Influence of Gas Composition on Recurrence of Atelectasis after a Reexpansion Maneuver during General Anesthesia

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Background: Atelectasis, an important cause of impaired gas exchange during general anesthesia, may be eliminated by a vital capacity maneuver. However, it is not clear whether such a maneuver will have a sustained effect. The aim of this study was to determine the impact of gas composition on reappearance of atelectasis and impairment of gas exchange after a vital capacity maneuver.

Methods: A consecutive sample of 12 adults with healthy lungs who were scheduled for elective surgery were studied. Thirty minutes after induction of anesthesia with fentanyl and propofol, the lungs were hyperinflated manually up to an airway pressure of 40 cmH2O. Fo2 was either kept at 0.4 (group 1, n = 6) or changed to 1.0 (group 2, n = 6) during the recruitment maneuver. Atelectasis was assessed by computed tomography. The amount of dense areas was measured at end-expiration in a transverse plane at the base of the lungs. The ventilation-perfusion distributions (VA/Q) were estimated with the multiple inert gas elimination technique. The static compliance of the total respiratory system (Cst) was measured with the flow interruption technique.

Results: In group 1 (FiO2 = 0.4), the recruitment maneuver virtually eliminated atelectasis for at least 40 min, reduced shunt (VA/Q < 0.005), and increased at the same time relative perfusion to poorly ventilated lung units (0.005 < VA/Q < 0.1; mean values are given). The arterial oxygen tension (PaO2) increased from 137 mmHg (18.3 kPa) to 163 mmHg (21.7 kPa; before and after 40 min, respectively; P = 0.028). In contrast to these findings, atelectasis reoccurred in 4 min after recruitment in group 2 (FiO2 = 1.0). Comparing the values before and after recruitment, all parameters of VA/Q were unchanged. In both groups, Cst increased from 57.1/55.0 ml·cmH2O−1 (group 1/group 2) to 70.1/67.4 ml·cmH2O−1 after the recruitment maneuver. Cst showed a slow decrease thereafter (40 min after recruitment: 61.4/66.0 ml·cmH2O−1), with no difference between the two groups.

Conclusions: The composition of inspiratory gas plays an important role in the recurrence of collapse of previously expanded atelectatic lung tissue during general anesthesia in patients with healthy lungs. The reason for the instability of these lung units remains to be established. The change in the amount of atelectasis and shunt appears to be independent of the change in the compliance of the respiratory system. (Key words: Anesthesia, general; Lung; Atelectasis; compliance; gas exchange. Measurement techniques; computed tomography; multiple inert gas elimination. Ventilation, mechanical; ventilation-perfusion ratio.)

GENERAL anesthesia regularly impairs gas exchange, and despite preventive measures such as an increase of the inspired oxygen fraction, this often results in a decreased oxygenation of blood.1,2 The formation of atelectasis with subsequent pulmonary shunt has been shown to be an important factor for the derangement of gas exchange.3,4 However, atelectasis may be reexpanded and virtually eliminated by hyperinflation of the lungs.5-11 We demonstrated that such a recruitment maneuver may result in a sustained effect on atelectasis, a decrease of pulmonary shunt, and a slightly improved oxygenation for at least 40 min.5 In that investigation, air was used for the recruitment maneuver, the lungs of all patients were ventilated with 40% O2 in nitrogen, and there was an increase in the perfusion to units with low ventilation-perfusion ratios (VA/Q) after the recruitment.

It has been shown that lung units with low VA/Q may be prone to collapse, particularly if high inspiratory concentrations of oxygen or other gases with a high solubility in blood are used.12 On the other hand, a single deep breath may result in release of surfactant,13,14 thus contributing to an improved alveolar stability and preventing lung collapse. We therefore tested the effect of low or high inspired oxygen concentration (FiO2 0.4 or 1.0, balance nitrogen) on the reappearance of atelectasis, a respiratory system collapse, and the general anesthetic method.

Materials

Study Population

Twelve consecutive surgical patients who participated in the study and were scheduled for general anesthesia were included. As judged by the clinical examination, no patient had any other condition in addition to the usual clinical findings. The study protocol was approved by the University Ethics Committee of the Karolinska Institute, and all patients gave their informed consent to the study.12 The sample size was calculated on the basis of previous studies.8,15 For each comparison between groups, a statistically significant difference between the group means was considered to be reached at a level of P < 0.05.

Anesthesia

On arrival, the patients were premedicated with 1 mg·kg−1 clorazepate, 1 mg·kg−1 atropine, and 1 mg·kg−1 midazolam. The anesthe-
of atelectasis, gas exchange, and compliance of the respiratory system after a vital capacity maneuver during general anesthesia.

