RE-EXPANSION OF ATELECTASIS DURING GENERAL ANAESTHESIA: A COMPUTED TOMOGRAPHY STUDY

H. U. ROTHEN, B. SPORRE, G. ENGBERG, G. WEGENIUS AND G. HEDENSTIERNA

SUMMARY

Formation of atelectasis is one mechanism of impaired gas exchange during general anaesthesia. We have studied manoeuvres to re-expand such atelectasis in 16 consecutive, anaesthetized adults with healthy lungs. In group 1 (10 patients), the lungs were inflated stepwise to an airway pressure (Paw) of 10, 20, 30 and 40 cm H$_2$O. In group 2 (six patients), three repeated inflations up to Paw = 30 cm H$_2$O were followed by one inflation to 40 cm H$_2$O. Atelectasis was assessed by analysis of computed x-ray tomography (CT). In group 1 the mean area of atelectasis in the CT scan at the level of the right diaphragm was 6.4 cm$^2$ at Paw = 0 cm H$_2$O, 5.9 cm$^2$ at 20 cm H$_2$O, 3.5 cm$^2$ at 30 cm H$_2$O and 0.8 cm$^2$ at 40 cm H$_2$O. A Paw of 20 cm H$_2$O corresponds approximately to inflation with twice the tidal volume. In group 2 the mean area of atelectasis was 9.0 cm$^2$ at Paw = 0 cm H$_2$O and 4.2 cm$^2$ after the first inflation to 30 cm H$_2$O. Repeated inflations did not add to re-expansion of atelectasis. The final inflation (Paw = 40 cm H$_2$O) virtually eliminated the atelectasis. We conclude that, after induction of anaesthesia, the amount of atelectasis was not reduced by inflation of the lungs with a conventional tidal volume or with a double tidal volume ("sigh"). An inflation to vital capacity (Paw = 40 cm H$_2$O) however, re-expanded virtually all atelectatic lung tissue. (Br. J. Anaesth. 1993; 71: 788-795)

KEY WORDS


It is well known that general anaesthesia impairs pulmonary gas exchange [1]. Much effort has been spent in analysing possible mechanisms, and 30 years ago the concept of progressive alveolar collapse during general anaesthesia with mechanical ventilation was put forward by Bendixen, Hedley-Whyte and Laver [2]. Atelectasis has been demonstrated in more recent studies, although the mechanism appears to differ from that which had been suggested initially. Thus, loss of respiratory muscle tone causing reduction in functional residual capacity and compression of dependent parts of the lungs was proposed, rather than absorption of gas behind closed airways [3]. A correlation has been shown between atelectasis and the magnitude of shunt [4, 5]. It is not known if the amount of atelectasis may be reduced by passive inflations of the lungs and at what extent of inflation this occurs.

The purpose of this study was to estimate, in anaesthetized subjects with healthy lungs, the change in the amount of atelectasis during inflations of the lungs up to a volume equal or close to vital capacity.

PATIENTS AND METHODS

Informed consent was obtained from each patient and the study was approved by the Ethics Committee of the University Hospital of Uppsala. We studied 16 consecutive patients (age range 28-61 yr; seven female) (table 1) undergoing elective neurosurgical procedures or laparoscopic surgery. No patient had cardiac or pulmonary disease. Patients were allocated to group 1 (recruitment manoeuvre by stepwise inflation) or group 2 (recruitment manoeuvre by repeated, similar inflations).

