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REVIEW

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The "baby lung" became an adult

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Take-home message: The baby lung was originally defined as the fraction of lung parenchyma that still maintains normal inflation with near normal mechanical characteristics despite at least a part of the well-aerated baby lung is inflamed. The baby lung is not an anatomical but a functional entity which changes dimensions and location with prone position and PEEP application. Particular attention should be paid to the general principles regulating the safety of ventilation during both mechanical ventilation and spontaneous breathing.

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Abstract The baby lung was originally defined as the fraction of lung parenchyma that, in acute respiratory distress syndrome (ARDS), still maintains normal inflation. Its size obviously depends on ARDS severity and relates to the compliance of the respiratory system. CO_2 clearance and blood oxygenation primarily occur within the baby lung. While the specific compliance suggests the intrinsic mechanical characteristics to be nearly normal, evidence from positron emission tomography suggests that at least a part of the well-aerated baby lung is inflamed. The baby lung is more a functional concept than an anatomical one; in fact, in the prone position, the baby lung ''shifts'' from the ventral lung regions toward the dorsal lung regions while usually increasing its size. This change is associated with

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better gas exchange, more homogeneously distributed trans-pulmonary forces, and a survival advantage. Positive end expiratory pressure also increases the baby lung size, both allowing better inflation of already open units and adding new pulmonary units. Viewed as surrogates of stress and strain, tidal volume and plateau pressures are better tailored to baby lung size than to ideal body weight. Although less information is available for the baby lung during spontaneous breathing efforts, the general principles regulating the safety of ventilation are also applicable under these conditions.

Keywords ARDS · Baby lung · Transpulmonary pressure · Prone position · Stress and strain

Introduction

Several diseases may produce the syndrome called acute respiratory distress syndrome (ARDS). The symptoms involved are impaired gas exchange, decreased lung compliance, increased lung weight and widespread involvement of the lung parenchyma. Whatever the cause of ARDS, the treatment of symptoms is mandatory to buy time for the resolution of the underlying process, such as pneumonia or sepsis. Possible harm from mechanical ventilation is understandable if the ARDS lung is modeled in two regions, one nearly normal and having dimensions similar to those of a healthy baby (Fig. 1). The restricted capacity of this 'baby lung' is primarily responsible for the mechanical characteristics described in ARDS. The second region, consolidated and collapsed, is primarily responsible for the impairment of oxygenation. This concept evolved over several decades: the baby lung, rather than an anatomical reality, became a functional status. Studies relating to prone positioning, positive end-expiratory pressure (PEEP), stress and strain, and spontaneous breathing may be considered according to the baby lung model, as we explore in this paper.

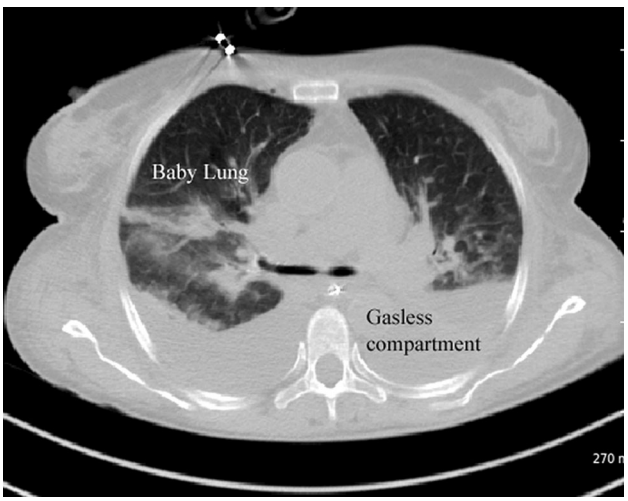

Fig. 1 A representative CT scan image of an ARDS patient showing that the ARDS lung can be modeled in one nearly normal region (having dimensions similar to those of a healthy baby) and a gasless region

Baby lung: anatomical characteristics

The concept of ''baby lung'' originated from observations on the first CT scan images obtained in ARDS patients, which showed that densities were preferentially distributed in the dependent lung regions while the non-dependent lung regions were relatively spared. This view deeply contrasts with the common belief, derived from antero-posterior imaging, that ARDS homogeneously involves the entire lung parenchyma. Quantitative analysis of the CT scan (see Fig. 2) [1] showed that the patho-anatomy of the ARDS lung is not homogeneously distributed but comprised of regions totally deprived of air and regions normally (or almost normally) aerated. Only a small proportion of the lung is ventilated, and this small (baby) lung has to fulfill the physiological needs of an adult body.

