Airway closure, atelectasis and gas exchange during general anaesthesia†

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Summary

Airway closure and the formation of atelectasis have been proposed as important contributors to impairment of gas exchange during general anaesthesia. We have elucidated the relationships between each of these two mechanisms and gas exchange. We studied 35 adults with healthy lungs, undergoing elective surgery. Airway closure was measured using the foreign gas bolus technique, atelectasis was estimated by analysis of computed x-ray tomography, and ventilation–perfusion distribution ($\dot{V}_A/\dot{Q}$) was assessed by the multiple inert gas elimination technique. The difference between closing volume and expiratory reserve volume (CV–ERV) increased from the awake to the anaesthetized state. Linear correlations were found between atelectasis and shunt ($r=0.68$, $P<0.001$), and between CV–ERV and the amount of perfusion to poorly ventilated lung units ("low $\dot{V}_A/\dot{Q}$", $r=0.57$, $P=0.001$). Taken together, the amount of atelectasis and airway closure may explain 75% of the deterioration in Pa_{O_2}. There was no significant correlation between CV–ERV and atelectasis. We conclude that in anaesthetized adults with healthy lungs, undergoing mechanical ventilation, both airway closure and atelectasis contributed to impairment of gas exchange. Atelectasis and airway closure do not seem to be closely related. (*Br. J. Anaesth.* 1998; **81**: 681–686).

Keywords: anaesthesia, general; airway, pressure; lung, atelectasis; lung, gas exchange; ventilation, ventilation–perfusion

Impairment of gas exchange during general anaesthesia with mechanical ventilation is a well known finding.[1][2] In several recent investigations, the formation of atelectasis with subsequent pulmonary shunting was shown to be related to impairment of gas exchange.[3][4] In addition to shunt, ventilation–perfusion ($\dot{V}_A/\dot{Q}$) mismatch and perfusion to lung units with poor ventilation in relation to their respective perfusion ("low $\dot{V}_A/\dot{Q}$") have been demonstrated during anaesthesia.[4] This low $\dot{V}_A/\dot{Q}$ increases with age.[4] Thus poorly ventilated lung units contribute more to impairment of gas exchange in older patients compared with younger ones. The cause of low $\dot{V}_A/\dot{Q}$ is not clear. One possibility is airway closure. It was demonstrated initially in awake subjects[5] and has been shown to increase in magnitude with age.[6][7] It has also been shown in anaesthetized subjects,[8–11] but its extent and influence on gas exchange have not been fully agreed upon. Airway closure occurs during deep expiration, beginning in the dependent lung regions. If closure (or marked narrowing, see also below) of airways occurs during a normal breath (i.e. above functional residual capacity), ventilation in affected lung regions is impaired, and this should result in a $\dot{V}_A/\dot{Q}$ mismatch with low $\dot{V}_A/\dot{Q}$. By combining measurements of airway closure, atelectasis and $\dot{V}_A/\dot{Q}$ distribution, further insight into the mechanisms of impairment of gas exchange may be obtained.

Therefore, in this study, we have elucidated the relationships between airway closure, atelectasis, $\dot{V}_A/\dot{Q}$ distribution and impaired oxygenation during general anaesthesia with mechanical ventilation.

Patients and methods

A total of 35 patients were included in the study (table 1). Another 14 patients refused to participate (i.e. refusal rate of 29%). No patient had cardiac or pulmonary disease on the basis of history and clinical examination. Some data from the same patients (concerning atelectasis and gas exchange) have been presented previously.[12–14] The study was approved by the Ethics Committee of the University Hospital of Uppsala, Sweden and informed consent was obtained from each subject.

In awake patients, measurements were obtained with the subject lying supine on the CT table and breathing air. Computed tomography of the lungs was performed, and ventilation–perfusion distribution and airway closure were measured. Anaesthesia was induced, and all measurements were repeated after a minimum of 20 min of anaesthesia (see below). Finally, the patient was moved from the x-ray department to the operating theatre to undergo surgery.

