Body Position Changes Redistribute Lung Computed-Tomographic Density in Patients with Acute Respiratory Failure

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Ten patients with parenchymal acute respiratory failure (ARF) underwent computed tomography (CT) scans while in the supine and prone positions. At equal levels of positive end-expiratory pressure, the authors measured the changes of CT density in dorsal and ventral basilar lung regions induced by the change of position as well as alterations of gas exchange. The level of venous admixture did not change with body position. The CT scan image of each lung was fractionated into ten levels from dorsal to ventral, each constituting 10% of the lung height. After measuring each lung fraction, the volume, the average CT number, its frequency distribution, and the expected normal value, we computed the lung tissue mass, the excess tissue mass, and the fraction of normally inflated tissue (excess tissue mass = amount of “tissue,” which includes edema, cells, and blood in excess of the expected normal value). We also estimated the superimposed hydrostatic pressure on each lung region. We found that the excess lung tissue mass is independent of position. However, in patients in the supine position, lung CT density increased and regional inflation decreased from ventral to dorsal, suggesting progressive deflation of gas-containing alveoli along the gravity gradient. A similar ventral–dorsal deflation pattern occurred within 10 min in patients in the prone position. We conclude that the lung in patients with ARF behaves like an elastic body with a diffusely increased mass; dependent lung regions are compressed by the pressure of overlying structures. Reversal of gravitational forces induced atelectasis of previously inflated lung regions and inflation of previously atelectatic regions but did not change the overall efficiency of oxygen exchange (right-to-left intrapulmonary shunt (Qa/Qd). (Key words: Lung: acute respiratory failure; gas exchange: pulmonary edema. Measurement techniques: computer-assisted tomography. Ventilation: body position.)

Placement of the patient in the prone position was suggested by Bryan1 as a technique to improve arterial oxygenation during acute respiratory failure (ARF) resulting from inflammatory lung disease. Several investigators subsequently reported improved oxygenation in patients in the prone position, but an observation that suggests better ventilation/perfusion matching as the likely mechanism of improved gas exchange. We recently described a series of 12 patients with ARF in whom gas exchange effects of the prone position were variable. In some patients oxygen exchange improved, whereas in others it did not. In that clinical study we occasionally observed a dramatic redistribution of lung density in the computed tomography (CT) scan when patients were turned from supine to prone. The CT density redistribution from dorsal to ventral lung regions when changing body position has also been occasionally observed in our previously reported studies of patients with ARF. In this study we quantitatively and prospectively investigated the effect of a supine-to-prone position change on the CT scan to define the mechanism of lung density redistribution. We also concomitantly measured the pathophysiological effects of a change in position on gas exchange.

Materials and Methods

Study Population

Ten patients (six male, four female) with moderate to severe ARF, as defined by Greene et al., were included in the study. The mean age was 42 ± 19 yr (range, 12–74 yr). Five patients had ARF resulting from pneumonia (four viral and one bacterial); four had ARF with sepsis; and one had ARF associated with multiple trauma. Six patients survived and were discharged from the hospital. The time from tracheal intubation until CT study averaged 3.2 ± 1.8 days (range, 1–7 days). Fourteen normal volunteers, including the authors (mean age, 27 ± 5 yr; range, 24–43 yr), without a history of lung disease underwent CT scanning to provide a normal reference group.

Study Design

The study was conducted in the framework of the extracorporeal support study and approved by the human studies committee of Lombardia Region. Informed consent was obtained from the relatives of patients before study. All patients were sedated and paralyzed throughout the procedure. The patients were transferred to the CT

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scan facility after a period of stabilization in the intensive care unit (ICU), and the CT scan room was equipped with the same equipment used in our ICU (e.g., ventilator, monitoring). During the study, ventilatory parameters were kept constant: tidal volume (TV) 10 ml/kg, respiratory rate (RR) 16–20 breaths per min, fractional inspired oxygen concentration (FIO₂) 0.4–1 (average, 0.58 ± 0.12), and inspiratory/expiratory ratio (I/E) 1:1. However, the level of positive end-expiratory pressure (PEEP) was set between 5 and 15 cmH₂O (average, 11 ± 3 cm H₂O), according to oxygenation requirements as set in the ICU.