Quantitatively, the baby lung includes the voxels comprised in the interval between −900 and −500 Hounsfield units (HU) [2], which describe the densities of the voxel, where −900 indicates 90 % gas and 10 % tissue and −500 indicates 50 % gas and 50 % tissue. In normal lungs, the voxels included in this interval (the well-inflated tissue) correspond to a weight of about 600–700 g, while in ARDS they may account for 1/3 or even 1/5 of this value. Quantitative analysis of the CT scan provided a powerful tool to compare the in vivo anatomy with the physiological variables commonly measured in the ARDS definition. The most important finding was the discovery that the aerated ARDS lung was not stiff but small, with near normal intrinsic mechanical characteristics [2]. The ratio of the respiratory system compliance to the residual healthy ('baby') lung (expressed as a fraction of the expected healthy lung volume) is roughly 1:1, i.e. 20 mL/cmH_2O compliance corresponds roughly to 20 % open ventilatable lung, 50 mL/cmH_2O to 50 %, and so on. The relationship between the

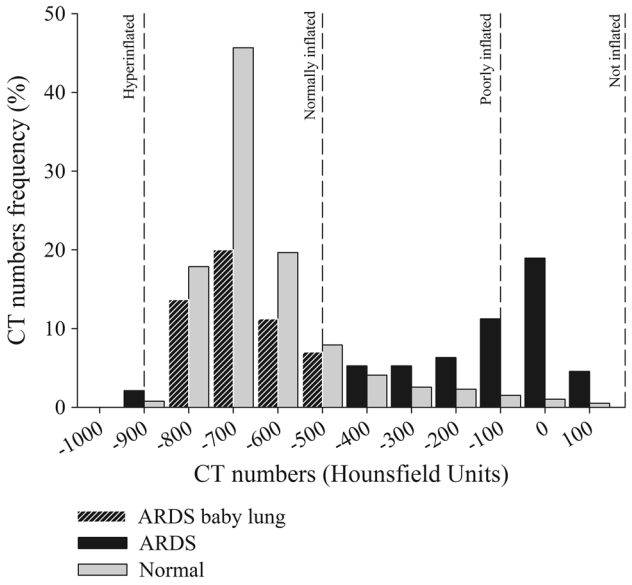

Fig. 2 The CT frequency distribution as a function of CT number compartments distribution of patients with ARDS (*black bars*) and normal subjects (*gray bars*). The *diagonal patterned bars* indicate the baby lung in ARDS patients. The zones of hyperinflated, normally, poorly and not-inflated lung are shown (*vertical dashed lines*)

baby lung and gas exchange provided further insight into flow distribution in ARDS, indicating that the hypoxic vasoconstriction is still very active in the ARDS lung [3].

Baby lung and inflammation

Although the specific compliance of the baby lung is nearly normal, regional quantitative analysis of CT scans showed that excess tissue mass, at least in severe ARDS, is present both in non-dependent and in dependent regions, suggesting that dependent densities primarily represent atelectasis due to the increased lung weight, which squeezes the gas out of the most dependent pulmonary units. Therefore, the question arises: if the parenchyma of the baby lung is mechanically normal, is it inflamed or simply edematous?

An answer may be provided by positron emission tomography (PET) which helps to explore the different properties and functions of the lung tissue by both imaging and quantitative approaches [4] (Fig. 3). In fact, PET can assess lung regional vascular permeability by measuring the plasma escape rate of 68Gallium-transferrin. By this technique, it has been shown that extravascular lung water is diffusely increased during ARDS, lobar pneumonia, and acute cardiogenic pulmonary edema. One study [5] showed that in ARDS not just the non-ventilated regions but also the baby lung bears one of the hallmarks of tissue inflammation: increased permeability of the endothelial–epithelial barrier. At variance with density and extravascular lung water, which both increase according to a vertical, gravitational gradient, increased permeability was evenly distributed throughout the entire ARDS lung and abnormally high in all isogravitational regions [6].

PET also allows investigation of the regional distribution of inflammation, using 18Fluor-deoxy-glucose (18FDG), an analogue of glucose, which is taken up by cells in proportion to their metabolic activity. During an inflammatory process, several cellular types can effectively uptake 18FDG, including macrophages [7], eosinophils [8] and alveolar cells [9], with predominant uptake occurring by neutrophils [10]. In several animal studies, PET with 18FDG has been shown to closely mirror the presence of inflammatory infiltrate [7] caused by various types of lung injuries [11], including aggressive ventilation [12].

