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New concepts of atelectasis during general anaesthesia

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At the beginning of the last century, Pasteur described postoperative pulmonary atelectasis,[58] analysed postoperative pulmonary complications (PCC; see below) and noted: 'when the true history of postoperative lung complications comes to be written, active collapse of the lung, from deficiency of inspiratory power, will be found to occupy an important position among determining causes'.[59] Indeed, atelectasis occurs regularly during general anaesthesia induction,[46] persists postoperatively[44] and may contribute to significant morbidity[7 8] and additional healthcare costs.[43]

This review article will review the mechanism of perioperative atelectasis, discuss its clinical significance and describe preventive measures.

Gas exchange and general anaesthesia

In 1964, Nunn[56] showed that during routine anaesthesia and spontaneous ventilation, gas exchange was altered by shunt and uneven ventilation perfusion ratios. He concluded that to ensure the maintenance of a normal arterial P_{O_2}, the alveolar P_{O_2} had to be as high as 200 mm Hg and this required an inspired oxygen concentration ($F_{I_{O_2}}$) of 35%. Since then it has been commonly accepted that all general anaesthesia should use at least 30–35% oxygen.

Atelectasis and general anaesthesia

Atelectasis was early suspected as a cause of impaired oxygenation during general anaesthesia. Bendixen and colleagues[5] postulated that spontaneous ventilation without periodic deep breaths may lead to progressive atelectasis, with increased shunting and decreased pulmonary compliance, and that these changes were reversible by hyperinflation of the lungs. They showed that general anaesthesia without supplemental oxygen reduced $P_{a_{O_2}}$ by 22% and compliance by 15% and that three successive hyperinflations of the lungs restored both arterial oxygen tension and lung compliance to control values, suggesting that periodic deep breaths prevented progressive atelectasis and shunting.

In the 1980s, atelectasis was shown by computed tomography (CT) in anaesthetized patients—neonates as well as adults.[7 14] Lung densities were seen in anaesthetized children and were called 'confluent high absorptive areas' but such areas were not found in the scans performed under sedation.[14] In 1985, Brismar and colleagues[7] showed that within 5 min of induction of anaesthesia, crest-shaped changes of increased density appeared in the dependent regions of both lungs (Fig. 1). In 1989, Hedenstierna and colleagues[33] also found densities in anaesthetized animals, with the same location and attenuation as in anaesthetized humans.[57] Microscopy showed that the densities were atelectatic lung regions. It was concluded that these densities in dependent regions during anaesthesia were caused by atelectasis.

Since then atelectasis has been studied extensively. Atelectasis on a CT scan is defined as pixels with attenuation values of –100 to +100 Hounsfield units (HU).[47] These occur in the most dependent parts of the lungs and are found in almost 90% of all patients who are anaesthetized.[47] They develop with both i.v. and inhalational anaesthesia and whether the patient is breathing spontaneously or is paralysed and ventilated mechanically.[88] The only anaesthetic so far tested that has not produced atelectasis is ketamine,[93] although when the patient was paralysed, atelectasis also appeared in these subjects. On the contrary, epidural anaesthesia caused no or little atelectasis and no change in shunting, ventilation/perfusion (V_A/Q) matching or oxygenation.[71 91]

Good correlations have been found between gas exchange impairment and the amount of atelectasis (r=0.93 for atelectasis and intrapulmonary shunt; r=0.99 for atelectasis and oxygenation).[35] Most atelectasis occurs near the diaphragm in the supine patient and less towards the apex (Fig. 2).[72] In most patients the atelectasis may not appear severe, but collapsed lung comprises four times more lung tissue than aerated regions.[72] Thus, in the average patient the atelectasis may contain 15–20% of the lung tissue close to the diaphragm and about 10% of the total lung tissue. In

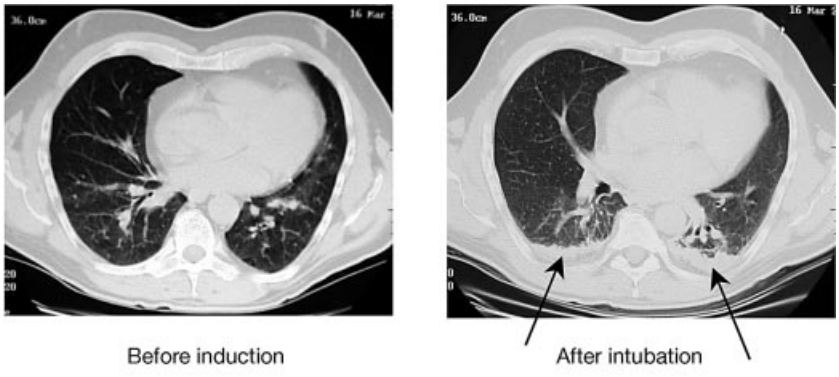

Fig 1 Examples of CT scans of a patient with healthy lungs, before and after induction of anaesthesia. The CT slices are 1 cm above the level of the right diaphragm. Arrows indicate lung densities, thought to represent atelectasis (from Rusca and colleagues[84]).

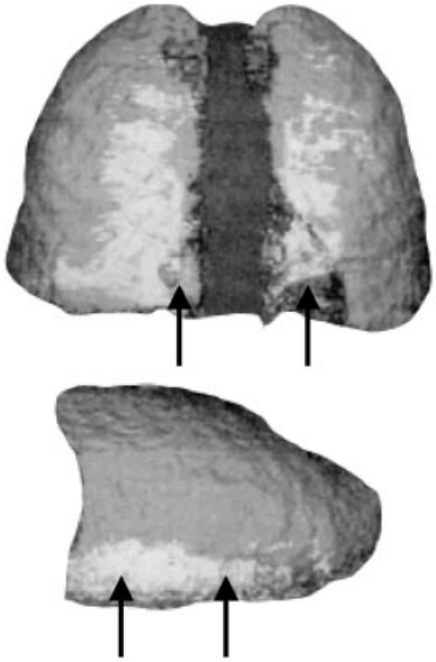

Fig 2 Two-dimensional representation of a volume image from an anaesthetized subject. The surface of the lung is shown in shades of grey and atelectasis is shown in white. The anteroposterior view (top) shows the right and left lungs, with a visible cardiac shadow (arrow). The lateral view (bottom) shows the atelectatic regions in the most dependent lung (from Warner and colleagues[98]).

extreme cases almost half the lung can be collapsed during anaesthesia, before any surgery has taken place or commonly after cardiac surgery![90] Atelectasis can persist for two days after major surgery[45] but disappears within 24 h after laparoscopy in non-obese subjects.[19]

After cardiac surgery with cardiopulmonary bypass (CPB) atelectasis is more prominent than after other forms of surgery, even thoracotomies. In an animal model, extensive atelectasis was seen 1 h after CPB, which was well correlated with intrapulmonary shunt.[49] In man, prominent atelectasis in the dorsal part of the lungs has been found on the first day after cardiac surgery.[90]

Measurement of atelectasis

Atelectasis is not seen on conventional chest radiograph unless it becomes massive.[47] Since 1980, atelectasis has been examined by CT scanning in awake[32] or anaesthetized lung-healthy patients,[29 32 33 47] in the extremes of age (paediatric populations[43 86 87] and patients over 80 years of age[30]), in morbidly obese patients,[19 89] in patients with chronic obstructive lung disease,[31] smokers,[89] and in patients with adult respiratory distress syndrome (ARDS).[65]

The CT scan method starts with a frontal scoutview of the chest to define the borders of the lungs and to guide the settings for subsequent scans. One or more transverse CT scans are then made. To avoid excessive radiation, if successive examinations are planned, only one or two transverse slices are done. If only one CT scan is to be done then the whole lung can be studied. This shows a limitation of the method, because a single level may not reflect the entire lung. The level most often used is 1 cm above the right diaphragm, equivalent to the interventricular septum (Fig. 1). This level appears to be the best compromise between the most affected bases of the lungs and the less affected apex[7 78] and the amount of atelectasis measured at this level correlates well with gas exchange impairment.[6 55 71]

The images are analysed by computer. The entire right and left lungs can be selected as a region of interest by drawing the external boundaries of the lungs at the inside of the ribs and the internal boundaries along the mediastinal organs. The total area of the lungs is measured by including pixels with density values between –1000 and +100 HU (Fig. 3). Densities considered to indicate atelectasis are identified in dependent lung regions and outlined manually. Atelectasis is then calculated by including all pixels within these regions with HU between –100 and +100. Manual delineation of atelectasis has only a small bias compared with computerized evaluation.[47]

With this technique, the partial volume effect may interfere with the measurement of atelectasis.[17] Dense tissue adjacent to the lung can influence the average slice

