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Morbid obesity and postoperative pulmonary atelectasis : an underestimated problem

THESE

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Abstract

La perturbation de la mécanique ventilatoire induite par une anesthésie générale et un acte chirurgical est plus prononcée chez les patients obèses morbides (OM). Vu qu'une anesthésie générale va entraîner la formation d'atélectases pulmonaires chez des patients non obèses, nous avons émis l'hypothèse que la formation d'atélectases serait particulièrement significative chez des patients OM. Nous avons donc investigué la quantité et la résorption d'atélectases après une anesthésie générale chez des patients OM et chez des patients non obèses. Vingt patients OM ont donc bénéficié d'une anesthésie générale pour une gastroplastie laparoscopique et dix patients non obèses pour une cholécystectomie laparoscopique. Nous avons évalué la quantité d'atélectases pulmonaires par CT-Scan thoracique à trois temps différents : avant l'induction de l'anesthésie générale, immédiatement après l'extubation trachéale et 24 heures plus tard.

Déjà avant l'induction de l'anesthésie générale, les patients OM présentaient une quantité plus importante d'atélectases que les non obèses, quantité exprimée en pourcent de la surface pulmonaire totale (2.1% vs 1.0% , respectivement ; $P < 0.01$). Après l'extubation trachéale, la quantité d'atélectases avait augmenté dans les 2 groupes mais restait significativement plus importante dans le groupe OM (7.6% pour le groupe OM vs 2.8% pour les non obèses ; $P < 0.05$).

Vingt-quatre heures plus tard, l'importance des atélectases demeurait inchangée chez les patients OM, mais nous avons observé une résorption complète chez les patients non obèses (9.7% vs 1.9%, respectivement ; $P < 0.01$).

L'anesthésie générale a généré une quantité bien plus importante d'atélectases chez des patients obèses morbides que chez des non obèses. De plus, 24 heures après une anesthésie générale nous avons observé un *statu quo* quant à l'évolution de ces atélectases chez les patients OM, alors quelles disparaissent chez les patients non obèses.

Morbid Obesity and Postoperative Pulmonary Atelectasis: An Underestimated Problem

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Perturbation of respiratory mechanics produced by general anesthesia and surgery is more pronounced in morbidly obese (MO) patients. Because general anesthesia induces pulmonary atelectasis in nonobese patients, we hypothesized that atelectasis formation would be particularly significant in MO patients. We investigated the importance and resorption of atelectasis after general anesthesia in MO and nonobese patients. Twenty MO patients were anesthetized for laparoscopic gastroplasty and 10 nonobese patients for laparoscopic cholecystectomy. We assessed pulmonary atelectasis by computed tomography at three different periods: before the induction of general anesthesia, immediately after tracheal extubation, and 24 h later. Already before the induction of anesthesia, MO patients had more atelectasis, expressed in the percentage of the total lung area, than nonobese patients (2.1% versus 1.0%, respectively; $P < 0.01$). After tracheal extubation, atelectasis had increased in both groups but remained significantly more so in the MO group (7.6% for MO patients versus 2.8% for the nonobese; $P < 0.05$). Twenty-four hours later, the amount of atelectasis remained unchanged in the MO patients, but we observed a complete resorption in nonobese patients (9.7% versus 1.9%, respectively; $P < 0.01$). General anesthesia in MO patients generated much more atelectasis than in nonobese patients. Moreover, atelectasis remained unchanged for at least 24 h in MO patients, whereas atelectasis disappeared in the nonobese.

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General anesthesia may impair pulmonary gas exchange (1,2), resulting in decreased blood oxygenation (2). Pulmonary atelectasis is a major cause of these detrimental effects (3,4). These pulmonary atelectasis occur in 85%–90% of healthy adults within minutes after the induction of general anesthesia, and up to 15% of the entire lung may be atelectatic, particularly in the basal regions (5), resulting in a true pulmonary shunt of approximately 5%–10% of cardiac output (6).

During general anesthesia (7), as well as during the immediate postoperative period (8), morbidly obese patients (MO) are more likely to have significant impairments of pulmonary gas exchange and respiratory mechanics (9). Because awake MO patients already have severe alterations of their respiratory mechanics (10) (decreased chest wall and lung compliance, decreased functional residual capacity [FRC]), we hypothesized that these patients were particularly prone to intra- and postoperative atelectasis. The aim of our study was, therefore, to compare intra- and postoperative pulmonary atelectasis between MO and nonobese patients until postoperative Day 1.

