Spontaneous Effort Causes Occult Pendelluft during Mechanical Ventilation

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Rationale: In normal lungs, local changes in pleural pressure (Pₚ₉) are generalized over the whole pleural surface. However, in a patient with injured lungs, we observed (using electrical impedance tomography) a pendelluft phenomenon (movement of air within the lung from nondependent to dependent regions without change in tidal volume) that was caused by spontaneous breathing during mechanical ventilation.

Objectives: To test the hypotheses that in injured lungs negative Pₚ₉ generated by diaphragm contraction has localized effects (in dependent regions) that are not uniformly transmitted, and that such localized changes in Pₚ₉ cause pendelluft.

Methods: We used electrical impedance tomography and dynamic computed tomography (CT) to analyze regional inflation in anesthetized pigs with lung injury. Changes in local Pₚ₉ were measured in nondependent versus dependent regions using intrabronchial balloon catheters. The airway pressure needed to achieve comparable dependent lung inflation during paralysis versus spontaneous breathing was estimated.

Measurements and Main Results: In all animals, spontaneous breathing caused pendelluft during early inflation, which was associated with more negative local Pₚ₉ in dependent regions versus nondependent regions (−13.0 ± 4.0 vs. −6.4 ± 3.8 cm H₂O; P < 0.05). Dynamic CT confirmed pendelluft, which occurred despite limitation of tidal volume to less than 6 ml/kg. Comparable inflation of dependent lung regions during paralysis required almost threefold greater driving pressure (and tidal volume) versus spontaneous breathing (28.0 ± 0.5 vs. 10.3 ± 0.6 cm H₂O, P < 0.01; 14.8 ± 4.6 vs. 5.8 ± 1.6 ml/kg, P < 0.05).

Conclusions: Spontaneous breathing effort during mechanical ventilation causes unsuspected overstretch of dependent lung during early inflation (associated with reciprocal deflation of nondependent lung). Even when not increasing tidal volume, strong spontaneous effort may potentially enhance lung damage.

Keywords: acute lung injury; spontaneous breathing; transpulmonary pressure; pleural pressure; electrical impedance tomography
Creating a uniform increase in transpulmonary pressure \( (P_L) \) (3–7): the combination of these pressures, the \( P_L \) gradient \( (P_L = P_{aw} - P_{pl}) \), is the net pressure driving inflation of the lung (10). Therefore, spontaneous breathing, by making \( P_{pl} \) more negative, uniformly augments \( P_L \) for any given \( P_{aw} \).

Thus, spontaneous breathing is traditionally encouraged in patients receiving mechanical ventilation (11, 12) because it is thought to provide lung expansion at lower levels of \( P_{aw} \), a strategy that results in better local (especially dependent) lung aeration, thereby enhancing gas exchange and potentially improving hemodynamics (13).

Recently, techniques such as electrical impedance tomography (EIT) that allow visualization of regional lung inflation in real-time have become available (14). We report here a patient with acute respiratory distress syndrome (ARDS) receiving mechanical ventilation (11, 12) because it is thought to provide lung expansion at lower levels of \( P_{aw} \), a strategy that results in better local (especially dependent) lung aeration, thereby enhancing gas exchange and potentially improving hemodynamics (13).

TABLE 1. BASELINE PATIENT CHARACTERISTICS

<table>
<thead>
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<th>Age, yr</th>
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<tr>
<td>Sex</td>
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<td>Weight, kg</td>
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<tr>
<td>Surgery</td>
<td>Coronary artery bypass graft surgery with two internal thoracic arteries and two saphenous veins</td>
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<td>Amount of lost blood, ml</td>
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<td>−7.8</td>
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<td>Lactate, mg/dl</td>
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</table>