Details of the anaesthetic have been described previously.[12–14] Before induction, all patients received atropine 0.5 mg i.v. Anaesthesia was induced with fentanyl 1–2 μg kg⁻¹ and propofol 2 mg kg⁻¹ i.v.,

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Table 1 Patient characteristics (mean (SD), median [range] or number). BMI = Body mass index calculated as weight×height^{-2}

	Mean (SD)	Median [range]
Sex (M/F)	20/15	
Non-smoker/smoker	30/5	
Age (yr)	45 (11)	48 [19–66]
Weight (kg)	79 (14)	79 [53–103]
Height (cm)	173 (8.6)	175 [160–195]
BMI (kg m^{-2})	26.0 (3.5)	26.3 [18.3–33.2]

followed by a continuous infusion of propofol 4–8 mg kg^{-1} h^{-1}. The dose of these drugs was adjusted according to clinical signs of depth of anaesthesia (arterial pressure and heart rate). During induction, the lungs were ventilated manually via a face mask with 100% oxygen. To facilitate orotracheal intubation, patients received pancuronium 0.1 mg kg^{-1}; additional doses of 1–2 mg were given when needed. The lungs were ventilated mechanically at a rate of 10 bpm with 40% oxygen in nitrogen (Servo Ventilator 900C, Siemens Elema AB, Solna, Sweden). No positive end-expiratory pressure was used. Tidal volume was adjusted to maintain an end-tidal carbon dioxide concentration of approximately 4% (CO_2 analyser Eliza, Datex-Engström, Helsinki, Finland). The resulting tidal volume was 9 ± 1 ml kg^{-1}.

The electrocardiogram, arterial pressure (Riva-Rocci method) and peripheral arterial oxygen saturation (pulse oximetry: Biox 3740, Ohmeda, Louisville CO, USA) were monitored throughout the study.

CLOSING VOLUME

Closing volume (CV) was measured using the foreign gas bolus technique, with helium as the tracer gas.[5][6] A specially designed device, including a Fleisch pneumotachograph, flow-integrator, and helium dosing and measuring unit was used (Hebotest Godart, Einthoven, Netherlands). Details of this equipment have been described previously.[15] With the conscious, supine subject breathing air, spirometry values of a few normal breaths were recorded first. Starting from maximal expiration (residual volume, RV) the subject was asked to inspire maximally until vital capacity (VC). During the initial phase of this inspiration, a bolus dose (300 ml) of helium was added to the inspired gas. During the subsequent expiration, the concentration of helium in the expired gas was plotted against expired volume on an X–Y recorder (Model 50000, Bryans, Mitcham, UK). Both maximal inspiration and expiration were made at a rate of approximately 0.5 litre s^{-1}. The onset of phase IV on the helium concentration curve was taken as onset of airway closure, and the volume expired during phase IV to RV was defined as CV. Note that CV–ERV (expiratory reserve volume) is the volume above functional residual capacity (FRC) where airway closure starts (see also comments on methodology below). If CV–ERV is a negative number, onset of airway closure is below FRC. Two or more measurements were made to obtain a minimum of two acceptable curves (even flow during inspiration and expiration, inspired and expired volume equal within 5%).

In the anaesthetized subject, a 7 L syringe was used for manual ventilation of the lungs during measurements. The lungs were deflated to an airway pressure (Paw) of -15 cm H_2O, a level considered to reflect RV. After automatic injection of the helium bolus, the lungs were inflated to a Paw of 40 cm H_2O and immediately deflated at a constant flow rate of approximately 0.5 litre s^{-1} to RV. Paw was measured using a manometer (BOC Ohmeda, Louisville CO, USA) attached to the tracheal tube. Inflation up to 40 cm H_2O was taken to represent total lung capacity and VC was taken as the volume between this inflation and the assumed RV. Further analysis of CV was performed analogous to the procedure in awake subjects.

The mean of 2–3 readings of ERV, CV and VC were used for further statistical analysis. CV was expressed as volume (ml) and as percentage of expired VC (%VC).[16] To test if the reproducibility of the CV measurement was affected by recruitment of atelectasis by the VC manoeuvre, the difference between the first and subsequent measurements of airway closure during general anaesthesia were compared with the amount of atelectasis. Only patients with more than 5 cm^2 of atelectasis (see below) were included in this analysis.