Anaesthesia

No premedication was given. Anaesthesia was induced with propofol 2 mg kg$^{-1}$ and fentanyl 1-2 µg kg$^{-1}$ i.v., followed by a continuous infusion of propofol 3-5 mg kg$^{-1}$ h$^{-1}$. Patients received pancuronium 0.1 mg kg$^{-1}$ to facilitate tracheal intubation; additional doses of 1-2 mg were given when needed. The lungs were ventilated mechanically at a rate of 10 b.p.m. with 60% nitrogen in oxygen (Servo Ventilator 900D or 710, Siemens). The end-inspiratory pause was set at 0.6 s and the end-expiratory pressure was 0 cm H$_2$O (= ZEEP). Total ventilation was adjusted to maintain an end-expiratory carbon dioxide concentration of approximately 4% (CO$_2$ Analyzer Eliza, Engström). Ventilatory volumes and airway pressures were obtained from the ventilator. The total dynamic compliance of the respiratory system (Crs) was calculated as tidal volume divided by end-inspiratory airway pressure.

Computed tomography of the chest

The densities of the lungs were studied by computed tomography of the lungs (Somatom plus, Siemens). The subjects were in a supine position

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with the arms raised above the head. An antero-posterior scout view covering the chest was obtained at end-expiration, awake and after induction of anaesthesia. For each subsequent analysis, one CT scan in the transverse plane was performed at end-expiration, 0–1 cm above the top of the right diaphragm. In group 1, a second scan was taken 5 cm cranially. The scan time was 1 s, at 225 mA and 137 kV with a slice thickness of 8 mm. Because the position of the diaphragm may vary between awake and anaesthesia, the scan levels relative to the spine were not identical.

To obtain slices from approximately the same lung region during the various inflations of the lungs, the scan was moved caudally, assuming a caudal movement of the diaphragm of 15 mm with each 10 cm H₂O increment of airway pressure (Paw). To verify the extent of movement, an additional antero-posterior scout view was performed at maximal inflation (Paw = 40 cm H₂O) in patients in group 1. The distance between the 4th or 5th lumbar spine and the top of the right and left diaphragm and the distance from the diaphragm to the apex of the lung were measured on each scout view and used to calculate the movement of the diaphragm.

To identify the densities (considered to reflect atelectasis), a magnified image (approximately ×2.5) was made of the dorsal portion of the CT scan of both the right and left lung. The dorsal border between the thoracic wall and the dense areas was drawn manually, but the ventral border between aerated lung tissue and atelectasis was identified by the region of interest program. The atelectatic area was calculated by the computer, including elements between −100 and +100 Hounsfield units (HU). The extent of atelectasis was expressed in absolute values (cm²) and as a percentage of the total intrathoracic area (including the mediastinum) [4].

Ventilation/perfusion ratios and blood-gas analysis

The ventilation/perfusion ratios (VA/Q) were assessed by the multiple inert gas technique [8]. Isotonic saline with a mixture of six inert gases (sulphur hexafluoride, ethane, cyclopropane, enflurane, diethyl ether and acetone) was infused into a peripheral vein at a rate of 3.5 ml min⁻¹. Under steady state conditions, arterial blood and mixed expired gas samples were collected in duplicate for subsequent analysis by gas chromatography. Oxygen uptake (VO₂) was estimated using the Douglas-bag technique: expired gas was collected and mixed expired oxygen measured using a mass spectrometer. The cardiac output was estimated as 0.02 × VO₂, assuming an arterio-venous oxygen content difference of 50 ml/litre of blood. Mixed venous inert gas concentrations were computed from the arterial values using mass balance principles [9]. This approximation of cardiac output is rather imprecise; however, it has been shown that the indices of VA/Q mismatch (SD of logarithmic perfusion distribution (log SDQ) and SD of logarithmic volume distribution (log SDV)) remain essentially unaffected by such uncertainties [9]. By mathematical analysis, the VA/Q distributions were fitted to a 50-compartment model. The result giving the best fit of data (smallest residual sum of squares (RSS)) of the duplicate samples was used for further statistical analysis.

Perfusion of lungs with VA/Q < 0.005 was defined as “shunt” and perfusion of lungs with 0.005 < VA/Q < 0.1 was defined as “low VA/Q”.

