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Atelectasis during General Anaesthesia

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ABSTRACT

Gas exchange is regularly impaired during general anaesthesia. A major cause of this deterioration appears to be atelectasis. It was shown to be present in most adults immediately after the induction of general anaesthesia, and to result in pulmonary shunt.

In a series of investigations, the influence of gas composition on the formation of atelectasis and on gas exchange during the induction of general anaesthesia was studied. Furthermore, possible manoeuvres to re-expand atelectatic lung tissue during ongoing general anaesthesia with mechanical ventilation were investigated.

It was found that the composition of inspiratory gases plays an important role in the formation of atelectasis during induction of general anaesthesia in adults with healthy lungs. By using a lower than usual oxygen concentration in nitrogen, the early formation of atelectasis may be avoided. During ongoing general anaesthesia with mechanical ventilation, the amount of atelectasis remains unchanged during normal tidal breathing, but atelectatic lung tissue may be re-opened by a hyperinflation of the lungs. The composition of inspiratory gas plays an important role in the recurrence of collapse of the re-expanded lung tissue, too. Atelectasis can be seen within five minutes, if 100% oxygen is used. With low concentrations of oxygen in nitrogen however, most of the re-expanded lung tissue remains inflated for at least 40 minutes and the oxygenation of arterial blood is slightly improved.

INTRODUCTION

Gas exchange is regularly impaired during general anaesthesia with mechanical ventilation[1)2)]. This may result in a decreased oxygenation of blood[3)4)]. A major cause of this derangement appears to be atelectasis[5)], which cannot be seen on conventional chest x-rays[6)], but was demonstrated by computed tomography (CT) of the chest[7)8)].

Immediately after the induction of general anaesthesia, densities may be found in dependent parts of the lungs, both with spontaneous breathing[9)] and mechanical ventilation[8)10)]. It has been shown in animal experiments that these densities correspond to collapsed lung tissue[11)], they are therefore usually termed "atelectasis". Note that the Greek words "ateles" and "ektasis" signify "incomplete" and "extension", respectively[12)]. Strictly speaking, this refers to incomplete expansion of lung tissue in the newborn, only. However, "atelectasis" may also be used more loosely to describe "collapse of lung tissue"[13)].

Calculations show that 10-20% of the lung tissue in basal regions, i.e. adjacent to the

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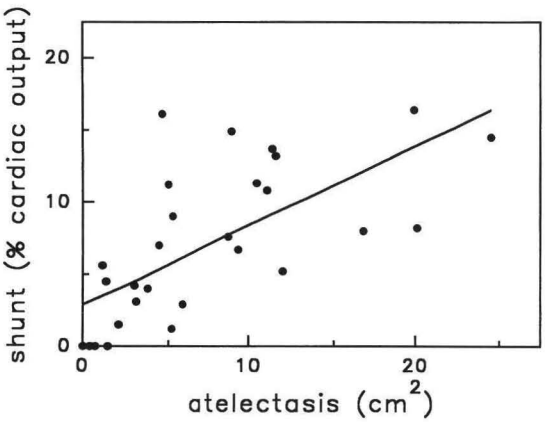

Figure 1 Atelectasis and pulmonary shunt during general intravenous anaesthesia (summary of data from previously published studies [15)51)52)]).

Atelectasis is estimated as densities (+100 to-100 HU) in a CT-scan 1 cm above the dome of the right diaphragm

shunt : perfusion to lung units with $V_A/Q < 0.005$

CO : cardiac output

A linear regression line is shown : shunt (% CO) = 2.9 + 0.6 * atelectasis (cm^2) ; R = 0.68, P = 0.0001

diaphragm, may be atelectatic[10]. Collapsed lung tissue is found in 85-90% of anaesthetized adults[10], and obese patients tend to have more atelectasis than lean ones[14)15)]. During ongoing anaesthesia, the amount of collapsed lung tissue increases slowly with time, only[16]. Atelectasis in turn causes pulmonary shunt (**fig. 1**)[8)17)18)]. It thus appears as if the early formation of atelectasis and pulmonary shunt were an unavoidable "adverse effect" of anaesthesia.

In addition to pulmonary shunt, impaired matching of ventilation and perfusion, becoming more important with age[18], contributes to the deterioration of the oxygenation of blood. The ventilation-perfusion mismatch can hardly be explained by atelectasis but requires another explanation. This may be airway closure, impeding ventilation of lung units. Airway closure was initially demonstrated in awake subjects[19] and was shown to increase in magnitude with age[20)21)]. It has also been demonstrated in anaesthetized subjects[22)-25)], but its extent and its influence on gas exchange have not been fully agreed upon. In this short review, only atelectasis and pulmonary shunt will be considered in more detail.