Materials and Methods

Study Population

Twelve consecutive patients scheduled for neurosurgical procedures or general surgery were included in the study (Table 1). Another four patients refused to participate in the study, i.e., the refusal rate was 25%. As judged by patient history and clinical examination, no patient had a cardiac or pulmonary disease. There were two smokers.

The study was approved by the Ethics Committee of the University Hospital of Upssala, and informed consent was obtained from each patient. The estimation of the sample size (n = 12) was based on previous studies, to detect a difference of 50% in the amount of atelectasis, at a P value of 0.05 and a power of 90%, between the two groups 40 min after re-expansion of atelectasis.

Anesthesia

On request, the patients received 0.04-0.08 mg·kg⁻¹ ketobemidon (Ketogan, an opioid) intramuscularly as premedication. Before anesthesia, 0.5 mg atropine was given intravenously. Anesthesia was induced with 1-2 µg·kg⁻¹ fentanyl and 2 mg·kg⁻¹ propofol intravenously, followed by a continuous infusion of 4-8 mg·kg⁻¹·h⁻¹ propofol. During the induction, the lungs were manually ventilated via a face mask, using 100% O₂. To facilitate orotracheal intubation, the patients received 0.1 mg·kg⁻¹ pancuronium, and additional doses of 1-2 mg were given when needed. The lungs were mechanically ventilated (Servo Ventilator 900C, Siemens) at a rate of 10 breaths/min with 40% O₂ in nitrogen during the following phase of the study (fig. 1). No positive end-expiratory pressure was used. The minute ventilation was adjusted to maintain an end-tidal carbon dioxide concentration of approximately 4% (carbon dioxide analyzer Eliza, Engström).

Study Protocol

The patients were assigned randomly (sealed envelopes) to two different groups. In group 1, FIO₂ was kept at 0.4, whereas in group 2, FIO₂ was switched to 1.0 during the recruitment maneuver (see below).

Three sets of measurements were performed (details are given below and in fig. 1): an estimation of atelectasis by computed tomography (CT) of the lungs, an estimation of the ventilation-perfusion relationship (VA/Q) with the multiple inert gas elimination technique (MIGET), and an estimation of the compliance of the total respiratory system (Crs) with the flow interruption technique. In the awake state, the measurements included CT of the lungs and the estimation of

<table>
<thead>
<tr>
<th>Group</th>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Gender</th>
<th>Smoking</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>BMI (kg·m⁻²)</th>
<th>Pao₂ Awake (mmHg)</th>
<th>Anesthesia Baseline (mmHg)</th>
<th>Atelectasis Anesthesia Baseline (cm²)</th>
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Anesthesia, baseline = 20 min after induction of anesthesia. M = male; F = female. FIO₂ = inspiratory oxygen fraction after recruitment maneuver. BMI = body mass index calculated as weight × height², normal average in white North-American, 42-44 years of age: 25.8 (men), 23.6 (women). Pao₂ = arterial oxygen partial pressure awake (FIO₂ = 0.2) and at anesthesia, baseline (FIO₂ = 0.4). Atelectasis: CT scan at the lung base, at anesthesia, baseline (note: atelectasis in all awake subjects = 0 cm²).
V/AQ. After 20 min of stable anesthesia, the CT scans and the V/AQ measurements were repeated, and the Cn was measured (= "anesthesia baseline"). This was followed by the recruitment maneuver (see below). The CT, V/AQ, and Cn measurements were repeated until 40 min after the recruitment (fig. 1). Finally, the patient was moved to the operating theater.

Recruitment Maneuver
To reexpand atelectasis, a recruitment maneuver was performed as described previously. Using a super syringe, the lungs were inflated with air three times up to an airway pressure (Paw) of 30 cmH2O (fig. 1). For the final inflation to a Paw of 40 cmH2O, either air (group 1) or 100% O2 (group 2) was used, and in group 2, FiO2 was changed to 1.0 on the ventilator for the succeeding study period. Each inflation was held for 15 s, and between each inflation, the lungs were ventilated for 1–2 min with the baseline settings of the ventilator. During the recruitment maneuver, the airway pressure was measured with a manometer (BOC, Ohmeda) attached to the endotracheal tube.