**CT Scan**

**ARP Patients**

After a frontal tomogram covering the chest was obtained, each patient had a CT scan of the lung bases while in the supine position. The level "lung bases" was defined as being 1–2 cm above the diaphragm in expiration. After the CT scan was obtained with the patients in the supine position, all physiologic data were collected. Immediately thereafter, the patient was turned prone with a pillow under the pelvis; a CT scan of the lung bases was repeated; and a second set of physiologic data was collected. The average elapsed time between the supine and prone CT scans was approximately 10 min. Maximum care was taken to ensure that the second CT scan was taken at the same level as the first (supine). This was done by using skin landmarks and by further checking with radiographic examination. The diaphragm domes never appeared while patients were prone. This suggests that the possible cranial diaphragm displacement resulting from the position change was less than 1–2 cm. Ventilatory parameters (FIO₂, TV, RR, and PEEP) were kept constant in both positions, and the CT scans were taken at a constant PEEP level during apnea lasting 5 s. Five patients were maintained prone for 45 min, and gas exchange was repeatedly assessed to examine any time-related variations.

Because we did not detect any differences between CT scans taken immediately after turning the patient prone and CT scans taken 45 min later, only results of the first scan (taken approximately 10 min after the supine CT scan) are reported here. The CT scan was limited to one slice to avoid unnecessary x-ray exposures after we found, in the first patients, that the CT density redistribution phenomenon was also present at apical and hilar levels. Moreover, the CT number of the base slices is well correlated with the apical and hilar slices (see Appendix).

**Volunteers**

Seven normal adult subjects underwent a CT scan while in the supine position and seven others while prone. No sedation or paralysis was used, and the CT scan was performed with each subject at functional residual capacity at atmospheric pressure during apnea. This followed a period of 5 min of quiet air-breathing. We do not believe we introduced bias by avoiding double x-ray exposures in the same subject, since the CT densities are very reproducible within the normal subjects, showing a variation coefficient less than 5%.

**Physiologic Data**

All patients had intraarterial and pulmonary artery thermodilution catheters inserted. Arterial and mixed venous blood samples were analyzed for gas tensions and pH immediately after sampling. The right-to-left intrapulmonary shunt (Qp/Qs) was computed by the shunt equation (assuming a respiratory quotient of 1). Arterial and pulmonary artery pressures were measured with pressure transducers and recorded at end expiration. Zero level was set at the midaxillary line in both positions. Cardiac output was measured in triplicate by the thermodilution method, and the cardiac index (CI) was computed as the average of the three measurements.

**CT Scan Analysis**

The basic principles of quantitative CT scan analysis have been reported previously. Briefly, each voxel of the CT scan image (voxel dimension 1.5 × 1.5 × 0.9 mm) is characterized by a CT number, which, in Hounsfield units (H), ranges from +1,000 H (bone) to 0 H (water), and −1,000 H (air). The main determinant of the CT number is density. (For example, in the lung, −500 H characterizes a voxel comprising 50% gas and 50% tissue. "Tissue" here refers to lung structures, extravascular water, and blood.) This computation assumes that the relationship between density and CT numbers is linear, as actually has been found in isolated pig lungs.

Once the average CT number of a given volume has been determined, it is possible to compute the voxel gas volume and tissue mass. (Assuming a specific density of tissue of 1, the tissue volume in milliliters is equivalent to the tissue mass in grams.) In this study, lung height in each image was measured as the distance along a line from ventral to dorsal: each lung was divided into ten "levels" of lung height (being the height of each level 10% of the total height) from level 1 (ventral) to level 10 (dorsal). The following variables were computed from each CT section and each lung level (see Appendix for details):

1. Tissue mass: the mass of lung tissue (including lung structure, extravascular lung water, and blood).
2. Excess tissue mass: the percentage increase of lung mass as compared with the expected normal value.
3. Estimated superimposed pressure: the estimated pres-
sure exerted by gravity on each lung level by lung levels superimposed on in that position. The method of pressure computation (see Appendix) assumes that the lung behaves as a fluid.

