Airway closure in anesthetized infants and children: influence of inspiratory pressures and volumes

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Background: Cyclic opening and closing of lung units during tidal breathing may be an important cause of iatrogenic lung injury. We hypothesized that airway closure is uncommon in children with healthy lungs when inspiratory pressures are kept low, but paradoxically may occur when inspiratory pressures are increased.

Methods: Elastic equilibrium volume (EEV) and closing capacity (CC) were measured with a tracer gas (SF6) technique in 11 anesthetized, muscle-relaxed, endotracheally intubated and artificially ventilated healthy children, aged 0.6–13 years. Airway closing was studied in a randomized order at two inflation pressures, 20 or 30 cmH2O, and CC and CC/EEV were calculated from the plots obtained when the lungs were exsufflated to >20 cmH2O. (CC/EEV ≥ 1 indicates that airway closure might occur during tidal breathing). Furthermore, a measure of uneven ventilation, multiple breath alveolar mixing efficiency (MBAME), was obtained.

Results: Airway closure within the tidal volume (CC/EEV ≥ 1) was observed in four and eight children (not significant, NS) after 20 and 30 cmH2O inflation, respectively. However, CC30/EEV > CC20/EEV in all children (P<0.001). The MBAME was 75±7% (normal) and did not correlate with CC/EEV.

Conclusion: Airway closure within tidal volumes may occur in artificially ventilated healthy children during ventilation with low inspiratory pressure. However, the risk of airway closure and thus opening within the tidal volume increases when the inspiratory pressures are increased.

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Key words: airway closure; sulfur hexafluoride; anesthesia; pediatric; multibreath washout; functional residual capacity.

It has been suggested that children undergoing surgery during general anesthesia may benefit from ventilation with high inspiratory pressures and large tidal volumes because this will reduce the incidence of atelectasis (1, 2). On the other hand, this type of ventilation may increase the risk of volu- and barotrauma (3, 4). High inspiratory pressure and large tidal volumes may thus produce cyclic opening and closing of unstable lung units (5). However, whether airway closure occurs in children below six years of age has not been studied, and it is not known if high inspiratory pressures would increase the tendency towards airway closing in children with normal lungs.

It is well known that lung collapse occurs within minutes after the induction of general anesthesia in children (6, 7) and adults predominantly as a result of a cranial shift of the diaphragm and breathing of high inspired oxygen concentration (8, 9). Some of this collapse can be regained or counteracted by lung recruitment by using low inspired oxygen concentration and by employing positive end-expiratory airway pressure (PEEP) (7, 10, 11). Nevertheless, anesthesia makes the lungs prone to collapse in infants and children (7).

Low inspiratory pressures may not open collapsed lung units (12), but if the inspiratory pressure is high enough to open the atelectatic airways it is likely that these unstable units will also collapse during the subsequent expiration.

We thus hypothesized that ventilation with a high inspiratory pressure and large tidal volumes produces cyclic opening and closing of unstable lung units.

To test this hypothesis we studied airway closure at two inspiratory pressure levels in healthy anesthetized children. We used pressure levels that would generally be viewed as ‘normal’ and ‘high’ (20 and 30 cmH2O, respectively) inspiratory pressures in normal healthy children during artificial ventilation.

Patients and methods
Eleven children, seven girls and four boys (0.6–12.8 years of age), scheduled for elective surgical pro-
cedures requiring general anesthesia and endotracheal intubation were studied (Table 1). Other than the reason for surgery, the children were healthy and had no signs of respiratory disease. The study was approved by the Human Studies Committee and parental consent was given in each case.

Procedure
Anesthesia was induced with i.v. thiopental and the patients were paralyzed with a non-depolarizing muscle relaxant (vecuronium 0.1 mg kg⁻¹), intubated with a cuffed endotracheal tube, and ventilated mechanically with 1% halothane in oxygen/nitrogen (FiO₂=0.6). The cuff of the endotracheal tube was not inflated. During mechanical ventilation the ventilator (Servo 900 C, Siemens-Elema, Solna, Sweden) was set at volume control, a rate of 20–30 min⁻¹, 25% inspiration, 10% end-inspiratory pause, 65% expiration, and ventilation was adjusted to give an end-tidal PCO₂ of 4–5 kPa. Electrocardiography (EKG), blood pressure, and pulse oxymetry saturation were monitored in all patients.