**Figure 1.** Electrical impedance tomography (EIT) waveforms in a patient with acute respiratory distress syndrome, spontaneous versus ventilator breaths. The patient was ventilated with assist pressure-controlled ventilation \( (IP, 5 \text{ cm H}_2\text{O}; f, 12 \text{ min}^{-1}; \text{PEEP}, 13 \text{ cm H}_2\text{O}; \text{triggering threshold}, −2 \text{ cm H}_2\text{O}) \). EIT data were recorded with spontaneous breathing (left) and without spontaneous breathing (right). The EIT image was divided into four zones, each covering 25% of the ventrodorsal diameter (zones 1–4). During controlled ventilation (under muscle paralysis), simultaneous inflation of each of the different lung regions was observed, although at differing inflation rates. In contrast, when spontaneous efforts were present, two observations were noted. First, in the initial stages of the breath spontaneous effort caused inflation of dependent lung regions (red in zone 4), which was greater with controlled breaths. Second, the early inflation in the dependent region was accompanied by concomitant (transient) deflation of nondependent region (red in zones 1 and 2), indicating movement of gas from nondependent to dependent lung regions: a pendelluft phenomenon. \( f = \text{respiratory frequency}; \text{IP} = \text{inspiratory pressure}; \text{PEEP} = \text{positive end-expiratory pressure.} \)

Creating a uniform increase in transpulmonary pressure \( (P_L) \) (3–7): the combination of these pressures, the \( P_L \) gradient \( (P_L = P_{aw} - P_{pl}) \), is the net pressure driving inflation of the lung (10). Therefore, spontaneous breathing, by making \( P_{pl} \) more negative, uniformly augments \( P_L \) for any given \( P_{aw} \).

We hypothesized that, in contrast to the uniform distribution of pressures exhibited in normal lung, negative \( P_{pl} \) generated by diaphragmatic contraction might not be transmitted all over the lung surface in the setting of lung injury, but rather would be transiently localized to dependent lung, and that such locally
concentrated $P_{pl}$ would cause pendelluft and expansion of the local dependent lung. Some of the results of these studies have been previously reported in the form of an abstract during 2013 American Thoracic Society International Conference (16).

METHODS

This study was approved by the ethics committee for clinical studies (patient report) and the ethics committee for experimental studies (animal study) at Faculdade de Medicina da Universidade de São Paulo. Detailed clinical data and experimental methods are described in the online supplement.

Patient Observations

A 40-year-old man developed postoperative hypoxemia while receiving mechanical ventilation in the intensive care unit after coronary artery bypass graft surgery. As part of comprehensive monitoring, EIT was used to visualize the regional distribution of inflation. EIT data were recorded using the Enlight impedance tomography monitor (Dixtal, São Paulo, Brazil), placed on the perimeter defining a cross-sectional plane of the thorax at the level of the fifth to sixth intercostal space (14, 17). Regional distribution of ventilation was analyzed with the subdivision of the thorax in four zones (ventrodorsal) in the presence or absence of spontaneous breathing effort.

Animal Experiments

Seven Landrace pigs were anesthetized with midazolam, ketamine, and fentanyl, and tracheotomized. An esophageal balloon catheter (SmartCath; Bicore, Irvine, CA) was inserted to measure the esophageal pressure ($P_{es}$) and its position validated as described previously (18). After establishing lung injury, animals were ventilated with assisted pressure-controlled ventilation at driving pressure of 5–15 cm H$_2$O to obtain a tidal volume of 6 ml/kg, respiratory rate of 20–30 breaths per minute, and pressure trigger of $-2$ cm H$_2$O. Positive end-expiratory pressure was set at “open-lung positive end-expiratory pressure” $-10$ cm H$_2$O. All animals preserved spontaneous breathing effort, with titrating sedatives. EIT data were recorded at the level of the sixth intercostal space and analyzed with the same subdivision as for the patient thorax (as above).

Pleural Pressure

Local changes in $P_{pl}$ were measured in nondependent versus dependent regions (of five animals) by balloon catheter occlusion (Pulmox, Inc., Redwood, CA) of subsegmental bronchi via a fiberoptic bronchoscope as follows: nondependent region, left B; dependent region, left lower lobe beyond D4. The pressure swings in the occluded subsegments were used as surrogates for the relative changes in local $P_{pl}$, as described previously (19). Simultaneous pressure recording of $P_{es}$ and $P_{pl}$ were performed, while preserving spontaneous effort. $P_{pl}$ was directly measured using a balloon-tipped catheter in five pigs. The catheter was surgically inserted between the dependent parts of the lung (dorsal aspect of the diaphragmatic surface) and the diaphragm. We simultaneously measured $P_{pl}$ and $P_{es}$ while preserving spontaneous effort.