Computed Tomography of the Lungs
Atelectasis was studied by CT (Somatom plus, Siemens). The subjects were in the supine position with arms above the head. A frontal scout view covering the chest was obtained at end-expiration, awake, and after induction of anesthesia. For each subsequent analysis, a CT scan in the transverse plane was performed at end-expiration, 1 cm above the top of the right diaphragm. An additional scan at the level of the hilum and at the apex of the lungs was taken after induction of anesthesia, immediately after the recruitment maneuver and 40 min thereafter. The scan time was 1 s at 255 mAs and 137 kV. The slice thickness was 8 mm, and a matrix of 512 × 512 was used, resulting in a pixel (picture element) of approximately 1.5 × 1.5 mm. The total x-ray exposure for each patient was 3 mSv (Millisievert; total average exposition in Sweden 5 mSv per year).

To identify atelectasis, a magnified image (approximately 3×) was made of the dorsal portion of the CT scan of both the right and the left lungs. The dorsal border between the thoracic wall and the dense area was drawn manually, whereas the ventral border between inflated lung tissue and atelectasis was identified by the region-of-interest (ROI) program. The atelectatic area, including all pixels with density values between −100 and +100 Hounsfield units (HU), was calculated by the computer. As an example, lung tissue with a density value of −100 HU represents a lung unit with 10% gas and 90% tissue. The amount of atelectasis was expressed in absolute values (cm²) and as percent of the total intrathoracic area (including the mediastinum). For details, see references 3 and 16.

To evaluate possible changes in regional lung volume in nondependent parts of the lungs, vessels that could be seen on consecutive scans were identified, and the angles between these vessels were measured. For this purpose, the CT scans were transferred to an image analysis software (Matlab, Mathworks, MA) for quantitative analysis of selected angles between vessels.

Ventilation and Perfusion Measurements
Throughout the study, arterial blood gas analysis (BGA) was performed in duplicate, blood samples were obtained from the radial artery before and after anesthesia and during the study period. For each subject, 1 cm of the right upper lobe was resected. The lungs were then isolated. Three sets of 20 ml saline were instilled into each lobe, which was ventilated with 100% O2 at a rate of 30 breaths per minute, for 2 min. After ventilation, the lung was resected and frozen.
ATELECTASIS DURING GENERAL ANESTHESIA

analysis software package (BioScan OPTIMAS, Edmonds, WA). This program allows the simultaneous analysis of several CT scans and the measurement of angles between lines (an example is given in fig. 2).

Ventilation-Perfusion Ratios and Blood Gas Analysis

The ventilation-perfusion ratios (VA/Q) were assessed with the multiple inert gas elimination technique. Isotonic saline with a mixture of six inert gases (sulphur hexafluoride, ethane, cyclopropane, enflurane, diethyl ether, and acetone) was infused continuously into a peripheral vein. Under steady-state conditions, arterial blood and mixed expired gas samples were collected in duplicate for subsequent analysis by gas chromatography (Hewlett Packard Gas Chromatograph 5880A and 5890). Oxygen uptake (VO2) and carbon dioxide elimination (VCO2) were estimated with the Douglas-bag technique, thus collecting expired gas and measuring the concentrations of mixed expired oxygen and carbon dioxide (Beckman OM-14 and Leybold-Heraeus). During anesthesia, only VO2 was measured directly, whereas VCO2 was calculated assuming the same respiratory quotient as measured awake. The cardiac output was estimated as 20×VO2, assuming an arteriovenous oxygen difference of 50 ml·l-1 blood and expressing VO2 in ml·min-1. The mixed venous inert gas concentrations were computed from the arterial and mixed expired values using mass balance principles. By mathematical analysis of the inert gas data, each VA/Q distribution was recovered in a 50-compartment model, and the result with the best fit of data (smallest remaining sum of squares, RSS) of each pair of duplicate samples was used for further statistical analysis. Intrapulmonary shunt (Qs/Qt) was defined as the fraction of total blood flow perfusing lung units with VA/Q < 0.005, and low VA/Q was defined as the fraction of total blood perfusion to lung units with 0.005 < VA/Q < 0.1. logSDQ, the standard deviation of the logarithmic distribution of perfusion, was calculated as a measure of the dispersion of blood flow distribution. logSDV is the standard deviation of the logarithmic distribution of ventilation. RSS for all measurements was on the average 2.0 and exceeded 6.0 in only two measurements, thus fulfilling the criteria as established by Wagner and West.

In two patients (one from each group), a thorough analysis of VA/Q was performed by so-called Monte Carlo simulation and by linear programming, as described previously. Thus, the inert gas data were analyzed taking into account both the experimental error of inert gas measurement and the uncertainty, inherent to the determination of VA/Q distributions by the multiple inert gas elimination technique.

Blood gas measurements were performed by standard technique (ABL-2, Radiometer).

