Pulmonary Densities during Anesthesia with Muscular Relaxation—A Proposal of Atelectasis

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Twenty patients (23-76 yr) were studied with regard to lung tissue changes prior to and following induction of general anesthesia with muscular relaxation, and another four subjects were studied for a longer period awake. The transverse thoracic area and the structure of the lung tissue were determined by computerized tomography. No abnormalities in the lung tissue were noted before anesthesia. Within 5 min after induction, including muscular relaxation, all subjects had developed crescent-shaped changes of increased density in the dependent regions of both lungs. They were largest in the most caudal segment (4.8 ± 0.8% of the transverse lung area, mean ± SE) and smaller in the cephalad exposures (3.4 ± 0.7% of the transverse area). The size of the densities showed no correlation to age. The densities did not increase after a further 20 min of anesthesia and were not affected by the inspiratory oxygen fraction. When the subjects were moved from the supine to the lateral position, the crescent-shaped densities disappeared in the nondependent lung and remained in the dorsal part of the dependent lung. The application of positive end-expiratory pressure of 10 cmH₂O eliminated or reduced the densities. The four awake subjects showed no lung densities after 90 min in the supine position. It is suggested that these crescent-shaped densities represent atelectases, which develop by compression of lung tissue rather than by resorption of gas. (Key words: Lung; airway closure; atelectasis; compression.)

GENERAL ANESTHESIA and mechanical ventilation causes a reduction of the resting lung volume (functional residual capacity, FRC) by 0.4-0.7 l,1-6 as a consequence of cephalad displacement of the diaphragm7,8 and a reduced transverse cross-sectional area of the thorax.8 Concomitantly, there is a displacement of about 0.3 l of blood from the thorax to the abdomen.5 The thoracic volume thus is reduced by as much as 0.75 l,6 a reduction that may be assumed to have effects on the lung tissue.

Indeed, before it became known that FRC was reduced during general anesthesia, Bendixen and co-workers9 suggested that during general anesthesia with controlled ventilation, oxygenation was impaired by the development of miliary atelectasis. This concept has been challenged by others,10,11 and conventional radiography during anesthesia failed to reveal any atelectasis. Since these studies, the technique of whole body computerized tomography (CT) has been introduced, offering new opportunities for detection of changes within the lung tissue. The present study was undertaken with the aim of determining whether induction and maintenance of general anesthesia with muscular relaxation and mechanical ventilation had any effects on the lung tissue. The observations were related to age, duration of anesthesia, airway closure, inspired oxygen fraction, body position, and positive end-expiratory pressure (PEEP).

Materials and Methods

The studies were performed on 24 subjects (15 men and nine women). Twenty of them were patients scheduled for abdominal surgery, requiring general anesthesia and intubation. All were free from cardiopulmonary disease as judged by clinical examination, chest roentgenograms, and electrocardiography. Their ages varied from 23 to 76 yr, with a mean of 50 yr. They had a normal body constitution, with a mean height of 169 cm (range 147-186 cm) and a mean weight of 71 kg (range 52-90 kg). Seven patients were smokers. All patients gave their informed consent to the study. In addition, four healthy, awake volunteers (authors of this paper) ranging in age from 29 to 43 yr and with normal heights and weights were studied. The investigation plan was approved by the Ethics Committee of Huddinge Hospital.

Anesthesia

Premedication was given on request to five patients, about 2 h before the preanesthesia measurements. Two patients received pethidine, 50 mg im, and three patients

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oxycodeine, 10 mg, and scopolamine, 0.4 mg, im. The other 15 patients were given no premedication. All patients received 0.25 mg of atropine intravenously approximately 10 min before the preanesthesia measurements. Anesthesia was induced with thiopentine, 5.6 ± 0.4 mg·kg⁻¹, and muscular relaxation was achieved with pancuronium bromide, 0.1 mg·kg⁻¹. Tracheal intubation was performed after preoxygenation. Anesthesia then was maintained with 0.5–1.0% inspired halothane, in oxygen/nitrogen (2:3) (n = 7) or in pure oxygen (n = 13). The patients were ventilated mechanically at a rate of 12 breaths/min with a Servo 900B Ventilator® equipped with a carbon dioxide analyzer (Siemens Elema, Stockholm, Sweden). Minute ventilation was adjusted to maintain the end-tidal carbon dioxide concentration at 4%. A heat and moisture exchanger was attached between the tracheal tube and the Y-piece of the respirator tubings.