The measurements of elastic equilibrium volume (EEV) and closing capacity (CC) lasted 15–30 min and were made with the patient supine and during continued muscle paralysis. Before the measurement, i.e. 10–15 min after induction of anesthesia, the cuff of the endotracheal tube was inflated and it was ascertained by auscultation and by comparing inspired and expired tidal volumes that no leakage was present. To allow normalization of CC data to absolute volumes, the CC measurements were preceded by EEV measurements.

To standardize lung volume history, the lungs were manually inflated to an airway pressure of approximately 20 cmH₂O before and after each EEV and CC determination. During EEV measurements, 5 cmH₂O of PEEP was applied during washin. To give time for the PEEP effect on lung volume to dissipate, which usually occurs within five breaths (13), PEEP was discontinued 0.5–2 min before starting washout. Data was stored on computer disks. Flow, volume, and airway pressure were continuously recorded on an ink-jet recorder.

Measurements
Elastic equilibrium volume was measured with an open tracer gas technique using sulfur hexafluoride (SF₆) as a tracer gas. The tracer gas concentration was measured by an infrared analyzer placed over a cuvette in the apparatus deadspace, and the flow was measured by a heated Fleisch pneumotachograph (Gould 00, 0 or 1) with a differential pressure transducer (Validyne, MP 45-1-871). The method has been described in detail elsewhere (14–16): SF₆ is washed in through a dispensing device, which mixes SF₆ in proportion with instantaneous inspiratory flow. In this way, a uniform inspired concentration is achieved even with a non-constant inspiratory flow. The wash-in continues until a stable end-tidal concentration of approximately 0.5% is attained. Sulfur hexafluoride washout is started by stopping tracer gas delivery between two inspirations, and is considered complete when the mean expired concentration is less than 0.001%. Signals representing flow and SF₆ concentration are fed into a personal computer that integrates the flow signal and gives an on-line display of inspired and expired volumes and of the tracer gas concentration in each breath. At the end of the washout, EEV is calculated as the volume of SF₆ washed out divided by the alveolar concentration at the end of the washin period. The value is converted to body

Table 1

<table>
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<tr>
<th>Patient no.</th>
<th>Age (y)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Sex</th>
<th>Reason for surgery</th>
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<td>F</td>
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<td>Anal atresia</td>
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<td>92</td>
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</tr>
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<td>103</td>
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<td>Meatoimi</td>
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<tr>
<td>8</td>
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<td>19</td>
<td>119</td>
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<tr>
<td>9</td>
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<td>28</td>
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<td>145</td>
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<td>38</td>
<td>155</td>
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<tr>
<td>SD</td>
<td>4.4</td>
<td>12.8</td>
<td>31.6</td>
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</table>

SD, standard deviation.
temperature and pressure of water vapour saturated gas (BTPS) conditions and apparatus deadspace is subtracted. Tidal volume, mean expired SF₆ concentration, end-tidal SF₆ concentration, and the SF₆ volume obtained in each expiration are stored on computer disks for later analysis of the washout curve.

In the present study, the apparatus deadspace was 8 ml with Fleisch pneumotachograph no. 00, 12 ml with no. 0, and 48 ml with no. 1. To ascertain that the lung volume was measured at zero alveolar pressure the expiration between washin and washout was prolonged to 3–5 s.

Closing was measured with the set-up shown in Fig. 1. The flow and SF₆ signals were obtained as in the previous set-up but the child was disconnected from the ventilator and connected to a 3-l syringe containing equal parts of oxygen and air. The measurements were done as follows: from EEV the lungs were deflated to a pressure of −20 cmH₂O, as assessed by a water manometer, at a level considered to reflect residual volume (RV), a bolus of 100% SF₆ (0.005 ml/ml EEV) was administered with a small syringe into the airway close to the tracheal tube, and the lungs were inflated to +20 cmH₂O and +30 cmH₂O. The order of inflation pressures was randomized. The computer registered the volumes. During the subsequent deflation to −20 cmH₂O, which was done slowly (the flow rate was 30 ml s⁻¹ for the youngest and 150 ml s⁻¹ for the oldest children) over 7–9 s to avoid dynamic compression of airways, signals representing expired tracer gas concentration and expired volume were processed by the computer and subsequently plotted on an X-Y recorder.