Further, the predicted VC (dependent on age, sex and height) for awake subjects was calculated according to Cotes.[17]

COMPUTED TOMOGRAPHY OF THE LUNGS

Atelectasis was studied by computed x-ray tomography (Somatom plus, Siemens, München, Germany) and has been described previously.[12][18] Subjects were in the supine position with their arms above the head. A frontal scout view, covering the chest, and a CT scan in the transverse plane, 1 cm above the top of the right diaphragm, were obtained at end-expiration (FRC), in the awake subject and after induction of anaesthesia. Scan time was 1 s, and slice thickness was 8 mm. With a matrix of 512×512, the resulting picture element (pixel) was approximately 1.5×1.5 mm.

To identify atelectasis, a magnified image of the dorsal portion of the CT scan of both the right and left lung was made. The dorsal border between the thoracic wall and the dense areas was drawn manually, whereas the ventral border between inflated lung tissue and atelectasis was identified by the region-of-interest (ROI) program. All pixels with attenuation values between -100 and $+100$ Hounsfield units (HU) were considered to represent atelectatic lung tissue.[18] For the logarithmic transformation (see results below), the smallest possible value of atelectasis was assumed to be 0.1 cm^2. This value represents the smallest amount that can reasonably be measured by CT.

VENTILATION–PERFUSION DISTRIBUTION AND BLOOD-GAS ANALYSIS

Ventilation–perfusion distribution ($\dot{V}A/\dot{Q}$) was assessed using the multiple inert gas elimination technique.[19][20] This method is based on the elimination and retention of a number of "inert" gases (usually six). Because pulmonary artery catheters were not acceptable for this study, a simplified method was applied, with measurements of inert gases in arterial blood and mixed expired air.[21] Using the Fick principle, cardiac output was estimated from oxygen con-
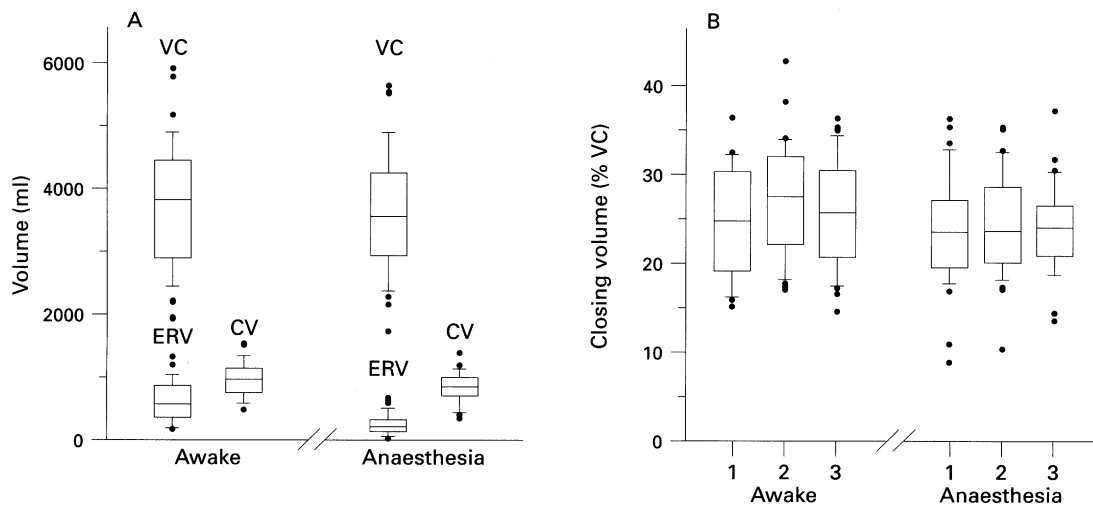
Figure 1 Box and whisker plots of lung volume measurements in awake and anaesthetized subjects. The box indicates the range between the 25th and 75th percentiles; the line marks the 50th percentile. Capped bars (whiskers) indicate the 10th and 90th percentiles and data outside these bars are shown as dots. For details on measurements, see text. A:VC = vital capacity, ERV = expiratory reserve volume and CV = closing volume. B: Consecutive measurements of CV (%VC) in awake and anaesthetized, supine subjects. Note that there was no difference between the first and third measurements, either awake or during anaesthesia.

sumption, assuming an arteriovenous oxygen content difference of 50 ml litre^{-1} blood,[14] and mixed venous inert gas concentrations were then computed using mass balance principles.[22] Approximation of cardiac output was imprecise but it has been shown that indices of $\dot{V}_A/\dot{Q}$ mismatch remain essentially unaffected by such uncertainties.[22] Further, shunt and low $\dot{V}_A/\dot{Q}$ may be estimated with reasonable accuracy.[14]