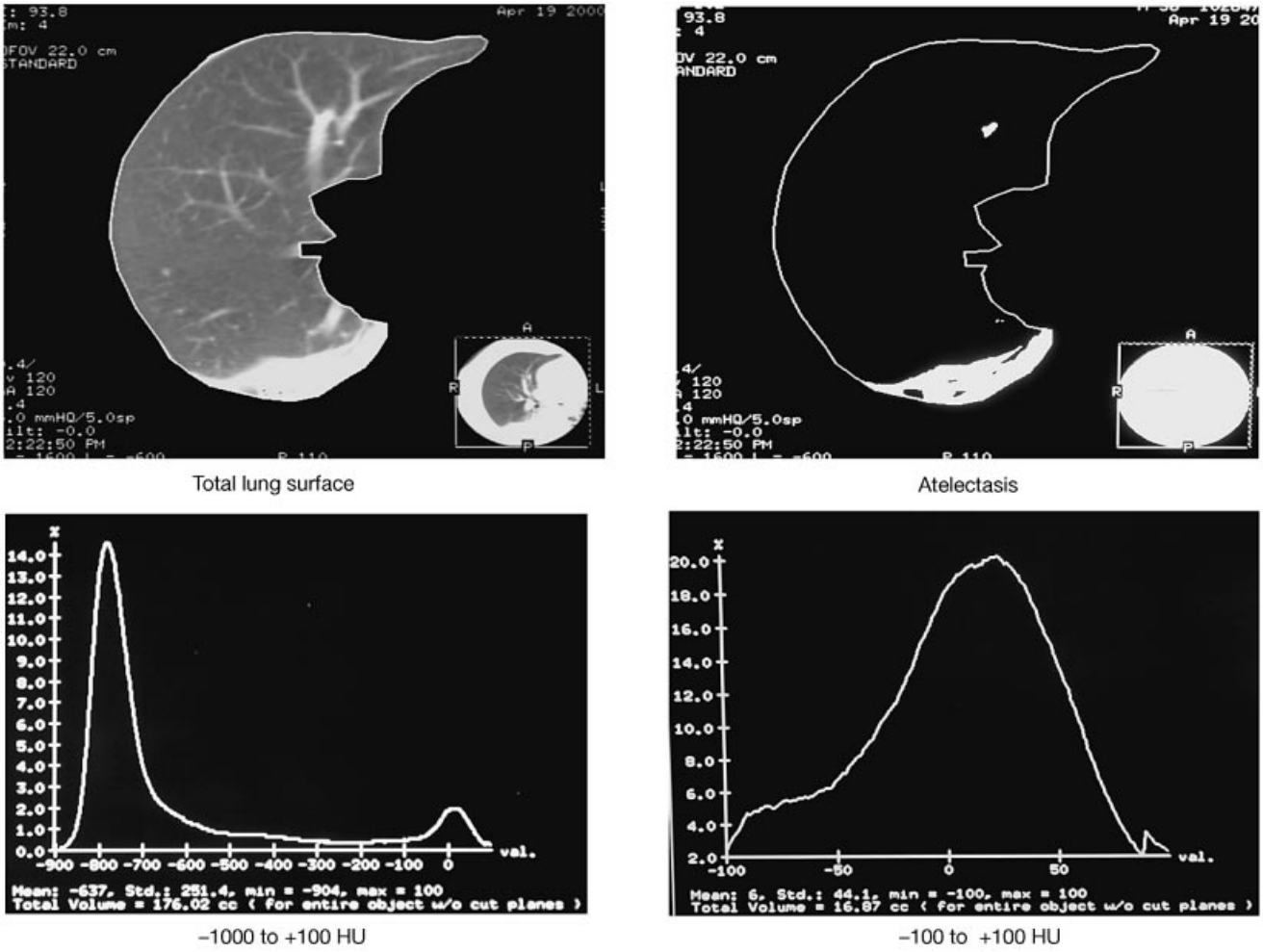

Fig 3 Measurement of atelectatic surface by CT of the lung at the level of the interventricular septum and corresponding histograms. The total lung surface has densities between −1000 and +100 HU. Atelectatic lung surface has densities between −100 and +100 HU (from Benoît and colleagues[6]).

density because of the limited spatial resolution of the system. The same pixel may contain both lung tissue and some adjacent dense tissue. This is called the partial volume effect and it becomes greater when the overall area of the lung is small. Therefore, this effect will be reduced on CT scans taken 1 cm above the right diaphragm where the lung area is great. Moreover, when studies are made of repeated CT scans, this systematic error should not interfere with changes seen over time.

Dependent lung densities have been shown to be atelectasis.[33 47] Nevertheless, blood is a component of lung tissue and has nearly the same density as atelectasis. Therefore, variations in the amount of blood during a study may alter or even explain dependent lung densities.[17] Variation in the amount of blood in a dependent lung region may occur with variation in intrathoracic pressure. However, basal densities are found during anaesthesia with stable intrathoracic pressure when $F_{I_{O_2}}$ is increased and thus cannot be explained only by variations in blood content.[81] In addition, no densities appear when pressure is changed while low oxygen concentration is used, suggesting that the amount of blood plays only a marginal role in appearance of dependent lung densities during general anaesthesia and that these densities even in human patients are atelectasis.

In most studies, dependent lung densities are called atelectasis. It would be better to be more descriptive and use the description lung densities in the title, summary and results, and only explain in the discussion that these densities are probably caused by atelectasis. Nevertheless, in this review lung densities will be considered as atelectasis.

Causes of atelectasis formation during general anaesthesia

Pulmonary atelectasis may be caused by a variety of factors, which have been classified into three basic mechanisms. Compression atelectasis occurs when the transmural pressure distending the alveolus is reduced. Absorption

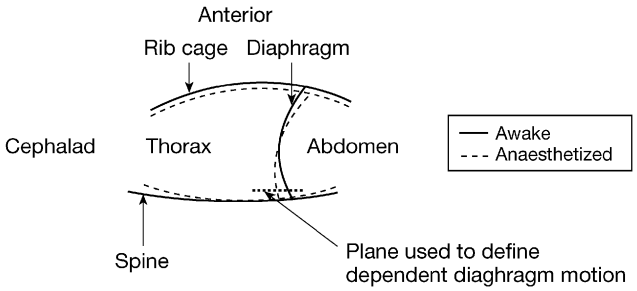

Fig 4 Diagram of a midsagittal section of the thorax while awake (solid lines) and while anaesthetized (dashed lines). Note the alteration in the position of the diaphragm (cephalad motion in the dependent portion; from Warner and colleagues[98]).

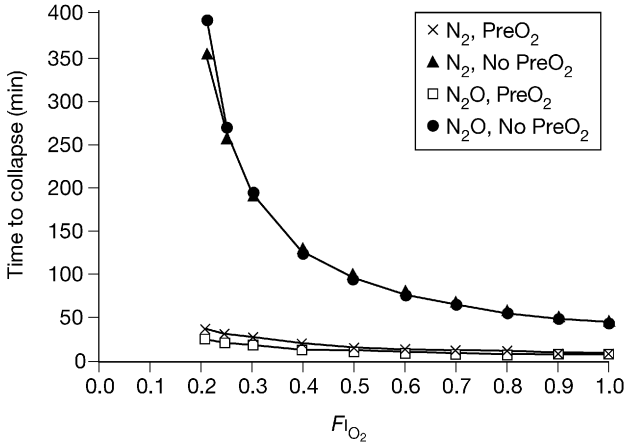

Fig 5 Time to collapse of an unventilated lung compartment. $PreO_2$=3 min of preoxygenation. The inert gas breathed after induction was either nitrogen or nitrous oxide. Collapse occurred more quickly after preoxygenation. The greater the inspired oxygen fraction after induction, the faster the collapse. Time to collapse is largely independent of whether the inspired gas mixture after induction contains nitrogen or nitrous oxide (from Joyce and colleagues[38]).

atelectasis occurs when less gas enters the alveolus than is removed by uptake by the blood. Loss-of-surfactant atelectasis occurs when the surface tension of an alveolus increases because of reduced surfactant action. Any of these factors may contribute to atelectasis during anaesthesia and the postoperative period.

