] studied 10 ARDS patients who were ventilated with a lung-protective strategy for 2 hours. This treatment was followed by 1 hour using the same lung protective strategy but with three consecutive sighs/minute at a plateau pressure of 45 cm H2O. This second period was followed by 1 hour of lung-protective strategy without sighs. During ventilation with sighs, PaO2/Fio2 and end-expiratory lung volume were increased, and venous admixture and PaCO2 were decreased. These effects returned to baseline within 30 minutes of sigh interruption, however [27]. Barbas et al [28] studied 10 ARDS patients ventilated with PEEP set 2 cm H2O above Pflex and 6 mL/kg tidal ventilation. These patients were then randomly assigned to receive either (1) three cycles of pressure-controlled ventilation (PCV) of 40 cm H2O for 6 seconds every 3 hours (and whenever necessary), or (2) three cycles of PCV of 40, 50, and 60 cm H2O for 6 seconds every 3 hours. In the latter group there was a further increase in PaO2/Fio2 ratio after 1 hour and 6 hours without hemodynamic impairment compared with the group limited to PVC of 40 cm H2O [28]. Patroniti et al [29] showed that the addition of 1 sigh/minute using the biphasic positive airway pressure mode (intermittent increment of PEEP up to 38 ± 3.2 cm H2O) improved gas exchange and lung volume and decreased respiratory drive in 13 patients who had early ARDS. These results suggest two major conclusions: sighs are effective in improving
oxygenation, and inspiratory pressures above 40 cm H\textsubscript{2}O are associated with further benefits in terms of oxygenation. The improvements in oxygenation obtained with sighs, however, occur at the cost of increased tidal recruitment and of repeated pulses of overdistension, not minimized to a single-shot application. For these reasons, the authors do not consider sighs a valid option in the search of an ideal ventilatory strategy.

Intermittent and stepwise high positive end-expiratory pressure levels with a fixed pressure control maneuver

Okamoto et al [30] studied the stepwise recruitment maneuver in clinically stable ARDS patients. After a baseline period using a PEEP of 10 cm H\textsubscript{2}O and 6 mL/kg tidal ventilation, the stepwise recruitment maneuver was applied, consisting of 2-minute steps of tidal ventilation with a fixed driving pressure of 15 cm H\textsubscript{2}O and progressive PEEP levels (25, 30, 35, 40, and 45 cm H\textsubscript{2}O) until full recruitment (defined as Pao\textsubscript{2} + PaCO\textsubscript{2} > 400 mm Hg ± 5% at Fio\textsubscript{2} of 1.0). The Pao\textsubscript{2} plus Paco\textsubscript{2} level increased from 178.4 ± 76.5 mm Hg to 487.8 ± 139.1 mm Hg. After 6 hours, the Pao\textsubscript{2} plus Paco\textsubscript{2} level remained at 521.4 ± 95.4 mm Hg with a PEEP titration strategy to keep the lung open. The PEEP titration strategy consisted of small, stepwise decrements of PEEP every 4 minutes until a drop in Pao\textsubscript{2} plus Paco\textsubscript{2} greater than 5% to 10% of the previous Pao\textsubscript{2} occurred. The PEEP level immediately preceding the level in which the Pao\textsubscript{2} plus Paco\textsubscript{2} drop occurred was the minimal PEEP that should maintain the benefit of the recruitment maneuver. In this study, the titrated PEEP maintained oxygenation after 6 hours (assessed in seven patients) [30]. All the patients were monitored with continuous intra-arterial blood analysis that showed that the high Pao\textsubscript{2} plus Paco\textsubscript{2} level could be maintained with a mean PEEP of 21 ± 5 cm H\textsubscript{2}O after the PEEP titration maneuver. If the PEEP was reduced below the titrated level, there was a decrease in Pao\textsubscript{2} within minutes.

Prone positioning as a recruitment maneuver and potential rescue therapy in severe acute respiratory distress syndrome

Prone positioning is an attractive adjunct to recruitment maneuvers because it applies and sustains a high recruiting force in the dorsal lung regions that are compressed in the supine position. This compression is caused by a higher local pleural pressure and by the weight of the overlying heart and mediastinal contents. Arterial oxygenation improves in 50% to 70% of ARDS patients placed in the prone position [31]. Despite this rationale, a randomized, controlled study indicated that placing ARDS patients into the prone position has no impact on survival [32]. In a post hoc analysis of this trial, Gattinoni et al [32] suggested that patients who had the most severe ARDS (defined as Pao\textsubscript{2}/Fio\textsubscript{2} < 88 mm Hg and a VT > 12 mL/kg) showed significant improvement (as measured by 10-day survival rate) with prone positioning.
Besides the low degree of confidence of post hoc analysis, in which some subgroups presented decreased mortality with prone positioning, the methodology of this study raises an important question. The intermittent use of the prone position incurs in the same risks as the use of insufficient PEEP after recruiting maneuvers. To be effective and protective, the prone positioning should be sustained for days or followed by a higher PEEP level after the patient is turned back to the supine position.