CHEST TOMOGRAPHY

The transverse lung area and the structure and density of the lungs were studied by CT scanning. The subject lay supine on a tomograph table (Siemens Somatom 2®). The arms were stretched cranially to allow the subject to be moved into the aperture of the tomograph. A frontal scout view covering the chest and the abdomen was obtained initially. Three CT slices in the transverse plane then were exposed, the lowermost at a level just above the top of the diaphragm. In the first seven subjects, exposures were made 5 and 10 cm cephalad to the first one. In most cases no changes were found in the most cephalad projection, and therefore the exposures were made 2.5 and 5 cm cephalad to the first one in the following 17 subjects. The exposure data were as follows: 115 mAs, exposure time 5 s, slice thickness 8 mm.

From the images thus obtained, the transverse areas of the thorax were calculated planimetrically using the computer connected to the tomograph. The area of each hemithorax was calculated separately by drawing a sagittal line through the middle of the mediastinum. The external boundary was drawn along the inside of the ribs at a location corresponding to the pleural space. Thus, the transverse areas of the chest did not include any tissue outside or between the ribs, but they did include the mediastinal organs.

The shape and location of a region of contrasting density were determined. A boundary was drawn around the densities, and the area was determined in absolute values (cm²). The area also was calculated as a fraction of the hemithorax area at that level. By multiplying the mean of the tomographic transverse areas by the height between them, the volume corresponding to the pulmonary density within the segment could be estimated.

Intravenous contrast injections (Isoopaque Cerebral® 1 ml·kg⁻¹) was given in order to see if the lung densities were perfused by blood. The contrast was given as a bolus injection into a peripheral vein, and an exposure was made 30 s later. Variations of the attenuation (absorption of x-ray radiation in the tissues) in the lung densities, in the aorta, and in subcutaneous fat prior to and after contrast injection were analyzed on the computer. The density was measured using a linear scale of attenuation units ranging from −1,000 (air) to +1,000 (bone). This enables a qualitative description of tissue perfusion but does not permit a quantitative analysis.

AIRWAY CLOSURE

Measurements of airway closure were performed by single-breath nitrogen washout in six patients during anesthesia. The lungs were inflated with oxygen from the resting position to an airway pressure of +45 cmH₂O (corresponding to an alveolar pressure at approximately 40 cmH₂O) by means of a 7-l syringe connected to a water-sealed spirometer (Bernstein) equipped with an electric output. The lungs then were deflated to a tracheal pressure of −20 cmH₂O. This procedure conforms with the guidelines of Leith. The nitrogen concentration in the expired gas was measured by a mass spectrometer (MGA 200®, Centronic) and the volume by the spirometer. Corrections were made for gas compression and phase shift between the nitrogen and volume signals. Under each condition, measurements were repeated until at least two acceptable curves had been obtained, i.e., until the inspiratory and expiratory flow rates had been maintained at 0.4–0.5 l, and the inspiratory and expiratory maximum volumes did not differ by more than 5%.

Statistics. Mean values ± SE were calculated. The significance of differences between results was tested by Student’s t test. Unpaired tests were used for comparisons between groups and paired tests for comparisons within groups. Correlation between variables was tested with linear regression analysis.

PROCEDURE

In the patients, measurements were performed in the supine position prior to and 5 min after induction of anesthesia with muscular relaxation. They all had been in the supine position for 10–20 min before the preanesthesia measurement. In five patients, measurements were repeated after 25 min of anesthesia. The effect of changing from the supine to the lateral position was studied in four patients. In another three patients, the effect of 5 min of PEEP of 10 cmH₂O was examined. Contrast was injected in four patients during anesthesia. They did not participate in other parts of the study. All measurements during anesthesia were carried out before
surgery. In the four healthy volunteers, measurements were made when they were awake and lay supine and breathed air for 1 h and subsequently oxygen for 30 min.

**Results**

**Awake—Anesthetized**

In the awake patients, no abnormalities in the lung tissue were observed, either from the frontal image or from the three transverse images.

After 5 min of anesthesia, all patients had developed crest-shaped changes of increased density in the dependent regions of both lungs (fig. 1). They appeared largest in the most caudal segment and smallest in the most cephalad one. In seven patients the most cephalad exposure was made 10 cm from the diaphragm. In this projection, one patient showed bilateral minimal changes, two patients showed unilateral minimal changes, and the other four subjects showed no changes at all. The frontal image revealed no changes in the lung tissue. The pulmonary densities had a volume of 36.2 ml (4.1%) and 36.5 ml (4.0%) of the lowermost 5.4 cm segment of the right and left hemithorax, respectively (including the CT slice thickness) (table 1).

**Age and Airway Closure**

An analysis of the possible relationship between the pulmonary densities and age showed no correlation between these variables.

Airway closure occurred at a mean of 0.27 ± 0.04 l above FRC in all six patients in whom airway closure measurements were made during anesthesia. However, one patient, the oldest studied, displayed airway closure above the tidal volume, suggestive of continuous airway closure and closed-off lung regions.