This tool was applied in ARDS patients, who underwent a combined CT/PET scan. In all patients, the inflammatory activity of the lungs was increased (sevenfold, on average) in comparison with controls. In this case, too, the baby lung showed a greater activity than controls and, in some cases, even a greater activity than the collapsed, non-aerated lung [13]. At variance from previous studies [12], 18FDG uptake was not normalized by the fraction of tissue from the corresponding regions, since if the increase in tissue fraction was caused by edema (as likely the case in ARDS), this would lead to an over-correction of 18FDG signal. However, only tissues of similar density were directly compared, hence avoiding the need for density-correction. A subsequent study investigated the association between mechanical ventilation and inflammatory activity of the baby lung. The uptake of 18FDG in the normally aerated tissue (the baby lung) was tightly correlated both with plateau pressure (with a steep increase for plateau pressure values above 26–27 cmH_2O) and with the ratio of tidal volume to end expiratory lung volume of the same regions [14]. These data suggested a causative role for both stress and strain in inflaming the ventilated baby lung. A recent CT-PET study, however, indicated that baby lung inflammation was primarily a function of ARDS severity. Unlike the baby lung of mild ARDS, no regions were spared the inflammatory reaction in severe ARDS [15].

Increasing the baby lung size

Prone position

At the beginning, the baby lung was considered as if it were an anatomically well-defined entity, with a definite