In brief, isotonic saline with a mixture of six inert gases (sulphur hexafluoride, ethane, cyclopropane, enflurane, diethyl ether and acetone) was infused continuously into a peripheral vein. Under steady state conditions, arterial blood and mixed expired gas samples were collected in duplicate for subsequent analysis by gas chromatography (Hewlett Packard Gas Chromatograph 5880A and 5890, Palo Alto CA, USA). By mathematical analysis of the inert gas data, each $\dot{V}_A/\dot{Q}$ distribution was recovered in a 50-compartment model and the result with the best fit of data (smallest remaining sum of squares, RSS) of each pair of duplicate samples was used for further statistical analysis. Intrapulmonary shunt was defined as the fraction of total blood flow perfusing lung units with $\dot{V}_A/\dot{Q} < 0.005$, and low $\dot{V}_A/\dot{Q}$ (= lung units with poor ventilation in relation to their perfusion) was defined as the fraction of total blood perfusion to lung units with $0.005 < \dot{V}_A/\dot{Q} < 0.1$. Log SDQ was calculated as the SD value of the logarithmic distribution of perfusion. This value is a measure of dispersion of blood flow distribution.

Blood-gas measurements were performed using a standard technique (ABL 300 Radiometer, Copenhagen, Denmark).

STATISTICAL ANALYSIS

Unless otherwise stated, mean (SD) values are presented. In addition, 95% confidence intervals (95% CI) are given where appropriate. To compare data between awake and anaesthetized subjects, the paired sample *t* test was used. The Pearson correlation coefficient was used to describe relationships between two different variables. To analyse relationships between

Table 2 Lung volume measurement, computed tomography of the lungs and gas exchange (mean (SD)) (see text for details). VC = Vital capacity; CV = closing volume; ERV = expiratory reserve volume; Atelectasis = dense areas (−100 to +100 HU), measured in a transverse CT scan at the dome of the right diaphragm; Thoracic area = cross-sectional chest area, measured in the same scan as atelectasis (for details see text); $F_{I_{O_2}}$ = fraction of inspired oxygen; Pa_{O_2}, Pa_{CO_2} = arterial partial pressure of oxygen and carbon dioxide, respectively; Shunt = $\dot{V}_A/\dot{Q} < 0.005$; low $\dot{V}_A/\dot{Q} = 0.005 < \dot{V}_A/\dot{Q} < 0.1$; log SDQ = SD of logarithmic distribution of perfusion; COest. = estimated cardiac output (see text)

	Awake	Anaesthetized	P
VC (ml)	3800 (1010)	3680 (1010)	0.2
ERV (ml)	650 (390)	300 (210)	<0.001
CV (ml)	980 (290)	870 (260)	0.034
CV (%VC)	25.9 (5.6)	24.1 (5.3)	0.09
CV–ERC (ml)	330 (410)	570 (290)	0.002
Atelectasis (cm2)	0.0 (0.0)	7.1 (6.6)	<0.001
Thoracic area (cm2)	347 (63)	325 (56)	<0.001
$F_{I_{O_2}}$	0.21	0.40	—
Pa_{O_2} (kPa)	12.5 (2.2)	19.8 (6.9)	—
Pa_{CO_2} (kPa)	5.2 (0.8)	4.5 (0.3)	<0.001
Shunt (% COest.)	0.1 (0.5)	6.5 (5.1)	<0.001
Low $\dot{V}_A/\dot{Q}$ (%COest.)	2.3 (3.3)	5.4 (8.7)	0.016
Log SDQ	0.76 (0.32)	1.08 (0.30)	<0.001
COest. (litre min^{-1})	4.8 (1.1)	3.8 (0.9)	<0.001

more than two variables, a stepwise multiple linear regression with analysis of residuals was performed. For all calculations, the SYSTAT 5.0 computer software package (SYSTAT, Evanston IL, USA) was used.

