Proximal and Tracheal Airway Pressures During Different Modes of Mechanical Ventilation: An Animal Model Study

G. Zobel, MD,1 D. Dacar, MD,2 and S. Rödl, MD1

Summary. Objective: To determine the differences between the mean proximal and tracheal airway pressures during 3 different modes of mechanical ventilation (MV) in an animal model of acute cardiac failure (CF) and respiratory failure (RF). Design: Prospective, randomized, crossover design. Setting: University research laboratory. Subjects: Twelve young pigs weighing 10–16 kg. Interventions: The experimental protocol consisted of 3 stable 30 min conditions: when ventricular and pulmonary function was normal (control), after the induction of acute cardiac failure by an β-blocking agent and after respiratory failure induced by repeated lung lavages. Modes of MV included controlled mechanical ventilation (CMV), high-frequency oscillation (HFO), and high-frequency jet ventilation (HFJV). Measurements and results: The tracheal mean airway pressure (Paw) was measured at the distal port of the Hi-lo jet tube using an air-filled pressure transducer. The mean transpulmonary Paw increased significantly from 0.41 ± 0.14 kPa during the control period to 1.15 ± 0.17 kPa (P < 0.0001) during the RF period. In all study periods both the proximal and tracheal Paw were lower during HFJV. There was no difference between the proximal and tracheal Paw during CMV and HFJV throughout the protocol. In the cardiac and respiratory failure periods the proximal Paw (CF, 1.45 ± 0.08 kPa; RF, 3.13 ± 0.27 kPa) was significantly higher than the tracheal Paw (CF, 1.04 ± 0.09 kPa, P < 0.01; RF, 2.18 ± 0.3 kPa, P < 0.01) with HFO. When ventilated by HFO, the mean external oscillatory amplitude was 4.33 ± 0.14 kPa and the intratracheal oscillatory amplitude was only 0.49 ± 0.06 kPa (P < 0.0001). Conclusion: HFJV provides adequate respiratory support at lower Paw than CMV and HFO. Proximal Paw closely reflects tracheal Paw during CMV and HFJV. However, with HFO great pressure differences between the proximal and tracheal airways are evident. Therefore, additional intratracheal airway pressure monitoring seems to be very useful for optimizing ventilator settings during HFO. Pediatr Pulmonol. 1994;18:239–243. © 1994 Wiley-Liss, Inc.

Key words: High frequency jet ventilation, high frequency oscillation, conventional mechanical ventilation, mean airway pressure, cardiac failure, respiratory failure.

INTRODUCTION

Mean airway pressure (Paw) is the major determinant of arterial oxygenation during both conventional mechanical ventilation (CMV) and high frequency jet ventilation (HFJV).1–2 In clinical practice Paw is usually measured proximally within the ventilator circuit. Recently Marini et al. in a review article concluded that Paw closely reflects mean alveolar pressure during CMV, except when flow-resistive pressure losses differ greatly between the inspiratory and expiratory phases of the ventilatory cycle. In addition, he stated that under conditions of passive inflation, Paw correlates with alveolar ventilation, arterial oxygenation, hemodynamic function, and barotrauma.3–4 In recent years HFO and HFJV have been introduced as alternative forms of respiratory support for infants and children with severe acute respiratory failure with or without pulmonary barotrauma.5–8 During HFJV adequate oxygenation and ventilation can be achieved at lower airway pressures, reducing the risk of pulmonary barotrauma.9 HFO has been introduced in two different forms. The low volume/pressure strategy used in neonates did not show any advantage over CMV.11 On the other hand, HFO has been utilized in a high volume/pressure mode to obtain alveolar recruitment and optimal lung volume. This approach might have beneficial effects on gas exchange and lung recovery.12 However, during this high volume/pressure strategy a much greater proximal Paw is required than during CMV.8 There are published data on the relationship between proximal, tra-

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cheal, and alveolar pressures during different modes of mechanical ventilation. However, to date no information on proximal and tracheal airway pressures during a high volume/pressure HFO strategy have been made available.