In summary,

Atelectasis during anaesthesia includes :
- 5-8% of lung surface in a CT-scan close to the diaphragm
- 10-20% of the total lung tissue
- 5-8% of lung blood flow

Atelectasis :
- develops promptly during induction of general anaesthesia
- increases slowly with time during ongoing anaesthesia

Poorly ventilated lung units (low V_A/Q)
- may be caused by airway closure or a change in regional ventilation.

MECHANISM OF ATELECTASIS FORMATION

Why does atelectasis occur? Three basic mechanisms have been proposed[26] : 1) Compression of lung tissue, 2) resorption of gas, 3) decreased function (or production) of surfactant.

Compression of lung tissue : With induction of general anaesthesia, there is a cranial shift of the diaphragm[27)28)] and a change in the rib cage configuration[29)-31)]. This may result in a compression of the most dependent parts of the lungs. Such atelectasis may therefore also be called gravity-dependent atelectasis[32]. There is more pulmonary shunt during general anaesthesia in obese patients[33], and this may be explained by a larger cranial-shift of the dia-

phragm in such patients, causing more atelectasis by compression. Vice versa, a caudal-shift of the diaphragm by phrenic nerve stimulation (PNS) during Halothane anaesthesia reduced the size of atelectasis[34].

Resorption of gas：It has been known for many years that a lung will collapse if it is not ventilated[35]. Moreover, breathing pure oxygen, may result in collapse of lung units with a low ventilation-perfusion ratio[36]. This holds true for other easily resorbed gases such as e.g. nitrous oxide (N_2O)[37)-40]. If the induction of anaesthesia results in an increased amount of lung units with no or poor ventilation[41)-43], and if such lung units are filled with an easily resorbed gas such as oxygen or nitrous oxide, this may result in lung collapse. The time until collapse of an entirely closed off lung unit has been estimated to be 6-9 hours if the unit contains air (21% oxygen in nitrogen)[40], whereas the time is about 3 hours if the unit contains 30% oxygen in nitrogen[44] and about 8 minutes if this unit contains 100% oxygen[40]. Because of the high solubility of N_2O, a lung unit may collapse even faster if it contains a mixture of N_2O and O_2[40].

Decreased function of surfactant：A change in the surface tension (e.g. caused by a lack of surfactant or by saline lavage) may cause alveolar instability and result in collapse of lung units[45)46]. Accordingly, a decreased function of surfactant, caused by general anaesthesia[47)48] or mechanical ventilation[48)-50], may result in atelectasis.

Because we were surprised by the rather slow reappearance of atelectasis after a re-expansion manoeuvre using air[51], the role of resorption of gas was analyzed in more detail. As a first step, the reappearance of atelectasis *during ongoing general anaesthesia* was studied in adults with healthy lungs[52].

Atelectasis was studied by computed x-ray tomography[8)10], and the ventilation-perfusion distribution (V_A/Q) was assessed with the multiple inert gas elimination technique[53)54]. Mechanical ventilation with a tidal volume of about 10 ml・kg^{-1} and no PEEP was used. To re-expand atelectasis, the lungs were inflated with air to an airway pressure of 40 cmH_2O (see below). This inflation was held for 15 seconds.

In patients, whose lungs were ventilaned with 40% oxygen in nitrogen, most of the re-expanded lung tissue remained inflated for at least 40 minutes. The pulmonary shunt was reduced by the recruitment manoeuvre, but low V_A/Q increased, with only a small but significant reduction of P_A-aO_2 as net effect. If 100% oxygen was used however, lung collapse reappeared within a few minutes, and as compared to pre-recruitment, V_A/Q was unchanged (**fig. 2**). This time course of atelectasis suggests that *resorption of gas* plays an important role in the recurrence of collapse in previously re-expanded atelectatic lung tissue. Such lung regions appear to be unstable, and the mechanisms that keep a lung unit open possibly are overcome by the forces caused by gas resorption. Further evidence that atelectasis is caused by resorption of gas, are the results of measurements of intravascular angles in non-dependent parts of the lungs. These angles were decreased by the recruitment manoeuvre, and they increased again thereafter. This indicates that competition for space may cause a compression of non-dependent parts of the lungs when dependent parts are expanded and an expansion of non-dependent 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 or even hyperinflated[11)55], thus fitting with a hypothesis of interdependence