Calibrations

The SF₆ analyzer was calibrated with a mixture containing 0.50±0.01% SF₆ (Alfax, Sweden). The linearity of the SF₆ reading was assessed by exposing the measurement system to SF₆ mixtures of a known composition, prepared by a precision gas mixer (Digamix G 18, H Wösthoff, Bochum, West Germany). The flow signal was calibrated before each measurement sequence with an accurate reciprocal pump using equal parts of air and oxygen. Zero adjustment of the flow signal was carried out before each individual measurement and the adjustment was repeated if the zero level had changed by more than 1 ml s⁻¹. A factor of 1.09 was used to convert the volumes and flow from ambient temperature and pressure of water vapour saturated gas (ATPS) to BTPS conditions.

Data analysis

At the end of the study, copies of the expiratory curves (Fig. 2) were placed in a random order, and analyzed by two independent observers. The closing point was taken to be the point at which upward departure occurred from a ‘best-fit’ line through the latter half of phase III (17, 18). The volume above closing

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*Fig. 1.* The system used for closing volume measurements. During inflation the pressure was controlled by a water manometer. The volume changes during the measurement procedure are shown in the insert. The dashed arrows indicate the flow of information. The apparatus deadspace between the tracheal tube and the tracer gas analyzer was 5 ml.

*Fig. 2.* Single-breath tracing obtained in a patient (aged 2.6 years) after inflation to 30 cmH₂O. The X-axis of the original tracing has been modified to show the absolute lung volumes. The volume corresponding to the inspiratory capacity found during inspiration identified the position of the elastic equilibrium volume (EEV) during expiration. Note the cardiac oscillations. The dots in the tracing are 0.4 s apart. CC, closing capacity; RV, residual volume.
point (VaCP) was noted. Corrections were made for the apparatus and airway deadspace and the SF₆ bolus. The deadspace volume was obtained from the first washout breaths during EEV measurements as the volume expired when the SF₆ concentration had reached 50% of the tracer gas concentration at the end of phase III (19). When calculating the CC, the deadspace value was corrected for the difference in the apparatus deadspace during EEV and the closing measurements. No correction was made for the effect of viscosity changes on pneumotachograph readings during the deflation-inflation maneuver: the gas mixture was nearly the same in the syringe and the lungs, and the resulting error in the CC values was estimated to be less than 1%.

Expiratory reserve volume (ERV) was all the volume between EEV and −20 cmH₂O. The vital capacity between −20 cmH₂O and +20 (30) cmH₂O (VC₂₀ and VC₃₀) was registered during both insufflation and exsufflation, and then compared.

The volume above EEV, i.e. the inspiratory capacity (IC), was defined as VC₂₀ and VC₃₀ minus ERV.

On the plotted curve, EEV was defined as the lung volume where the volume corresponding to the IC had been exsufflated (Fig. 1) and the CC was found to be IC₂₀ or IC₃₀ – EEV – VaCP.

To test whether the occurrence of airway closure could be predicted from other data, the slope of phase III, expressed as a change in tracer gas concentration (ΔSF₆%) per liter during deflation divided by mean SF₆% during phase III (ΔSF₆%/mean SF₆%) (20, 21), and an index of ventilation inhomogeneity, the multiple breath alveolar mixing efficiency (MBAME), were also calculated. Multiple breath alveolar mixing efficiency is defined as 100×TOideal/TOactual where TOideal is the ideal number of turnovers (cumulative expired volume/EEV) needed to wash 90% of EEV free of tracer gas, and TOactual is the actual number of volume turnovers (22). In the calculations of the MBAME, volume turnovers were corrected for deadspace (19). In a previous study in supine, awake adults without lung disease, 36±11 years of age, the mean MBAME (±SD) was 67±7%. In these patients, the mean MBAME was the same during subsequent anesthesia and mechanical ventilation (19). The normal MBAME values reported from nitrogen washout were 75±10% (22).

Statistics

A priori we estimated the number of patients needed to detect a significant difference in the primary endpoint, i.e. CC/EEV, between the two pressure levels to be 10. A posteriori power analysis using the mean value and the SD for the obtained difference gave α = 0.05 and β = 0.25, i.e. a power of 0.75.

The mean results obtained in each individual and for each inflation pressure were used for statistical analysis. Regression lines were calculated by the method of least squares. Significance of linear correlations was assessed with the t-test. The t-test for paired data was used to compare duplicate measurements and the difference between inspiratory and expiratory VC. Wilcoxon’s signed rank test was used to assess the effect on lung volumes and closing data when increasing the inflation pressure from 20 to 30 cmH₂O. Reproducibility is expressed as the coefficient of variation, i.e. as SD/m−1 (= D/(m·√2), where D is the absolute value of the difference between two observations and m is the mean. The difference in the number of children with CC/EEV > 1 between the two pressure levels was assessed with Fisher’s exact test.

