MEASUREMENT OF END-EXPIRATORY PRESSURE DURING TRANSTRACHEAL HIGH FREQUENCY JET VENTILATION FOR LARYNGOSCOPY

J. L. BOURGAIN, E. DESRUENNES, M. F. COSSET, G. MAMELLE, S. BELAICHE AND J. TRUFFA-BACHI

SUMMARY
An anaesthetic technique using high frequency jet ventilation has been proposed for direct laryngoscopy, but this may expose the patients to the risk of barotrauma. In order to assess this risk, we have measured end-expiratory airway pressure (EEP) through the injector using two three-way solenoid valves mounted in series. At the end of insufflation the first valve was switched off and the apparatus deadspace connected to atmosphere through a large exit port during an adjustable time (decompression time). Then the second valve was switched off and the injection line connected to a transducer, allowing measurement of EEP through the injector. The accuracy of this measurement was tested against airway pressure measured directly in the trachea (Pt) in a lung model. Provided that the decompression time was long enough (70 ms) and the apparatus deadspace was small (6 ml), the difference between EEP and Pt was less than 1 cm H₂O for frequencies up to 5 Hz. A clinical evaluation was performed in 64 patients under general anaesthesia before laryngoscopy. EEP correlated with end-expiratory pulmonary volume above apnoeic FRC inferred from abdominal and thoracic displacements. At jet frequencies up to 5 Hz, the correlations between these two variables were satisfactory (r > 0.88), suggesting that EEP is a good indicator of pulmonary overdistension.

KEY WORDS

High frequency ventilation (HFV) has been proposed as an alternative to conventional ventilation for laryngoscopy under general anaesthesia [1]. Different methods of tracheal access have been suggested: nasotracheal, orotracheal and transtracheal [2-5]. The cricothyroid membrane catheter is particularly interesting because it avoids intubation manoeuvres. Potential hazards of jet ventilation include barotrauma with subcutaneous emphysema, pneumomediastinum or pneumothorax [6-8]. The risk of barotrauma may be greater in patients with upper airway obstruction, a condition which is frequently found in patients requiring laryngoscopy.

Detecting hyperinflation is difficult when the trachea is not intubated; it involves either measurement of airway pressure or external spirometric methods [9, 10]. External spirometric methods possess technical and methodological difficulties in terms of linearity, frequency response and calibration [11].

Mean airway pressure has been reported to correlate well with mean pulmonary volume during high frequency jet ventilation (HFJV) [12], but its measurement requires the use of a special intratracheal cannula which may be inconvenient when the trachea is intubated with a small tracheal tube, or when HFJV is delivered via a transtracheal catheter. Measurement of this article is accompanied by an Editorial.

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airway pressure is possible via the injector at the end of expiration and if the end-expiratory pressure (EEP) is a good reflection of pulmonary distension, this would be a useful manoeuvre.

The aim of this study was to evaluate: the difference between EEP measured via the injector and through a tracheal catheter in a lung model; the accuracy of EEP as a reflection of pulmonary distension during HFJV in patients undergoing direct laryngoscopy; and the reliability of a device to stop the ventilator automatically when EEP reaches a preset value in order to avoid pulmonary distension.

METHODS

EEP measurement

The ventilator used (GR300, LSA, France) operated as described by Carlon and colleagues [13]. This device is solenoid operated and the electronic clock allows independent setting of ventilatory frequency and inspiratory:expiratory (I:E) ratio. The unique feature of this ventilator is the measurement of airway pressure through the injector catheter between each insufflation. Two three-way solenoid valves in series are used for this purpose (fig. 1). During insufflation the two valves are open. At the end of insufflation, the proximal valve is switched off and the apparatus deadspace connected to atmosphere. For 70 ms, compressed gas escapes into the atmosphere through a large port and the operating pressure returns quickly to baseline. Then the second solenoid valve is switched off and the injection line connected to a piezoelectric transducer, allowing measurement of dynamic end-expiratory airway pressure (EEP) via the injector. The frequency response of the transducer was flat up to 20 Hz. The technical characteristics of each valve are: internal diameter 3 mm, opening time 12 ms, closing time 17 ms. In case of system failure, valves close automatically.

Each EEP value is recorded by a microprocessor in the ventilator. The microprocessor allows successive inspirations only when EEP is less than a preset value (low mandatory pressure) which may be adjusted from 1 to 30 cm H₂O.