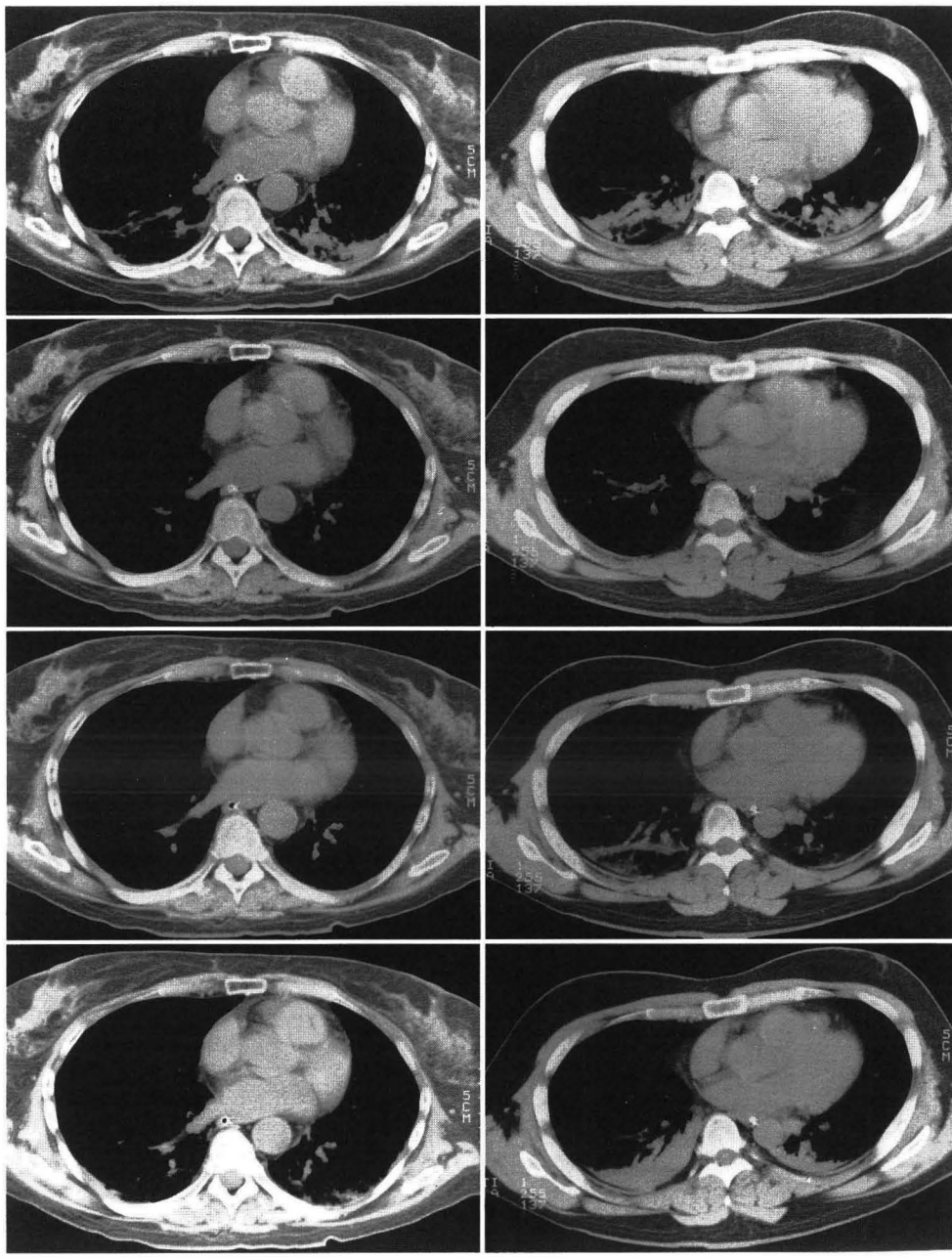

Figure 2 Computed tomography (CT) of the lungs in two patients. The CT was positioned in the supine patient one centimeter above the right diaphragm, and all scans were taken at end-expiration.

Window：500 HU (Hounsfield units), center-150 HU.

Left row：$FI_{O_2}=0.4$ before and after recruitment manoeuvre.

Right row：$FI_{O_2}=0.4$ before recruitment manoeuvre, $FI_{O_2}=1.0$ during and after recruitment manoeuvre.