P-values less than 0.05 were considered statistically significant. Data are presented as mean ± SD when not otherwise indicated.

Results

Lung volume measurements (Tables 2 and 3)

EEV was 20.2 mlkg⁻¹ and inspiratory VC₂₀ and VC₃₀ was 29±10 and 43.3±12.8 ml/kg, respectively.

The coefficient of variation for duplicate measurements was 3.0±2.4% (n=9, NS) for EEV and 3.3±2.6% and 4.2±3.7%, respectively, for the duplicate inspiratory and duplicate expiratory VC measurements (n= 16, NS).

The inspiratory VC₂₀ (n=18) and VC₃₀ (n=20) was

<table>
<thead>
<tr>
<th>Table 2</th>
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<tbody>
<tr>
<td>Elastic equilibrium volume and multiple breath alveolar mixing efficiency.</td>
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<tr>
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</tr>
<tr>
<td>1</td>
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<td>10</td>
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<tr>
<td>11</td>
</tr>
<tr>
<td>Mean</td>
</tr>
<tr>
<td>SD</td>
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</table>

EEV, elastic equilibrium volume; MBAME, multiple breath alveolar mixing efficiency; SD, standard deviation.
Table 3

Comparison of values obtained using two different inspiratory pressures.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Inflation pressure 20 cmH(_2)O</th>
<th></th>
<th>Inflation pressure 30 cmH(_2)O</th>
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</tr>
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<tbody>
<tr>
<td></td>
<td>VC(_{in})</td>
<td>VC(_{out})</td>
<td>CC/EEV</td>
<td>Slope phase III</td>
</tr>
<tr>
<td>1</td>
<td>139</td>
<td>122</td>
<td>&lt;RV</td>
<td>0.28</td>
</tr>
<tr>
<td>2</td>
<td>142</td>
<td>136</td>
<td>1.19</td>
<td>0.24</td>
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<tr>
<td>3</td>
<td>181</td>
<td>168</td>
<td>1.02</td>
<td>0.06</td>
</tr>
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<td>4</td>
<td>188</td>
<td>172</td>
<td>0.93</td>
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<td>429</td>
<td>431</td>
<td>0.78</td>
<td>0.09</td>
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<td>460</td>
<td>449</td>
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<td>584</td>
<td>580</td>
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<td>1701</td>
<td>0.86</td>
<td>0.03</td>
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<tr>
<td>Mean</td>
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<td>583.2</td>
<td>0.95</td>
<td>0.10</td>
</tr>
<tr>
<td>SD</td>
<td>553.7</td>
<td>523.9</td>
<td>0.11</td>
<td>0.08</td>
</tr>
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</table>

VC, vital capacity; CC, closing capacity; EEV, elastic equilibrium volume; RV, residual volume.

30.4±35.8 ml (6.0±4.5%, P≤0.001) and 29.3±38.6 ml (3.7±4.4%, P≤0.01) greater than the corresponding expiratory VC measurements.

These volume differences were thus similar and not significantly different. However, when relating the difference to age a significant connection was found: VC\(_{30}\)diff (ml) = 6.6×age (years) − 1.9, r = 0.75, P≤0.008.

Closing measurements

Mean tracer gas concentration during phase III was 0.26±0.09%. A phase IV phenomenon was observed in 10 of 11 children at an inflation pressure of 20 cmH\(_2\)O (i.e. one child had a closing point below RV), and in all 11 children at an inflation pressure of 30 cmH\(_2\)O. The coefficient of variation for VaCP values calculated from the two independent interpreters’ assessment of the closing point was 2.4±2.1% (n=36, NS). Closing capacity increased by 9.2±5.3% when the inflation pressure was increased from 20 to 30 cmH\(_2\)O (P≤0.005).

The CC/EEV ratio was over 1.0 in four of 11 children at an inflation pressure of 20 cmH\(_2\)O and in eight of 11 children at an inflation pressure of 30 cmH\(_2\)O (NS) (Fig. 3), and had no significant correlation to age, weight or height (Fig. 3). In the 10 children in whom both inflation pressures gave closure above RV, CC/EEV obtained at an inflation pressure of 20 cmH\(_2\)O was significantly correlated to CC/EEV obtained at an inflation pressure of 30 cmH\(_2\)O (r=0.92, P≤0.005) (Fig. 4).