Compliance of the Respiratory System

Estimations of static compliance of the respiratory system (Cst) were obtained using the technique of flow

![Fig. 2. Measurement of intravascular angles of pulmonary vessels present on subsequent computed tomography (CT) scans. Angles are given in degrees. CT scan is processed by image analysis software. (Left) After induction of anesthesia. (Right) Immediately after recruitment maneuver.](image-url)
interruption. Pressure and flow were measured in the ventilator on the inspiratory side and fed into a computer for on-line signal processing (Macintosh II/1x, with LabView 2, National Instruments). The mean value of two "inspiratory hold" maneuvers (2 s hold) was used for each point. \(C_{v} \) was calculated as tidal volume (\(V_{t}\)) divided by end-inspiratory pressure minus end-expiratory pressure. Thus, \(C_{v}\) reflects the elastic behavior of the lungs and the chest wall over the tidal volume range.

Statistical Analysis
Where not stated otherwise, mean values and standard deviations are presented. In addition, for major variables, 95% confidence intervals (95% CI) are given. To compare variables at different times within groups, we used the Friedman two-way ANOVA and the Wilcoxon's signed-rank test. To compare data between the two groups, the Mann-Whitney \(U\) test was used. Spearman's rank correlation coefficient was used to analyze relationships between variables. For all calculations, the SYSTAT computer software package (SYSTAT, Evanston, IL) was used.

Results
Baseline Awake and after Induction of Anesthesia
The CT scans in the awake patients displayed no abnormalities. All patients experienced atelectasis after induction of anesthesia, with an overall mean area of 8.0 ± 8.2 cm² (group 1 10.0 ± 7.1 cm², group 2 6.1 ± 9.3 cm²). Figure 3 shows an example in two patients, table 1 shows details of the data, and figure 4 gives a summary.

The ventilation-perfusion measurements are summarized below and in table 2. In the awake condition, a shunt (\(V_{a}/Q < 0.005\)) was found in only one patient (2.5% of cardiac output [CO]), the same patient also had a considerable amount of low \(V_{a}/Q\) (14.2% CO). On the average, low \(V_{a}/Q\) (0.005 < \(V_{a}/Q < 0.1\)) was 3.1 ± 4.2% CO. \(P_{a/2}\) measured while breathing air, exceeded 75 mmHg (10.0 kPa) in 11 of 12 patients (mean 98 ± 22 mmHg [13.0 ± 2.9 kPa]), and mean \(P_{a/2}\) was 59.0 ± 5.3 mmHg (5.2 ± 0.7 kPa). The patient with the rather marked derangement of \(V_{a}/Q\) and the low \(P_{a/2}\) was well premedicated and fell asleep during the measurement of \(V_{a}/Q\) "awake." Furthermore, he was obese and had the second highest body mass index (patient 8 in table 1).

After induction of anesthesia the pulmonary shunt was increased to 6.5 ± 5.2%, low \(V_{a}/Q\) increased to 5.3 ± 5.3%, \(P_{a/2}\) was 150 ± 55 mmHg (20.0 ± 7.4 kPa) at an \(F_{a/2}\) of 0.4, and \(P_{a/2}\) was 35.7 ± 2.3 mmHg (4.5 ± 0.3 kPa). The estimated mean cardiac output, used to calculate the \(V_{a}/Q\) distributions, was 4.4 ± 1.0 1·min⁻¹ in the awake subjects and 3.5 ± 0.81 1·min⁻¹ after induction of anesthesia.

The measurement of \(C_{v}\) yielded values in the normal range for patients whose trachea is intubated and whose lungs are mechanically ventilated (15) (group 1 57.1 ± 19.5 ml·cmH₂O⁻¹, group 2 55.0 ± 19.1 ml·cmH₂O⁻¹, fig. 4).

After Recruitment Maneuver
The recruitment maneuvers caused no clinically important adverse effects.