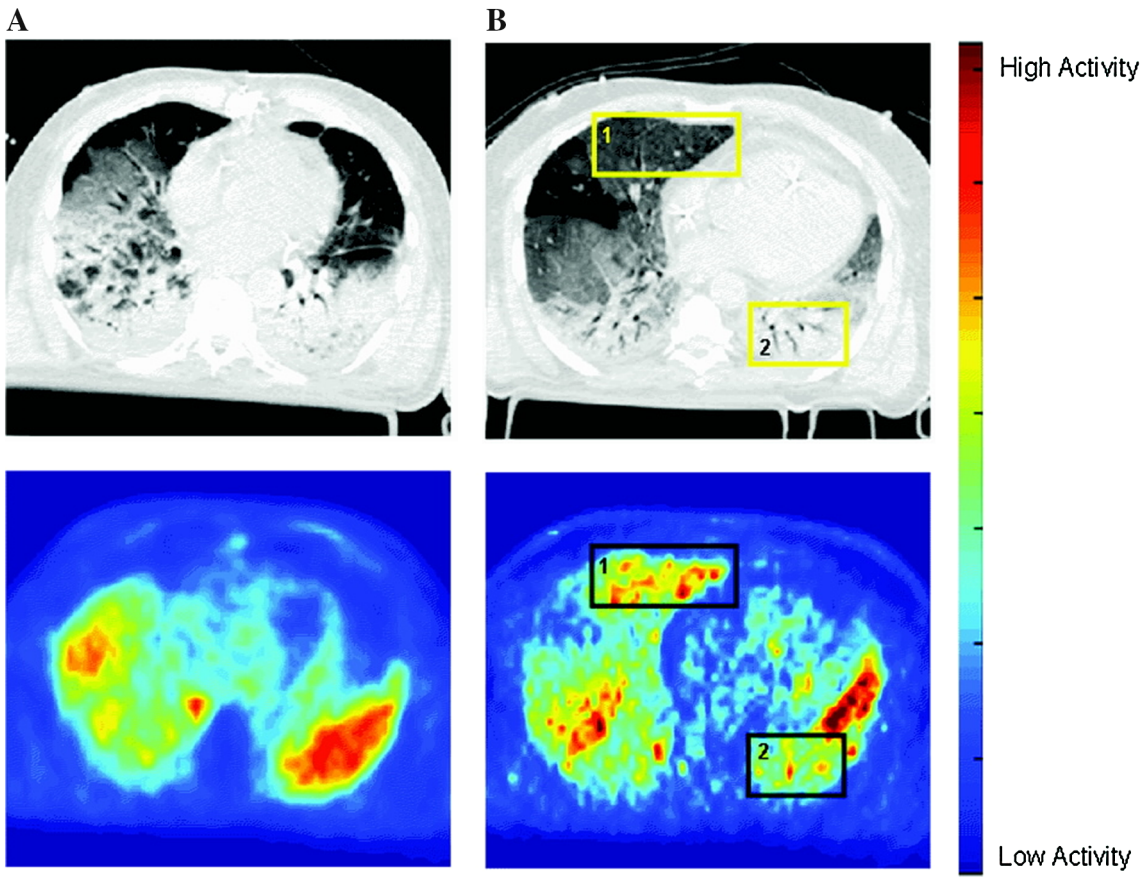

Fig. 3 Representative images of cross-registered computed tomography (at mean airway pressure) and [18F]-fluoro-2-deoxy-D-glucose (^{18}FDG) positron emission tomography from two patients with acute respiratory distress syndrome. Positron emission tomographic images represent the 18FDG uptake and the color scale represents radioactivity concentration. **a** ^{18}FDG distribution parallels that of the opacities detected on CT. **b** Intense ^{18}FDG uptake can be observed in normally aerated regions (*square 1*), while activity is lower in the dorsal, ''non-aerated'' regions of both lungs (*square 2*). Reproduced from [13]

spatial location (non-dependent) and with normal mechanical intrinsic characteristics. However, when the ARDS lung was studied by CT scan in the prone position (PP), it appeared that the densities redistributed from the dorsal to the ventral lung regions, leading to the concept of ''the sponge model''. Accordingly, the baby lung was no longer considered an anatomical entity but instead a functional one, which may quickly change its location and/or its dimensions with PP and/or application of PEEP (Fig. 4).

Regional mechanics of the baby lung

The anterior chest wall is more flexible than the posterior one. Therefore, assuming the PP increases total chest wall elastance, such changes affect trans-pulmonary pressures and lessen the pressure gradients that determine absolute lung volume as well as its topographic distribution. More uniform distribution of transpulmonary pressure by PP helps establish and sustain positional recruitment [16, 17] (Fig. 5). PP relieves cardiac compression of the supporting lung that lies beneath the heart in the supine position and may improve lymphatic drainage as the heart moves ventrally to a dependent position [18]. Such changes help to explain why PP improves shunt and aids resolution of hydrostatic edema [19, 20].

Yet, recruitment that occurs after turning neither fully explains proning-related improvements in oxygenation nor the reduced propensity to ventilator-induced lung injury (VILI). Establishing more uniform alveolar distension improves ventilation/perfusion ratios of acutely injured lungs [21]. Proning benefits oxygenation in the majority of cases and helps ventilation efficiency in a somewhat lesser proportion of patients [22]. Because perfusion distribution is only modestly modified by the