Compression atelectasis

The rapid formation of atelectasis on induction of anaesthesia, being detectable as soon as CT scans can be made, and the fast reappearance after discontinuation of PEEP suggested that the atelectasis was caused by compression of lung tissue rather than by resorption of gas behind occluded airways.[7] The finding that atelectasis could be reduced by phrenic nerve stimulation[34] provided further support to this hypothesis, as did the absence of atelectasis during ketamine anaesthesia.[93] The latter two studies may indicate that loss of inspiratory muscle tone is an important factor in atelectasis formation. It may thus be that the greater abdominal pressure is more easily transmitted into the thoracic cavity when the diaphragm has a reduced tone or is paralysed, as during anaesthesia. The classic study by Froese and Bryan[22] showed that diaphragm motion in spontaneously breathing normal volunteers is changed when the volunteers are paralysed with neuromuscular blocking agents. The authors concluded that in the supine position during spontaneous ventilation, the dependent part of the diaphragm had the greatest displacement. However, after neuromuscular block and positive pressure ventilation, exactly the opposite was seen: the non-dependent part had the greatest displacement. Also, Krayer and colleagues,[41] using CT scans, found altered diaphragmatic motion during general anaesthesia and mechanical ventilation. In addition, Warner and colleagues[98] found alterations in the end-expiratory position of chest wall structures during general anaesthesia (Fig. 4), and Reber and colleagues[74] showed that general anaesthesia induced a cephalad displacement of the most dorsal part of the diaphragm.

Thus, compression atelectasis occurs during general anaesthesia and is caused by chest geometry and diaphragm position and motion.

Absorption atelectasis

Absorption atelectasis can occur by two mechanisms. The first mechanism is complete airway occlusion, which creates a pocket of trapped gas in the distal lung unit. The pressure in the pocket initially is close to atmospheric pressure. Mixed venous blood continues to perfuse the pocket, and since the sum of the gas partial pressures in the mixed venous blood is subatmospheric, gas uptake from the pocket by the blood continues and the pocket collapses.[46] The rate of absorption of gas from an unventilated lung area increases with an increasing $F_{I_{O_2}}$ (Fig. 5).[37]

The second mechanism is when the inspired V_A/Q ratio is less than a critical value. If the inspired V_A/Q ratio of a lung unit is reduced, a point is reached where the rate at which inspired gas entering the alveolus is exactly balanced by gas uptake from the alveolus into the blood. This point is known as the critical V_A/Q ratio.[15] If the inspired V_A/Q ratio is less than this, the lung unit will collapse. This is likely when $F_{I_{O_2}}$ is high and the gas uptake is large. Conversely, a reduction in the amount of atelectasis is seen when lower concentrations of oxygen are used at induction,[73 83] during maintenance of general anaesthesia,[80] or just before extubation.[6]

Loss-of-surfactant atelectasis

Recurrence of atelectasis within 5 min after a vital capacity manoeuvre (VCM; see below) at $F_{I_{O_2}}$=1.0[81] or immediately after removal of PEEP at $F_{I_{O_2}}$=0.4[7 35] suggests an instability

in the alveoli that have been collapsed. It is possible that atelectasis, once formed, impedes surfactant function so that such a region is prone to collapse again after having been reopened. A VCM may promote surfactant production or release, and distribution of surfactant over the alveolar surface may cause a longer lasting protection against new collapse.[50] Indeed, it has been shown that large gasps increase the proportion of active forms of alveolar surfactant.[58]

In summary, all three mechanisms (compression, absorption and loss of surfactant) may contribute to atelectasis formation during general anaesthesia. Absorption and compression are the two mechanisms most implicated in perioperative atelectasis formation. Indeed, Rothen and colleagues[80] have shown that intrapulmonary shunt is correlated to the amount of atelectasis and that poorly ventilated lung units ('low VA/Q') are correlated with airway closure measured by the difference in closing volume and expiratory reserve volume (CV–ERV). There is no correlation between CV–ERV and atelectasis. Taken together, the amount of atelectasis and airway closure may explain 75% of the deterioration in gas exchange seen during general anaesthesia.

Factors influencing atelectasis formation

Fraction of inspired oxygen

High oxygen concentration has often been associated with atelectasis formation. When an $F_{I_{O_2}}$ of 1.0 is used after a VCM, atelectasis recurs within 5 min.[81] On the other hand, when 40% oxygen is used, atelectasis will not recur for at least 40 min.[79 81] In order to avoid atelectasis formation, lower oxygen concentration has been used during induction of general anaesthesia. With 100% oxygen, shunt increased from 0.3% to 6.5%, with atelectasis formation corresponding to an area of 8.0 cm^2. With 30% oxygen, shunt increased to only 2.1%, with minimal atelectasis (0.2 cm^2).[83] Without any preoxygenation, no atelectasis was seen directly after induction, but when $F_{I_{O_2}}$ was increased to 1.0 before intubation, atelectasis appeared.[73 82] Moreover, increasing $F_{I_{O_2}}$ at the end of surgery to 1.0 before extubation will also favour atelectasis formation, persisting in the postoperative period.[6] These results suggest that the composition of inspired gas is important in atelectasis formation during general anaesthesia. A smaller $F_{I_{O_2}}$ may increase the risk of hypoxaemia if airway management is difficult, and therefore the use of lower $F_{I_{O_2}}$ at induction of anaestheisa has not been recommended. Moreover, the standard use of 30–40% oxygen during general anaesthesia has been challenged recently. Using 80% oxygen compared with 30% reduces the incidence of postoperative nausea and vomiting from 30% to 17%[27] and ondansetron is no more effective than supplemental oxygen in preventing postoperative nausea and vomiting.[25] It has also been shown that 80% oxygen as compared with 30% oxygen during general anaesthesia augments antimicrobial and pro-inflammatory responses in alveolar macrophages.[40] Increased antimicrobial function may be beneficial for pulmonary defence. Perhaps even more importantly, use of high oxygen concentrations ($F_{I_{O_2}}$=0.8) during colorectal resection halved the incidence of surgical wound infection compared with an $F_{I_{O_2}}$ of 0.3.[26] The cost of supplemental oxygen is trivial, so the use of 80% oxygen may be an economical way to reduce postoperative infections.

Akca and colleagues[2] found that the use of 80% rather than 30% oxygen for colon resection did not affect the incidence and severity of atelectasis or gas exchange efficiency. Preoxygenation with 80% oxygen is associated with only 0.8% of atelectasis directly after intubation, compared with 6.8% following preoxygenation with 100% oxygen. However, the time to reach 90% oxygen saturation during apnoea is decreased by more than 1 min compared with 100% oxygen (307 s *vs* 391 s, respectively).[18]

Such studies suggest that an $F_{I_{O_2}}$ of 0.8 may offer advantages during general anaesthesia despite the potential effect on atelectasis formation, particularly since when PEEP is used after a VCM, atelectasis does not recur despite the use of 100% oxygen.[55]

Obesity

In 1987, Strandberg and colleagues[89] found a weak correlation (r=0.34) between obesity [calculated by Broca's index: weight (kg)/(height (cm) –100)] and the area of lung densities seen directly after induction of anaesthesia. More recently, it has been shown that during general anaesthesia, morbidly obese patients had more atelectasis than non-obese patients. Atelectasis persisted for at least 24 h in morbidly obese patients whereas it disappeared in the non-obese (Fig. 6).[19] Functional residual capacity (FRC) is lower in morbidly obese patients, the alveolar–arterial oxygenation gradient (A–aD_{O_2}) is increased and intra-abdominal pressure is higher.[62–64] The different mechanics of the respiratory system and the hypoxia found in the morbidly obese patients are largely explained by a reduction in lung volume by increased intra-abdominal pressure.[62] When PEEP was applied, respiratory function improved in morbidly obese patients but not in non-obese patients,[66] and the reverse Trendelenburg position improves oxygenation and lung mechanics in morbidly obese patients.[67]

In morbidly obese patients, avoiding atelectasis formation may be particularly difficult but at the same time particularly important.

Chronic obstructive pulmonary disease

In contrast, patients with chronic obstructive pulmonary disease develop only a small shunt and almost no atelectasis during anaesthesia. However, they develop a more severe VA/Q mismatch. Hyperinflation of the lungs may make