Results

AWAKE

In awake, supine subjects, VC was 3800 (1010) ml. This corresponds to 89 (17) % of the predicted VC in sitting subjects.[17] There was no significant difference between the first and succeeding measurements of CV (fig. 1, right). Mean CV was 980 (290) ml or 25.9 (5.6) % VC. The difference between CV and ERV (CV–ERV) was 330 (410) ml (range −1080 to 910 ml) (table 2, fig. 1, left).
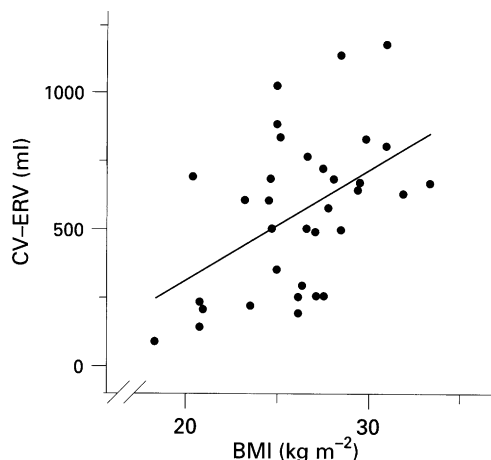
Figure 2 Body constitution and airway closure during general anaesthesia with mechanical ventilation. BMI = Body mass index, a measure of body constitution. CV–ERV = closing volume–expiratory reserve volume. A linear regression line is shown: CV–ERV (ml) = $-490 + 41 \times$ BMI (kg m^{-2}); $r = 0.49$, $P = 0.003$.

Table 3 Correlation coefficient of linear regressions between two variables during general anaesthesia (for definitions, see text)

Variable			r	P
Age	*vs*	CV %VC	0.38	0.024
Age	*vs*	Log SDQ	0.45	0.008
CV–ERV	*vs*	Atelectasis	0.32	0.092
CV–ERV	*vs*	Low $\dot{V}A/\dot{Q}$	0.57	0.001
CV–ERV	*vs*	Shunt + low $\dot{V}A/\dot{Q}$	0.63	<0.001
CV–ERV	*vs*	Log SDQ	0.51	0.003
Atelectasis	*vs*	Shunt	0.68	<0.001

CT scans revealed no atelectasis or other lung pathology. Data on gas exchange and ventilation–perfusion distribution are given in table 2.

GENERAL ANAESTHESIA

Airway closure and atelectasis

Mean VC in anaesthetized subjects was 3680 (1010) ml (fig. 1, left). This is equivalent to 98 (14) % of VC while awake. There was no increase in CV from the first to the second and third measurements (fig. 1, right). Mean CV was 870 (260) ml or 24.1 (5.3) % VC, and mean CV–ERV was 570 (290) ml (range 90–1190 ml). Compared with awake, CV–ERV increased by 250 (440) ml (95% CI 100–410 ml, $P = 0.002$) (table 2).

The relationship between airway closure and body constitution (BMI) is presented in figure 2. The characteristics of the linear regression between airway closure and age are given in table 3.
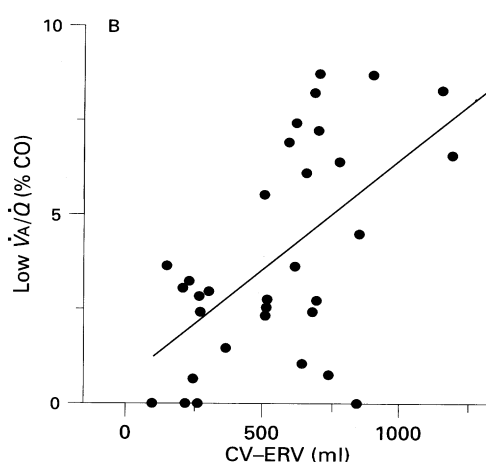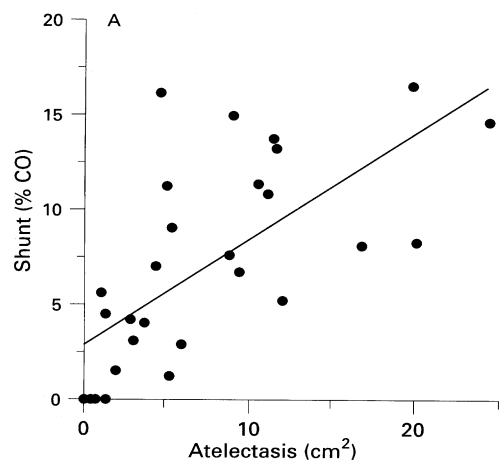
The mean amount of atelectasis at the base of the lungs was 7.1 (6.6) cm^2 (mean and 95% CI, calculated after logarithmic transformation: 3.4–1.9–6.2 cm^2). There was no correlation between airway closure and atelectasis (table 3).