Arterial blood-gas tensions were measured using the ABL-2 (Radiometer). The alveolar–arterial oxygen partial pressure difference (PAO₂ − PAO₂) was calculated according to:

\[
(PAO₂ − PAO₂) = \frac{FIO₂ × (PB − PH₂O) − (PAO₂/RQ) − PAO₂}{PAO₂ − PB}
\]

where PB = barometric pressure; PH₂O = water vapour pressure; RQ = respiratory quotient, assumed to be 0.8.

Lung volumes

In the awake, supine patient, the vital capacity was measured according to standard procedures. In the anaesthetized patient, we estimated the vital capacity by a procedure analogous to that used for airway closure during anaesthesia [10]. The lungs were inflated first to Paw 40 cm H₂O and then deflated to Paw −15 cm H₂O (BOC manometer, Ohmeda, attached to the tracheal tube). To avoid interference with the recruitment manoeuvre and with the estimation of atelectasis, this vital capacity recording was performed at the end of the measurement series for each individual patient. For all the above measurements, a Fleisch pneumotachograph (Godart, Eindhoven Netherlands) was used.

Two or more measurements were made to obtain a minimum of two acceptable readouts and the mean of two or more readings was used for statistical analysis.

General procedure and recruitment manoeuvre

In the x-ray department, the infusion of inert gases was started and a catheter was inserted in a radial artery. The first CT scan and the measurement of vital capacity were then performed. After 40 min of infusion, samples were obtained for the gas exchange measurements. The patient was then anaesthetized. After 20 min of stable anaesthesia, gas exchange measurements and CT scans at end-expiration were repeated.

In the first 10 patients (group 1) a stepwise inflation of the lungs was performed (fig. 1.): with a super syringe, the lungs were inflated to Paw 10 cm H₂O. This pressure was held for about 15 s while the two CT scans were taken. This procedure was repeated subsequently with inflations to 20 cm H₂O, then
Group 1: stepwise inflation

\[
P_{aw} (\text{cm } H_2O): 0 \rightarrow 10 \rightarrow 20 \rightarrow 30 \rightarrow 40
\]

\[
15 \text{ s} \uparrow 3-5 \text{ min} \]

\[
15 \text{ s} \uparrow 3-5 \text{ min} \]

\[
15 \text{ s} \uparrow 3-5 \text{ min} \]

\[
15 \text{ s} \uparrow 3-5 \text{ min} \]

\[
15 \text{ s} \uparrow 3-5 \text{ min} \]

Group 2: repeated inflation

\[
P_{aw} (\text{cm } H_2O): 0 \rightarrow 30 \rightarrow 30 \rightarrow 30 \rightarrow 40
\]

\[
15 \text{ s} \uparrow 3-5 \text{ min} \]

\[
15 \text{ s} \uparrow 3-5 \text{ min} \]

\[
15 \text{ s} \uparrow 3-5 \text{ min} \]

\[
15 \text{ s} \uparrow 3-5 \text{ min} \]

\[
15 \text{ s} \uparrow 3-5 \text{ min} \]

FIG. 1. Recruitment manoeuvre and timing of CT of the lungs (1) (time intervals not to scale). Inflation to the various airway pressures maintained for 15 s.

30 cm H\textsubscript{2}O and finally 40 cm H\textsubscript{2}O. Between each inflation, the lungs were ventilated for 3–5 min at the baseline setting of the ventilator. As mentioned above, for each increment of 10 cm H\textsubscript{2}O, the scan level was moved 15 mm caudally, and the position of the diaphragm at an inflation to 40 cm H\textsubscript{2}O was checked with a final scout view. After the measurement of the “vital capacity anaesthetized”, the patient was moved to the operating theatre.

In group 2 the lungs were inflated in a similar manner, but starting with three inflations up to 30 cm H\textsubscript{2}O and finally with inflation to Paw 40 cm H\textsubscript{2}O (fig. 1). Between inflations the lungs were ventilated for 3–5 min at the baseline setting of the ventilator. CT scans were taken at end-inspiration, as for group 1, but only the scan at the level of the diaphragm was performed in group 2. Finally, vital capacity anaesthetized was measured and the patient was moved to the operating theatre.

