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Review article

Atelectasis during anesthesia: Can it be prevented?

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Introduction

Anesthesia is regularly accompanied by an increased venous admixture that averages 8% [1], but that can exceed 25%–30% in some cases. The venous admixture can be further analyzed by the multiple inert gas elimination technique, which allows the construction of a virtually continuous distribution of ventilation/perfusion ratios ($\dot{V}_A/\dot{Q}$). Thus, the major feature during anesthesia is shunt, i.e., perfusion of essentially non-ventilated lung units, and sometimes lung regions that are poorly ventilated in comparison to their blood flow ("low $\dot{V}_A/\dot{Q}$") [2–4].

Atelectasis during anesthesia

Ten to fifteen years ago, atelectasis was demonstrated in anesthetized patients, including neonates as well as adults [5,6]. The atelectasis can be demonstrated by computed X-ray tomography but is invisible on conventional chest X-ray (Fig. 1). It is located in the most dependent parts of both lungs and appears in almost 90% of all patients who are anesthetized [7]. It develops whether the anesthesia is intravenous or inhalational, and whether the patient is breathing spontaneously or is paralyzed and ventilated mechanically [8]. The atelectasis is largest near the diaphragm in the supine patient and decreases in size towards the apex [9]. A three-dimensional reconstruction of the atelectasis is shown in Fig. 2. The atelectasis covers approximately 5% of the transverse pulmonary area near the diaphragm, with a large variation from zero to 10%–15%. In the average patient, the atelectasis may not look too impressive. However, it should be remembered that the collapsed area comprises 4-times more lung tissue than the aerated regions. Thus, in the average patient the atelectasis comprises about 15%–20% of the lung tissue near the diaphragm and about 10% of the total lung tissue [9]. In extreme cases almost half the lung can be collapsed during anesthesia, before any surgery has taken place!

The magnitude of the shunt correlates well with the size of the atelectasis [10]. That the shunt is located in dependent lung regions corresponding to the location of atelectasis has also been demonstrated by single photon emission computed tomography (SPECT) [11]. An interesting finding is that the atelectasis does not increase with the age of the patient, nor does the shunt increase with age [12]. This may appear surprising, since gas exchange impairment mostly worsens when the patient gets older [13]. However, perfusion of poorly ventilated lung regions ("low $\dot{V}_A/\dot{Q}$") increases with age [12]. The dependence of the shunt and low $\dot{V}_A/\dot{Q}$ on age is shown in Fig. 3. It can also be seen that venous admixture increases with age, since it will be affected both by the shunt and low $\dot{V}_A/\dot{Q}$. The cause of low $\dot{V}_A/\dot{Q}$ has not been fully established. However, airway closure increases with age and may be a major contributor to impaired ventilation [14]. Moreover, closure of airways above functional residual capacity (FRC), i.e., during an ordinary breath, seems to be more common during anesthesia than in the awake state [15], although some controversy exists.

Causes of atelectasis

The atelectasis appears promptly after induction of anesthesia, or can be seen as soon as it has been possible to make a computed tomographic (CT) scan [6]. Moreover, positive end-expiratory pressure (PEEP) can reopen collapsed lung tissue, but as soon as PEEP is discontinued, the atelectasis reappears within 1 min [16]. The rapid formation of atelectasis after induction of anesthesia and discontinuation of PEEP may suggest that a major cause of atelectasis is the compression of lung tissue rather than slow absorption of gas behind