Ventilation–perfusion distribution and Pa_{O_2}

Data on ventilation–perfusion distribution and oxygenation of arterial blood are given in table 2. There was a correlation between pulmonary shunt and atelectasis (fig. 3, left) and between airway closure (expressed as CV–ERV) and low $\dot{V}A/\dot{Q}$ (table 3, fig. 3, right). The correlation coefficient r was slightly greater when total perfusion to lung units with either poor or no ventilation (low $\dot{V}A/\dot{Q}$ + shunt) was related to airway closure (table 3).

A linear correlation was found between Pa_{O_2} on the one hand and shunt and perfusion to poorly ventilated lung units (low $\dot{V}A/\dot{Q}$) on the other ($r = 0.94$, $P < 0.001$). There also was a linear correlation between Pa_{O_2} and airway closure ($r = 0.59$, $P < 0.001$). Adding atelectasis as a further variable to this analysis gave a greater correlation coefficient and therefore an increased coefficient of determination (r^2):

$$Pa_{O_2}(kPa) = 29 - 2.9 \times \ln(\text{atelectasis (cm}^2))$$
$$- 0.0085\,(CV - ERV\,(ml)); r = 0.86, P < 0.001.$$

where Pa_{O_2} = arterial partial pressure of oxygen, measured at $F_{I_{O_2}} = 0.40$, and ln (atelectasis) = natural logarithm of atelectasis.

Figure 3 A: Atelectasis and shunt during general anaesthesia with mechanical ventilation. Atelectasis = amount of densities (+100 to −100 HU) in a CT scan 1 cm above the dome of the right diaphragm (cm^2); shunt = perfusion to lung units with $0.005 < \dot{V}A/\dot{Q} < 0.1$; CO = cardiac output. A linear regression line is shown: shunt (% CO) = 2.9 + 0.55×atelectasis (cm^2); $r = 0.68$, $P < 0.001$. B: Airway closure and ventilation–perfusion distribution mismatch during general anaesthesia with mechanical ventilation. CV–ERV (see fig. 2); low $\dot{V}A/\dot{Q}$ = perfusion to lung units with $0.005 < \dot{V}A/\dot{Q} < 0.1$; CO = cardiac output. A linear regression line is shown: low $\dot{V}A/\dot{Q}$ (% CO) = 0.68 + 0.006 × (CV–ERV (ml)); $r = 0.57$, $P = 0.001$.

Finally, Pa_{O_2} was also related to age and body constitution (BMI), resulting in the following equation:

$$Pa_{O_2} \; (kPa) = 53 - 0.16 \times age \; (yr) - 1.0 \times BMI \; (kg \; m^{-2})$$
$$r = 0.61, \; P = 0.001.$$

Discussion

We have shown that impaired oxygenation during uneventful anaesthesia in patients with healthy lungs can be explained by shunt and perfusion to units with poor ventilation in relation to their perfusion ("low $\dot{V}_A/\dot{Q}$"). The results also suggest that airway closure is a likely mechanism of low $\dot{V}_A/\dot{Q}$ during anaesthesia. Atelectasis is associated with shunt, as proposed in earlier investigations[3 4] and confirmed in this study. Together, atelectasis and airway closure can explain 75% of the impairment of arterial oxygenation.[1]

COMMENTS ON METHODOLOGY

Measurements of lung volumes, both in awake and anaesthetized subjects, yielded results similar to previous investigations.[8 16] The difference from predicted VC in the awake subject[17] may be explained by differences in body position (supine *vs* sitting).

Estimation of airway closure is based on either a resident gas technique (nitrogen washout) or a foreign gas bolus technique. A steep increase in the gas concentration at the end of expiration from VC (termed onset of phase IV) is considered to indicate an abrupt increase in the amount of airway closure in dependent regions of the lung.[5] Accordingly, the volume above RV at which phase IV begins is termed CV. Despite the fact that the term "airway closure" might suggest a single clearly defined event, it should be remembered that a certain amount of airway closure may exist before the onset of phase IV.[23] Further, a marked increase in airway resistance[24 25] or flow limitation[23] may contribute to the onset of phase IV. Despite these limitations, strong evidence exists for the occurrence of true airway closure, although it may not be the sole mechanism responsible for the onset of phase IV (for a detailed review see Rehder[26]).