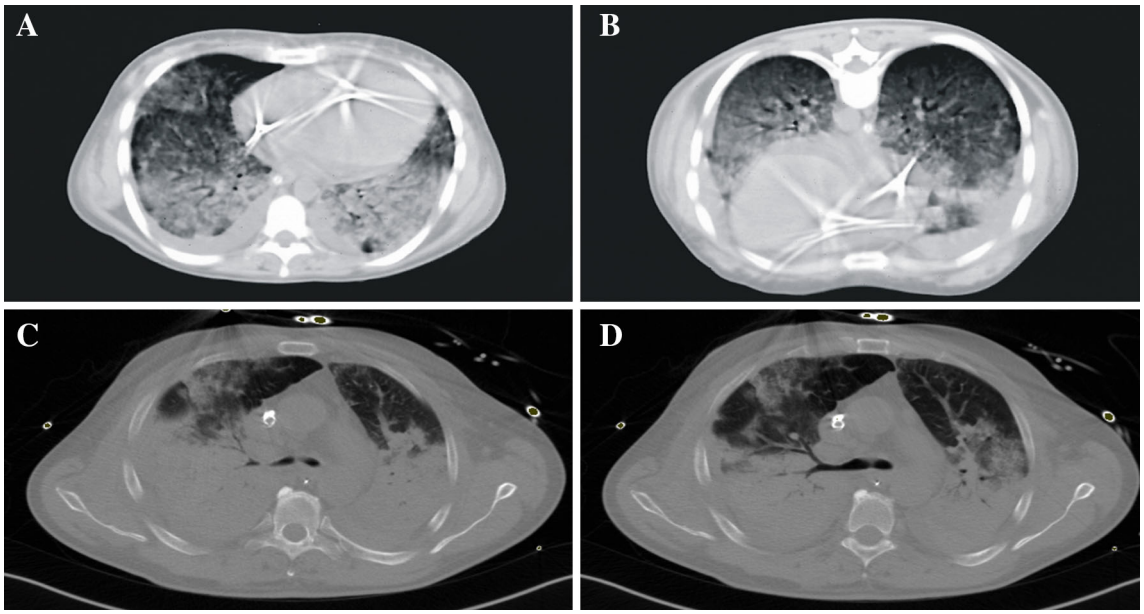

Fig. 4 Examples of widening the baby lung by prone position (**a**, **b**) and by increasing PEEP from 5 cmH_2O (**c**) to 15 cmH_2O (**d**). Note that recruitment in general proceeds from dorsal to ventral regions in the prone position and from ventral to dorsal by pressure increase

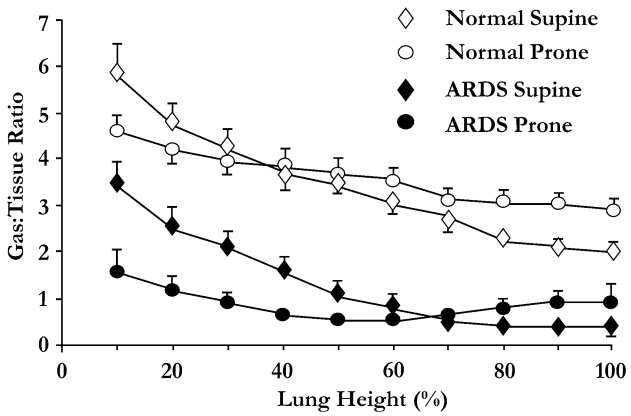

Fig. 5 The gas:tissue ratio as a function of lung height in supine (*rhombus*) prone (*circles*) positions. The *open symbols* refer to normal lungs of 14 patients and the *filled symbols* to lungs of 20 ARDS patients. The height = 0 refers to the ventral surface in the supine position and to the dorsal surface in the prone position. The height scale direction is from nondependent (0) to dependent (100). Reproduced from Ref. [74]

inversion of position [23], change in the distributional pattern of ventilation appears to be of greater importance. Resolving disparities of regional alveolar distension may also help prevent zonal over-distension and inappropriate redirection of blood flows from inflated to collapsed units. The latter phenomenon tends to occur when raising PEEP and mean airway pressure in the supine position. Right ventricular afterload may benefit from reduced over-distension, improved oxygenation, and recruitment of vascular beds [24, 25].

Proning and ventilator-induced lung injury

Ventilator-induced lung injury arises from repeated application of high mechanical forces that either tear fragile tissue directly or initiate signaling that culminates in inflammatory change [26]. Stress is augmented at the interfaces between open and closed (or anatomically non-inflatable) tissues, and these stress-focusing junctions (as well as VILI) prevail in gravitationally dependent zones [14]. These same units are subjected to surfactant-depleting tidal cycles of airspace opening and closure and to shearing forces that exceed those experienced by lung units that remain continuously open. Recruitment that occurs during proning enlarges the baby lung, reduces the number of high stress junctions and portends benefit from repositioning [22]. By reducing the number of interfaces between open and closed units, as well as by moderating transpulmonary forces, the regional excursions of mechanical tension (effective alveolar driving pressures) are lessened, especially in the dorsal zones. Finally, airway bio-fluids and secretions may directly inhibit surfactant viability or production, impede the ease of airspace opening, or predispose to lung infection [27]. Airway drainage, regional mechanics, and vascular filling are each improved by PP. Perhaps in part for this reason, hemorrhagic pulmonary edema and inflammation that result from adverse patterns of ventilation are differentially affected by positioning. In both healthy canine lungs [28] and those pre-injured by infused oleic acid, proning may reduce dorsal hemorrhage, edema and inflammation, whereas nondependent zones are relatively spared in both positions [28, 29].

In addition to attenuating regional transpulmonary disparities of force, PP confers a secondary benefit simply by improving the P/F ratio, thereby reducing the need for potentially iatrogenic interventions to sustain it. Improved oxygenation by PP may allow reductions of FiO_2, mean airway pressure, and pulmonary vascular resistance, thereby lowering the risk of injury to mechanically stressed membranes [30] and/or right ventricular afterload [31].