To assess the validity of EEP measurement through the injector, we used a lung model (Manley lung ventilator performance analyser). The static compliance of the lung model was 66 ml cm H₂O⁻¹ and the downstream resistance was minimal. The end-expiratory lung volume was 500 ml. EEP was compared with the pressure measured through a tracheal catheter (Pt), the tip of which was placed 5 cm downstream from the tip of the injector and connected directly to a quartz transducer (HP 1290); the frequency response of the pressure measurement system was flat up to 30 Hz. The driving pressure (3.8 bar), inspired oxygen concentration (F1O₂ = 1) and I:E ratio (0.54) were kept constant. Five ventilatory frequencies were studied (1, 2, 3, 4 and 5 Hz), three injector diameters (2, 1.7 and 1.3 mm i.d.) and two apparatus deadspaces (6 ml and 1.8 ml). All these settings and designs were tested in two lung configurations: with minimal airway resistance, and with a resistance (8 cm H₂O litre⁻¹ s⁻¹) located at the proximal tip of the tracheal tube (to simulate upper airway obstruction).

Clinical study

We studied 64 patients (59 males) aged 59 (SD 9) yr, undergoing endoscopy for upper airway cancer. We excluded patients with contraindications to cricothyroid puncture (local infection, neoplastic extension near the puncture point, anticoagulant therapy) and patients with severe cardiac disease.

The study was approved by our local Ethics Committee and all patients gave informed consent.

Diazepam 0.15 mg kg⁻¹ and atropine 0.007 mg kg⁻¹ were given i.m. 1 h before laryngoscopy. Anaesthesia was induced with an i.v. bolus dose of propofol 2 mg kg⁻¹ or methohexisone 3 mg kg⁻¹ in combination with fentanyl 0.10-0.15 mg. Muscle relaxation was produced with vecuronium 0.08 mg kg⁻¹. Anaesthesia was maintained with propofol 10 mg kg⁻¹ h⁻¹ or methohexisone 7 mg kg⁻¹ h⁻¹ infused continuously from a syringe pump. Vecuronium was given in intermittent bolus doses of 1 mg when necessary as indicated by monitoring of neuromuscular transmission. After induction of anaesthesia, the cricothyroid membrane was punctured, 2% lignocaine 2 ml was injected and a 14-gauge, 10-cm long catheter (Seldicath, Plastimed France) was inserted into the trachea. Depending on the individual length of the trachea, the tip of the injector was located usually 3 or 4 cm above the carina; this was confirmed by the surgeon during endoscopy in the first patients. HFJV was started immediately. Driving pressure 3.8 bar, F1O₂ = 1 and I:E ratio 0.54 were kept constant. The internal volume of the connecting tube was 6 ml. The patient's
trachea was not intubated and airway patency was maintained by lifting the jaw.

Measurements were performed before endoscopy and recorded on an ES1000 Gould chart recorder. To assess changes in pulmonary volumes, two 50-cm mercury strain gauges (LG 500 Sega Electronique, France) were strapped to the patient at nipple and umbilical levels to measure rib cage and abdominal displacements. Frequency response of the strain gauges was flat up to 15 Hz and linearity was satisfactory for displacements smaller than 4 cm (± 1 %).

The classical method of strain gauge calibration using a syringe could not be used [14], and we therefore used a method derived from that described by Rouby and colleagues [9]. Lungs were insufflated five or six times through the injector for 175 ms at 4-s intervals, with the upper airway occluded firmly by neck flexion and manual closure of mouth and nose. Under these conditions there was no entrained volume and the displacements of the thorax and abdomen were caused only by the injected volume. Simultaneous recording of thoracic and abdominal displacements gave the coefficient of calibration of the two strain gauges by averaging five steps. The presence of leaks was detected easily by absence of a plateau after each step, suggesting incomplete upper airway occlusion. At the end of the study, when the patient was awakened, the injector was connected to a dry spirometer and the injector outflow was measured for 1 min at a frequency of 2 Hz and I:E ratio 0.54 (insufflation time 175 ms). The minute volume was corrected to BTPS conditions and divided by the frequency to obtain the injected volume during the patient calibration procedure. For all patients, the injected volume was found to vary from 105 ml to 122 ml (mean 114 (sd 4) ml).

After the calibration procedure, the study began. Throughout the study, the patients' jaw was lifted forward manually. The ventilatory frequency (1, 2, 3, 4 or 5 Hz) was changed in a random sequence. For each frequency, the thoracic and abdominal pulmonary volumes greater than FRC (dFRC) and end-expiratory pressures were measured after ventilation of the lungs for 30 s.

All data are expressed as mean (sd) and were compared using analysis of variance followed by a modified t test and linear regression analysis. \( P < 0.05 \) was considered significant.

RESULTS

An example of simultaneous recording of EEP and \( Pt \) is shown in figure 2. In the lung model, injector diameter, volume of the connecting tube
and frequency affected the difference between EEP and $P_t$ (table I). This difference increased when the injector diameter decreased or when the apparatus deadspace increased. Using a 2-mm i.d. injector, this difference was less than 1 cm H$_2$O, irrespective of frequency or apparatus deadspace. With a 1.7-cm i.d. injector, the difference was greater than 1 cm H$_2$O at frequencies greater than 3 Hz with the large connecting tube. The difference became appreciable with the 1.3-mm i.d. injector, reaching 5 cm H$_2$O at 5 Hz with the large connecting tube. The addition of an upstream resistance did not change the gradient between EEP and $P_t$ (table II).