As it was not known in advance at which inflation a significant change in the extent of atelectasis would be attained, it was necessary to investigate the two groups sequentially. Thus a randomized procedure was precluded.

Statistics

Unless stated otherwise, mean values and SD are presented. Within each group, the significance of the difference between the atelectasis at different inflations was tested using Friedman two-way ANOVA and Wilcoxon’s signed ranks test. Spearman’s rank correlation and multiple stepwise linear regression were used to analyse relationships between different variables. For all calculations, the SYSTAT computer software package (SYSTAT, Evanston IL, U.S.A.) was used.

RESULTS

Awake

Overall, the measurements in the awake patients were in the normal range (table II).

Only two patients, one in each group, had a small amount of densities in the dependent part of the caudal scan. The CT scans displayed no other abnormalities.

The measured retention and excretion of the inert gases resulted in technically good ventilation/perfusion distributions. The fit of the ventilation/perfusion data, expressed as RSS, averaged 3.7 and did not exceed 6 in 13 of 16 measurements, thus fulfilling the criteria established by Wagner and West [11]. A small shunt up to 1% was found in three patients. The partial pressure of oxygen in arterial blood exceeded 10.0 kPa in all patients but one (overall mean 12.3 (1.9) kPa) and \(P_{aCO_2}\) was in the normal range (mean 5.2 (1.0) kPa).

Anaesthesia

The main findings after induction of anaesthesia are summarized in table II. The intrathoracic area at the level of the diaphragm (scan 1) decreased by a mean of 4 (8)%. Fourteen patients developed atelectasis; the mean area was 7.0 cm\textsuperscript{2} (mean of all 16 patients), corresponding to 2.1% of the total intrathoracic area. There was a correlation between body constitution as expressed by body mass index (BMI) and the extent of atelectasis (fig. 2).

Compared with the awake, supine position, the top of the left and right diaphragm moved cranially on average by 10 (11) mm \((P = 0.0002, \text{sign test})\) and 9 (11) mm \((P = 0.001, \text{sign test})\), respectively. The \(V_a/Q\) measurements were technically good with a greatest RSS of 4.5 and mean RSS 1.4. The increase in log sdQ from awake 0.78 to anaesthesia 0.95 reflects a broadening of the \(V_a/Q\) mode. The shunt (perfusion of regions with \(V_a/Q < 0.005\)) increased in 13 patients (mean of all 16 patients: 6.6 (5.3)%), and an increase in low \(V_a/Q\) (0.005 < \(V_a/Q\) < 0.1) was found in 11 patients (mean of 16 patients: 2.4 (2.4)%). In five patients, low \(V_a/Q\) decreased or remained unchanged, but in these patients the sum of shunt plus low \(V_a/Q\) increased with anaesthesia.
Table II. Lung CT, lung mechanics and gas exchange data awake and during anaesthesia (mean (SD)). Scan 1 = at level of right diaphragm; scan 2 = 5 cm cranially. VT = Tidal volume; VC = Vital capacity (for details of VC measurement, see text); Crs = Dynamic compliance of the respiratory system during mechanical ventilation; shunt = Vd/Q < 0.005; low Vd/Q = 0.005 < Vd/Q < 0.1; CO = Cardiac output; (Pawxygen - Pawoxygen) = Alveolar-arterial oxygen partial pressure difference. Median 3.3 cm², range 0–20.1 cm² (skewed data); † median 1.5 cm², range 0–9.2 cm² (skewed data); ‡ median 9.8 cm², range 5.1–12.0 cm² (skewed data); N.s. = not studied. Movement of diaphragm in relation to position awake, supine: positive value = cranial movement. *FiO₂ awake = 0.21; FiO₂ anaesthesia = 0.40