Immediately after recruitment, atelectasis was virtually eliminated in both groups (0.0 ± 0.1 and 0.1 ± 0.2 cm² in groups 1 and 2, respectively). In group 1 (\(F_{a/2} = 0.4\)), there was a slow increase in the amount of atelectasis, which reached about one-sixth of the initial area of atelectasis 40 min after the recruitment (5 min 0.2 ± 0.2 cm²; after 40 min 1.6 ± 2.1 cm², 95% CI 0 to 3.8 cm²; \(P = 0.028\) vs. anesthesia baseline). In group 2 (\(F_{a/2} = 1.0\)), the amount of atelectasis was increased to a value close to pre-recruitment 5 min after the recruitment maneuver (5.3 ± 8.7 cm², 95% CI 0 to 16.5 cm², no significant difference vs. anesthesia baseline), and there was a further slow increase during the rest of the study period (after 40 min 7.9 ± 9.7 cm², no significant difference vs. anesthesia baseline). See figure 3 for an example in two patients and figure 4 for a summary of the data. The mean intrathoracic area in the awake subject, expressed as percentage of the intrathoracic area in the awake subject, showed only small, insignificant changes from anesthesia baseline to 0 and 40 min after recruitment both in group 1 (91.3 ± 5.6%/94.5 ± 4.9%/92.0 ± 5.2%) and in group 2 (90.5 ± 10.4%/92.0 ± 3.9%/90.8 ± 9.7%). Pulmonary vessels, suitable for measurements of angles, could be identified on subsequent CT scans of four patients in group 1 and of all six patients in group 2. Between anesthesia baseline and the first measurement immediately after the recruitment, there was a decrease of the intervascular angles in all but two patients (\(P = 0.013\)), the mean decrease being 12 ± 7%/11 ± 10% (group 1/2). As compared to the baseline value before recruitment, the mean angles remained decreased in group 1 by 9 ± 6% and 11 ± 10% at 5 and 40 min after
atelectasis was virtually absent (0.1 and 0.1 ± 0.1% in group 1 and 2, respectively). In group 1, the amount of atelectasis was one-sixth of the amount in group 2. The recruitment was 1.6 ± 2.1 ml · cmH₂O⁻¹, and the mean difference was 0.1 ml · cmH₂O⁻¹. In group 2, the difference was 5.3 ± 8.7 ml · cmH₂O⁻¹, and the mean difference was 0.4 ml · cmH₂O⁻¹. During the first period (after 40 min), the atelectasis was not different from that in the group 1. In the second period (after 40 min), the atelectasis was not different from that in group 1. The recruitment in group 1 was higher than that in group 2. The recruitment in group 1 was 2.0 ± 1.0 ml · cmH₂O⁻¹, and the mean difference was 0.2 ± 0.3 ml · cmH₂O⁻¹. In group 2, the difference was 4.0 ± 3.5 ml · cmH₂O⁻¹, and the mean difference was 0.4 ± 0.3 ml · cmH₂O⁻¹. During the first period (after 40 min), the atelectasis was not different from that in the group 1. In the second period (after 40 min), the atelectasis was not different from that in group 1. The recruitment in group 1 was higher than that in group 2.

Footnote: The atelectasis was virtually absent (0.1 and 0.1 ± 0.1% in group 1 and 2, respectively). In group 1, the amount of atelectasis was one-sixth of the amount in group 2. The recruitment was 1.6 ± 2.1 ml · cmH₂O⁻¹, and the mean difference was 0.1 ml · cmH₂O⁻¹. In group 2, the difference was 5.3 ± 8.7 ml · cmH₂O⁻¹, and the mean difference was 0.4 ml · cmH₂O⁻¹. During the first period (after 40 min), the atelectasis was not different from that in the group 1. In the second period (after 40 min), the atelectasis was not different from that in group 1. The recruitment in group 1 was higher than that in group 2. The recruitment in group 1 was 2.0 ± 1.0 ml · cmH₂O⁻¹, and the mean difference was 0.2 ± 0.3 ml · cmH₂O⁻¹. In group 2, the difference was 4.0 ± 3.5 ml · cmH₂O⁻¹, and the mean difference was 0.4 ± 0.3 ml · cmH₂O⁻¹. During the first period (after 40 min), the atelectasis was not different from that in the group 1. In the second period (after 40 min), the atelectasis was not different from that in group 1. The recruitment in group 1 was higher than that in group 2.

In group 1, shunt decreased from 7.2 ± 5.2% CO at anesthesia baseline to 1.3 ± 2.2 and 2.3 ± 2.6% CO 20 and 40 min after the recruitment maneuver (P = 0.028 and 0.028, if compared to anesthesia baseline), respectively. Low VA/Q increased from 6.7 ± 2.1% CO to 11.0 ± 3.5 and 8.0 ± 3.7% CO (P = 0.028 and 0.5, if compared to anesthesia baseline). In group 2, there was no significant change in any of the measured parameters of the ventilation-perfusion distribution. At 40 min after the recruitment, shunt was 6.4 ± 6.6%, and low VA/Q was 3.5 ± 2.5% CO. Further details of...
Fig. 4. Atelectasis and compliance of the total respiratory system (Crs) before and after recruitment maneuver. Data are mean ± SD. Group 1 (P0.8 after recruitment = 0.4): open bars. Group 2 (P0.8 after recruitment = 1.0): hashed bars. (Top) Atelectasis versus time. Atelectasis is estimated as amount of dense areas (<100 to >100 HU) in a computed tomography slice 1 cm above the right diaphragm. *P = 0.028 (vs. atelectasis after induction of anesthesia, within group). \( \dagger P = 0.2 \) (comparing groups 1 and 2, after induction of anesthesia). (Bottom) Compliance versus time. \( \ast P = 0.028 \) (vs. compliance after induction of anesthesia, within group). \( \dagger P = 1.0 \) (comparing groups 1 and 2, after induction of anesthesia).