Methods

After local ethics committee approval and written, informed consent, 30 ASA physical status I–III patients aged 20 to 60 yr and scheduled for a laparoscopic procedure were enrolled into the study. The estimation of the sample size was based on previous studies. This size was calculated to detect a difference of 50% in atelectasis between the groups, with $P = 0.05$ and a power of 80%. Exclusion criteria were previous cardiac or pulmonary disease, except sleep apnea syndrome; carotid stenosis; and a history of vascular neurologic disorders. One group consisted of 20 MO patients (with a body mass index (BMI) of >35 kg/m^2); 10 nonobese patients (BMI <30 kg/m^2) were enrolled into the second group. The scheduled laparoscopic

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procedure was gastric bypass or gastric bending for the MO group and cholecystectomy for the nonobese subjects.

No premedication was given. General anesthesia was induced after 5 min of breathing 100% oxygen with 2 mg/kg of propofol and 0.75 μg/kg of remifentanil during the first 45 s, followed by an infusion of 0.1–0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Maintenance of anesthesia was obtained with desflurane and remifentanil. The dosage of these drugs was adjusted to achieve a clinically adequate depth of anesthesia. During induction, the lungs were ventilated manually via a face mask with 100% oxygen. To facilitate orotracheal intubation, patients received 0.2 mg/kg of cisatracurium; additional doses of 1–4 mg were given when needed. The patients were mechanically ventilated with 50% oxygen in nitrogen with a tidal volume of 10 mL/kg in the nonobese patients and 10 mL/kg of ideal body weight for the MO group. The respiratory rate was adjusted to maintain an end-tidal carbon dioxide concentration of 35–45 mm Hg with an inspiratory/expiratory ratio of 1:2. A positive end-expiratory pressure of 6 cm H_2O was applied in both groups. At the end of surgery, any residual effect of the muscle relaxant was reversed by 2.5 mg of neostigmine and 0.25 mg of glycopyrronium. Postoperative analgesia was provided by 2 g of propacetamol, 30 mg of ketorolac, and 0.1 mg/kg of morphine given 30 min before the end of the surgical procedure. Ten minutes before extubation, all patients were given 100% oxygen. After extubation, all patients were spontaneously breathing with a face mask (providing a fraction of inspired oxygen [F_{IO_2}] of 0.5) for 2 h or more when required. Postoperative analgesics consisted of propacetamol 2 g four times per day and ketorolac 30 mg three times per day. Metamizole 500 mg three times per day was added if needed in both groups.

During the surgical procedure, the peritoneum was insufflated with CO_2 by a WOLF gas insufflator (Treier Endoscopie, Berommunster, Switzerland) up to an intraperitoneal peak pressure of 15 mm Hg. Patients were excluded from the study if the procedure was converted to laparotomy.

Measurements consisted of computed tomography (CT) performed at three different periods: before the induction of general anesthesia, immediately after awakening and tracheal extubation, and 24 h later. Before each CT, a front scout view was obtained, and three sections of 5 mm at 120 kV and 150 mA were acquired at end-expiratory position at the level of the interventricular septum with a lung algorithm (GE Light Speed; General Electric, Milwaukee, WI). The CT data were transferred on a GE Advantage Windows station.

Each right and left lung surface was manually extracted, and a window setting of −1000 to +100 Hounsfield units (HU) was selected to assess the total lung surface. A threshold of −1000 to 500 HU was applied to quantify the amount of normally ventilated lung, a second threshold of −500 to −100 was chosen to establish the surface of poorly ventilated lung, and third threshold of −100 to +100 HU was set to measure the surface of atelectatic lung area. The right and left lung surfaces of pulmonary atelectasis were summed and reported to the total lung surface. The amount of atelectatic tissue was expressed as a percentage of the total lung area (excluding the heart and the great vessels).

Data were compared by using analysis of variance, the unpaired Student's *t*-test, and Dunnett's test. $P < 0.05$ was considered significant. Values are expressed as mean ± SD. The statistical package used was JMP (Version 3.1.5; SAS Institute, Cary, NC).

Results

There were differences between the two groups in the following demographic data: BMI, ASA status, and anesthesia duration (Table 1). The male/female ratio and the age of the patients showed no significant difference between the two groups.