<table>
<thead>
<tr>
<th></th>
<th>Awake</th>
<th>Anaesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atelectasis scan 1 (cm²)</td>
<td>0.0 (0.1)</td>
<td>5.7†</td>
</tr>
<tr>
<td>Atelectasis scan 2 (cm²)</td>
<td>0.0 (0.0)</td>
<td>2.0††</td>
</tr>
<tr>
<td>Diaphragm left (mm)§</td>
<td>—</td>
<td>10 (12)</td>
</tr>
<tr>
<td>Diaphragm right (mm)§</td>
<td>—</td>
<td>9 (13)</td>
</tr>
<tr>
<td>VT (ml kg⁻¹)</td>
<td>3.9 (0.8)</td>
<td>4.1 (0.8)</td>
</tr>
<tr>
<td>VC (litre)</td>
<td>—</td>
<td>75 (19)</td>
</tr>
<tr>
<td>Crs (ml cm H₂O⁻¹)</td>
<td>1.8 (2.4)</td>
<td>4.7 (4.7)</td>
</tr>
<tr>
<td>Shunt (%CO)</td>
<td>0.0 (0.1)</td>
<td>0.2 (0.5)</td>
</tr>
<tr>
<td>low Vd/Q (%CO)</td>
<td>1.8 (2.4)</td>
<td>1.1 (2.5)</td>
</tr>
<tr>
<td>(Pawxygen - Pawoxygen) (kPa)*</td>
<td>0.9 (1.9)</td>
<td>10.3 (7.5)</td>
</tr>
</tbody>
</table>

Group 1

Awake

Anaesthesia

Atelectasis scan 1 (cm²)

0.0 (0.1)

9.0†‡

Atelectasis scan 2 (cm²)

0.0 (0.0)

N.s.

Diaphragm left (mm)§

10 (12)

10 (10)

Diaphragm right (mm)§

9 (13)

8 (7)

VT (ml kg⁻¹)

3.9 (0.8)

3.7 (0.7)

VC (litre)

—

3.5 (0.7)

Crs (ml cm H₂O⁻¹)

75 (19)

60 (15)

Shunt (%CO)

0.2 (0.5)

9.9 (4.8)

low Vd/Q (%CO)

1.1 (2.5)

1.6 (1.0)

(Pawxygen - Pawoxygen) (kPa)*

10.3 (7.5)

15.9 (5.5)

Stepwise inflation (group 1)

During stepwise inflation, the extent of atelectasis decreased significantly only at airway pressures of 30 and 40 cm H₂O (table III, fig. 3). At Paw 40 cm H₂O, only one patient still showed atelectasis. Details for each patient are shown in figure 4; figure 5 shows the CT scans from one patient. The vital capacity anaesthetized (mean 4090 (850) ml) corresponded closely to the vital capacity awake, supine (mean 3940 (780) ml). This inflation to Paw = 40 cm H₂O may therefore also be called a vital capacity manoeuvre.

In order to obtain an estimate of the magnitude of the inflation necessary to diminish atelectasis, the airway pressure reducing the atelectatic area of scan 1 by 50% ( = Paw₅₀) was determined. In the individual plots of airway pressure vs atelectatic area, a quadratic regression curve was fitted to the data (fig. 4). Paw₅₀ was then estimated directly from the graph. The multiple linear regression between Paw₅₀ and age, BMI and Crs showed the following relationship:

\[
Paw_{50} = 29.9 - age \times 0.18 + BMI \times 0.94 - Crs \times 0.21
\]

\[r = 0.973, \text{ analysis of variance: } P = 0.001\]

There was a correlation between shunt and atelectatic area (r = 0.66, P = 0.006).

At FiO₂ 0.4, mean arterial oxygen partial pressure was 19.6 (7.4) kPa.

Fig. 2. Atelectatic area during anaesthesia and body mass index. r = 0.66; P = 0.010.