Recruitment

The amount of non-aerated and poorly aerated lung mainly determines the gas exchange impairment observed in ARDS. The use of PEEP keeps open newly recruited and previously non-aerated regions, thus enlarging the baby lung and improving oxygenation. The effects of PEEP on alveolar recruitment have been assessed using pressure–volume curves of the respiratory system and CT scan methods [32–34]. Irrespective of how recruitment is measured, an important concept is that the recruitment process occurs throughout inspiration, following the trajectory of the pressure–volume relationship of the respiratory system [33, 35, 36]. Therefore, what can remain open with PEEP is the tissue that was opened by the preceding inspiration. In other words, if the previous end-inspiratory pressure is low, the recruitment obtained at a given PEEP is less. This is supported by the finding that de-recruitment that results from decreasing tidal volume (VT) can be reversed by increasing PEEP [37].

With CT scanning, it has been shown that the amount of alveolar opening–closing decreases when PEEP increases [38]. At PEEP levels above 10–15 cmH_2O and end-inspiratory plateau airway pressures (Pplat) above 30 cmH_2O, the amount of tissue reopening and collapsing during the tidal cycle was minimal and the VT was redistributed more homogeneously between dependent and non-dependent regions.

In a series of studies on ARDS patients, Puybasset and coworkers performed extensive measurements on lung CT scans, gas exchange, hemodynamics and mechanics at zero end-expiratory pressure and PEEP 10 cmH_2O [39–41]. According to CT scan distribution of densities, they described three types of patterns: lobar, diffuse and patchy. Those with a diffuse, as compared to the lobar pattern, had a significantly lower functional residual capacity and a significantly higher amount of non-aerated lung volume. A diffuse versus lobar density pattern, a lower PaO_2/FiO_2, and less proportion of aerated lung, identified patients with higher mortality risk. The decrease in non-aerated lung with PEEP 10 cmH_2O was almost negligible (−82 mL) in patients with lobar pattern, whereas the highest decrease in non-aerated lung (−383 mL) was observed in those with a diffuse pattern.

The overall effects of PEEP on recruitability, however, are complex. The percentage of potentially recruitable lung, as analyzed with CT scans performed up to 45 cmH_2O airway pressure, was highly variable in acute lung injury/ARDS patients [42]. About 24 % of the lung could not be recruited even at 45 cmH_2O, and lung recruitment occurring from PEEP 5–15 cmH_2O correlated well with the percentage of potentially recruitable lung. Interestingly, patients with a high percentage of potentially recruitable lung had higher mortality rates than those with a low percentage. As recruitability increased with severity, it follows that the smaller the baby lung at low pressure, the greater would be its relative increase when PEEP is increased. A further analysis by Caironi and coworkers [43], showed that, in patients with a higher percentage of potentially recruitable lung, raising PEEP from 5 to 15 markedly decreased the amount of opening–closing lung tissue, but PEEP did not produce any effect in patients with a lower percentage of potentially recruitable lung. Analysis of the association between mortality and determinants of VILI showed that the amount of opening–closing was independently associated with an increased risk of death. A recent study [44] has shown that, among different bedside PEEP selection methods in ARDS patients, the one based on oxygenation criteria was most closely related to lung recruitability. Taken together, these data [42–44] suggest high PEEP levels should be used in more severe ARDS but not in patients with a lower percentage of potentially recruitable lung. In other words, the smaller the baby lung at low pressure the greater the PEEP to be applied.

Recently, Cressoni et al. [45] showed that the greater the ARDS severity, the greater the lung inhomogeneity. Such inhomogeneities were mainly distributed at the interface between lung regions with high and normal CT density, i.e. at the ‘‘boundary zones’’ of the baby lung, which were more numerous in non-survivors than in survivors and were always ‘‘inflamed’’, as shown by PET [15]. The authors proposed that lung inhomogeneity acts as a stress raiser, and they calculated that these regions are exposed to a stress which almost doubles the transpulmonary pressure applied. Such a mechanism (stress raising) can further magnify VILI. Interestingly, the extent of inhomogeneity was associated with the fraction of poorly aerated lung tissue, and it decreased when PEEP was increased.

Are there clinical data to suggest which PEEP level is most appropriate in ARDS? In the ExPress study [46], the use of low VT with high PEEP (about 15 cmH_2O, individually titrated to reach a Pplat of 28–30 cmH_2O) tended to increase mortality in acute lung injury patients when compared to low VT and moderate PEEP. Conversely, the low VT/high PEEP strategy decreased mortality in sicker ARDS patients. A secondary post hoc analysis of randomized PEEP trials further supports the selective use of high PEEP levels in severe ARDS patients, i.e. those with

smaller baby lungs [47]. Our understanding of the effects of PEEP in the baby lung is maturing. The new clinical challenge is to determine whether a PEEP titration strategy based on sound physiological and morphological principles, and which can be easily used at the bedside (using measurements such as dead space or driving pressures), improves outcomes in ARDS.