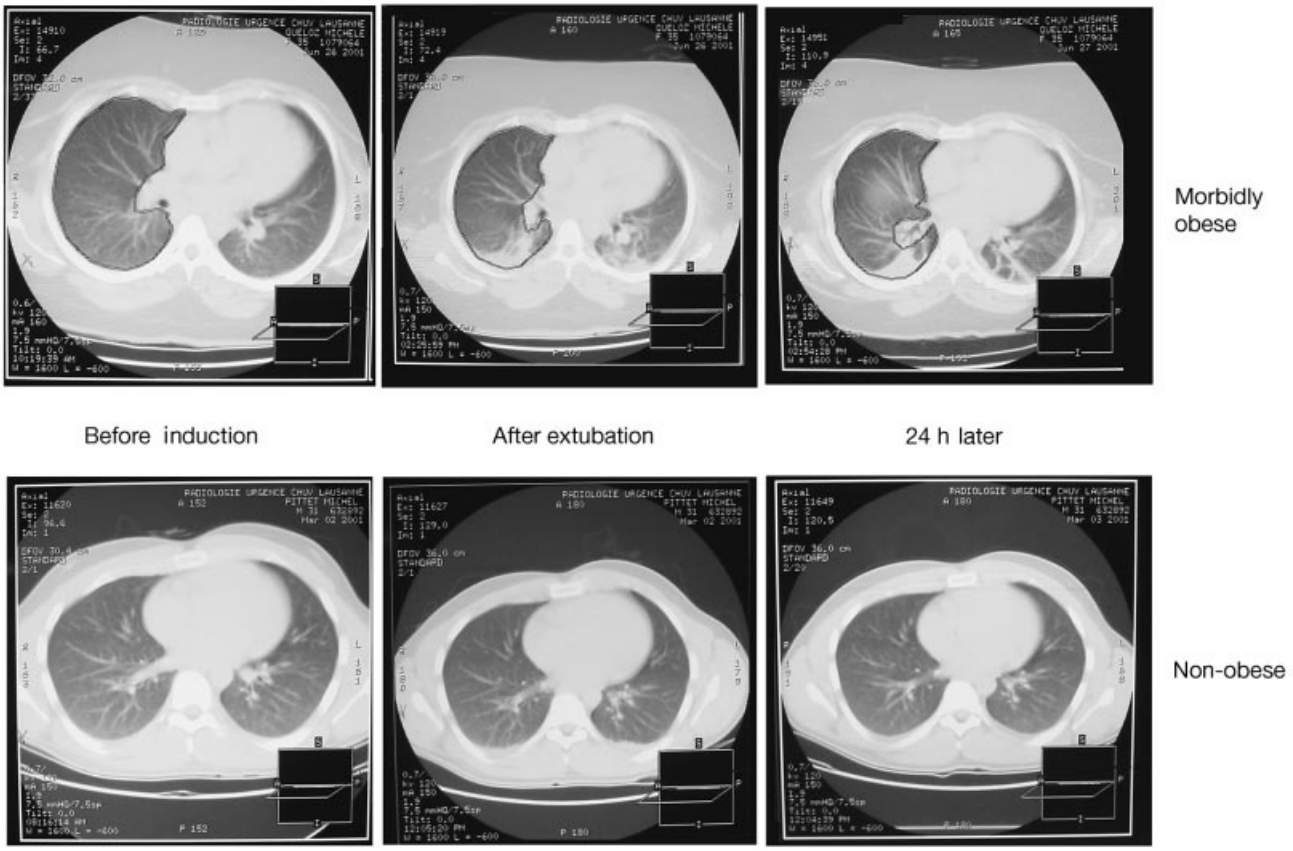

Fig 6 Samples of CT scans of a morbidly obese and a non-obese patient before anaesthesia, after extubation and 24 h later. These slices were taken at the level of the interventricular septum (from Eichenberger and colleagues[19]).

them resist collapse or airway closure may prevent gas from leaving the alveoli (gas trapping).[31]

Other factors

Atelectasis during anaesthesia is found in all ages, from the newborn[14 86] to patients over 80 years of age.[30] Interestingly, the magnitude of atelectasis seems to be independent of age in adults, and 80-year-old patients have no more atelectasis than younger patients.[30] In children, densities appear rapidly in the dependent lung regions following induction of anaesthesia, while atelectasis was not seen in sedated children.[43 87 88] This is important because these atelectasis may obscure pulmonary metastases in 68% of children.[86]

In contrast to the circumstances in adults, atelectasis occurs even when preoxygenation is avoided and $F_{I_{O_2}}$<0.4 is used intraoperatively.[87] Atelectasis in the dependent regions of the lung in children during anaesthesia cannot be explained by reabsorption of oxygen alone. Although the inward recoil of a child's lungs is similar to that of young adults, the outward recoil of the chest wall is less.[16] This results in a decrease in the FRC, and the less negative intrathoracic pressure increases the tendency both to airway closure and to the development of atelectasis.[4 23]

Less contribution of the rib cage to ventilation has been demonstrated in children during halothane anaesthesia.[95] In infants, contraction of the diaphragm may cause paradoxical inward movement of the highly deformable chest wall, which could reduce ventilatory efficiency and increase diaphragmatic fatigue. Closing volume is greater in young children, in whom the elastic supporting structure of the lung is incompletely developed. This puts an infant at greater risk for atelectasis since airway closure can occur even during tidal breathing. The infant's lung is less compliant in relation to the chest wall: the net effect is a lower resting volume (FRC) than that seen in adults. Moreover, in children, PEEP (5 cm H_2O) maintained throughout anaesthesia is able to recruit all the available alveolar units and promote the disappearance of atelectatic areas in dependent pulmonary regions.[87]

Importance of atelectasis on patient outcome

Since most of atelectasis appearing during general anaesthesia resolves within 24 h after surgery[19] one may argue that there is no need to prevent or study atelectasis since it may have no long-lasting effects. Indeed, often the lung dysfunction is transient and normal lung function resumes soon after anaesthesia and surgery. Nevertheless, patients do

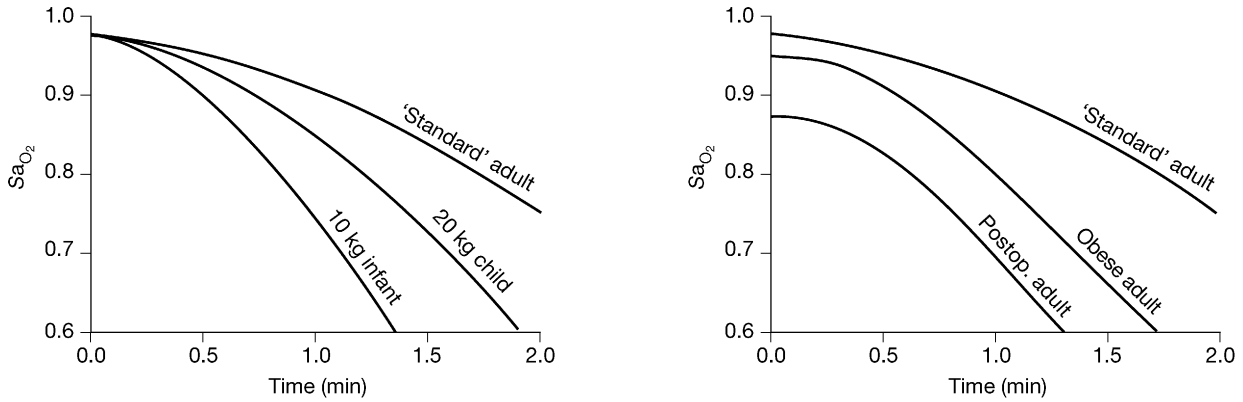

Fig 7 Changes of arterial oxygen saturation (Sa_{O_2}) during apnoea in children, in an obese adult and in a 'typical' postoperative patient compared with the 'standard' adult (from Farmery and colleagues[20]).

develop perioperative respiratory complications.[8 11] Since the number of anaesthetic procedures in the Western world is considerable (60–70 000 occasions per million inhabitants, with more than half of these being general anaesthesia), even a small fraction of complications results in a large number of patients.

Some pulmonary complications occur during or immediately after anaesthesia, mainly hypoxaemia, and some will occur later, mainly pneumonia.

Perioperative hypoxaemia

Mild-to-moderate hypoxaemia, defined as an arterial saturation of 85–90%, occurs in approximately half of all patients undergoing elective surgery and can last from a few seconds to up to 30 min.[54] More alarming is the fact that about 20% of the patients may suffer from severe hypoxaemia (oxygen saturation <81% for up to 5 min) during anaesthesia [54] and 13% in the post-anaesthesia care unit (PACU).[53] Thirty-three percent of hypoxaemic events occur during induction of anaesthesia, one-third intraoperatively and one-third during awakening and in the PACU.[11] Nowadays, more frequent peripheral arterial saturation monitoring may reduce intraoperative hypoxaemic events compared with the 1980s.

Hypoxaemia during induction of anaesthesia

In the UK during the 1980s, three pregnant women died annually during induction of general anaesthesia because of failure to ventilate or intubate. It is estimated that difficulty in airway management during induction of anaesthesia accounts for 600 patient deaths per year.[39] Difficult airway management is not easily anticipated; therefore this complication may arise during every anaesthesia induction. During apnoea, oxygenation depends on the oxygen stores, which are small and are mainly in three compartments: the lungs, plasma and red cells. The normal store of oxygen is approximately 1500 ml, and may be increased to 3700 ml with preoxygenation with 100% oxygen.[99] Half of this increase is from the increase in the oxygen concentration in the FRC. Therefore, prevention of atelectasis formation, which diminishes FRC, during induction of anaesthesia is important for all patients.