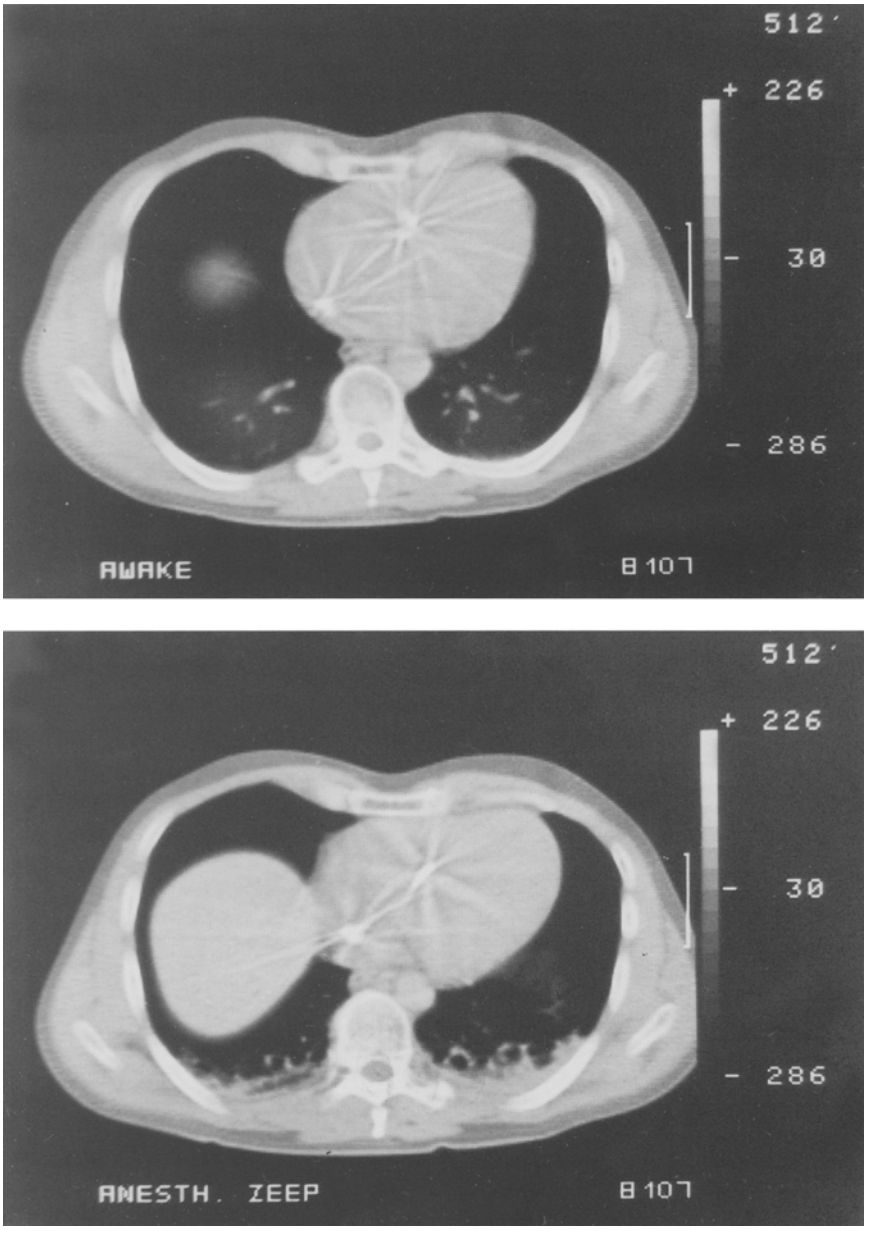

Fig. 1. Computed tomographic scans of a patient's lungs (at a level above the right hemidiaphragm) while awake (*top panel*) and during general anesthesia with mechanical ventilation (*bottom panel*). Note the appearance of atelectasis in the dependent part of the lungs after induction of anesthesia

occluded airways. A similar conclusion was drawn by Morimoto et al. [17] in a study of anesthetized, mechanically ventilated rabbits. They injected air into the abdomen (pneumoperitoneum) in order to displace the diaphragm cranially and to reduce lung volume. This caused atelectasis in dependent lung regions which the authors called "gravity-dependent atelectasis". In a subsequent study, Tomiyama et al. [18] demonstrated by means of xenon wash-out technique that the dependent lung regions in the rabbit model were rapidly ventilated just prior to their collapse. The authors concluded that such ventilation fitted with reduced alveolar volume, followed by collapse, rather than airway closure and absorption of gas behind occluded airways. In humans anesthetized with ketamine that allowed the maintenance of respiratory muscle tone, no atelectasis developed until the patient was paralyzed and mechanically ventilated [19]. Tensing the diaphragm by phrenic nerve stimulation reduced the atelectasis in anesthetized patients [16]. All these findings fit with the concept of compression or gravity-dependent atelectasis.