Baby lung and mechanical ventilation

Tidal volume and plateau pressure

In clinical practice, tidal volume is scaled to patient ideal body weight (IBW), predicted from the patient height (VT/IBW) to avoid excessive strain of the lung parenchyma. Relatively high tidal volumes, quantified as 12 mL/kg IBW or greater, cause un-physiological strain in the size-reduced ARDS lung. In turn, excessive strain causes, through mechanical transduction, increased local inflammation and inflammatory cytokines which likely contribute to increased mortality. Halving the harmful tidal volume may decrease mortality but predisposes to CO_2 retention (permissive hypercapnia) which, when moderate, is generally well tolerated and possible beneficial. Several clinical studies have analyzed the effects of lower versus higher values of VT/IBW. Among these outcome studies testing different tidal volumes, only the ARDS Network study, which compared the two extreme values (6 vs. 12 mL/kg) in 861 patients, showed a significant mortality reduction in the lower VT group (31 vs. 40 %) [48]. Although other studies did not find any significant mortality reduction [49–51] when testing VT values intermediate within the 6–12 mL/kg interval, low VT ventilation (6 ml/k IBW ventilation) has been widely accepted by the scientific community. Over-distension, however, has been described in the non-dependent lung regions even at 6 ml/kg IBW [52]. Although the ideal (or predicted) body weight was introduced as a better surrogate than measured weight to adjust for variations in lung size, it is evident that this approach does not perform optimally well in ARDS. In a man with an ideal body weight of 70 kg, the aeratable lung volume, i.e. the baby lung, may range from 200 to 300 ml to more than 1000 ml, depending on the ARDS severity. Referring to the gas volume of the baby lung instead of to the body weight is more physiologically adequate as it better reflects its distortion (strain) during ventilation. In fact, strain is defined as the ratio between tidal volume and the baseline gas volume of the baby lung. In the example above, the estimated strain induced by a tidal volume of 6 ml/kg IBW would range between 2.1 and 0.42—values found in animal studies to be lethal (i.e. leading to death) and innocent (without any harm), respectively. Therefore, a safer mechanical ventilation strategy should consider the size of the baby lung instead of body weight [53]. More recently, a post hoc analysis of 3562 patients with ARDS enrolled in nine previously reported randomized trials [54], compared the relative predictive power of tidal volume normalized to estimated lung compliance (driving pressure) with tidal volume normalized to ideal body weight. The former index performed considerably better, suggesting that compliance—an indicator of baby lung size—is a better surrogate than ideal body weight when tailoring tidal volume to prevent excessive strain.

The same line of reasoning may be applied when considering the stress, i.e. the pressure, which develops within lung structures to counteract the applied transpulmonary pressure. Therefore the stress equals the transpulmonary pressure (with opposite sign). The widely accepted surrogate for stress is the airway plateau pressure, and 30 cmH_2O is the suggested limit [55]. A simple analysis of the relationship of transpulmonary pressure (P_L) and airway pressure (P_{AW}) shows how this suggestion is questionable. In fact, the same P_{AW} is associated with a wide range of P_L due to different ratios of chest wall elastance (E_W) to respiratory system elastance (E_{RS})

$$P_L = P_{AW} \times \frac{E_W}{E_{RS}}$$

A post hoc analysis by the ARDS network could not identify a safe upper limit for plateau pressures in patients with ALI/ARDS, as values even lower than 30 cmH_2O might cause lung damage [56]. These data indicate that at 30 cmH_2O the baby lung may be very close to its total lung capacity or very far from it. Depending on chest wall elastance, transpulmonary pressure associated with 30 cmH_2O can be estimated to vary between 28 and 8 cmH_2O [53]. In animal studies, these levels are lethal or innocent, respectively [57].

Baby lung and spontaneous breathing

Although awareness of the baby lung concept has encouraged significant improvement in ventilator management, the same reasoning has not always been followed when the patient makes breathing efforts, as during noninvasive ventilatory support and when allowing some spontaneous breathing activity during the early phase of ARDS.