The dFRC inferred from abdominal displacements correlated well with dFRC inferred from thoracic displacements for all ventilatory frequencies (table III). Correlation coefficients were greater than 0.90 and the slopes were close to the line of identity. Values of dFRC obtained by either thoracic or abdominal gauges were similar. For all patients, EEP correlated well with dFRC measured by either thoracic or abdominal strain gauges (for example $r = 0.90$ and $r = 0.89$, respectively, at 2 Hz) (fig. 3). EEP increased when ventilatory frequency increased while dFRC remained stable. The increase in EEP with frequency was constant in each patient and did not exceed 3 cm H$_2$O from 1 to 5 Hz (mean 1.7 (SD 1.1) cm H$_2$O). The individual variations in EEP varied from 0 to 9.4 cm H$_2$O. Nevertheless, a correlation between dFRC and EEP was present for each frequency and all the correlation coefficients exceeded 0.88 (table III).

The effect of an upper airway obstruction is

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**TABLE I. Differences between EEP and $P_t$ (cm H$_2$O) with two sizes of connecting tube, three injector diameters and five ventilatory frequencies, in the lung model**

<table>
<thead>
<tr>
<th>Connecting tube</th>
<th>Injector diameter (mm)</th>
<th>Ventilatory frequency (Hz)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large (6 ml)</td>
<td>2</td>
<td>-0.02</td>
<td>0.05</td>
<td>0.3</td>
<td>0.3</td>
<td>0.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.7</td>
<td>0</td>
<td>0.3</td>
<td>0.7</td>
<td>1.1</td>
<td>1.8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.3</td>
<td>0.05</td>
<td>1.2</td>
<td>2.3</td>
<td>3.7</td>
<td>5.3</td>
<td></td>
</tr>
<tr>
<td>Small (1.8 ml)</td>
<td>2</td>
<td>-0.1</td>
<td>-0.05</td>
<td>-0.1</td>
<td>0.1</td>
<td>0.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.7</td>
<td>-0.1</td>
<td>0.2</td>
<td>0.4</td>
<td>0.5</td>
<td>0.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.3</td>
<td>0</td>
<td>0.4</td>
<td>0.8</td>
<td>1.1</td>
<td>1.6</td>
<td></td>
</tr>
</tbody>
</table>

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**TABLE II. Differences between EEP and $P_t$ (cm H$_2$O) after addition of an upstream expiratory resistance simulating an upper airway obstruction in the lung model. These values are similar to those reported in table I**

<table>
<thead>
<tr>
<th>Connecting tube</th>
<th>Injector diameter (mm)</th>
<th>Ventilatory frequency (Hz)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large (6 ml)</td>
<td>2</td>
<td>0.1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>1.7</td>
<td>0.1</td>
<td>0.2</td>
<td>0.5</td>
<td>0.9</td>
<td>1.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.3</td>
<td>0.05</td>
<td>1.0</td>
<td>2.0</td>
<td>3.2</td>
<td>4.8</td>
<td></td>
</tr>
<tr>
<td>Small (1.8 ml)</td>
<td>2</td>
<td>-0.2</td>
<td>-0.2</td>
<td>-0.3</td>
<td>-0.4</td>
<td>-0.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.7</td>
<td>0</td>
<td>0.2</td>
<td>0.2</td>
<td>0.3</td>
<td>0.6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.3</td>
<td>0.05</td>
<td>0.8</td>
<td>1.1</td>
<td>1.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table III. Values of dFRC and EEP (mean (SD)) in the 64 patients measured at five ventilatory frequencies using the thoracic (RC) and the abdominal (AB) strain gauges. The correlation coefficient r of the regression lines between dFRC AB and dFRC RC and between dFRC RC and EEP are presented.