The coefficient of variance for duplicate measurements of the MBAME was 5.2±5.3% (n=11, NS). The MBAME was not correlated to age, CC/EEV ratios, or the difference between inspiratory and expiratory vital capacity.

The slope of phase III became steeper when the in-

![Fig. 3. The closing capacity/elastic equilibrium volume (CC/EEV) ratio. The ratio was higher than 1.0 in four of the 11 children when the inflation pressure was 20 cmH\(_2\)O, and in eight of the 11 children when the inflation pressure was 30 cmH\(_2\)O. One patient did not have closing over residual volume (X).](image1)

![Fig. 4. The closing capacity/elastic equilibrium volume (CC/EEV) ratios with the inflation pressures of 20 and 30 cmH\(_2\)O were significantly correlated. One patient did not have closing over residual volume (X). The line of identity is shown.](image2)
flation pressure was increased from 20 to 30 cmH2O (p<0.003). No correlation was found between the slope of phase III and the CC/EEV or the MBAME.

Discussion

This study shows that: (1) airway closure within tidal volumes might occur in healthy children during artificial ventilation at peak inspiratory pressures of 20 cmH2O, and (2) the risk of airway closure and thus also of opening and closing within the tidal volume increases when the inspiratory pressure is increased. Before discussing these findings and their clinical implications some methodologic issues need to be addressed.

This study’s EEV measurement method has been used in studies in neonates, children and adults (14–16). The measurements agree well with actual volumes in lung models and with body plethysmography and nitrogen washout measurements in adults (14, 15). The reproducibility in the present study is similar to our findings in earlier studies.

It should be noted that although the accuracy of the EEV measurements is important when calculating the CC/EEV ratio, it has no effect on whether the CC/EEV is greater than 1 or not, because this is determined by the difference between the inspiratory VC and the ERV measurements and the identification of the closing point (CP). The tracer gas bolus technique has been used by several earlier investigations with anesthetized patients (23, 24).

The SF6 bolus produced an obvious phase IV phenomenon during 21 of the 22 measurements. The identification of CP was less obvious in some patients but the agreement in the measurements between the two independent observers suggests that this was not an important source of error.

Nevertheless, our technique has several limitations. First, tracer gas washout techniques cannot detect firmly collapsed or consolidated lung regions. Second, the estimation of airway closure was done under quasi-dynamic conditions and not under normal tidal breathing. Although these conditions differ, it seems unlikely that airway closure would occur during tidal breathing if it cannot be demonstrated with our technique. On the other hand, if the method indicates closing above EEV, it may very well exist during tidal breathing. We also assumed that there is an equilibrium between opening and closing during tidal breathing. Thus if the method found an airway closing above EEV we assumed that the airway opening also occurred above EEV.

The study was designed to compare two insufflation pressure-levels: 20 cmH2O and 30 cmH2O. However, these pressures generated large volumes: a mean volume of 20 ml kg-1 at 20 cmH2O and a mean volume of 36 ml kg-1 at 30 cmH2O. Thus, even 20 cmH2O in this setting gave a volume almost twice that of a large normal tidal volume. This was probably because the children had healthy lungs with a high compliance and a long duration of the insufflation, approximately 2–3 s, which is two- to six-fold the normal inspiratory time. However, it is important to realize that it is not the tidal volume in itself that opens collapsed or closed lung units: it is the trans-pulmonary pressure in the terminal airways near the collapsed lung unit. Theoretically, a trans-pulmonary pressure of approximately 16 cmH2O is needed to open the atelectatic lung regions (25), and to open all the atelectatic regions induced by anesthesia in adults a mean airway pressure of 40 cmH2O is required (12).

It is well recognized that rapid exsufflation may result in dynamic compression and gas trapping. To avoid this we exsufflated the lungs over 7–9 s, which gave a flow rate of 30 ml s-1 for the youngest and of 150 ml s-1 for the oldest patients; this is well below the flow rate recommended for single-breath closing tests in children (200–300 ml s-1; 18). Still, we believe that some air trapping did occur during the exsufflation and that this is the main reason why the insufflated volume was somewhat larger than the exsufflated volume during the closing maneuver. Thus, we do not think that this was as a result of the gas exchange and oxygen consumption during this short maneuver, but more likely because of the airway closure with the entrapment of air distal to the closed airways.