Fig. 3. Atelectatic area and stepwise inflation of the lungs (individual values (●) and mean (——)). A: CT scan at level of right diaphragm. B: CT scan 5 cm above right diaphragm.
TABLE III. Atelectasis with stepwise inflation of the lungs (one patient excluded (no atelectasis at Paw = 0 cm H$_2$O))

<table>
<thead>
<tr>
<th>Airway pressure (cm H$_2$O)</th>
<th>Scan 1 atelectasis (cm$^2$)</th>
<th>Scan 2 atelectasis (cm$^2$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Median</td>
</tr>
<tr>
<td>0</td>
<td>6.4</td>
<td>3.7</td>
</tr>
<tr>
<td>10</td>
<td>6.4</td>
<td>4.2</td>
</tr>
<tr>
<td>20</td>
<td>5.9</td>
<td>3.8</td>
</tr>
<tr>
<td>30</td>
<td>3.5</td>
<td>1.4</td>
</tr>
<tr>
<td>40</td>
<td>0.8</td>
<td>0.0</td>
</tr>
</tbody>
</table>

**Fig. 4.** Relative change in atelectatic area with stepwise inflation of the lungs for each individual patient, with a quadratic regression curve, calculated by the least squares method.

where age is in years; BMI = body mass index (kg m$^{-2}$); $C_{rs}$ = total compliance of the respiratory system (ml cm H$_2$O$^{-1}$).

During anaesthesia, the top of the left and right diaphragm was moved caudally between the position at $P_{aw} = 0$ cm H$_2$O (functional residual capacity (FRC)) and $P_{aw} = 40$ cm H$_2$O with means of 62 (14) mm and 70 (15) mm, respectively.

**Repeated inflation (group 2)**

With the first inflation to 30 cm H$_2$O, the amount of atelectasis was approximately halved (table IV, fig. 6). The second inflation resulted in an additional reduction of about 33%. With the third inflation to the same airway pressure, however, the further reduction in atelectasis was small (about 10%). The final inflation, to 40 cm H$_2$O (= vital capacity manoeuvre), eliminated the atelectasis.

With both recruitment manoeuvres, no clinically important adverse effects were observed.

**DISCUSSION**

The aim of this study was to estimate, in anaesthetized patients with healthy lungs, the degree of inflation of the lungs at which the amount of atelectasis may be reduced.

Computed tomography of the lung has become very helpful in analysing various aspects of lung pathophysiology, especially during anaesthesia [3] and in intensive care [12], and was therefore a
suitable technique to use in our study of atelectasis during inflation of the lung. However, it is well known that the atelectasis which appears after induction of anaesthesia is located mostly in dependent, basal parts of the lung of the supine patient [4]—a fact which was confirmed in our study, the caudal scans showing larger atelectasis than the cranial one. Thus positioning of the CT scan is critical to estimation of the amount of atelectasis: gradual cranial movement of the scan level may lead to a false conclusion of a reduction of atelectasis. In order to draw valid conclusions about changes in atelectasis, it is therefore important to visualize the same areas of lung tissue in subsequent scans. Our
between the subjects, and by the fact that BMI may influence body build, expressed as a wide range of BMI values. In our study, the correlation between BMI and atelectasis, has not been described before, although a significant but weak correlation with another measure of body constitution, Broca's index, was shown by Strandberg and others [16]. In our study, the correlation was probably favoured by the rather large variability in body build, expressed as a wide range of BMI between the subjects, and by the fact that BMI may be a better reflection of body constitution than Broca's index [6]. This correlation fits well with the theory of compression atelectasis which states that the atelectasis in the dependent lung is mostly the result of cranial shift of the diaphragm in conjunction with a change in thoraco-abdominal configuration [3]. It is likely that this cranial shift is more marked in subjects with a heavier abdominal content.

**Stepwise inflation**

A marked reduction in atelectasis with stepwise inflation of the lungs occurred only at an airway pressure greater than 20 cm H$_2$O. Considering the average total compliance in patients in group 1 (75 ml cm H$_2$O$^{-1}$) an airway pressure of 20 cm H$_2$O results from approximately a doubling of the "conventional" tidal volume. This implies that a doubling of the tidal volume (often termed "sigh"), maintained for 15 s, does not significantly reduce either atelectasis or shunt. It has been known for many years that $P_{aO_2}$ may not necessarily be improved by a sigh [17].