Already before anesthesia induction, there was significantly more atelectasis in MO than in nonobese patients (2.1% ± 1.2% versus 1.0% ± 0.4%, respectively; $P < 0.01$) (Fig. 1). After tracheal extubation, the amount of pulmonary atelectasis had significantly increased in both groups but remained greater in the MO patients (MO patients, 7.6% ± 4.1% versus 2.8% ± 2.2%; $P < 0.05$) (Figs. 1 and 2). Twenty-four hours later, pulmonary atelectasis had returned to baseline in nonobese patients (1.9% ± 0.9%) but remained increased in MO patients (9.7% ± 6.4%; $P < 0.01$) (Figs. 1 and 2). Within MO patients, no correlation was found between BMI and the amount of atelectasis ($R^2 = 0.0042$ after extubation; $R^2 = 0.0001$ 24 h later) (data not shown). Furthermore, when the two groups were pooled, there was no correlation between the BMI and the amount of pulmonary atelectasis at the three different times (before induction, after extubation, and 24 h later). Moreover, there was no correlation between anesthesia duration and the amount of pulmonary atelectasis after the extubation and 24 h later ($R^2 = 0.0211$ and $R^2 = 0.154$, respectively).

Discussion

The major finding of this study is that MO patients develop more atelectasis during general anesthesia than nonobese patients. Moreover, 24 hours after the end of the surgical procedure, atelectasis persists in MO patients, whereas complete resorption occurs in nonobese patients.

Table 1. Patient Characteristics and Procedure

Variable	Morbid obese patients ($n = 20$)	Nonobese patients ($n = 10$)	*P* value
Male/female	4/16	3/7	NS
Body mass index (kg/m²)	46.5 ± 7.0	24.0 ± 2.3	<0.05
Age (yr)	39.5 ± 10.0	47.5 ± 13.7	NS
ASA status (I/II/III)	(0/15/5)	(4/6/0)	<0.05
Anesthesia duration (min)	140.5 ± 54.3	72.6 ± 20.7	<0.001

NS = not significant.

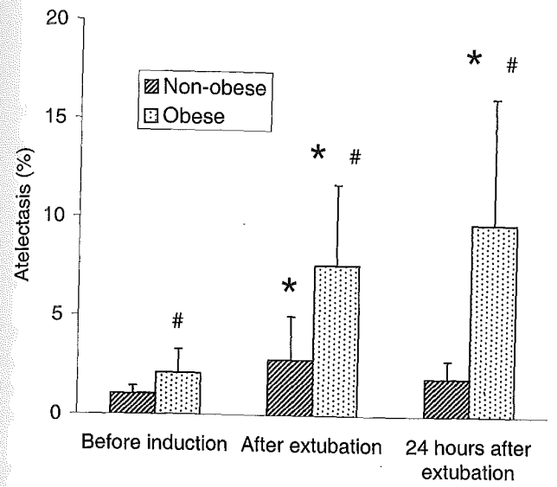

Figure 1. Comparison of the percentage of pulmonary atelectasis between morbidly obese and nonobese patients at the three study times (before anesthesia induction, after extubation, and 24 h later). *$P < 0.05$ compared with before induction (within group); #$P < 0.05$ compared with the control group (between groups).

Only one previous study has evaluated the correlation between obesity and atelectasis formation (11). It found a weak correlation between Broca's index [$weight_{kg}/(height_{cm} - 100)$] and the atelectatic surface ($R^2 = 0.12$; $P < 0.05$). In our study, there was also a weak correlation between the amount of atelectasis and BMI when both groups were pooled, but not when the two groups were studied separately. Because of the design of our study, the BMI was not a continuous variable, and therefore we had to study this correlation in each group.

In general, compared with nonobese patients, functional residual capacity (FRC) is markedly decreased in MO patients, the alveolar-arterial oxygenation gradient is increased, and intraabdominal pressure is higher (7,8,12). This has been shown during general anesthesia and during surgical procedures (12), as well as after surgery in sedated/paralyzed MO patients (7,8). The authors postulated that the hypoxemia and the marked alterations of the mechanical properties of the respiratory system seen in the MO patients were largely explained by a reduction in lung volume because of excessive unopposed intraabdominal pressure (7). This was confirmed by another study showing an improvement of respiratory function in MO patients, but not in nonobese patients, when 10 cm H_2O of positive end-expiratory pressure was applied (13). In our study, the surgical procedure was performed by laparoscopy with an intraperitoneal pressure of 15 mm Hg in both groups. Therefore, the intraabdominal pressure was probably not different between the two groups during the surgical procedure, and it was increased in the MO group only during general anesthesia before and after the surgical procedure. Moreover, it has been shown that the reverse Trendelenburg position, which was the position of both groups for all surgical procedures, improves oxygenation and lung mechanics in MO patients (14). We therefore consider the increased intraabdominal pressure as not only the explanation for the larger amount of atelectasis seen in the MO group, but also the reason why the decreased lung volume already shown in these patients (12) also participated in atelectasis formation.