Regional Inflation

To estimate the magnitude of local lung inflation caused by spontaneous effort, we determined the pressures required to inflate a region of dependent lung with positive pressure (i.e., ventilator, airway driving pressure only) versus spontaneous effort (i.e., ventilator + diaphragmatic contraction, airway driving pressure + the change in $P_{pl}$) in five pigs. Then, after measuring the local (dependent) impedance change (delta $Z$) during assisted ventilation (representing local tidal strain) at a fixed ventilator driving pressure of 10 cm H$_2$O, we paralyzed the animal and commenced controlled ventilation with progressive increments in driving pressure, until the same delta $Z$ developed in the dependent lung.

Figure 2. Electrical impedance tomography (EIT) waveforms in experimental lung injury, spontaneous versus ventilator breaths. In an anesthetized pig model of acute lung injury assist pressure-controlled ventilation ($IP$, $15$ cm H$_2$O; $f$, $25$ min$^{-1}$; $PEEP = 13$ cm H$_2$O; triggering threshold, $-2$ cm H$_2$O) was used. The EIT image was divided into four zones, each covering 25% of the ventrodorsal diameter (zones 1–4). During controlled ventilation (under muscle paralysis), simultaneous inflation of each of the different lung regions was observed, although at different inflation rates. In contrast, when spontaneous efforts were present, two observations were noted. First, in the initial stages of the breath, spontaneous efforts caused inflation of dependent lung regions (red in zones 3 and 4), which was greater with controlled breaths. Second, the early inflation in the dependent region was accompanied by concomitant (transient) deflation of nondependent region (red in zone 1), indicating movement of gas from nondependent to dependent lung regions; because this was not associated with alterations in tidal volume it indicates a pendelluft phenomenon.

This finding was always present during spontaneous breathing efforts in all animals with experimental lung injury. $f$ = respiratory frequency; $IP$ = inspiratory pressure; $PEEP$ = positive end-expiratory pressure.
Dynamic Computed Tomography Scans

For the validation of regional distribution of ventilation and pendelluft detected by EIT, dynamic computed tomography (CT) scans were performed in one representative pig to evaluate the absolute movement of air within thick slices close to the diaphragm, and in additional thick slices above the carina. Dynamic CT scans were performed over 10-second intervals without interrupting mechanical ventilation, using the same ventilator settings as recorded in EIT. For each slice, we assessed the regional ventilation at nondependent one-third versus dependent one-third of the ventrodorsal chest diameter.

Statistical Analysis

Intergroup differences were evaluated using one-way repeated measurement analysis of variance followed by Dunnett test. Two-condition comparisons (Ppl measurements) were done by unpaired t test. Statistical significance was considered where P less than 0.05.

RESULTS

Observations from the Patient with Lung Injury

During controlled mechanical ventilation with muscle paralysis (i.e., no spontaneous effort), we observed simultaneous inflation of different lung regions, although the rates of inflation differed across the lung (Figure 1, right; see Video E1 in the online supplement). In contrast, when spontaneous effort was present (i.e., without neuromuscular paralysis) we observed two key differences (Figure 1, left; see Video E2). First, in the initial stages of the breath (until peak inspiratory flow was reached, after completion of the “triggering phase”) spontaneous effort inflated dependent lung regions to a greater extent (~1.5–2 fold) than with controlled ventilation (i.e., in the presence of muscle paralysis). Second, the early inflation in the dependent region was accompanied by concomitant (transient) deflation in the nondependent region, suggesting the existence of pendelluft (i.e., movement of alveolar air from one region of lung to another, without significant gain in tidal volume). Importantly, aside from deflections in the pressure-time tracing, no differences were noted with conventional respiratory monitoring (e.g., tidal volume, minute ventilation, plateau pressure) during spontaneous breathing efforts as compared with controlled ventilation under muscle paralysis (Table 1).