A greater oxygen store allows a greater margin of safety, with more time for airway management. Time to a 90% oxygen saturation is longest when anaesthesia induction is done with 100% oxygen, although this is associated with significant atelectasis formation.[18] However, such atelectasis can be prevented by application of an end-expiratory positive pressure during anaesthesia induction despite the use of 100% oxygen[84] and this will prolong the time to desaturation by more than 2 min (unpublished observations). In patients at increased risk of rapid desaturation, greater oxygen stores would be especially useful. In a mathematical model, effective preoxygenation in a 'standard adult' results in a time to decrease arterial saturation to 85% of 502 s.[20] This is reduced to 180 s in the 10-kg infant and 171 s in the morbidly obese (Fig. 7).

Hypoxaemia during awakening and in the PACU

Transport from the operating room to the PACU is another period particularly at risk for hypoxaemic events. During this transport, patients may be without monitoring of oxygen saturation and without supplemental oxygen. At arrival in the PACU, 20% of patients may have an oxygen saturation <92% and in 10% the saturation may be <90%.[51] Age and obesity increase the risk. In the PACU within 3 h of surgery, 7% of patients will have at least one episode of desaturation <90%, and 3% will desaturate to <85%. This proportion is greater for thoraco-abdominal procedures, when more than half of the patients will have oxygen saturation <90% and 20% of patients will have severe hypoxaemia (<85%).[102] Despite the use of 40% oxygen given by face mask, 15% of patients will have an oxygen saturation below 92% lasting more than 30 s.[85] During a

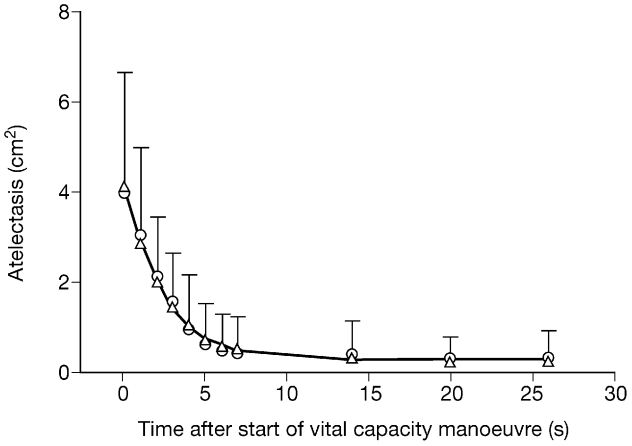

Fig 8 Atelectasis before (time=0) and during the vital capacity manoeuvre. Mean values and SD (error bars) are shown. An exponential decay curve is fitted to individual data (from Rothen and colleagues[77]).

stay in the PACU, 25% of all patients will have at least one episode of desaturation.

Children, particularly young children, are also subject to hypoxaemia in the immediate postoperative period.[11 101] On arrival in the PACU, 50% of children will have an arterial saturation <95% and 8% will be <90%. If the transport time was greater, the number of subjects with oxygen saturation levels <95% increased.[21]

In a large study with more than 24 000 patients, 0.9% had an hypoxaemic event in the PACU requiring a specific intervention other than only supplemental oxygen.[75] Hypoxaemic events appear to prolong stay in the PACU, cause more intensive care admissions, and increase the incidence of cardiac complications. In another study, elderly cardiovascular patients (more than 80 years old) who sustained mild hypoxaemia (longer than 5 min) or severe desaturation (<80%) after surgery were more likely to experience silent myocardial ischaemia.[24] Other studies have shown that postoperative hypoxaemia is linked with ECG abnormalities[76] or delirium.[1]

There is no clear evidence that atelectasis is the cause of all these postoperative hypoxaemic events. Respiratory depression from residual anaesthetic may contribute. In a typical postoperative scenario, hypovolaemia, reduced cardiac output, anaemia, increased V_A/Q mismatch, increased shunt, hypoventilation and reduced alveolar volume can all contribute to more rapid onset of hypoxaemia (Fig. 7).[20]

It seems likely that preventing atelectasis formation during the whole perioperative period will increase the oxygen stores of the body. This increase in oxygen stores may reduce postoperative hypoxaemia. This may be particularly important in aged, obese and unfit patients.

Postoperative pulmonary complications

In studies on postoperative pulmonary complications (PPC), atelectasis and pneumonia are often considered together because the changes associated with atelectasis may predispose to pneumonia. A continuum exists from non-infectious PPC (atelectasis) to infectious PPC (exacerbation of chronic bronchitis or pneumonia). In studies of non-cardiac surgery, the frequency of PPC and cardiac complications (which have always received more attention) are comparable. For example, in adult men after elective abdominal surgery, PPC are more frequent than cardiac complications (estimated rates of 9.6% and 5.7%, respectively) and are associated with a longer hospital stay and greater healthcare costs.[44] Pulmonary complications account for 24% of deaths within 6 days of surgery.[8 9] Postoperative pneumonia is associated with a 30–46% mortality rate, and pneumonia causes 30–60% of the infections related to mortality.[42 61] Some patients have increased risk of PPC. Obese patients have a 25–30% risk of developing PPC.[9 94] PPC are more common after some types of surgery, for example oesophagectomy (17–50% PPC)[13 28] or major head and neck surgery (15% PPC).[52] Despite the lack of direct evidence of a correlation between atelectasis and pneumonia, reducing or avoiding atelectasis may diminish PPC, particularly in some patients[94] or after some types of surgery[28] and thus improve outcome.

Prevention of atelectasis formation

A VCM can completely abolish atelectasis that develops after induction of general anaesthesia.[78] Lung inflation to an airway pressure of 20 cm H_2O did not affect atelectasis; an airway pressure of 30 cm H_2O reduced atelectasis; only with a pressure of 40 cm H_2O maintained for 15 s is atelectatic lung tissue fully re-expanded. This pressure is equivalent to inflation to vital capacity, and thus this manoeuvre has been called the VCM. More recently, it has been shown that this manoeuvre needs to be maintained for only 7–8 s in order to re-expand all previously collapsed lung tissue (Fig. 8).[77] This manoeuvre not only has a 'cosmetic' effect on CT scans but also improves oxygenation. When the inspired oxygen concentration was 40%, Pa_{O_2} increased from 17.7 kPa to 22.2 kPa after the VCM.[77] The safety of this inflation manoeuvre has been questioned but no adverse haemodynamic or pulmonary effects have been reported. In animal experiments, repeated VCM had no deleterious pulmonary effects as measured by extravascular lung water, pulmonary clearance of ^{99m}Tc-DTPA (which is a marker of the functional integrity of the alveolocapillary barrier) and light microscopy.[48]

Tusman and colleagues[97] studied an alternative manoeuvre. They increased both PEEP to 15 cm H_2O and tidal volume to either 18 ml kg^{-1} or to a volume that caused a peak airway pressure of 40 cm H_2O, and maintained this for 10 breaths. PEEP was then decreased stepwise to 5 cm H_2O and tidal volume reduced to 9 ml kg^{-1}. This procedure increased Pa_{O_2}, which persisted for 120 min. The same method (Fig. 9) was also successful in augmenting arterial oxygenation during one-lung ventilation.[95]

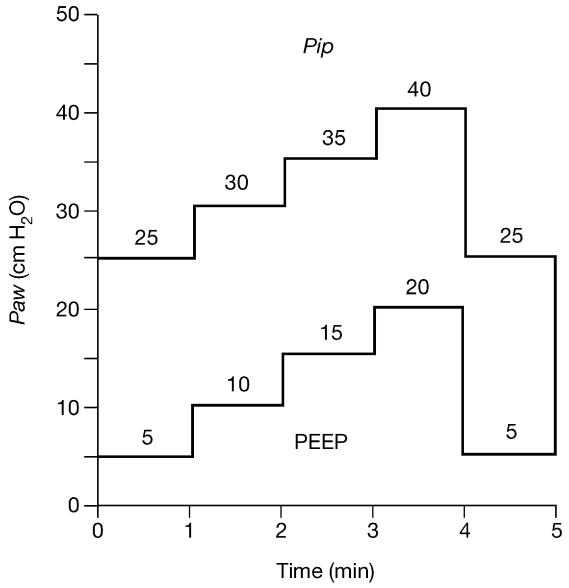

Fig 9 Schematic representation of the alveolar recruitment strategy. In pressure control ventilation, the pressure amplitude of 20 cm H_2O remains constant throughout the manoeuvre. Each pressure step is maintained for 1 min. After airway pressures of 40/20 cm H_2O, the pressures are reduced to 30/10 cm H_2O. The initial settings are then resumed. (Paw, pulmonary airway pressure; Pip, peak inspiratory pressure; from Tusman and colleagues[96]).