The authors have found that some severe cases of ARDS that do not respond to recruitment maneuvers will demonstrate some slow improvement in oxygenation after a few hours of prone positioning. Additionally, the combination of recruiting maneuvers and prone positioning sometimes results in dramatic responses in patients unresponsive to recruitment maneuvers in supine position.

Potential tools for evaluation of recruiting maneuvers and positive end-expiratory pressure titration

Computed tomography

Borges et al [22] evaluated eight ARDS patients by thoracic CT scan, gas exchange, and hemodynamics after 6 minutes of ZEEP. This treatment was followed by CPAP of 40 cm H₂O for 40 seconds and PEEP set 2 cm H₂O above Pflex during 6 mL/kg tidal ventilation. Following a series of progressive recruitment maneuvers, subsequent CT scans were taken at higher PEEP levels (25 cm H₂O), always at exhalation. Keeping a fixed driving pressure (15 cm H₂O) during PCV (pressure controlled ventilation), the PEEP level was increased in 5-cm H₂O increments from 25 cm H₂O to 45 cm H₂O, with corresponding inspiratory plateau pressures of 40, 45, 50, 55, and 60 cm H₂O. The CT scans revealed that the percent mass of nonaerated tissue (ie, collapsed tissue) decreased from 63.7% at ZEEP to 28.6% after an open-lung approach and to 4.7% after the highest recruitment maneuver. The Pao₂/Fio₂ ratio also increased from 92 mm Hg at ZEEP to 257 mm Hg after the open-lung approach and to 394 mm Hg after recruitment [22].

Although CT has contributed importantly to the understanding of ARDS physiopathology and remains the most relevant tool for research, it lacks monitoring properties and requires that the patient be transported to the radiology department. Because there is not yet a better alternative the clinical setting, an initial CT scan might be performed to determine if the lung is recruitable.

Electrical impedance tomography

Kunst et al [33] evaluated regional P–V curves of the thorax using electrical impedance tomography (EIT) before and after repeated-lavage injury was induced in anesthetized pigs. Low continuous-flow P–V curves were constructed until an airway pressure of 50 cm H₂O was achieved. The
electrical impedance signal was detected using 16 standard electrocardiography electrodes placed around the thorax and was transmitted to a device that produces two-dimensional images of changes in impedance. The EIT image was divided into anterior and posterior sections and then was subdivided into four regions along the vertical axis. By analyzing the Pflex of the regional P–V curves, the authors found a higher Pflex in the posterior region of the thorax (29.5 cm H$_2$O versus 21.5 cm H$_2$O in the anterior region), as expected according to the superimposed-pressure theory. The EIT upper Pflex could be determined in the anterior part of the thorax in all of the animals. In the posterior region, however, no upper Pflex could be determined until a pressure of 50 cm H$_2$O was reached. This finding indicates a distinct behavior in different regions of the lungs, with upper lung zones always submitted to higher degrees of overdistension.

After lung injury was induced, there was a significant increase in the ratio of impedance swings (caused by tidal ventilation) in the anterior versus in the posterior thoracic regions (from 1.75 ± 0.63 to 4.51 ± 2.22). This finding suggests that substantial alveolar collapse had occurred in the posterior lung regions, with regional ventilation diverted to the nondependent lung. After performing two different types of recruitment maneuvers (open-lung approach and open-lung concept), the anterior/posterior ratio of impedance swings decreased from 4.51 ± 2.22 to 1.1 ± 0.3 (open-lung approach) and 1.2 ± 0.3 (open-lung concept). This finding showed that recruitment maneuvers can open the lung and homogenize the ventilation, at least in a lavage model of lung injury [34].

Using EIT in six ARDS patients, Sipmann et al [35] found a Pflex of 16 cm H$_2$O in the anterior part of the lung, in contrast to 24 cm H$_2$O for the posterior region. The total lung P–V curve showed a Pflex of 16 cm H$_2$O [35]. Additionally, Borges et al [36] showed that the EIT provided information about regional values of opening and closing pressures, allowing a better assessment and optimization of lung ventilation in ARDS patients (Fig. 8).

Electrical impedance tomography has the unique capability of imaging regional ventilation dynamically. For this reason, it might be a useful tool for monitoring recruitment maneuvers and PEEP titration at the bedside. The EIT device is still unavailable for clinical use, however. Improvements in the hardware and software are still under way, as are developments in the band of skin electrodes.

Other promising uses of EIT include the measurement of extravascular lung water [37] and the bedside assessment of ventilation: perfusion ratios at different regions of the lung [38].

**Future directions and perspectives**

In the near future, bedside monitoring of regional ventilation with EIT may help determine the optimal mechanical ventilation strategy for ARDS patients (Fig. 8). High-speed and real-time monitoring might allow clinicians
to recruit ARDS lungs more safely and to adjust the ideal PEEP levels simultaneously with the optimization of tidal ventilation [39]. This new proposal for optimizing ventilation in ARDS patients (ie, avoiding derecruitment and minimizing the stretch of the lung parenchyma by titration of a homogenous ventilation according to EIT) must still be tested in a prospective, randomized, clinical trial. In spite of the physiologic rationale, the overall patient outcome is still the best measure of efficacy.

References