Experimental Studies

Regional distribution of inflation: lung injury versus normal lungs.

Using EIT, in injured lungs, we demonstrated that in the presence of neuromuscular paralysis there was heterogeneous but simultaneous inflation in the dependent and in the nondependent lung regions (Figure 2, right; see Video E3), as observed in our patient.

Also in injured lungs, spontaneous breathing effort resulted in initial inflation of dependent lung with concomitant deflation in

| TABLE 2. RESPIRATORY MEASUREMENTS AT ELECTRICAL IMPEDANCE TOMOGRAPHY RECORDING AND SIMULTANEOUS RECORDING OF PLEURAL PRESSURE AND ESOPHAGEAL PRESSURE |
|-------------------------------|----------------|----------------|
| Respiratory Variables at Electrical Impedance Tomography Recording (n = 7) | Normal Lung (before lung injury) | Spontaneous Breathing | Muscle Paralysis |
| Body weight, kg | 31.2 ± 4.1 | 7.26 ± 0.11 |
| pH | | |
| PaO2/FiO2, mm Hg | 79.4 ± 61.2 | 46.2 ± 14.7 |
| PaCO2, mm Hg | 14.1 ± 25.3 |
| Dynamic compliance of respiratory system, ml/cm H2O | 14.4 ± 3.7 | 14.1 ± 3.5 |
| Tidal volume, ml/kg | 14.0 ± 7.1 | 9.7 ± 1.3 | 5.2 ± 2.0 |
| Change in esophageal pressure, cm H2O | 3.4 ± 0.9 | 5.1 ± 3.5 | 2.4 ± 0.6 |
| Change in pleural pressure, cm H2O | 6.0 ± 2.3 | 9.6 ± 4.5 | 9.2 ± 5.8 |
| Peak transpulmonary pressure, cm H2O | 13.0 ± 4.2 | 25.3 ± 4.2 |
| Peak flow, L/s | 0.73 ± 0.08 | 0.69 ± 0.28 | 0.51 ± 0.17 |

Lung Regions

<table>
<thead>
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<th>Nondependent Lung</th>
<th>Dependent Lung</th>
</tr>
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<tbody>
<tr>
<td>Airway occlusion manometry (n = 5)</td>
<td></td>
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<tr>
<td>Change in esophageal pressure, cm H2O</td>
<td>−6.5 ± 3.1</td>
</tr>
<tr>
<td>Change in pleural pressure, cm H2O</td>
<td>−6.4 ± 3.8</td>
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<tr>
<td>Direct measurement (n = 5)</td>
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<tr>
<td>Change in esophageal pressure, cm H2O</td>
<td></td>
</tr>
<tr>
<td>Change in pleural pressure, cm H2O</td>
<td></td>
</tr>
</tbody>
</table>

Note that during airway occlusion manometry, we presented the each Pp measured at the same time as the nondependent Pp swing measurement and the dependent Pp swing measurement, respectively.

* P < 0.05 compared with others.
† P < 0.05 compared with change in pleural pressure in nondependent lung.
‡ P < 0.01 compared with change in esophageal pressure.
nondependent inflation (Figure 2, red; see Video E4). Synchronous inflation and deflation occurred without significant difference in exhaled tidal volume between spontaneous effort and muscle paralysis (5.9 ± 2.1 vs. 5.2 ± 2.0 ml/kg; P = 0.13) (Table 2), suggesting transfer of gas within the lung, in this case from nondependent (deflating) to dependent (inflating) regions. This observation is consistent with pendelluft, as observed in our patient. The inflation of dependent lung (zones 3 and 4) and simultaneous deflation of nondependent lung (zone 1) extended well beyond the triggering phase and lasted until esophageal pressure reached its most negative levels (Figure 2, red). Such pendelluft-type ventilation was observed in all lung-injured pigs. Continuous recording of EIT and Ppl was made during the transition from spontaneous effort to complete muscle paralysis (after a bolus of succinylcholine chloride) (Figure 3); this demonstrated that the extent of the pendelluft was proportional to the intensity of the respiratory effort.