The application of a PEEP of 10 cm H_2O has been tested in several studies and will consistently reopen collapsed lung tissue.[7 32 92] However, some atelectasis persists in most patients. Further increases in PEEP level could re-expand this persistent atelectasis but PEEP may not be ideal. Firstly, shunt is not reduced and the arterial oxygenation is not always improved. Persistent shunt may be explained by the redistribution of blood flow towards the most dependent parts of the lung when intrathoracic pressure is increased, so that residual atelectasis lung receives a larger share of the pulmonary blood flow when PEEP is applied.[100] The increased intrathoracic pressure will also impede venous return and reduce cardiac output. This will decrease venous oxygen tension and augment the impact of shunted blood and perfusion of poorly ventilated regions on arterial oxygenation. Secondly, the lung may re-collapse rapidly after discontinuation of PEEP. Within 1 min after cessation of PEEP the collapse is as large as it was before the application of PEEP.[34] However, PEEP applied immediately after a VCM will completely prevent recurrence of atelectasis, even when 100% oxygen is used.[55]

During induction of anaesthesia, application of PEEP (6 cm H_2O) can prevent formation of atelectasis[84] and can increase the margin of safety before intubation. Application of PEEP (10 cm H_2O) in morbidly obese patients is also very effective for the prevention of atelectasis during induction.[12]

Clarke and colleagues[10] compared four treatments (manual inflations, large tidal volumes, PEEP, and pressure control inverse ratio ventilation [IRV]), using the A–a$D\mathrm{O}_2$ as the measure of atelectasis during anaesthesia, to determine if any of these treatments affected atelectasis after anaesthesia. PEEP and IRV were most effective in reducing intraoperative A–a$D\mathrm{O}_2$, and no treatment affected postoperative A–a$D\mathrm{O}_2$. However, since increasing $F\mathrm{I}_{\mathrm{O}_2}$ even for a few minutes before extubation can cause atelectasis, this could explain why no difference was seen after anaesthesia in Clarke's study.

Large tidal volumes (22 ml kg^{-1}) do not improve oxygenation in morbidly obese patients during general anaesthesia.[3] With these large tidal volumes, peak inspiratory airway pressure increased to 38 cm H_2O but with an end-inspiratory airway pressure of only 28 cm H_2O. This plateau pressure is far from the 40 cm H_2O airway pressure applied for 10 s that will relieve atelectasis in non-obese patients. The pressure generated in these obese patients was probably insufficient to re-expand the atelectasis and improve gas exchange.

Allowing spontaneous breathing during mechanical ventilation, even as little as 10–20% of the total ventilation, improves gas exchange. This can be done with airway pressure release ventilation (APRV) or biphasic positive airway pressure (BiPAP), as it is frequently called in Europe. Putensen and colleagues[69] found better oxygenation in animals with APRV than with conventional mechanical ventilation. Patients with ARDS showed similar improvement.[68] The long-term effects of APRV (72-h ventilation) in patients with acute lung injury were compared with control patients receiving pressure-controlled, time-cycled mechanical ventilation.[70] Patients receiving APRV had greater respiratory compliance, $P\mathrm{a}_{\mathrm{O}_2}$ and cardiac output. APRV was associated with fewer days of ventilation (15 days with APRV *vs* 21 days with pressure-controlled ventilation). The better cardiopulmonary function was attributed to recruitment of collapsed lung units. Spontaneous breathing contributed only 10–20% of total ventilation in these studies. Perhaps gas exchange would also improve with this technique during general anaesthesia in normal patients.

Use of the BiPAP system with inspiratory and expiratory positive airway pressure set at 12 and 4 cm H_2O, respectively (12/4) to treat obese patients for the first 24 h after gastroplasty significantly reduced pulmonary dysfunction, indicated by forced vital capacity, forced expiratory volume in 1 s (FEV_1) and oxygen saturation, and pulmonary function recovers more rapidly (Fig. 10).[36] With lower BiPAP pressure (8/4 cm H_2O) or only supplemental oxygen via a face mask, the pulmonary dysfunction was more severe and lasted longer. Atelectasis was not measured in this study but it is possible that the positive airway pressure applied after extubation could reduce atelectasis, explaining in part the improved lung mechanics.

By combining some of these techniques, it could be possible to prevent atelectasis formation during general anaesthesia.

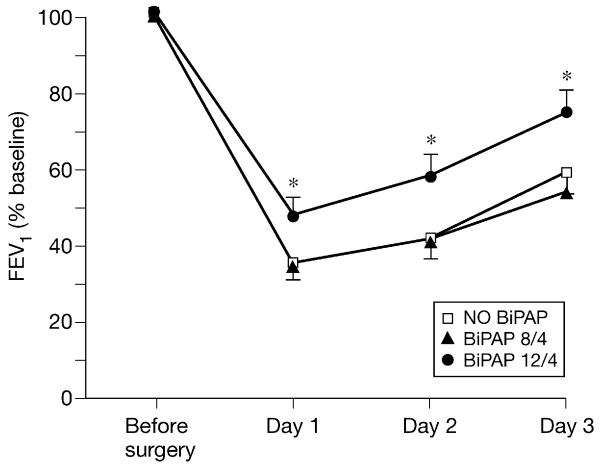

Fig 10 Effect of bi-level positive airway pressure (BiPAP) on FEV_1. *$P<0.05$ compared with the other two groups (from Joris and colleagues[36]).

Conclusion

Atelectasis that develops during general anaesthesia may lead to perioperative pulmonary complications. Prevention of atelectasis formation is therefore an important goal.

References

1 Aakerlund LP, Rosenberg J. Postoperative delirium: treatment with supplementary oxygen. *Br J Anaesth* 1994; **72**: 286–90

2 Akca O, Podolsky A, Eisenhuber E, *et al.* Comparable postoperative pulmonary atelectasis in patients given 30% or 80% oxygen during and 2 hours after colon resection. *Anesthesiology* 1999; **91**: 991–8

3 Bardoczky GI, Yernault JC, Houben JJ, d'Hollander AA. Large tidal volume ventilation does not improve oxygenation in morbidly obese patients during anesthesia. *Anesth Analg* 1995; **81**: 385–8

4 Beardsmore CS, Stocks J, Helms P. Elastic properties of the respiratory system in infants. *Eur Respir J Suppl* 1989; **4**: 135–9

5 Bendixen HH, Hedley-White J, Laver MB. Impaired oxygenation in surgical patients during general anesthesia with controlled ventilation. A concept of atelectasis. *N Engl J Med* 1963; **269**: 991–6

6 Benoît Z, Wicky S, Fischer J-F, *et al.* The effect of increased $F_{I_{O_2}}$ before tracheal extubation on postoperative atelectasis. *Anesth Analg* 2002; **95**: 1777–81

7 Brismar B, Hedenstierna G, Lundquist H, *et al.* Pulmonary densities during anesthesia with muscular relaxation – a proposal of atelectasis. *Anesthesiology* 1985; **62**: 422–8

8 Brooks-Brunn JA. Postoperative atelectasis and pneumonia. *Heart Lung* 1995; **24**: 94–115

9 Brooks-Brunn JA. Predictors of postoperative pulmonary complications following abdominal surgery. *Chest* 1997; **111**: 564–71

10 Clarke JP, Schuitemaker MN, Sleigh JW. The effect of intraoperative ventilation strategies on perioperative atelectasis. *Anaesth Intens Care* 1998; **26**: 262–6

11 Cote CJ, Goldstein EA, Cote MA, Hoaglin DC, Ryan JF. A single-blind study of pulse oximetry in children. *Anesthesiology* 1988; **68**: 184–8

12 Coussa M, Proietti S, Frascarolo P, Spahn D, Magnusson L. Continuous positive airways pressure prevents atelectasis formation during induction of general anaesthesia in morbidly obese patients. *Swiss Med Wkly* 2002; **132**: 53

13 Crozier TA, Sydow M, Siewert JR, Braun U. Postoperative pulmonary complication rate and long-term changes in respiratory function following esophagectomy with esophagogastrostomy. *Acta Anaesthesiol Scand* 1992; **36**: 10–15

14 Damgaard-Pedersen K, Qvist T. Pediatric pulmonary CT-scanning. Anaesthesia-induced changes. *Pediatr Radiol* 1980; **9**: 145–8

15 Dantzker DR, Wagner PD, West JB. Proceedings: Instability of poorly ventilated lung units during oxygen breathing. *J Physiol* 1974; **242**: 72

16 Davis GM, Coates AL, Dalle D, Bureau MA. Measurement of pulmonary mechanics in the newborn lamb: a comparison of three techniques. *J Appl Physiol* 1988; **64**: 972–81

17 Drummond GB. Computed tomography and pulmonary measurements. *Br J Anaesth* 1998; **80**: 665–71

18 Edmark L, Enlund M, Kostova-Aherdan K, Hedenstierna G. Atelectasis formation and apnoea tolerance after pre-oxygenation with 100%, 80%, or 60% oxygen. *Anesthesiology* 2001; **95**: A1330