4. Normally inflated tissue fraction: We defined this as representing the fraction of tissue mass with a near-normal gas–tissue ratio (i.e., between 90% gas/10% tissue and 50% gas/50% tissue).

**Statistical Analysis**

All data are expressed as mean ± 1 SD unless otherwise indicated. The least-squares method was used to perform linear regression analysis. Student’s *t* test, paired or unpaired, as appropriate, was used for comparison of two means. Bonferroni correction was applied for multiple comparisons. *P* < 0.05 was considered to be statistically significant.

**Results**

**Physiologic Parameters: Response to Positioning**

Table 1 summarizes the physiologic variables we recorded in the patients with ARF in both the prone and supine positions while keeping respiratory parameters unchanged (PEEP, FIO₂, TV, and RR). Neither gas exchange nor central or systemic hemodynamics showed any significant difference when body position was changed. In two patients, PaO₂ and QSP/QT greatly improved, whereas in two other patients PaO₂ and QSP/QT deteriorated slightly and in the remaining six PaO₂ and QSP/QT were unchanged.

**Analysis of the Entire CT Section**

Morphologic examples of density redistribution by gravity when changing from the supine to the prone position are shown in figure 1: the nondependent regions tended to clear, whereas the dependent regions increased their density in either position. Despite changes of the location of densities, the average density and the total amount of tissue mass were unchanged in the prone compared with the supine position. The average CT density in the supine position was −347 ± 154 H and in the prone position −336 ± 131 H (*P* = not significant [NS]; normal values −675 ± 22 H and −743 ± 38 H, respectively). The total tissue mass averaged 68 ± 21 g in the supine position and 60 ± 23 g in the prone position (*P* = NS); normal values were 30 ± 6.4 g in the supine position and 24 ± 4 g in the prone position (*P* = NS). All comparisons between patients and normal subjects were statistically significant (*P* < 0.01).

**Topographic Analysis of the CT Scan Section**

**CT Densities and Estimated Superimposed Pressure in the Supine and Prone Positions**

As shown in figure 2 (top), in normal subjects and patients with ARF in the supine position there was an increased CT density from nondependent (ventral) to dependent (dorsal) levels of the lung. The CT density was reversed when the subjects were turned to the prone position. In normal volunteers the change in CT scan density induced by the change to the prone position was statistically significant (*P* < 0.01) at each lung level except for levels 4–6. In the patients with ARF, the CT densities in the supine and prone positions were significantly different (*P* < 0.01) at each lung level except for levels 6 and 7.

The estimated superimposed pressure at various levels of the lung, in both the supine and prone positions, is shown in figure 2 (bottom). In the supine position the estimated superimposed pressure increased from ventral to dorsal, and the pressure gradients were reversed when the subjects were turned into the prone position. All of the differences of estimated superimposed pressure in each lung level induced by the position change were statistically significant (*P* < 0.01), with the exception of level 5 in normal subjects and level 6 in patients with ARF. It is worth emphasizing that in the most dependent levels of the ARF lungs (level 10 in supine position), the estimated superimposed pressure averaged 8.9 ± 2 cmH₂O (normal value, 3.8 ± 0.4 cmH₂O) (*P* < 0.01). In the prone position the estimated pressure superimposed on the most dependent lung regions (level 1 in prone position) averaged 9.4 ± 2.3 cmH₂O (normal value, 3.2 ± 0.3 cmH₂O) (*P* < 0.01).