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Discussion

This study confirms that reexpansion of atelectasis during general anesthesia in patients with healthy lungs may have a sustained effect with respect to atelectasis, shunt, and \( \text{Pao}_2 \): if the lungs are ventilated with 40% \( \text{O}_2 \) in nitrogen. If 100% \( \text{O}_2 \) is used, however, lung collapse reappears within a few minutes, and as compared to prerecruitment, the ventilation-perfusion relationship is essentially unchanged after the recruitment. This time course of atelectasis suggests that gas resorption plays an important role in the recurrence of collapse in previously reexpanded atelectatic lung tissue. Such lung regions appear to be unstable, and the mechanisms

Table 2. Gas Exchange Data

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that keep a balance in the forces causing atelectasis.

Methodology

The amount of postoperative atelectatic regions in dependent lung regions was measured by chest radiography in dependent and nondependent lungs. In one CT scan after induction of general anesthesia, the total atelectatic regions were estimated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated. The CT scan was performed 30 minutes after induction of anesthetic, and the atelectatic regions were calculated.
Atelectasis During General Anesthesia

Table 2. Gas Exchange Awake, after Induction of Anesthesia, and after Recruitment Maneuver

<table>
<thead>
<tr>
<th></th>
<th>Log SDQ 1</th>
<th></th>
<th>Log SDV 1</th>
<th></th>
<th>PaO2 (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1</td>
<td>Group 2</td>
<td>Group 1</td>
<td>Group 2</td>
<td></td>
</tr>
<tr>
<td>Awake</td>
<td>0.92 ± 0.20</td>
<td>0.65 ± 0.52</td>
<td>0.55 ± 0.07</td>
<td>0.55 ± 0.25</td>
<td>89 ± 7</td>
</tr>
<tr>
<td>Anesthesia, baseline</td>
<td>1.34 ± 0.18</td>
<td>0.93 ± 0.52</td>
<td>0.60 ± 0.12</td>
<td>0.55 ± 0.08</td>
<td>137 ± 33</td>
</tr>
<tr>
<td>20 min after recruiting</td>
<td>1.43 ± 0.14</td>
<td>0.94 ± 0.38</td>
<td>0.67 ± 0.11</td>
<td>0.57 ± 0.12</td>
<td>153 ± 27</td>
</tr>
<tr>
<td>40 min after recruiting</td>
<td>1.35 ± 0.14</td>
<td>0.94 ± 0.38</td>
<td>0.69 ± 0.07</td>
<td>0.59 ± 0.13</td>
<td>163 ± 23</td>
</tr>
</tbody>
</table>

PaO2 = arterial oxygen tension (note: awake PaO2 = 21, anesthesia PaO2 = 4.5, except for group 2 at 20/40 min after recruiting, where FIO2 = 1.0). P = 0.028 (vs. anesthesia, baseline).

Methodologic Aspects

The amount of atelectasis was estimated by CT. Methodologic details concerning the analysis of dense regions in dependent parts of the lungs have been described in detail elsewhere. Atelectasis that appears after induction of general anesthesia is located mostly in dependent, basal parts of the lung. To avoid excessive exposure to radiation, we decided to perform only one CT scan per condition under investigation. At three occasions, an additional scan at the level of the hilum and the apex of the lungs was taken. As in previous investigations, the amount of atelectasis at these additional levels was always smaller as compared to the CT scan close to the dome of the diaphragm. With respect to the time course of atelectasis, the analysis of these additional scans provided no further information and therefore is not presented in this paper.

The ventilation-perfusion distribution in the lungs was analyzed with the multiple inert gas elimination technique. This method is based on the elimination and retention of a number (usually six) of "inert" gases. To calculate these parameters, the measurement of cardiac output and the concentrations of the "inert" gases in mixed expired air, mixed venous blood, and arterial blood is necessary. Some of these parameters thus necessitate the use of a pulmonary artery catheter. However, a simplified methodology can be applied, requiring measurements in arterial (or venous) blood and in mixed expired air only. We decided to use this latter technique as it was not considered justifiable to apply the more invasive procedure of pulmonary artery catheterization to the subjects eligible for this study.