Top panel：after induction of anesthesia, second panel：immediately after recruitment, third panel：5 minutes after recruitment, bottom panel：40 minutes after recruitment

Note the early reappearance of atelectasis in the patient receiving inspired gas with $FI_{O_2}=1.0$.

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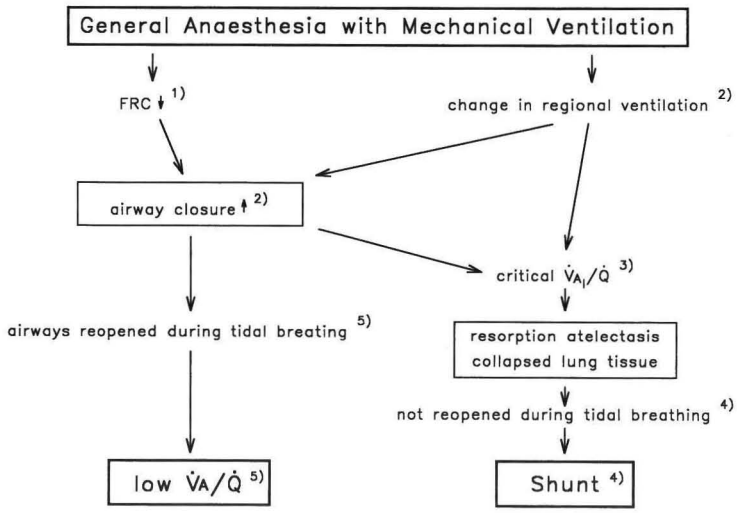

Figure 3 Putative mechanisms leading to hypoxaemia during general anaesthesia with mechanical ventilation. Explanations, see text.

between adjacent lung units.

In a further study, the influence of gas composition upon the initial formation of atelectasis *during induction of general anaesthesia* was analyzed[56]. The same methods as described above were used for that investigation. Interestingly, only a very small amount of atelectasis was found after the induction of general anaesthesia in subjects whose lungs were ventilated with 30% oxygen in nitrogen. Furthermore, the amount of atelectasis during general intravenous anaesthesia increased about three times more slowly, when 30% oxygen in nitrogen was used, compared to 100% oxygen. Thus, during the induction of general anaesthesia in patients with healthy lungs, *gas composition* plays an important role for atelectasis formation and the changes in ventilation-perfusion relationships. By using a mixture containing a poorly resorbed gas such as 30% oxygen in nitrogen, the early formation of atelectasis may be avoided.

Based on the above considerations, the following model of the relationships between the impairment of gas exchange, atelectasis, and airway closure is proposed (**fig. 3**)：

1) General anaesthesia results in a decrease of functional residual capacity (FRC)[57].

2) Concomitantly, there is an increased amount of airway closure during normal tidal breathing, resulting in an increased amount of poorly ventilated lung units[22)23)41].

3) Whether a lung unit collapses, depends primarily on its V_{A_I}/Q and the composition of the gas used for its ventilation[39)52)56]. A further factor may be the property of surfactant[58].

4) Once collapsed, the lung unit will stay atelectatic during normal tidal breathing (see below), and pulmonary shunt will ensue[8)18].

5) Closed airways on the other hand may be reopened during normal tidal breathing[59], and low V_A/Q may reflect such poorly ventilated lung units.

In summary,

During anaesthesia：

- atelectasis results from an increased amount of poorly ventilated lung units or lung units excluded from

ventilation with subsequent resorption for gas.

- An imbalance of forces retracting and expanding lung tissue and a dysfunction of surfactant may be contributing factors.

PREVENTION AND TREATMENT OF ATELECTASIS DURING GENERAL ANAESTHESIA

What conclusions may be drawn, based on the above presented findings?

Atelectasis during induction of general anaestesia

In clinical practice, the lungs are often ventilated with 100% oxygen during the induction of general anaesthesia. Although on long lasting severe hypoxemia was seen in our study when 30% oxygen in nitrogen was used during the induction of general anaesthesia, we are well aware of the risks of omitted ventilation with 100% oxygen. As discussed recently, preoxygenation increases the margin of safety[60)61)]. On the other hand, this appears to be paid for by promoting lung collapse, which occurs in 85% to 90% of all adults and results in pulmonary shunt. Our findings suggest that using 100% oxygen during a standard anaesthesia induction should be reevaluated and alternative approaches should be looked for. Alternatively, a re-expansion by manual hyperinflation should be considered, when atelectasis is presumed to be the main cause of a disturbed gas exchange during general anaesthesia with mechanical ventilation[15)]. Such a situation could be for example at hand, when hypoxemia is present during general anaesthesia in an adipose patient, where the development of a large amount of atelectasis and shunt can be expected[15)33)].