In contrast, in normal lungs (i.e., before lung injury), we observed simultaneous inflation of different lung regions during spontaneous breathing (see Figure E1), consistent with fluid-like behavior of the lung.

**Regional distribution of inflation in lung injury: dynamic CT.** Representative fast dynamic CT acquisitions in an experimental animal corroborated the EIT data, confirming the induction of pendelluft by spontaneous breathing (Figure 4; see Figure E2B). Fast dynamic CT acquisitions also revealed that spontaneous breathing effort caused early local deflation of the nondependent regions in already aerated lung during inspiration (Figure 4, green). Simultaneously, transient local recruitment of dependent regions was observed because of pendelluft (i.e., tidal recruitment) (Figure 4, red). No pendelluft was observed during controlled mechanical ventilation (see Figure E2A). The CT studies also showed that, when present, the intra-slice movement of air (from nondependent to dependent zones) was most pronounced in lung slices close to the diaphragm (see Figure E2B, red) than in sections located further cephalad from the diaphragm.

**Plural pressure.** Spontaneous effort was associated with a large vertical gradient of Ppl swings, from nondependent to dependent regions. The swings in Ppl were larger in dependent regions, as compared with nondependent regions (−13.0 ± 4.0 vs. −6.4 ± 3.8 cm H2O; P < 0.05) (Table 2). The changes in Pes were similar to the changes in Ppl observed in nondependent lung regions, but smaller than the Ppl changes in dependent regions, indicating that ∆Pes significantly underestimated ∆Ppl in dependent regions. Furthermore, with spontaneous effort, directly measured swings in Ppl were significantly larger in dependent lung than swings in Ppl (−14.9 ± 3.1 vs. −7.1 ± 2.1 cm H2O; P < 0.01) (Table 2), corroborating the indirect measurements with intrabronchial balloon catheter. A sample of Ppl waveform in dependent regions versus a simultaneous change in Pes is illustrated in Figure 5.

**Magnitude of local inflation caused by spontaneous effort.** To achieve comparable tidal inflation of dependent lung regions, passive inflation (positive pressure only) of the lung required a threefold increase in airway driving pressures when compared with airway driving pressures used during spontaneous effort (28.0 ± 0.5 vs. 10.3 ± 0.6 cm H2O; P < 0.001). In contrast, similar levels of local delta transpulmonary pressures were observed during spontaneous versus controlled ventilation (peak ∆P; 22.3 ± 3.9 vs. 24.9 ± 1.7 cm H2O; P = 0.14) when achieving comparable inflation of the dependent lung (Figure 6, upper).

During spontaneous effort, the large tidal excursion in the dependent lung (as indicated by EIT) was associated with no changes in the (small; 5.8 ± 1.6 ml/kg) global tidal volume. During controlled ventilation, to achieve comparable inflation of the dependent lung, a significantly larger global tidal volume (14.8 ± 4.6 ml/kg; P < 0.05) (Figure 6, lower) was required. This was related to the presence of pendelluft during spontaneous breaths, which transiently inflated the dependent lung, but transiently deflated the nondependent lung.

**DISCUSSION**

This is an early description of a pendelluft phenomenon in a patient with lung injury, consisting of shifts of alveolar air from nondependent to dependent lung regions without a change in tidal volume, and caused by spontaneous breathing effort in the presence of mechanical ventilation.

This study has two important implications. First, lung-protective ventilation strategy (i.e., the limitation of plateau pressure and tidal volume) cannot prevent unsuspected local overstretch of the dependent lung because of pendelluft when moderate to strong spontaneous effort is preserved during mechanical ventilation. Second, the phenomenon occurs because, in contrast to normal lung, the injured lung does not exhibit fluid-like behavior, and in this case the transmission of local changes in Ppl is heterogeneous: a large vertical pressure gradient in Ppl swings. In this context, a single spatial measurement (e.g., esophageal pressure monitoring) does not represent an overall change in Ppl.