19 Eichenberger A-S, Proietti S, Wicky S, *et al.* Morbid obesity and postoperative pulmonary atelectasis: an underestimated problem. *Anesth Analg* 2002; **95**: 1788–92

20 Farmery AD, Roe PG. A model to describe the rate of oxyhaemoglobin desaturation during apnoea. *Br J Anaesth* 1996; **76**: 284–91

21 Fossum SR, Knowles R. Perioperative oxygen saturation levels of pediatric patients. *J Post Anesth Nurs* 1995; **10**: 313–19

22 Froese AB, Bryan AC. Effects of anesthesia and paralysis on diaphragmatic mechanics in man. *Anesthesiology* 1974; **41**: 242–55

23 Gerhardt T, Bancalari E. Chestwall compliance in full-term and premature infants. *Acta Paediatr Scand* 1980; **69**: 359–64

24 Gill NP, Wright B, Reilly CS. Relationship between hypoxaemic and cardiac ischaemic events in the perioperative period. *Br J Anaesth* 1992; **68**: 471–3

25 Goll V, Akca O, Greif R, *et al.* Ondansetron is no more effective than supplemental intraoperative oxygen for prevention of postoperative nausea and vomiting. *Anesth Analg* 2001; **92**: 112–17

26 Greif R, Akca O, Horn EP, Kurz A, Sessler DI. Supplemental perioperative oxygen to reduce the incidence of surgical-wound infection. Outcomes Research Group. *N Engl J Med* 2000; **342**: 161–7

27 Greif R, Laciny S, Rapf B, Hickle RS, Sessler DI. Supplemental oxygen reduces the incidence of postoperative nausea and vomiting. *Anesthesiology* 1999; **91**: 1246–52

28 Griffin SM, Shaw IH, Dresner SM. Early complications after Ivor Lewis subtotal esophagectomy with two-field lymphadenectomy: risk factors and management. *J Am Coll Surg* 2002; **194**: 285–97

29 Gunnarsson L, Strandberg A, Brismar B, *et al.* Atelectasis and gas exchange impairment during enflurane/nitrous oxide anaesthesia. *Acta Anaesthesiol Scand* 1989; **33**: 629–37

30 Gunnarsson L, Tokics L, Gustavsson H, Hedenstierna G. Influence of age on atelectasis formation and gas exchange impairment during general anaesthesia. *Br J Anaesth* 1991; **66**: 423–32

31 Gunnarsson L, Tokics L, Lundquist H, *et al.* Chronic obstructive pulmonary disease and anaesthesia: formation of atelectasis and gas exchange impairment. *Eur Respir J* 1991; **4**: 1106–16

32 Hachenberg T, Lundquist H, Tokics L, Brismar B, Hedenstierna G. Analysis of lung density by computed tomography before and

during general anaesthesia. *Acta Anaesthesiol Scand* 1993; **37**: 549–55

33 Hedenstierna G, Lundquist H, Lundh B, *et al.* Pulmonary densities during anaesthesia. An experimental study on lung morphology and gas exchange. *Eur Respir J* 1989; **2**: 528–35

34 Hedenstierna G, Tokics L, Lundquist H, *et al.* Phrenic nerve stimulation during halothane anesthesia. Effects of atelectasis. *Anesthesiology* 1994; **80**: 751–60

35 Hedenstierna G, Tokics L, Strandberg A, Lundquist H, Brismar B. Correlation of gas exchange impairment to development of atelectasis during anaesthesia and muscle paralysis. *Acta Anaesthesiol Scand* 1986; **30**: 183–91

36 Joris JL, Sottiaux TM, Chiche JD, Desaive CJ, Lamy ML. Effect of bi-level positive airway pressure (BiPAP) nasal ventilation on the postoperative pulmonary restrictive syndrome in obese patients undergoing gastroplasty. *Chest* 1997; **111**: 665–70

37 Joyce CJ, Baker AB, Kennedy RR. Gas uptake from an unventilated area of lung: computer model of absorption atelectasis. *J Appl Physiol* 1993; **74**: 1107–16

38 Joyce CJ, Williams AB. Kinetics of absorption atelectasis during anesthesia: a mathematical model. *J Appl Physiol* 1999; **86**: 1116–25

39 King TA, Adams AP. Failed tracheal intubation. *Br J Anaesth* 1990; **65**: 400–14

40 Kotani N, Hashimoto H, Sessler DI, *et al.* Supplemental intraoperative oxygen augments antimicrobial and pro-inflammatory responses of alveolar macrophages. *Anesthesiology* 2000; **93**: 15–25

41 Krayer S, Rehder K, Vettermann J, Didier EP, Ritman EL. Position and motion of the human diaphragm during anesthesia-paralysis. *Anesthesiology* 1989; **70**: 891–8

42 Kroenke K, Lawrence VA, Theroux JF, Tuley MR. Operative risk in patients with severe obstructive pulmonary disease. *Arch Intern Med* 1992; **152**: 967–71

43 Lam WW, Chen PP, So NM, Metreweli C. Sedation versus general anaesthesia in paediatric patients undergoing chest CT. *Acta Radiol* 1998; **39**: 298–300

44 Lawrence VA, Hilsenbeck SG, Mulrow CD, *et al.* Incidence and hospital stay for cardiac and pulmonary complications after abdominal surgery. *J Gen Intern Med* 1995; **10**: 671–8

45 Lindberg P, Gunnarsson L, Tokics L, *et al.* Atelectasis and lung function in the postoperative period. *Acta Anaesthesiol Scand* 1992; **36**: 546–53

46 Loring SH, Butler JP. Gas exchange in body cavities. In: Farhi LE, Tenney SM, eds. *Handbook of Physiology. Section 3*, *The Respiratory System. Volume 4*, *Gas Exchange*. Bethesda, Maryland: Am Physiol Soc, 1987; 283–95

47 Lundquist H, Hedenstierna G, Strandberg A, Tokics L, Brismar B. CT-assessment of dependent lung densities in man during general anaesthesia. *Acta Radiol* 1995; **36**: 626–32

48 Magnusson L, Tenling A, Lemoine R, *et al.* The safety of one, or repeated, vital capacity maneuvers during general anesthesia. *Anesth Analg* 2000; **91**: 702–7

49 Magnusson L, Zemgulis V, Wicky S, *et al.* Atelectasis is a major cause of hypoxemia and shunt after cardiopulmonary bypass: an experimental study. *Anesthesiology* 1997; **87**: 1153–63

50 Mason RJ. Surfactant secretion. In: Robertson B, van Golde LMG, Batenburg JJ, eds. *Pulmonary Surfactant. From Molecular Biology to Clinical Practice*. Amsterdam: Elsevier, 1992; 295–312

51 Mathes DD, Conaway MR, Ross WT. Ambulatory surgery: room air versus nasal cannula oxygen during transport after general anesthesia. *Anesth Analg* 2001; **93**: 917–21

52 McCulloch TM, Jensen NF, Girod DA, Tsue TT, Weymuller EA Jr. Risk factors for pulmonary complications in the postoperative head and neck surgery patient. *Head Neck* 1997; **19**: 372–7

53 Moller JT. Anesthesia related hypoxemia. The effect of pulse oximetry monitoring on perioperative events and postoperative complications. *Dan Med Bull* 1994; **41**: 489–500

54 Moller JT, Johannessen NW, Berg H, Espersen K, Larsen LE. Hypoxaemia during anaesthesia—an observer study. *Br J Anaesth* 1991; **66**: 437–44

55 Neumann P, Rothen HU, Berglund JE, *et al.* Positive end-expiratory pressure prevents atelectasis during general anaesthesia even in the presence of a high inspired oxygen concentration. *Acta Anaesthesiol Scand* 1999; **43**: 295–301

56 Nunn JF. Factors influencing the arterial oxygen tension during halothane anaesthesia with spontaneous respiration. *Br J Anaesth* 1964; **36**: 327–41

57 Nyman G, Funkquist B, Kvart C, *et al.* Atelectasis causes gas exchange impairment in the anaesthetised horse. *Equine Vet J* 1990; **22**: 317–24

58 Oyarzun MJ, Iturriaga R, Donoso P, *et al.* Factors affecting distribution of alveolar surfactant during resting ventilation. *Am J Physiol* 1991; **261**: 210–17

59 Pasteur W. Massive collapse of the lung. *Lancet* 1908; 1351–3

60 Pasteur W. Active lobar collapse of the lung after abdominal operations. *Lancet* 1910; **2**: 1080–3

61 Pedersen T, Viby-Mogensen J, Ringsted C. Anaesthetic practice and postoperative pulmonary complications. *Acta Anaesthesiol Scand* 1992; **36**: 812–18