However, two recent observations have made the explanation of atelectasis formation during anesthesia more complex. First, collapsed lung tissue can be re-expanded by a vital capacity maneuver (see also below), but if the lungs are ventilated with pure oxygen they rapidly re-collapse within 5 min after the vital capacity maneuver [20]. If, on the other hand, the lungs are

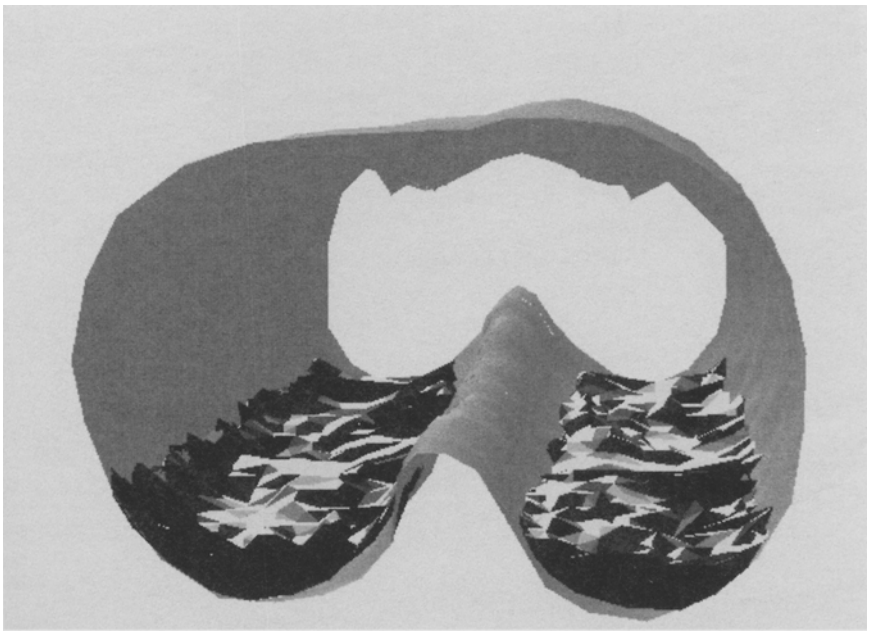

Fig. 2. Three-dimensional reconstruction of the chest wall and the dependent atelectatic regions of the lungs from base to apex. Note that the atelectasis decreases towards the apex

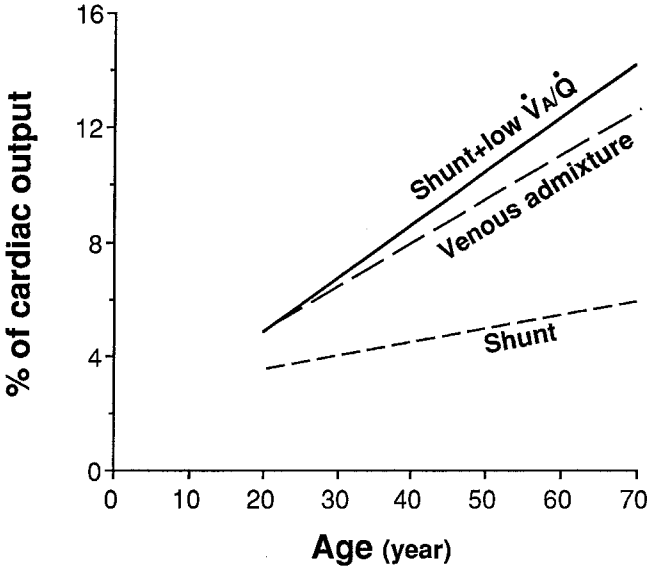

Fig. 3. Shunt, the sum of shunt and perfusion of regions with low ventilation/perfusion ratios ($\dot{V}_A/\dot{Q}$), and venous admixture against age, during anesthesia. Note the appearance of increased shunt during anesthesia (independent of age) and the increasing perfusion of low $\dot{V}_A/\dot{Q}$ regions with age both awake and during anesthesia. From [12] with permission

ventilated with 40% O_2 in nitrogen after the re-expansion, the lungs remain open with little or no atelectasis formation for half an hour or longer. Second, if anesthesia is induced without "preoxygenation" and ventilation is given with 30% O_2 in nitrogen, little or no atelectasis is formed [21]. These observations underscore the importance of the inspired oxygen fraction, which suggests that the rate of absorption of gas from the alveoli may play an important role in the formation of atelectasis. However, the breathing of oxygen alone for more than half an hour does not promote atelectasis [6]. If the oxygen breathing is done at a reduced lung volume, atelectasis may ensue, as suggested by the reduction in compliance and oxygenation in healthy volunteers during chest strapping [22]. Also, ketamine anesthesia did not promote atelectasis despite induction during oxygen breathing, as mentioned above [19]. Ketamine has been shown not to reduce FRC [23]. Thus, for atelectasis to occur during anesthesia, there must be both a reduced respiratory muscle tone with lowered FRC and ventilation with high fractions of oxygen, at least for a period of time. Atelectasis is thus an effect of both the compression of lung tissue and resorption of gas.