The difference seen between the two groups at 24 hours in our study may have several explanations. First, it has been shown that FRC in MO patients was diminished and even worsened when they were in the supine position (15). This may explain why, in our study, before general anesthesia, MO patients already had more atelectasis than lean patients. Therefore, during the first postoperative night, this phenomenon will recur with persisting or even increased atelectasis surface. Second, despite the same type of surgical procedure, MO patients generally remain longer and are more immobilized in their beds than nonobese patients. Indeed, it is easier for the lean patients to mobilize themselves and for the nursing staff to stimulate them. Early mobilization certainly contributed to the rapid disappearance of atelectasis in the nonobese group. Third, the surgical procedure was twice as long in the MO group than in the nonobese group. However, no correlation was found between the duration of surgery and the percentage of atelectasis, which is confirmed by two studies showing no progressive decrease of the lung volume in normal-weight and obese subjects with the duration of general anesthesia

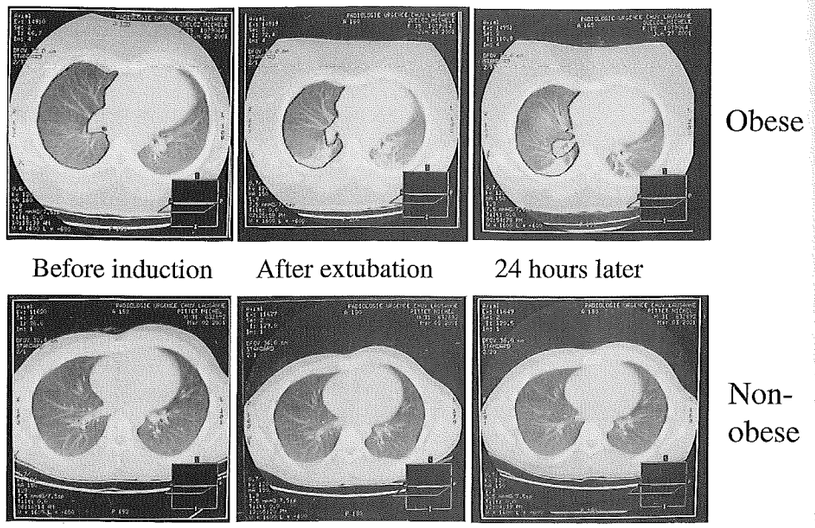

Figure 2. Samples of computed thoracic tomography scans of one morbidly obese and one nonobese patient before anesthesia induction, after extubation, and 24 h later. These slices were realized at the level of the interventricular septum.

(16,17). Therefore, the duration of the surgical procedure is not a main factor explaining atelectasis formation. Fourth, analgesia was the same in both groups. Because no postoperative opiates were given, postoperative respiratory depression might not have resulted from the analgesic regimen. Although the same surgical procedure was performed in both groups, one might argue that laparoscopic gastric bypass, or banding versus laparoscopic cholecystectomy, would produce a different intensity of postoperative pain. This difference between the two groups would then explain the difference in the amount of atelectasis seen 24 hours after tracheal extubation. Nevertheless, previous studies investigating different postoperative analgesic techniques, to improve respiratory efficiency, failed to show any or showed only a minor benefit of improving postoperative pain (18,19).

Consequently, the above-mentioned causes seem to explain only partially the difference seen at 24 hours. Therefore, the most likely explanation for this difference is obesity by itself.

The method of atelectasis measurement by CT scan is established (5,6). To avoid excessive radiation exposure, only the level of the interventricular septum was chosen. The interventricular septum level may not be representative of the whole lung, but it appeared to be a compromise between the most affected bases of the lungs and the less affected apex.

F_{IO_2} was similar in both groups during general anesthesia and the immediate postoperative period. The MO patients needed a supplement of oxygen by face mask for a longer period of time (F_{IO_2} 0.5). This longer exposure to 50% oxygen may have contributed to the longer persistence of atelectasis in the MO group. Low F_{IO_2} has no influence on atelectasis formation (20), and, therefore, the longer exposure to 50% oxygen had no effect on the amount of atelectasis seen in our study.

The increased amount of atelectasis found in MO patients explains, at least partially, postoperative pulmonary complications. Therefore, it seems important to further investigate any techniques for avoiding atelectasis formation in this high-risk group.

We conclude that pulmonary atelectasis appearing during general anesthesia will resolve in nonobese patients within hours. However, MO patients will already show atelectasis on the morning of surgery before any medication has been given. They will develop much more atelectasis, which will persist and even tend to increase, 24 hours after tracheal extubation.

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