Thus, mixed venous "inert" gas concentrations were computed using mass balance principles, and cardiac output was estimated from the oxygen consumption. This approximation of cardiac output is imprecise, but it has been shown that indexes of Va/Q mismatch (e.g., logSDQ and logSDV) remain essentially unaffected by such uncertainties. Errors in cardiac output measurements, however, may be transmitted to other parameters of Va/Q, including shunt, low Va/Q, and mean Va/Q. Thus, a quantitative estimation of these parameters is less reliable. However, if cardiac output is con-
stant during the study period, relative changes in shunt and low V̅a/Q can be estimated. This may be seen from the formula used to calculate mixed venous partial pressures (Pv) of an “inert” gas from the arterial partial pressure (Pa) and the partial pressure in mixed expired gas (Pe) \( P_{v} = P_{a} + \left( \frac{(P_{r} - P_{v})}{\lambda} \right) \), where \( \lambda \) blood gas partition coefficient, V̅e: minute ventilation, and Q̅r: cardiac output.

With V̅e, Q̅r, and \( \lambda \) unchanged, changes in Pa and Pe will transmit directly to changes in Pv. Variations in shunt and low V̅a/Q during unchanged anesthesia therefore will not merely reflect errors in measurement but may be interpreted as changes of shunt or low V̅a/Q. Because oxygen uptake did not vary during anesthesia in those cases in which repeated measurements were made, we assume that CO was more or less constant during the study. Moreover, an analysis of the data of our investigation showed that a variation of cardiac output by 25% will result in a rather modest variation of shunt and low V̅a/Q by about 20% of the measured values.

Finally, Monte Carlo simulation and linear programming showed that low V̅a/Q could be separated from shunt and that the V̅a/Q distribution in patient 5 of group 1 (no or minor atelectasis 20 min after the recruitment) was significantly different from that in patient 9 of group 2 (with atelectasis 20 min after the recruitment). This lends support to our conclusion that the reappearance of atelectasis after a recruitment maneuver affects the V̅a/Q distribution.

**Mechanism of Atelectasis Formation**

The findings of this study are in accordance with previous investigations with respect to atelectasis and ventilation-perfusion relationship (V̅a/Q) before and after induction of general anesthesia with mechanical ventilation. Furthermore, the changes in the amount of atelectasis and V̅a/Q after the recruitment maneuver in the patients ventilated with a F\(_{1,2,3}\) of 0.4, yielded results similar to a recently performed study. The very fast reappearance of densities in the group ventilated with 100% \( O_{2} \) after the recruitment maneuver, however, deserves further discussion.

That resorption of gas may play a role in the formation of atelectasis during anesthesia has been discussed on theoretical grounds and in a number of experimental and clinical studies, although it was not possible to demonstrate lung collapse on conventional x-ray. The role of this mechanism for the formation of atelectasis during general anesthesia remained unclear until now. In the current investigation, the fast reappearance of atelectasis in the group ventilated with 100% \( O_{2} \) suggests that gas resorption is a major factor for a renewed collapse of previously reopened lung tissue.

Collapse by gas resorption may be present in a lung unit if there is a total stop of gas flow to this unit (thus resulting in trapped gas) or if the expired ventilation of this lung unit falls to zero (critical V̅a/Q). In the current study, the amount of atelectasis and shunt was reduced by the recruitment maneuver, the amount of lung units with low V̅a/Q increased at the same time. Therefore, the amount of units with a critical V̅a/Q probably increased, too. It may be assumed that units with a low V̅a/Q are the ones that were collapsed and caused shunt before the recruitment. Therefore, they are located in dependent parts of the lungs. As the preinspiratory lung volume in supine subjects is markedly smaller in dependent than in nondependent parts of the lungs, the gas used for an inflation up to vital capacity will be distributed predominantly to dependent lung units, too. Thus, if 100% \( O_{2} \) is used for this maneuver, the concentration of oxygen will increase mostly in such lung units.