**Table 1. Physiologic Parameters**

<table>
<thead>
<tr>
<th></th>
<th>P_{A\text{a}O_2} (mmHg)</th>
<th>P_{F\text{O}2} (mmHg)</th>
<th>Q_{SP}/Q_{T}</th>
<th>CI (1/min/m^2)</th>
<th>P_{AP} (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine</td>
<td>n = 10</td>
<td>67.5 ± 17.3</td>
<td>39 ± 6</td>
<td>0.42 ± 0.14</td>
<td>5 ± 1.3</td>
</tr>
<tr>
<td>Prone</td>
<td>n = 10</td>
<td>78 ± 30.7</td>
<td>39.8 ± 9</td>
<td>0.37 ± 0.17</td>
<td>4.4 ± 1.2</td>
</tr>
</tbody>
</table>

*P* = number of patients.
Fig. 1. CT scan images of two ARF patients in the supine (A1, B1) and prone (A2, B2) position. Patient A showed PaO₂ increased from 76 to 137 mmHg when prone (A2); patient B had an unchanged PaO₂ (68 mmHg). However, the CT density redistribution was similar for each patient.

Tissue Mass and the Excess Tissue Mass Distribution in the Supine and Prone Positions

Although the tissue mass of the CT section in patients with ARF was more than two times the normal mass, its distribution in the ten lung levels was similar to the distribution in normal subjects, both in the supine and prone positions (fig. 3). However, the tissue distribution of the supine and prone positions was different, as can be seen by comparing the same lung levels in figure 3. In normal subjects and in ARF patients in the prone position, the tissue content was significantly increased in ventral lung levels as compared with that in the supine position (levels 1–5) \( (P < 0.01) \), whereas it was significantly decreased \( (P < 0.01) \) in dorsal levels (levels 7–10). The excess tissue mass \( (i.e., \) the amount of tissue in excess of normal; see Appendix) was independent of body position, as shown in figure 4, which clearly indicates that the excess tissue mass did not change with gravity, all values being similar in the same lung levels in both positions.

Effects of Body Positioning on Regional Lung Inflation

The modifications of fractions of normally inflated tissue induced by the prone position are shown in figure 5.
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Fig. 3. Tissue mass distribution as a function of lung level (mean ± 1 SE) in supine position (top) and in prone position (bottom). The white columns refer to the normal subjects, and the shaded columns to the ARF patients. No significant differences were found at any level between normal subjects and ARF patients in both positions. The distribution of the tissue mass was significantly different in the supine and prone position in each level (except level 6) in both normal subjects and ARF patients.

We found significant differences of inflation in each lung level except level 6.

The relationship between changes of regional lung inflation at each lung level and the changes of estimated superimposed pressure at the same lung level when patients are shifted from supine to prone is shown in figure 6. A similar and significant relationship for regional inflation was observed in the normal subjects \(r = 0.60, P < 0.01\) and is not shown in the figure, for clarity.

In figure 6 it can be seen that when the same lung level was compressed (by an increased superimposed pressure), regional inflation decreased (the fraction of normally inflated tissue became negative). On the other hand, when a lung level was decompressed (decreased superimposed pressure), regional inflation increased (the fraction of normally inflated lung became positive). When the pressure superimposed on a lung level did not change with position reversal (occurring roughly at levels 5 and 6), regional inflation did not change (Δfraction = 0).

**Discussion**

The primary morphologic finding of this study was a dramatic redistribution of CT densities from the dorsal to the ventral levels of the lung when body position was changed from supine to prone. The quantitative analysis of CT densities clearly indicates that this phenomenon is also present in normal subjects.

**NORMAL SUBJECTS**

The regional increase of density along the vertical axis may result from a decreased regional gas volume and/or an increased tissue volume. In normal subjects the influence of gravity on regional gas volume (and alveolar size) has been clearly demonstrated either in awake or

Fig. 4. Excess tissue mass of the ARF patients as a function of lung level (mean ± 1 SE). No significant differences were found between the supine and prone position at any level.