**Inspired Oxygen Fraction and Time**

To determine the question whether gas resorption played any role in producing the crests (cf., results concerning airway closure above), seven patients were given 40% oxygen in nitrogen and nine patients 99% oxygen as the inspiratory gas (plus 0.5–1.0% halothane). The two groups were of comparative ages. There was no difference in the size of the crest-shaped densities after 5 min of anesthesia between the two groups (table 2). Possible time dependence of these changes also was studied in five patients by performing the tomography again after 25 min of anesthesia with 99% oxygen, halothane, and muscular relaxation. The pulmonary density

| Table 1. Mean Transverse Areas (±SE) of Crest-shaped Pulmonary Densities in Two CT-scan Slices of the Thorax (The Transverse Areas of the Corresponding Right and Left Hemithoracic Segments also Are Given. Measurements Performed after 5 Min of Anesthesia in the Supine Position [n = 16]) |
|---------------------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|
| **Slice 1 (At Top of Diaphragm)** | **Slice 2 (5 cm Cephalad to 1)** | **Segment between Slices I and II** |
| Total Area (cm²) | Density Area (cm²) | Relative Density Area (%) | Total Area (cm²) | Density Area (cm²) | Relative Density Area (%) | Relative Density Volume (%) |
| Right hemithorax | 174.2 ± 7.5 | 8.2 ± 2.4 | 4.8 ± 0.8 | 153.0 ± 7.6 | 5.2 ± 1.0 | 3.4 ± 0.7 | 4.1 ± 0.7 |
| Left hemithorax | 178.1 ± 7.4 | 8.3 ± 1.4 | 4.7 ± 0.8 | 161.0 ± 7.3 | 5.2 ± 1.0 | 3.3 ± 0.7 | 4.0 ± 0.7 |
changes did not increase in size or density after 25 min of anesthesia (table 3).

Time dependence and oxygen dependence of possible changes in lung structure in awake subjects were studied in four healthy volunteers. One transverse CT slice, just cephalad to the diaphragm, was exposed immediately after the subject had been placed in the supine posture and, again, after 1 h of air breathing. The subject, still supine, then breathed 100% oxygen for 30 min, and at the end of this period a third corresponding CT scan was obtained. None of these subjects displayed any abnormalities in the lung tissue at the first measurements, and no changes were noted after 1 h of air breathing or an additional half hour on oxygen. Thus, neither any time-dependent changes nor any changes dependent on oxygen were observed in spontaneously breathing, awake subjects.

**Supine-Lateral Position**

In four patients, measurements were made after 5 min of anesthesia with halothane in 99% oxygen in the supine position and immediately after repositioning the patient to the right lateral position. In the supine position, all four patients displayed changes in the dependent regions of both lungs, as described above. Repeated measurements immediately after altering the position of the patient to right lateral showed that the densities in the right lung persisted within the dorsal region, although this region was no longer the most dependent one. The previous changes in the left lung, which was now the upper one, had diminished or disappeared (table 4, fig. 2). After another 10 min of anesthesia in the lateral position, the changes that previously had been observed in the lowermost part of the lung, corresponding to the dorsal region, had disappeared or diminished. On the other hand, new crest-shaped densities now were noted in the newly dependent region of the dependent lung in three of the four cases.

**Positive End-Expiratory Pressure**

The above results were obtained at the resting lung level with zero end-expiratory airway pressure. In three patients a PEEP of 10 cmH₂O was applied. After 5 min of ventilation with PEEP, the crest-shaped densities had diminished or disappeared. After a further 10 min of ventilation with zero end-expiratory pressure, they had returned (table 5, fig. 3). The findings were similar in all three patients.

**Contrast Injections**

The injection of contrast caused an increased attenuation in the lung densities of 37 attenuation units on average. In dorsal parts of the lung close to the densities, the increase was 21 units and in the most ventral parts it was 14 units. In the aorta the increase was 78 units, and in subcutaneous fat it was 10 units. The increase of attenuation is caused by the contrast in the blood vessels and thus suggests that the tissues, including the pulmonary densities, are perfused.

**Discussion**

In this CT study it was found that on induction of general anesthesia with muscular relaxation and mechanical ventilation, crest-shaped changes of increased density rapidly developed in the dependent lung regions. They did not progress with time nor when the oxygen
fraction in the inspired gas was increased. When the patient was turned on his side, the densities disappeared in the nondependent lung, but they persisted within the dorsal (no longer the most dependent) region in the dependent lung. The densities could be eliminated or reduced by increasing the lung volume by the application of PEEP, and they reappeared within 10 min after discontinuation of PEEP. These observations suggest (but do not prove) that the crest-shaped changes represent atelectatic regions, the increased density being

![Image 2](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931412/ on 01/10/2019)

![Image 3](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931412/ on 01/10/2019)

**Fig. 2.** Transverse CT scans of the thorax during anesthesia with the patient supine (upper panel) and then turned onto his right side (lower panel). Note that in the latter position the densities have disappeared from the upper lung.