Standard physiologic explanations indicate that lung exhibits fluid-like behavior while inflating (1–7). This is based on the
observations in normal lung, in which diaphragmatic contraction triggers local changes in $P_{pl}$ that are uniformly transmitted across the entire lung surface (3–7). This principle explains, in part, why $P_{es}$ is used as a surrogate of overall $P_{pl}$ to optimize lung distention, an approach that may improve outcome in patients with ARDS (18, 20).

In the current study we confirmed that spontaneous effort during mechanical ventilation in healthy (uninjured) lungs resulted in almost simultaneous inflation throughout the lung. Although inflation of different regions occurred at different rates, inflation was almost simultaneous in all areas; there was no significant concomitant deflation (see Figure E1). In addition, airway occlusion tests performed in healthy lung demonstrated that changes in $P_{es}$ closely matched changes in $P_{aw}$ (and local changes in $P_{pl}$ matched changes in $P_{aw}$) (see Figure E3). Thus, these observations indicate simultaneous generalization of local $P_{pl}$ changes throughout normal lung, supporting the concept of fluid-like behavior in the (normal) lung (1–7).

However, the pattern of lung inflation was different in the presence of lung injury. In the injured lung pendelluft occurred. It resulted from the development of a more negative swing in $P_{pl}$ in the dependent lung than in the nondependent lung. Acute lung injury involves tissue inflammation and fluid-filled alveoli in a heterogeneous distribution; thus, some areas are well aerated and others collapse or fill with fluid and inflammatory cells (15). Such dense tissue may behave less like a fluid and more as a frame of solid areas resisting shape deformation. In this setting, part of the mechanical energy generated by the diaphragmatic contraction is exerted on local lung deformation rather than being transmitted to the rest of the lung; this results in imperfect elastic anisotropic inflation. Because atelectasis in ARDS tends to be greater in the dependent and peridiaphragmatic lung regions (21), such responses might be more marked in these areas. Supporting this idea is the finding that even in normal lung, extreme levels of phrenic nerve stimulation, which results in exaggerated diaphragmatic contraction, can cause more negative change in $P_{pl}$ at the diaphragmatic surface than in other parts of the lung (3, 5, 6). Thus, even the normal lung may depart from its fluid-like behavior at extreme levels of deformation. We speculate that during injury, this phenomenon is exacerbated and, even at moderate levels of muscle effort, the physical properties of the lung cannot compensate (i.e., with sufficient rapidity) for dynamic shape changes produced by diaphragmatic contraction.

An important issue of clinical relevance is that this effect is not detectable using conventional monitoring. In the current study, demonstration of the effect required dynamic imaging (i.e., EIT or dynamic CT). In addition, although esophageal monometry ($P_{es}$) is becoming a more standard clinical tool for estimation of $P_L$ or $\Delta P_L$ (18, 20), this technique significantly
underestimated the PL in the dependent lung (Figure 5), and would therefore not detect this potentially harmful phenomenon. Furthermore, the concomitant inflation and deflation that resulted in air exchange from nondependent to dependent lung segments resulted in little net change in tidal volume. Hence, pendelluft would not be suspected from monitoring gas flow at the trachea. Finally, low tidal volume ventilation did not prevent the phenomenon. Despite limitation of tidal volume to less than 6 ml/kg, strong spontaneous effort resulted in unsuspected local overstretch of the dependent lung because of pendelluft, leading to tidal recruitment in dynamic CT acquisitions (Figure 4); indeed, matching this degree of regional overstretch during neuromuscular paralysis required an overall tidal volume of 15 ml/kg (i.e., a highly injurious lung stretch).