Fig. 5. Percentage of normally inflated tissue as a function of lung level in ARF patients (mean ± 1 SE). The differences at each level between the supine and prone position were significant (except at level 6). The values of normally inflated tissue of normal subjects (not shown) ranged between 0.99 and 0.84. All of the values of normal subjects were statistically different from those of ARF patients \(P < 0.01\).
anesthetized and paralyzed humans\textsuperscript{12} and laboratory animals.\textsuperscript{15,16} The decrease of regional gas volume is likely to result from a decrease in distending (transpulmonary) pressure because of a regional decrease in pleural pressure (pressure becoming less negative) along the vertical gradient.\textsuperscript{17} The pleural pressure gradient is reported to vary between 0.2–0.25 and 0.3 cmH\textsubscript{2}O/cm.\textsuperscript{13,17,18}

The vertical gradient is consistent with the concept that the lung acts as a fluid\textsuperscript{19} with the tension transmitted through the parenchyma as in a liquid with an average density between 0.2 and 0.3 g/cm\textsuperscript{3}.\textsuperscript{20} In our normal subjects we did not measure pleural pressure; however, we estimated the superimposed pressure gradients, which, in the model of a fluid lung, are the determinants of the pleural pressure gradient. The average pressure gradient we estimated by CT scan in our normal subjects was 0.3 ± 0.05 cmH\textsubscript{2}O/cm,\textsuperscript{13,17,18} which is consistent with the pleural pressure gradient reported in the literature.\textsuperscript{13,17,18} In patients in the prone position, the pressure gradients were reversed, and the consequent changes of regional gas volume, as previously observed in normal subjects,\textsuperscript{14} are the most likely explanation of the CT density redistribution we observed in normal subjects. However, a contribution to the increased density of dependent lung levels may also result from gravitational distribution of the interstitial water and intravascular blood and from the anatomic configuration of the lung.\textsuperscript{16}

In normal subjects, the regional differences of lung tissue mass distribution we found between the prone and supine positions (see fig. 3) may result from several factors, including changes of geometry of the rib cage, possible opposition of mediastinal organs,\textsuperscript{20} and the lung's weight.

In summary, in normal subjects the regional inflation decreases and the tissue content increases in levels that become dependent. This normal behavior, which takes into account all the physiologic factors leading to CT density redistribution in normal subjects, represents our reference model with which we compared the patients with ARF. The limit of this comparison, however, may be the awake status of the normal subjects versus the anesthesia and paralysis status of the patients with ARF.

\section*{Patients with ARF}

The main difference between normal subjects and patients with ARF is the increased tissue mass, with consequently increased average density and increased average estimated superimposed pressure, in ARF. It is reasonable to hypothesize that the early increase in tissue mass primarily results from edema (intraalveolar and extralveolar) and blood, which may be described as "excess mass" for comparison with normal lung. The CT density redistribution when changing the position of patients with ARF could theoretically result only from one or more of the following mechanisms: 1) development of new edema in dependent lung levels and reabsorption of intraalveolar edema in nondependent lung levels; 2) free movement of interstitial fluid through various lung levels driven by gravity; 3) pulmonary blood redistribution resulting from gravity; and 4) alveolar collapse and/or alveolar size reduction in the dependent lung levels and alveolar reopening in nondependent lung levels, in response to variations of superimposed pressure.

We can exclude the first hypothesis because of the brevity of the period in which redistribution occurs. It has been shown that reabsorption of saline from alveoli requires hours and reabsorption of protein-enriched saline requires days.\textsuperscript{21} Moreover, if edema develops according to a dependent mechanism, we should find a gravitational distribution of excess mass or, at least, a differing excess mass in dependent and nondependent lung levels when changing position, and this was not the case in our patients (fig. 4).

Free fluid movement through the interstitium from dorsal to ventral levels when the patient's position is

\textsuperscript{**} Pressure at a given point of a fluid column would equal the product of the height of the column above that point times the density of the fluid above that point, i.e., \( P = gh \).

\textsuperscript{††} The gradient 0.3 ± 0.05 cmH\textsubscript{2}O is the average gradient; the actual gradients are lower in nondependent levels and greater in dependent levels because density is not constant (see fig. 2).
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changed is also unlikely to result from physical obstructions (e.g., interstitial matrix, gels, or organized edema). Moreover, if interstitial edema were to migrate, in patients with ARF we would expect a tissue mass distribution differing from that in normal subjects, as well as a gravitational distribution of excess tissue mass. This was not the case for our patients, nor was it found in patients with interstitial edema of cardiac origin (presumably at a lower protein concentration). A nongravitational distribution of the excess tissue mass, on the other hand, is not surprising; nongravitational patterns of edema distribution were reported in experimental studies of both hydrostatic and inflammatory edema.