The rapid collapse of lung tissue after discontinuation of PEEP and the long-lasting effect of a vital capacity maneuver on the patency of the lung tissue may appear as contradictory consequences of two re-expansion maneuvers. The cause is not clear, but a possible mechanism is the effect of surfactant on the stability of the alveoli. Thus, we hypothesize that a lung region that has been collapsed and reopened by PEEP is inherently unstable because of lack or inactivation of surfactant in the previously collapsed region. When the PEEP is discontinued the region rapidly re-collapses. The effect of a vital capacity maneuver is different. It has been shown that the production or release of surfactant is increased by a large inflation of the lung, and it may also be that the surfactant is squeezed out onto the alveolar and terminal bronchial walls [24]. By this means the lung is not only reopened, but it has also become more stable.

In summary, there seem to be rather complex and interactive mechanisms behind atelectasis formation during anesthesia. These mechanisms are loss of respiratory muscle tone causing reduced FRC and promoting the transmission of abdominal pressure into the thoracic cavity, gas resorption in small alveolar units, and impaired surfactant function in previously collapsed lung regions.

Prevention of atelectasis during anesthesia

The procedures that can be followed in order to prevent atelectasis or to reopen collapsed tissue are much the same as have been discussed in the section on the causes of atelectasis. However, here they will be analyzed with a view to their feasibility in clinical praxis and possible negative effects. The procedures that will be discussed are: (1) PEEP, (2) maintenance or restoration of respiratory muscle tone, (3) recruitment maneuvers, and (4) minimization of pulmonary gas resorption.

PEEP

The application of PEEP of 10 cm H_2O has been tested in several studies and will consistently reopen collapsed lung tissue [6,10,16]. However, some atelectasis persists in most patients. Further increase in the PEEP level may have reopened this tissue. However, PEEP appears not to be the ideal procedure. First, shunt is not reduced and the arterial oxygenation not improved on average in larger groups. This was already demonstrated in 1974 by Hewlett et al., who warned against the "indiscriminate use of PEEP in routine anesthesia" [25]. The maintenance of shunt may be explained by the redistribution of blood flow towards the most dependent parts when intrathoracic pressure is increased, so that any persisting atelectasis in the bottom of the lung receives a larger share of the pulmonary blood flow than without PEEP [26]. The increased intrathoracic pressure will also impede venous return and lower cardiac output. This results in a lower venous oxygen tension for a given oxygen uptake, which will augment the desaturating effect of shunted blood and perfusion of poorly ventilated regions on the arterial oxygenation [27]. Second, the lung re-collapses rapidly after discontinuation of PEEP. Within 1 min after this cessation of PEEP, the collapse is as large as it was before the application of PEEP [16]. This instability of the lung may be attributed to impeded surfactant function, as discussed above. This means that in order to bring the patient over the perioperative period without lung collapse, PEEP must be maintained without interruption. PEEP should also be maintained during the wake-up and early postoperative periods.

Maintenance of muscle tone

The use of an anesthetic that allows maintenance of respiratory muscle tone will prevent atelectasis formation. As mentioned above, ketamine does not cause atelectasis as long as it is used as the only anesthetic agent or in combination with other drugs that do not interfere with the respiratory muscle function. However, if muscle relaxation is required, atelectasis will appear as with other anesthetics [19].

Another possibility is to restore respiratory muscle function which can be achieved, at least partly, by diaphragm pacing. This was tested by applying phrenic nerve stimulation, which did reduce the atelectatic area [16]. However, the effect was small and it can be argued that the technique is too complicated to become routine during anesthesia and surgery [19].

Recruitment maneuvers

The use of a sigh maneuver, or a double tidal volume, has been advocated to reopen any collapsed lung tissue [28]. However, the atelectasis is not affected by an ordinary tidal volume to an end-inspiratory airway pressure of 10 cmH_2O nor by a deep sigh with an airway pressure to +20 cmH_2O [29]. Not until an airway pressure of 30 cmH_2O was reached did the atelectasis decrease to approximately half the initial value. For a complete reopening of all collapsed lung tissue an inflation pressure of 40 cmH_2O was required, and the breath was held for 15 s [29]. Such a large inflation and subsequent expiration down to −20 cmH_2O corresponded to a vital capacity measured during spontaneous breathing with the patient awake. Although approved for lung function studies in anesthetized subjects [30], it may be argued that such a maneuver carries the risk of causing baro/volo-trauma [31]. Another procedure was therefore tested with repeated inflations of the lung to an airway pressure of +30 cmH_2O. However, this caused only minor further opening of lung tissue after the first maneuver [29]. A full vital capacity maneuver with an inflation to +40 cmH_2O therefore seems necessary to completely reopen the lung.