<table>
<thead>
<tr>
<th>Ventilatory frequency (Hz)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>dFRC RC (ml)</td>
<td>162 (180)</td>
<td>191 (173)</td>
<td>190 (157)</td>
<td>182 (147)</td>
<td>161 (139)</td>
</tr>
<tr>
<td>dFRC AB (ml)</td>
<td>154 (168)</td>
<td>171 (169)</td>
<td>172 (163)</td>
<td>161 (151)</td>
<td>145 (131)</td>
</tr>
<tr>
<td>EEP (cm H(_2)O)</td>
<td>0.9 (1.7)</td>
<td>1.5 (2.0)</td>
<td>1.9 (2.1)</td>
<td>2.1 (2.1)</td>
<td>2.6 (2.0)</td>
</tr>
<tr>
<td>r dFRC AB vs dFRC RC</td>
<td>0.92</td>
<td>0.95</td>
<td>0.94</td>
<td>0.92</td>
<td>0.90</td>
</tr>
<tr>
<td>r EEP vs dFRC RC</td>
<td>0.94</td>
<td>0.90</td>
<td>0.92</td>
<td>0.88</td>
<td>0.90</td>
</tr>
</tbody>
</table>

shown in figure 4. When obstruction was complete, expiration was not possible. The ventilator insufflated the lungs and both EEP and dFRC increased. When EEP reached the low mandatory pressure, the ventilator could not perform the next insufflation. In case of partial upper airway obstruction, the next insufflation was performed when Pt was less than the mandatory pressure. In both situations, EEP could not exceed the preset low mandatory pressure.

**DISCUSSION**

Measurement of EEP via the injection catheter is a reflection of Pt if the gas compressed in the ventilator deadspace escapes completely at the end of the decompression time; for a given decompression time this depends on injector diameter and ventilator deadspace volume. This volume is influenced mainly by the length and internal diameter of the connecting tube. The influence of injector diameter on EEP–Pt difference may be explained by the fact that part of the gas compressed in the ventilator escapes through the injector. The larger the diameter, the larger is the amount of gas which escapes through the injector. In our configuration, a decompression time of 70 ms, a ventilator deadspace of 6 ml and a 14-gauge injection catheter were adequate to allow satisfactory measurement of EEP up to 5 Hz. Using the same ventilator in similar patients, satisfactory gas exchange has been obtained with a 2-mm injector, ventilatory frequency 2 Hz and I:E 0.54 ratio [15]. Therefore, the safety margin of EEP measurement was good for injector diameters larger than or equal to 1.7 mm. Obviously, the decompression time must be shorter than the expiratory time and this limit gives the maximal frequency available for a given I:E ratio. As expected, the addition of an upstream resistance did not affect the relationship between Pt and EEP.

During positive pressure ventilation in anaesthetized patients, measurement of lung volumes by external devices depends on Konno and Mead's principle [16]. In such a system, the displacements of each compartment are proportional and the volume inferred from each displacement must be equivalent. This was confirmed in our study, as measurements of dFRC by the two strain gauges were similar.

The use of HFJV has been reported to increase...
FIG. 4. Simultaneous recording of expiratory airway pressure measured through the injector and thoracic displacement. Low mandatory pressure is represented as a dashed line. When the airway obstruction was complete, end-expiratory pressure increased up to the value of low mandatory pressure. Then, the next insufflation was not performed until the airway pressure was less than the mandatory pressure.

pulmonary volume to greater than the apnoeic FRC [9]. This “PEEP-effect” is primarily a result of intrapulmonary gas trapping. External spirometry has been suggested as a method of monitoring overdistension during HFJV [10]. This procedure requires individual calibration to obtain quantitative measurements, but the calibration procedure is time consuming and requires a computer to calculate changes in dFRC in millilitres. In view of the excellent correlation which we found between dFRC and EEP, it appears that continuous measurement of end-expiratory pressure may be easier and provides a good indication of overdistension at frequencies up to 5 Hz. Similar results have been reported previously in an animal study [17] and in isolated lung [18]. Measurement of EEP did not require individual calibration and the ability to make this measurement through the injector was particularly useful in the absence of a tracheal tube.

The frequency dependence of EEP has been described previously [18]. In our study, it may be related to the increase in gradient between EEP and end-expiratory alveolar pressure. At any frequency, the relationship between EEP and dFRC was present for all patients and the value of EEP may indicate the degree of pulmonary distension. In the presence of bronchial obstruction, there may be a large difference between Pt and alveolar pressure; in this situation, EEP would not be an accurate guide to pulmonary volume [19]. Further studies are needed in the use of this monitoring in patients with chronic obstructive pulmonary disease.

Control of jet ventilation by the adjustment of EEP may be appropriate in situations which predispose to barotrauma. The system operates during each cycle and allows a quick response to an increase in EEP. In the presence of upper airway obstruction, the risk of increased airway pressure is limited because of decreased ventilatory frequency and minute ventilation (as shown in figure 4), but hypoventilation may develop.

We conclude that, provided certain technical precautions are taken, measurement of EEP through the injection catheter may provide a satisfactory guide to end-expiratory pulmonary volume during transtracheal HFJV. Consequently, continuous monitoring of EEP may detect pulmonary overdistension during laryngoscopy. In the presence of increased airway pressure, the automatic control of ventilatory frequency and minute ventilation reduces the risk of barotrauma, although at the expense of potential hypoventilation.

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AIRWAY PRESSURE DURING HFJV

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