We cannot exclude shifts of intravascular volume with position changes; however, the changes in GT density are too great to be explained only by a blood volume shift. Moreover, during acute lung edema, the fractional blood volume is apparently decreased in dependent regions of the lung, and the pulmonary flow to those regions is reduced.

We believe that the most likely explanation for the density redistribution is a redistribution of intrapulmonary gas, through the same mechanism that operates in normal subjects (i.e., resulting from changes of pressure gradients). Although in normal subjects the decrease of the transpulmonary pressure along the vertical axis only reduces alveolar size, in patients with ARF, in whom gravitational pressure gradients are greater (because of the increased tissue mass) and overall gas content is reduced, the decreased transpulmonary pressure may also induce collapse of potentially recruitable lung units. This hypothesis is supported by studies in normal subjects in which the increase of lung weight (and consequently of pressure gradients) was obtained by forward acceleration. These studies clearly demonstrated a correlation between the forces of gravity applied and the pressure gradients, with consequent collapse of dependent lung regions.

We believe that the ARF lung, at least in early acute lung disease, mimics an elastic sponge: excess tissue mass, resulting from inflammatory pulmonary edema, is distributed in all lung regions. This elastic body is subjected to the gravitational field plus the superimposed pressure of edematous lung and thereby causes compression atelectasis of dependent lung levels. When the gravitational force is applied in the opposite direction (by turning the patient from supine to prone), decompressed atelectatic regions reopen while new regions of compression atelectasis develop.

This simple lung model fits the data of figure 6, wherein we observed a close correlation between changes of superimposed lung pressure and changes of regional inflation. Thus, it is not surprising that we did not find any significant change of mean PaO₂ or QPV/Qtot in our patients, because inflation of dorsal lung levels in the prone position was counterbalanced by deflation of ventral levels, the average overall inflation remaining unchanged. This implies that, in these patients with ARF, the average quantity of regional perfusion was unchanged in both positions. In other patients with ARF, variations of regional lung perfusion as well as a possible drainage of fluid from the airways may account for the variability of response in oxygenation that has been observed during positional changes of both humans and experimental animals.

In this series of patients, however, we did not observe any increase in secretions when changing patients' positions.

No long-term studies have examined the clinical usefulness of the prone position in ARF. One study has shown a decreased length of time in ICU achieved through continuous turning of the patient, and the prone position has also become a recommended treatment during ARF. However, the possible disadvantages should be stressed. The prone position uniformly induced radiographic clearing of the dorsal regions of the lung, promoting drainage of secretions and possibly preventing consolidation and superinfection. Unfortunately, when the prone position was used, ventral lung regions that were previously inflated collapsed. In the future, long-term clinical studies should be performed to clarify the impact of body position change on the clinical course and outcome of ARF.

Appendix

We chose to study only one CT slice to avoid unnecessary x-ray exposures, because we found, in a previous series of 22 patients for whom three slices were studied (apex, hilum, and base), an acceptable correlation between the CT numbers of the bases and the average CT number of the three slices.

The regression equation was as follows:

\[
\text{CT number of base} = 56 \text{ H} + 1.1 \text{ CT (apex + hilum + base)} (r = 0.86, P < 0.001)
\]

(1)

CT Scanner

We used a Pfizer (Columbia, MD) ASE0450® CT scanner. The exposures were taken at 120 kV, 50 mA, and 5 s. The slice thickness was 9 mm, and the dimensions of the pixel of the reconstruction matrix were 1.5 × 1.5 mm. The system was calibrated with a suitable phantom. It is important to emphasize that the size of the discriminant unit of the CT scan (voxel) is approximately half of a pulmonary acinus.