62 Pelosi P, Croci M, Ravagnan I, *et al.* Respiratory system mechanics in sedated, paralyzed, morbidly obese patients. *J Appl Physiol* 1997; **82**: 811–18

63 Pelosi P, Croci M, Ravagnan I, *et al.* The effects of body mass on lung volumes, respiratory mechanics, and gas exchange during general anesthesia. *Anesth Analg* 1998; **87**: 654–60

64 Pelosi P, Croci M, Ravagnan I, Vicardi P, Gattinoni L. Total respiratory system, lung, and chest wall mechanics in sedated-paralyzed postoperative morbidly obese patients. *Chest* 1996; **109**: 144–51

65 Pelosi P, Crotti S, Brazzi L, Gattinoni L. Computed tomography in adult respiratory distress syndrome: what has it taught us? *Eur Respir J* 1996; **9**: 1055–62

66 Pelosi P, Ravagnan I, Giurati G, *et al.* Positive end-expiratory pressure improves respiratory function in obese but not in normal subjects during anesthesia and paralysis. *Anesthesiology* 1999; **91**: 1221–31

67 Perilli V, Sollazzi L, Bozza P, *et al.* The effects of the reverse Trendelenburg position on respiratory mechanics and blood gases in morbidly obese patients during bariatric surgery. *Anesth Analg* 2000; **91**: 1520–5

68 Putensen C, Mutz NJ, Putensen-Himmer G, Zinserling J. Spontaneous breathing during ventilatory support improves ventilation-perfusion distributions in patients with acute respiratory distress syndrome. *Am J Respir Crit Care Med* 1999; **159**: 1241–8

69 Putensen C, Rasanen J, Lopez FA. Ventilation-perfusion distributions during mechanical ventilation with superimposed spontaneous breathing in canine lung injury. *Am J Respir Crit Care Med* 1994; **150**: 101–8

70 Putensen C, Zech S, Wrigge H, *et al.* Long-term effects of spontaneous breathing during ventilatory support in patients with acute lung injury. *Am J Respir Crit Care Med* 2001; **164**: 43–9

71 Reber A, Bein T, Hogman M, *et al.* Lung aeration and pulmonary gas exchange during lumbar epidural anaesthesia and in the lithotomy position in elderly patients. *Anaesthesia* 1998; **53**: 854–61

72 Reber A, Engberg G, Sporre B, *et al.* Volumetric analysis of aeration in the lungs during general anaesthesia. *Br J Anaesth* 1996; **76**: 760–6

73 Reber A, Engberg G, Wegenius G, Hedenstierna G. Lung aeration. The effect of pre-oxygenation and hyperoxygenation during total intravenous anaesthesia. *Anaesthesia* 1996; **51**: 733–7

74 Reber A, Nylund U, Hedenstierna G. Position and shape of the diaphragm: implications for atelectasis formation. *Anaesthesia* 1998; **53**: 1054–61

75 Rose DK, Cohen MM, Wigglesworth DF, DeBoer DP. Critical respiratory events in the postanesthesia care unit. Patient, surgical, and anesthetic factors. *Anesthesiology* 1994; **81**: 410–18

76 Rosenberg J, Rasmussen V, von Jessen F, Ullstad T, Kehlet H. Late postoperative episodic and constant hypoxaemia and associated ECG abnormalities. *Br J Anaesth* 1990; **65**: 684–91

77 Rothen HU, Neumann P, Berglund JE, *et al.* Dynamics of re-expansion of atelectasis during general anaesthesia. *Br J Anaesth* 1999; **82**: 551–6

78 Rothen HU, Sporre B, Engberg G, Wegenius G, Hedenstierna G. Re-expansion of atelectasis during general anaesthesia: a computed tomography study. *Br J Anaesth* 1993; **71**: 788–95

79 Rothen HU, Sporre B, Engberg G, Wegenius G, Hedenstierna G. Re-expansion of atelectasis during general anaesthesia may have a prolonged effect. *Acta Anaesthesiol Scand* 1995; **39**: 118–25

80 Rothen HU, Sporre B, Engberg G, Wegenius G, Hedenstierna G. Airway closure, atelectasis and gas exchange during general anaesthesia. *Br J Anaesth* 1998; **81**: 681–6

81 Rothen HU, Sporre B, Engberg G, *et al.* Influence of gas composition on recurrence of atelectasis after a reexpansion maneuver during general anesthesia. *Anesthesiology* 1995; **82**: 832–42

82 Rothen HU, Sporre B, Engberg G, *et al.* Prevention of atelectasis during general anaesthesia. *Lancet* 1995; **345**: 1387–91

83 Rothen HU, Sporre B, Engberg G, *et al.* Atelectasis and pulmonary shunting during induction of general anaesthesia – can they be avoided? *Acta Anaesthesiol Scand* 1996; **40**: 524–9

84 Rusca M, Wicky S, Proietti S, *et al.* Continuous positive airways pressure prevents atelectasis formation during induction of general anaesthesia. *Anesthesiology* 2001; **95**: A1331

85 Russell GB, Graybeal JM. Hypoxemic episodes of patients in a postanesthesia care unit. *Chest* 1993; **104**: 899–903

86 Sargent MA, McEachern AM, Jamieson DH, Kahwaji R. Atelectasis on pediatric chest CT: comparison of sedation techniques. *PediatrRadiol* 1999; **29**: 509–13

87 Serafini G, Cornara G, Cavalloro F, *et al.* Pulmonary atelectasis during paediatric anaesthesia: CT scan evaluation and effect of positive endexpiratory pressure (PEEP). *Paediatr Anaesth* 1999; **9**: 225–8

88 Strandberg A, Tokics L, Brismar B, Lundquist H, Hedenstierna G. Atelectasis during anaesthesia and in the postoperative period. *Acta Anaesthesiol Scand* 1986; **30**: 154–8

89 Strandberg A, Tokics L, Brismar B, Lundquist H, Hedenstierna G. Constitutional factors promoting development of atelectasis during anaesthesia. *Acta Anaesthesiol Scand* 1987; **31**: 21–4

90 Tenling A, Hachenberg T, Tyden H, Wegenius G, Hedenstierna G. Atelectasis and gas exchange after cardiac surgery. *Anesthesiology* 1998; **89**: 371–8

91 Tenling A, Joachimsson PO, Tyden H, Wegenius G, Hedenstierna G. Thoracic epidural anesthesia as an adjunct to general anesthesia for cardiac surgery: effects on ventilation–perfusion relationships. *J Cardiothorac Vasc Anesth* 1999; **13**: 258–64

92 Tokics L, Hedenstierna G, Strandberg A, Brismar B, Lundquist H. Lung collapse and gas exchange during general anesthesia: effects of spontaneous breathing, muscle paralysis, and positive end-expiratory pressure. *Anesthesiology* 1987; **66**: 157–67

93 Tokics L, Strandberg A, Brismar B, Lundquist H, Hedenstierna G. Computerized tomography of the chest and gas exchange measurements during ketamine anaesthesia. *Acta Anaesthesiol Scand* 1987; **31**: 684–92

94 Tseuda K, Debrand M, Bivins BA, Wright BD, Griffen WO. Pulmonary complications in the morbidly obese following jejunoileal bypass surgery under narcotic anesthesia. *Int Surg* 1980; **65**: 123–9

95 Tusiewicz K, Bryan AC, Froese AB. Contributions of changing rib cage–diaphragm interactions to the ventilatory depression of halothane anesthesia. *Anesthesiology* 1977; **47**: 327–37

96 Tusman G, Bohm SH, Melkun F, *et al.* Alveolar recruitment strategy increases arterial oxygenation during one-lung ventilation. *Ann Thorac Surg* 2002; **73**: 1204–9

97 Tusman G, Bohm SH, Vazquez de Anda GF, do Campo JL, Lachmann B. 'Alveolar recruitment strategy' improves arterial oxygenation during general anaesthesia. *Br J Anaesth* 1999; **82**: 8–13

98 Warner DO, Warner MA, Ritman EL. Atelectasis and chest wall shape during halothane anesthesia. *Anesthesiology* 1996; **85**: 49–59

99 West JB. *Pulmonary Pathophysiology*. Baltimore: Williams and Wilkins, 1987

100 West JB, Dolley CT, Naimark A. Distribution of blood flow in isolated lung: relations to vascular and alveolar pressure. *J Appl Physiol* 1964; **19**: 13–24

101 Xue FS, Huang YG, Tong SY, *et al.* A comparative study of early postoperative hypoxemia in infants, children, and adults undergoing elective plastic surgery. *Anesth Analg* 1996; **83**: 709–15

102 Xue FS, Li BW, Zhang GS, *et al.* The influence of surgical sites on early postoperative hypoxemia in adults undergoing elective surgery. *Anesth Analg* 1999; **88**: 213–19