To summarize, a recruitment maneuver with oxygen results in an increase of lung units with low V̅a/Q, prone to fast collapse because of increased oxygen content within such units. In a computer model of absorption atelectasis, the time to collapse in a lung unit filled with 100% \( O_{2} \) and excluded from ventilation has been estimated to be about 8 min. This collapse may be faster if a mixture of nitrous oxide and oxygen is used. According to the same study, this time will be about 3 h if a lung unit was filled with 50% \( O_{2} \) in nitrogen before exclusion from ventilation. Whether a decreased function of surfactant, as suggested during general anesthesia, or other, yet unknown factors play an additional role for instability of lung units remains to be shown.

Further evidence that this atelectasis is caused by resorption of gas is suggested by the results of our measurements of intravascular angles in nondependent parts of the lungs. These angles were decreased by the recruitment maneuver, and they increased again thereafter. This indicates that competition for space may cause a compression of nondependent parts of the lungs when dependent parts are expanded and an expansion of nondependent parts when dependent parts of the lungs collapse. In two other studies, it was found that lung units in the immediate vicinity of atelectatic tissue were well aerated, even after ventilation with an intermediate \( F_{1,2,3} \).

**Compliance**

The compliance of the lung is not measurable, change in \( C_{a} \) was increased by the recruitment maneuver, similarly in both atelectasis groups. Furthermore, the change between the control group and the recruitment maneuver groups showing almost no atelectasis was associated with the second, and a small increase in \( C_{a} \) despite the final collapse of most lung units.

These findings are important because the lung compliance that may be decreased due to the recruitment maneuver that is performed in the control group may not be measurable in the study groups, characterized by low lung compliance. It has been suggested that alveolar wall rigidity may be the factor responsible for the decreased lung compliance. Even though the compliance in the control group and the recruitment maneuver groups was not measured, the findings support the hypothesis that the recruitment maneuver may be associated with the decrease in lung compliance.

In conclusion, this investigation of atelectasis in a group of patients receiving general anesthesia indicates that gas resorption plays a major role in the formation of atelectasis. Further studies are needed to elucidate the mechanisms involved in this process and to determine the role of gas resorption in the development of atelectasis during general anesthesia.
Atelectasis during general anesthesia

The compliance of the respiratory system ($C_v$) did not show the same course as atelectasis. In both groups, $C_v$ was increased and atelectasis was decreased by the recruitment maneuver, but thereafter $C_v$ decreased similarly in both groups, whereas the behavior of atelectasis was different between groups 1 and 2. Furthermore, there appears to be no close relationship between the amount of atelectasis and the relative change in $C_v$ before and immediately after the recruitment maneuver (fig. 5). For example, there were subjects showing an increase in $C_v$, by one-third and having almost no atelectasis. On the other hand, the subject with the second largest amount of atelectasis had only a small increase in $C_v$ with the recruitment maneuver, despite the fact that the atelectasis was eliminated.

These findings indicate that atelectasis is not the only cause of decreased compliance. Other mechanisms that may be involved are changes in thoracic configuration, changes in the micromechanics of acinus or alveolar walls, an altered function of surfactant, and an altered function of surfactant. It has been shown that a single stretch of alveolar type II cells may be followed by a stimulation of surfactant secretion that is sustained for up to 30 min. This time scale is fairly similar to that of the behavior of compliance in the current investigation. The compliance measurement was made over the tidal volume range, which makes it sensitive to the presence of poorly ventilated lung regions. Regions with low $V_{A}/Q$ were more common in group 1 (with little atelectasis) than in group 2, and they may have balanced the effect of atelectasis on compliance.

In conclusion, atelectasis as found during general anesthesia in patients with healthy lungs may be expanded by deep inflations of the lungs, but resorption of gas plays an important role in the recurrence of collapse in such lung units. Therefore, gas exchange may be improved only by a recruitment maneuver, if the lungs are ventilated with a gas mixture containing a poorly resolved gas such as nitrogen. The change in the amount of atelectasis and shunt appears to be independent of the change in total compliance of the respiratory system. The reason for the instability of lung units, leading to atelectasis with induction of anesthesia, remains to be established.

References


Background: Recent studies have suggested that propofol may be a safe anesthetic in critically ill and postcardiac surgery patients. The present study was designed to evaluate the safety and efficacy of propofol in the ICU setting.

Methods: Forty-two patients were enrolled in the study. They were randomized to receive either propofol (n=21) or alfentanil (n=21). The patients were monitored for 72 h after surgery. The primary outcome measure was the rate of adverse events. The secondary outcome measures were the incidence of hypertension, tachycardia, and hypotension.

Results: No significant differences were observed between the two groups in terms of the rate of adverse events. The incidence of hypertension, tachycardia, and hypotension was similar in both groups.

Conclusion: The results of this study suggest that propofol is a safe and effective anesthetic in the ICU setting.


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