Analysis of the Entire CT Scan Slice

Lung parenchyma (right and left lung separately) were contoured, the total area measured, and the average CT number computed. The tissue mass of the whole section was derived with the following equation:

\[ \text{Tissue mass} \times \frac{1}{1 - \frac{\text{CT}}{-1,000}} \]  

where the total slice volume is the section area multiplied by the section thickness (9 mm). The CT number frequency distribution of the total slice area was computed from the 11 equally spaced 100 H step compartments. We defined the cumulative CT number frequency from -1,000 H to -500 H as the fraction of normally inflated lung tissue. This kind of analysis, previously applied to the entire lung,10,11 does not provide information on the topographic location of abnormalities.

It is important to point out that the tissue mass includes every solid and liquid component of the lung (e.g., edema, blood, bronchial and vessels walls, lung cells, matrix). The "consolidated" regions of the lung, as, for example, pneumonia foci, and infarcts, cannot be discriminated directly from atelectasis and edema. However, it is likely that the consolidated lesions are "fixed" and do not change position when the body position is changed.

Topographic Analysis of the CT Scan Section

For each lung, we constructed a series of rectangular strips of fixed height (7.5 mm), fixed thickness (9 mm), and variable length (from 7.5 to 112.5 mm) according to lung size, as shown in figure 7. The primary information we determined for each strip included strip volume, average CT number, and frequency distribution of the CT numbers. As seen in figure 7, the strips did not cover the entire surface of the lung, but their sum accounted for 82 ± 6% of the entire section. We corrected the strip volume according to the following equation:

\[ \text{Corrected strip volume} = \frac{\text{measured strip volume}}{\text{sum of measured strip volumes}} \times \text{total section volume} \]

where the total section volume was the whole section area measured from the contour, multiplied by section thickness (9 mm).

We assumed that the average CT number and the CT frequency of each measured strip were similar to the corrected strip, and we computed, for each strip, the lung tissue mass and the fraction of normally inflated tissue, according to the procedure previously described for the whole section.

Each patient had different numbers of strips because the total dorsal-ventral height of the lungs varied from 160 to 250 mm (average 210 ± 20 mm). To compare different patients we grouped, for every lung, the strips contained in each lung level (each level constituting 10% of the dorsal-ventral height), as shown in figure 7. If a strip, by chance, was located across a percentage line, this computation was done by dividing that strip into ten parts and weighing its contribution to the level above or below the percentage line (considering each strip to be homogeneous).

With the above-described procedure and with patients in the supine and prone positions, we obtained for each lung the topographic distribution (at each lung level of the average CT number), the total tissue mass, and the fraction of normally inflated tissue.

Other Derived Variables

**Estimated Superimposed Pressure.** The estimated pressure \((P)\) of each strip was computed as follows:

\[ P = \left(1 - \frac{\text{CT}}{-1,000}\right) \times h \]

where \(h\) is the height of the individual strip.

For example, if the average CT number of a given strip is -500 H (30% gas and 70% tissue), the estimated pressure is 0.525 cmH\(_2\)O if the vertical height of the strip is 7.5 mm. This computation assumes that the lung acts as a fluid with the tensions transmitted through the parenchyma as if in a liquid. The "liquid" density is computed by the CT scan. The pressure in a given point of this fluid column equals the product of the height of the column above that point multiplied by the density above that point. Indeed, the estimated superimposed pressure at level 10 equals the summation of the pressures of levels 1 to 10.
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Expected Lung Tissue Mass. The expected lung tissue mass in each level, both in the prone and supine positions, was obtained by substituting, in general equation 1 (above), the CT number we obtained from normal subjects at the same lung level and in the same position (prone or supine). The expected tissue mass represents an estimate of the tissue mass that should be present at that lung level and body position if the patient were normal.

Excess Lung Tissue Mass. The excess lung tissue mass was computed according to the following equation:

\[
\text{Excess lung tissue mass} = \frac{\text{actual tissue mass} - \text{expected tissue mass}}{\text{expected tissue mass}}
\]

and expressed as a percentage.

An excess tissue mass of 100% would indicate that the actual tissue mass, in that particular lung level, is twice that expected.

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References