In theory, baby lung size should be the same at a given transpulmonary pressure, whether it is generated by a positive pressure, by a negative pressure (and therefore the respiratory muscles) or by a combination of the two. The validity of this principle was illustrated in an early animal model of VILI [58]. During spontaneous breathing, however, negative pleural pressure swings will have different vascular effects than during controlled mechanical ventilation, resulting in higher filling

pressures of the heart and in the pulmonary circulation [59]. This vascular congestion may increase the risk of VILI [60, 61]. In addition, the measurement of esophageal pressure may poorly reflect the distribution of regional pleural and transpulmonary pressures. Under the action of the diaphragm and of other accessory muscles, the regional pleural pressures may have a wider distribution than during controlled mechanical ventilation, where the gradient of pleural pressure is mostly dictated by gravity. Local variations of distending pressure may help explain the observation of *pendelluft* (i.e., regional redistribution of ventilation during the tidal cycle), among injured lung regions during spontaneous breathing [62]. In such cases, the risk of VILI may be poorly reflected by the global transpulmonary pressure.

Experimental and clinical data

Sustained hyperventilation induced by severe acidosis may lead to acute lung injury in animals [63]. In experimental models, however, allowing spontaneous breathing seems to have different consequences, depending on the severity of lung injury [64]. In the case of severe lung injury, spontaneous breathing appears to worsen damage while the opposite may happen in those with mild injury. These differences may reflect an increase in transpulmonary pressure and tidal volume during spontaneous ventilation, associated with high regional distending pressures and increases of trans-vascular pressure. A series of studies conducted in patients, confirmed by a multicenter trial, strongly suggested that the systematic administration of neuromuscular blockers can reduce biomarkers of inflammation and improve survival in patients with ARDS [65, 66]. Neuromuscular blockade was also the first treatment to show a significant reduction in the rate of pneumothorax (11.7 vs. 4.0 %). Of note, even the seminal study on low tidal volume did not show any reduction of barotrauma [48]. These results seem to have been determined primarily by data from patients with PaO_2/FiO_2 at or below 120 mmHg [65]. Although these results are not fully understood, they suggest a possible negative effect of spontaneous breathing on local distending pressures, as discussed above, by high ventilatory drive-caused asynchronies such as breath stacking and double cycling, or by reverse triggering. [67, 68]. In this situation, clinicians should carefully monitor the results of patient–ventilator synchronization in terms of the resulting tidal volume and transpulmonary pressure. Clinicians must be aware of the physiological differences between volume-controlled and pressure controlled ventilation on both trans-pulmonary pressure and patient comfort [69]. The use of non- synchronized pressure-targeted modes in this situation may help to maintain some spontaneous breathing activity with minimal risks, offering an interesting compromise [70].

Noninvasive ventilatory support

In non-intubated patients, gauging the severity of lung injury presents problems because respiratory mechanics are difficult to assess. Noninvasive ventilation has been frequently attempted in lung-injured patients as a way to avoid intubation, with various degrees of success [71, 72]. Recently, another technique of noninvasive ventilatory support in the form of high-flow heated and humidified oxygen therapy has been tested in patients with acute hypoxemic respiratory failure mostly resulting from severe infectious pneumonia [73]. Patients with PaO_2/FiO_2 at or below 200 mmHg were less likely to need intubation than those treated by conventional masks. Overall, patients treated by high-flow nasal oxygen had a lower hospital and 90-day mortality. Such surprising results clearly need confirmation. However, one possibility to explain the benefit in survival with this simple and well-tolerated technique may be a reduction in ventilatory needs due to a washout of anatomic dead space. Although not clearly demonstrated, it is conceivable that a reduction in tidal volume and/or in minute ventilation imposed on the baby lung (due to dead space wash-out or improved comfort) could explain this observed decrease in mortality.

Conclusions

The size of the baby lung determines tissue compliance and directly dictates the mechanical properties of the ARDS lung. For a given tidal volume, therefore, stress and strain relate to baby lung size. Physical characteristics such as mechanical homogeneity and dimension of the baby lung may be deeply affected by PP and PEEP. The positive effects of these two maneuvers appear inversely related to the baby lung size. Although less well documented, these same rules are likely to apply when spontaneous breathing efforts are made. The baby lung concept may help to understand and implement safe mechanical or spontaneous ventilation during the different stages of ARDS.

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Compliance with ethical standards

Conflicts of interest None.

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