Minimizing gas resorption

As mentioned above, ventilation of the lungs with pure oxygen after a vital capacity maneuver that had reopened previously collapsed lung tissue resulted in a rapid reappearance of atelectasis [20]. If, on the other hand, ventilation was made with 40% O_2 in nitrogen, atelectasis reappeared slowly and even 40 min after the vital capacity maneuver only 20% of the initial atelectasis had reappeared (Fig. 4). Thus, ventilation during anesthesia should be done with a moderate in-

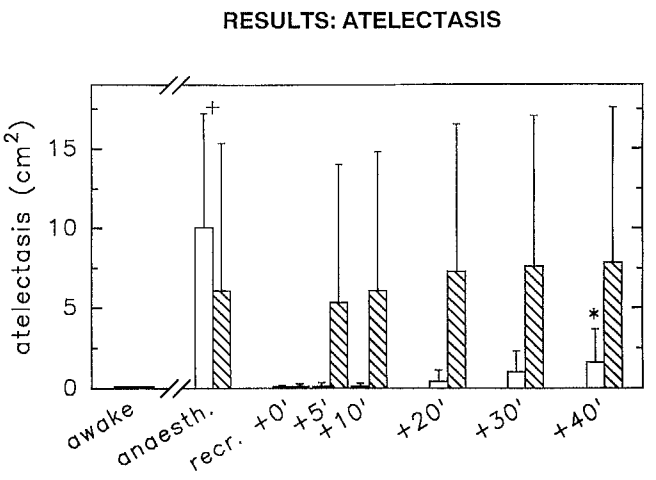

Fig. 4. Atelectasis in anesthetized and mechanically ventilated patients before and for 40 min after a recruitment maneuver (inflation of the lung to an airway pressure of +40 cmH_2O and breath holding for 15 s, followed by an expiration to functional residual capacity). The *open columns* show the results of a recruitment maneuver with air in 6 patients followed by 40 min of ventilation with a fractional inspiratory oxygen (FiO_2) of 0.4. The *hatched columns* show a recruitment maneuver followed by ventilation with 100% oxygen in another 6 patients. Note that after the recruitment maneuver, there was essentially no atelectasis in either group. With an FiO_2 of 0.4, atelectasis returned slowly. In the oxygenated group, atelectasis reappeared 5 min after the recruitment maneuver to almost the same extent as before the inflation. $^*P = 0.012$; $^+P = 0.4$. Reproduced from [20] with permission

spired oxygen fraction (e.g., 0.3–0.4) and be increased only if arterial oxygenation is compromised.

Moreover, avoidance of the preoxygenation procedure during induction of anesthesia more and less eliminated the atelectasis formation during anesthesia [21]. If the preoxygenation period was prolonged from a standard 2–3 min to 4–5 min, atelectasis increased further in size [32]. Thus, avoidance of the preoxygenation or at least lowering of the inspired oxygen fraction during the induction phase will reduce or avoid the formation of atelectasis during the subsequent anesthesia. It is obvious that the lowering of the inspired oxygen fraction may increase the risk of hypoxemia in a difficult and prolonged intubation. However, the present findings call for a re-evaluation of the present standard procedures for inducing anesthesia. It may be that induction of anesthesia with an inspired oxygen fraction of 50%–80% is enough to ensure safe oxygenation. Moreover, the obligatory use of pulse oximeters during anesthesia in many countries makes it easy to detect dangerous hypoxemia. A vital capacity maneuver immediately after the intubation of the airway, followed by new vital capacity maneuvers every 30–40 min or so, will keep the lung open during the anesthesia and into the postoperative period. It is the opinion of the author that such concepts deserve the attention of the anesthetists and that they be tested in a larger trial.

In summary, atelectasis is produced in most patients during anesthesia and is a major cause of impaired oxygenation. The causative mechanisms seem to be loss of respiratory muscle tone and gas resorption. Avoidance of high inspired oxygen fractions during induction and maintenance of anesthesia, and intermittent "vital capacity" maneuvers prevent or reduce the atelectasis formation.

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