However, as shown in this investigation, a vital capacity manoeuvre may open almost all atelectatic lung tissue and should therefore markedly diminish the amount of shunt. This interpretation of our data agrees with findings in an earlier study, in which the shunt in anaesthetized patients was reduced successfully with a "hyperinflation" ($P_{aw} = 40$ cm H$_2$O, maintained for 40 s) of the lungs [18]. In the same investigation it was shown also that inflations to smaller pressures were not effective. Comparable findings were presented recently in a study of patients after open heart surgery [19]. Significant recruitment was found only at PEEP of 10–15 cm H$_2$O, resulting in end-inspiratory pressures of about 30 cm H$_2$O. It should be noted, however, that similar manoeuvres may be less successful in long standing atelectasis [20].

We tried to determine if there is a relationship between the amount of inflation necessary to reduce atelectasis and general parameters of the patient's constitution. For this purpose, the airway pressure required to reduce atelectasis by 50% was derived. An analysis of the multiple linear regression between $P_{aw50}$ and BMI, age and Crs during anaesthesia was carried out. This relationship showed that the patients who needed a greater $P_{aw}$ to reduce atelectasis had a large BMI, were younger and had small Crs, but this must be interpreted cautiously because of possible time-dependent factors relating to the inflation manoeuvres, and the small number of patients in this study.

**Repeated inflation**

The first inflation to $P_{aw}$ 30 cm H$_2$O reduced atelectasis to the same extent as the inflation of the "stepwise manoeuvre" with the corresponding pressure. However, further inflations to the same airway pressure gradually showed a less marked effect. This is in agreement with the results of an earlier study [19], with no further change in the volume of recruited lung units and no change in static compliance of the respiratory system after the third increase in PEEP. This finding suggests that the
critical opening pressure or the time constants are not equal in all units of a lung, even if those units are located in the same anatomical region—the dependent part of the lungs in our patients—or, expressed differently: in a given lung, not all alveoli may be opened by the same airway pressure. It has yet to be established if the recruited lung tissue remains aerated over time and if this recruitment is accompanied by a similar decrease in or even disappearance of, pulmonary shunt and by a significant decrease in \( P_{H_2O} - P_{a_0} \).

Does the favourable effect of a vital capacity manoeuvre on atelectasis, as described in these findings, have a clinical application? Two possible adverse effects have to be borne in mind: barotrauma and disruption of the alveolo-capillary barrier. The possibility of barotrauma may not be excluded completely; however, it should be stressed that the amount of inflation we used was in accordance with the guidelines proposed by Leith [21]. It has been shown recently that in small animals periods of hyperinflation as short as 2 min may alter microvascular permeability, resulting in increased extra-vascular lung water [22]. Hyperinflation was defined in that study as ventilation to a preset peak airway pressure of 35 mm Hg (≈ 48 cm H\(_2\)O), corresponding to a tidal volume of 46 ml kg\(^{-1}\). However, it is not known at which amount of inflation clinically significant alterations in lung tissue occur in human adults [23]. Furthermore, the vital capacity manoeuvre, as used in our study, results in a volume not larger than the awake, supine vital capacity. We therefore believe that such a manoeuvre still may be used in healthy adults having adequate clinical indication, such as significant hypoxaemia during general anaesthesia in an obese patient, in whom the development of atelectasis is likely, as shown above by the close relationship between body mass index and amount of atelectasis.

In summary, we found that atelectasis during general anaesthesia was not reduced by inflation of the lungs with a conventional tidal volume, or with a double tidal volume (sigh). With even larger inflation volumes, more than two repeated inflations to the same airway pressure added little to the recruitment of normal lung tissue. After an inflation to vital capacity, however, virtually all atelectatic lung tissue was re-expanded.

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