**Table 5.** The Effect of Positive End-expiratory Pressure on Pulmonary Densities (Relative Volume (%) of Corresponding Hemithoracic Segments) *(n = 3)*

<table>
<thead>
<tr>
<th>Patient</th>
<th>End-expiratory Pressure</th>
<th>0 cm H2O (10 min)</th>
<th>10 cm H2O (5 min)</th>
<th>0 cm H2O (10 min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right hemithorax</td>
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<td>0.3</td>
<td>1.1</td>
</tr>
<tr>
<td></td>
<td>F</td>
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<td>10.7</td>
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<tr>
<td></td>
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</tbody>
</table>

**Fig. 3.** Transverse CT scans of the thorax during anesthesia without and with a positive end-expiratory pressure (PEEP) of 10 cm H2O (upper and middle panels, respectively) and 10 min after discontinuation of PEEP (lower panel). Note the disappearance of densities in dependent regions during PEEP and their reappearance after discontinuation of PEEP.
attributed to loss of gas-filled spaces and subsequent collapse of lung tissue. A conceivable alternative explanation could be fluid accumulation in the pleural space or in the lung. However, pleural fluid would not have remained in the same lung area on rotation of the patient but would have moved to the new dependent region immediately. The results of the contrast injections where the densities increased their attenuation considerably more than normal lung tissue but less than blood in the aorta will support the idea that the densities include perfused lung tissue. Fluid accumulation in the lung tissue (or in the pleural space) hardly would result in an increased density to such an extent. The diminution of the crests on inflation of the lung by PEEP also would support the idea of atelectasis as the cause of the lung tissue changes. This observation must be interpreted with caution, however. The increase in pulmonary pressure most likely reduces the intrathoracic blood volume, concomitantly forcing blood to dependent lung regions. The net effect may be expected to be a decreased blood volume in dependent lung regions.

The amount of lung tissue involved in the proposed atelectatic process was approximately 36 ml in the lung segment that was studied. Since the densities continued into the more cephalad segments, the total volume can be roughly estimated to 50 ml in each lung. Since these volumes constitute nonexpanded lung tissue, their inflation by air should result in much larger volume. This volume cannot be predicted with any precision. The average tissue volume constitutes about one-tenth of the total lung volume, but the dependent regions will be less inflated than the average.

The rapid development of the densities on induction of anesthesia, the lack of dependence on the inspired oxygen fraction, and the absence of continuous airway closure (except in one patient) make it unlikely that they represent reabsorption atelectasis. Rather, the prompt occurrence of the densities raises the question whether they are produced by compression, gas being expelled from alveoli and airways by supernormal external pressure. Rehder et al. have shown that there is an increased gradient in regional lung volume from the top to bottom of the lung during anesthesia, fitting in with an increase in the vertical pleural pressure gradient from the awake value of 0.2–0.4 cmH2O/cm vertical distance to approximately 1 cmH2O/cm. Further support for a higher pleural pressure gradient also has been obtained by the construction of separate pressure–volume curves of the lungs in the lateral position during anesthesia. Interestingly, an increased vertical pleural pressure gradient has been observed on relaxation of the diaphragm from a voluntarily tense state in awake subjects. Further diaphragmatic relaxation may result in a further increase in the pleural pressure gradient. Such relaxation appears likely during anesthesia, as indicated by the cephalad shift of the diaphragm. The vertical abdominal pressure gradient can be assumed to be around 1 cmH2O/cm, and on complete relaxation of the diaphragm this gradient may be transmitted to the thoracic space.

The decrease in lung compliance that occurs shortly after induction of anesthesia also might, at least in part, be explained by the development of atelectasis. The possible contribution of atelectasis to this decrease is difficult to evaluate, however. The simultaneous events of atelectasis, reduction of transverse chest areas, and cephalad shift of the diaphragm, all will have an impact on the lung volume, making an individual analysis of a single factor impossible.

In conclusion, the present study has demonstrated densities in dependent lung regions during general anesthesia with muscle paralysis. They appeared early during anesthesia in all patients irrespective of age and could be reduced or eliminated by PEEP. The densities did not increase in size with time, and there was no difference whether the patients were ventilated with 40 or 99% oxygen. It is suggested that the densities represent compression atelectasis. To what extent this phenomenon affects gas exchange and whether it leads to postoperative atelectasis remains to be speculated at present.

References

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