Overall ventilator-induced lung injury consists of important regional elements (22) and it is possible that such regional components could be exacerbated by this pendelluft effect. For example, transient overstretch of dependent region could cause or worsen injury in more atelectatic lung, causing exacerbation of tidal-recruitment as obvious in dynamic CT acquisitions (Figure 4). Indeed, a previous paper clearly disclosed that spontaneous breathing effort associated with high PL caused tidal recruitment and worsened experimental lung injury, despite the use of low tidal volume ventilation (23). In addition, cycles of transient deflation/inflation of the nondependent regions could worsen already aerated (and potentially hyperinflated) lung. Such mechanisms could explain a recent report that muscle paralysis early in the course of severe ARDS leads to a better survival, and less barotrauma, notwithstanding the use of comparable tidal volumes and plateau pressures (24–26).

Important potential influences on changes to thoracic impedance during the respiratory cycle include cephalocaudal displacement of the lung during the respiratory cycle and pulmonary-cardiac blood volume change. Approximately 10-cm spans of lung were sampled with EIT; thus, the bulk of a whole lobe would be in measurement range of EIT at all stages of the respiratory cycle. In addition, dynamic, thin-slice (5 mm) CT ensured that we underestimated the ΔPl in the dependent lung (Figure 5), and would therefore not detect this potentially harmful phenomenon. Furthermore, the concomitant inflation and deflation that resulted in air exchange from nondependent to dependent lung segments resulted in little net change in tidal volume. Hence, pendelluft would not be suspected from monitoring gas flow at the trachea. Finally, low tidal volume ventilation did not prevent the phenomenon. Despite limitation of tidal volume to less than 6 ml/kg, strong spontaneous effort resulted in unsuspected local overstretch of the dependent lung because of pendelluft, leading to tidal recruitment in dynamic CT acquisitions (Figure 4); indeed, matching this degree of regional overstretch during neuromuscular paralysis required an overall tidal volume of 15 ml/kg (i.e., a highly injurious lung stretch).

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compared the same anatomic slices at the end of inspiration and of expiration. Notwithstanding slight cephalocaudal movement of lung with respiration, the broad sampling range used in EIT and the thin sampling slice used in dynamic CT precludes movement artifact. Finally, we carefully excluded the heart and diaphragm from regions of interest, and CT analysis was based on absolute (not relative) air content, thereby excluding artifact from changes in intrathoracic blood volume.

A potential concern is that $P_L$ during spontaneous breathing does not directly reflect transalveolar pressure (i.e., alveolar pressure = $P_{aw}$ – resistive pressure). However, the resistive pressure generated is negligible. We demonstrated (Figure 6, upper) that at equal local tidal volume (delta $Z$), the $\Delta P_L$ generated during paralysis versus spontaneous breaths were similar, and thus at that level of delta $Z$ the resistive pressure must be negligible. Note that during paralysis, $P_L$ = transalveolar pressure because we have zero flow conditions at the end inspiration.

It is important to emphasize that spontaneous breathing caused pendelluft whether triggering was with pressure triggering or flow triggering (see Figure E4). The flow-time curves indicate that pendelluft is apparent after inflow has begun, after triggering is complete. In addition, simultaneous EIT and pressure recording during transition from spontaneous effort to paralysis demonstrate that the degree of pendelluft is proportional to the intensity of spontaneous effort (Figure 3). These observations suggest that triggering is associated with, but not the mechanism of, pendelluft. Nonetheless, we anticipate that during spontaneous breathing, triggering delay or auto positive end-expiratory pressure could augment pendelluft.

Although we demonstrated extreme, transient degrees of regional stretch, additional experiments are needed to determine whether such overstretch actually caused localized lung injury. Indeed, it is possible that the phenomenon may selectively inflate dependent (atelectatic) lung regions thereby augmenting overall lung recruitment, especially when lung injury is mild, spontaneous effort is modest, and $P_L$ can be maintained at low levels (12, 23, 27).

In conclusion, this study describes a new phenomenon during mechanical ventilation whereby spontaneous breathing during mechanical ventilation causes unsuspect, transient overstretching of dependent lung regions with concurrent deflation of nondependent lung.

Author disclosures are available with the text of this article at www.atjournals.org.

References