For measurement of airway closure in anaesthetized subjects, the foreign gas bolus technique is more appropriate than the nitrogen washout technique because of less dependency on the vertical lung volume gradient[16] and on variations in the inspiratory manoeuvre.[27] Further, rapid onset of absorption atelectasis[14] may interfere with CV measurements if the VC manoeuvre is performed with 100% oxygen, as used in the nitrogen washout technique. In our investigation, using the foreign gas (helium) bolus technique and room air for the VC manoeuvre, there was no obvious relationship between amount of atelectasis and variability between the two or three measurements of CV during general anaesthesia. This agrees with the finding that previously collapsed lung tissue is reopened within the first few seconds of a VC manoeuvre (Neumann and colleagues, unpublished observations) and with the fact that most of the re-expanded lung tissue remains aerated for more than 40 min if air is used for the VC manoeuvre and if the lungs are ventilated with 40% oxygen in nitrogen thereafter.[13] Lung tissue, re-opened during the initial phase of the first measurement procedure, is accessible and thus is included during all subsequent measurements of airway closure.

AIRWAY CLOSURE

The significant increase in airway closure (expressed as CV–ERV) from awake to anaesthesia confirms earlier investigations,[9 16] but some studies found no difference in CV–ERV between the awake state and anaesthesia.[10] Appropriate explanations for these different results are lacking.

In anaesthetized subjects, there was a correlation between airway closure (CV–ERV) and body constitution, expressed as body mass index (BMI). Obese patients tended to have more airway closure during anaesthesia, and airway closure was greater in older patients, which is similar to findings in conscious subjects.[7]

AIRWAY CLOSURE, ATELECTASIS AND GAS EXCHANGE

Pulmonary shunt was greater in anaesthetized subjects whose airway closure (although measured while awake) occurred above FRC.[28] Our data, with both variables measured in anaesthetized subjects, did not confirm such a close relationship between shunt and airway closure. However, there was a significant linear correlation between airway closure (expressed as CV–ERV) and low $\dot{V}_A/\dot{Q}$. Subjects with a larger CV above ERV (CV–ERV) had greater perfusion to poorly ventilated lung units. There also was a linear correlation between airway closure and other measures of ventilation–perfusion distribution mismatch (log SDQ or low $\dot{V}_A/\dot{Q}$ + shunt). These findings suggest that airway closure may reflect a major part of lung units subject to severe ventilation–perfusion mismatch.

A correlation between airway closure and Pa_{O_2} during general anaesthesia has been found previously.[8 11] These investigations concluded that airway closure contributed to pulmonary dysfunction, but may not be the most important mechanism.[11] This was confirmed by our investigation. Thus the more airway closure, the worse the oxygenation of arterial blood. Adding atelectasis to the linear regression equation markedly increased the correlation coefficient. Our data showed that 75% of the variation in Pa_{O_2} was accounted for by atelectasis and airway closure.[29] If one considers the many other factors (e.g. cardiac output, oxygen consumption) that may influence Pa_{O_2}, this agreement is quite high. Therefore, we conclude that both atelectasis and airway closure are important determinants and may explain most of the impairment of gas exchange during general anaesthesia in patients with healthy lungs undergoing mechanical ventilation.

Based on the above findings, the "ideal patient", with no atelectasis and no airway closure during normal tidal breathing, will have an estimated Pa_{O_2} of 36 kPa at an $F_{I_{O_2}}$ of 0.4. This hypothetical value is in accordance with that described recently for subjects with no shunt and no $\dot{V}_A/\dot{Q}$ mismatch.[30]

In summary, in adults with healthy lungs, both airway closure and atelectasis contributed to impairment of gas exchange during general i.v. anaesthesia with mechanical ventilation. There was a close rela-

tionship between airway closure and disturbance of ventilation–perfusion distribution. Atelectasis and shunt impaired gas exchange, but the amount of atelectasis did not appear to be related closely to the amount of airway closure. Impairment of gas exchange was more pronounced in older and obese patients.

Acknowledgements

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