Atelectasis during ongoing general anaesthesia

Already several years ago, measures to treat atelectasis during mechanical ventilation or general anaesthesia have been suggested. In 1930, intermittent hyperventilation was proposed to avoid "hypostatic congestion" and bronchopneumonia in mechanically ventilated patients (at that time, the patients were ventilated in a negative pressure chamber!)[62)]. Another early study, emphasizing the importance of intermittent deep breaths, was published by Mead and Collier in 1959[63)], and one should not forget the often cited investigation by Bendixen et al (1963), stressing again the application of intermittent deep breaths[5)]. In addition, a single deep breath may result in the release of surfactant[64)65)]. This may contribute to an improved alveolar stability, prevent lung collapse and result in an increased compliance of the lungs.

As a consequence, a 'sigh'-function is still an option in some anaesthesia-respirators[66)]. However, no advantage in terms of Pa_{O_2} and compliance could be found by routinely administering sighs to patients on prolonged mechanical ventilation[67)68)]. And, above all, it is not known at what extent of inflation the amount of atelectasis may be reduced by passive inflation of the lungs. This question was addressed recently[15)]. In that investigation, the lungs were inflated either with a "stepwise procedure" up to an airway pressure (P_{aw}) of 10, 20, 30 and finally 40 cmH_2O or with a "repeated procedure" three times up to a P_{aw} of 30 and finally up to 40 cmH_2O. Each inflation was held for 15 seconds and between each inflation, the lungs were ventilated for 1-2 minutes with the baseline settings of the ventilator.

It was found that the amount of atelectasis is not changed during normal tidal breathing, nor with a double tidal volume ('sigh', P_{aw}=

20 cmH_2O). However, atelectasis was re-expanded by an inflation of the lungs up to an airway pressure of 40 cmH_2O, held for 15 seconds. The volume, needed for an inflation up to a P_{aw} of 40 cmH_2O corresponds to the vital capacity awake, supine. We therefore propose to call this inflation up to P_{aw} of 40 cmH_2O a "vital capacity manoeuvre". Remember that a pressure of 4-5 cmH_2O, only, is required to open a closed airway of about 1 mm inner diameter[69)70)]. Furthermore, the characteristics of the opening-pressure vs. diameter relationship also are dependent on the properties of surfactant[58)70)]. Obviously, once a lung unit is collapsed (atelectatic), the pressure necessary to open it is higher than the pressures, which are necessary to open collapsed airways. This may have implications when comparing effects of airway closure and atelectasis on gas exchange and when analyzing measures (e.g. PEEP or changes in tidal volume) to influence atelectasis and airway closure.

Does the favorable effect of a vital capacity manoeuvre on atelectasis have a clinical application? Two possible complications have to be borne in mind: Barotrauma and disruption of the alveolo-capillary barrier. The possibility of a barotrauma may not completely be excluded, however it should be stressed that the amount of inflation, as used in this study, is in accordance with the guidelines proposed by Leith[71)]. Concerning the alveolo-capillary barrier, it has recently been shown that in small animals periods of hyperinflation as short as two minutes may alter the microvascular permeability, resulting in an increased extravascular lung water[72)]. Hyperinflation was defined in that study as ventilation to a preset peak airway pressure of 35 mmHg (=48 cmH_2O), corresponding to a tidal volume of 46 $ml \cdot kg^{-1}$. However, it is not known at which amount of inflation clinically significant alterations of the lung tissue occur in human adults[73)]. Furthermore, the vital capacity manoeuvre covers a volume not larger that vital capacity awake, supine. We therefore believe, that such a manoeuvre still may be used in lung healthy adults, having an adequate clinical indication. Such a situation could be a singnficant hypoxemia during general anaesthesia in an adipose patient, where the development of atelectasis is rather probable[15)].

In summary,

In adults with healthy lungs:

- during induction of general intravenous anaesthesia, the composition of inspiratory gases plays an important role in the formation of atelectasis;
- during ongoing anaesthesia with mechanical ventilation, the amount of atelectasis remains unchanged during normal tidal breathing, but
- atelectatic lung tissue may be re-expanded by a hyperinflation of the lungs, and
- the composition of inspiratory gas plays an important role in the recurrence of collapse of previously re-opened lung tissue.

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