The mechanism for this may be as follows: before the closing maneuver the patient was disconnected from the ventilator and connected to the closing volume measurement system. The 5 cmH2O of PEEP was thus discontinued and the lungs therefore slowly emptied to EEV. In some patients this may have resulted in airway closure without air entrapment. When the lungs where subsequently inflated to 20 or 30 cmH2O airway pressure, the closed airways opened, but during the subsequent exsufflation airways closed more rapidly and air was entrapped, resulting in a somewhat smaller expiratory VC. This interpretation agrees with the findings of Sigurdsson and co-workers (26) who observed by analyzing pressure-volume curves that the lungs lost more volume when 5 cmH2O of PEEP was discontinued (over 6 s) in anesthetized patients with normal lungs, than could be expected on the basis of the reduction in PEEP. At 5 cmH2O the lost lung volume was approximately 160
ml. Most of the lung volume was regained when the airway pressure was again increased above 20 cmH2O. If this explanation were correct, one would expect the CC to decrease by about 30ml with decreasing exsufflation rate in our study.

We found that the CC/EEV ratio was >1 in four of the 11 children when the insufflation pressure was 20 cmH2O. This indicates that airway closing might occur during tidal breathing when end-inspiratory airway pressure is 20 cmH2O. However, our measurement was performed during quasi-dynamic conditions. Furthermore, EEV was estimated from the starting point of the maneuver. As we found that the exsufflated air was about 30ml, i.e. 4–6% less than the insufflated air, we can assume that this air was trapped in the residual volume and that the ‘dynamic elastic equilibrium volume’ could have been located at a higher level than we had estimated. This fact is important because in the four children that had a CC/EEV > 1 with the closing maneuver performed at 20 cmH2O, CC/EEV was very near 1, indicating that a small under-estimation of the location of the elastic equilibrium volume would indeed change the ratio to below 1. Moreover, as mentioned, 20 cmH2O insufflation gave a volume almost twice that of a normal large tidal volume. If instead we had adjusted the insufflation pressure to obtain a 10–15 ml kg\(^{-1}\) inspired volume, which is similar to a normal tidal volume, the insufflation pressure would have been lower. A lower airway pressure would open fewer, if any, closed units and we would probably have found few, if any, signs of closing above EEV. As also discussed, the finding that most children had no airway closure above EEV does not exclude the possibility that the children had atelectatic lung regions. Our results only indicate that these regions did not open and close when 20 cmH2O of airway pressure was applied and withdrawn, suggesting that this is not likely to occur during tidal breathing using the same inspiratory pressure. Hence, from our data we can deduce that airway closing and opening might occur but are probably not common during artificial ventilation at low inspiratory airway pressures. This is also supported by the fact that we did not find any correlation between MBAME and CC/EEV. Closing of airways within tidal breathing would theoretically give a low MBAME.

The CC/EEV ratio did increase when the insufflation pressure was increased to 30 cmH2O. This agrees with findings by Holz and coworkers in awake adults (27) and Hedenstierna in anesthetized adults (28). The difference between insufflated and exsufflated volume was numerically the same at an insufflation pressure of 30 cmH2O as at 20 cmH2O. This implies that increased closing tendency was not a result of the closure of small airways with increased trapping of air because this would have increased the difference between insufflated and exsufflated air. Instead, we believe that it was caused by the opening of the collapsed unstable lung units during insufflation, which again closed totally above EEV during exsufflation. Hence, high inspiratory pressures might cause the cyclic opening of the collapsed lung units with subsequent closing. Repeated opening and closing of lung units generates extremely high shear forces in the lung parenchyma (29).

In adult patients with normal lungs ventilated short-term with 15 ml kg\(^{-1}\) tidal volumes, Wrigge and coworkers did not find any mechanic or biochemic indication of lung injury (30). However, in their study the mean peak inspiratory pressure was only 16 cmH2O, which supports our notion that a higher pressure is needed to open closed lung units and thus induce cyclic opening and closing of lung regions and lung injury.

Conclusion

Our study indicates that opening and closing of lung units might occur, but is probably not common, at artificial ventilation at an inspiratory pressure of 20 cmH2O. However, as it seems to be more regular when the airway pressure is increased to 30 cmH2O, we believe that it is pertinent to also avoid such a high inspiratory pressure in healthy children.

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