The aim of this experimental study was to show the relationship of proximal and tracheal airway pressures during different modes of MV, including CMV, HFO, and HFJV, when cardiopulmonary function was normal, during acute cardiac failure (CF) and during respiratory failure (RF).

MATERIALS AND METHODS

The protocol was approved by the Institutional Animal Research Committee and the care of the animals was in accordance with guidelines for ethical animal research.

In 12 pigs of either sex weighing 10–16 kg, premedicated with azaperone (8 mg/kg) and atropine (0.02 mg/kg), anesthesia was induced with ketamine (10 mg/kg) and thereafter maintained by a continuous infusion of fentanyl (0.15 μg/kg/min), pentobarbital sodium (4 mg/kg hr), and pancuronium (0.3 mg/kg/hr). After tracheostomy and intubation with a 5.5 mm-i.d. Hi-Lo cuffed tube containing a jet ventilation injection port and a port for measuring airway pressure at the distal end 1 cm above the distal tip of the endotracheal tube (Hi-Lo jet tube®, National Catheter Corp., Malineckrodt, Glen Falls, NY). Initially a venous line was inserted into the right subclavian vein for nutrition and anesthesia. A 7.5 Fr thermistor catheter (Edwards Swan-Ganz oximeter TD catheter, Edwards Critical Care Division, Irvine, CA) was placed into the pulmonary artery by peripheral cutdown of the external jugular vein. A 4 Fr O2 saturation catheter (Edsab® double lumen O2 Sat II catheter, Edwards Critical Care Division) was inserted via the right common carotid artery and placed into the thoracic aorta for continuous arterial oximetry. A short 16-gauge catheter (Abbo cath®, Abbott Ireland LTD, Sligo, Republic of Ireland) was placed into the femoral artery for continuous pressure recording. Cardiac output was measured every hour using the thermodilution technique and the mean of three thermodilution runs. A saline-filled 7 Fr feeding tube with side-holes (B. Braun Melsungen AG, Melsungen, Germany) was placed at the level of the distal third of the esophagus for esophageal pressure monitoring.

The tracheal airway pressure (Paw) was measured at the distal port of the Hi-Lo jet tube using an air filled pressure transducer (Monitoring Kit NM-081-D®, Peter von Berg Medizintechnik, Kirchseeon, Germany). The proximal (ventilator measured) airway pressure during CMV and HFO was measured within the ventilator circuit close to the endotracheal tube. During HFJV the ventilator airway pressure line was connected with a T-piece connected with the Hi-Lo jet tube®. Two oxygen saturation monitors (Baxter Sat2 Oximeter®) were used for continuous arterial and mixed venous oximetry. Arterial and mixed venous blood samples were taken for measuring PaO2, Pco2, and pH using an automatic blood gas system (AVL 99s®, AVL Corp., Graz, Austria) every half an hour.

Mechanical Ventilation

CMV was applied in the volume controlled mode with a tidal volume of 12 mL/kg, a respiratory rate of 20/min, I/E ratio of 1:2, PEEP of 2–4 cm H2O, and an FiO2 of 0.4 (Veolar®, Hamilton Comp., Rhaizins, Switzerland).

For HFJV an Acutronic Jet respirator (Acutronic AMS-1000®, Acutronic Medical Systems AG, Switzerland) was used. The jet pulse was applied to the jet port of the Hi-Lo jet tube. Driving pressure was set at 3–4 lb/in², respiratory rate at 150/min, and I/E ratio at 1:2. The proximal end of the endotracheal tube was connected to a T-attachment through which 21% oxygen was delivered at a constant flow of 5 L/min with a threshold resistor in the expiratory limb.

For HFO the Sensor Medics 3100A respirator® (Sensor Medics Corporation, CA, USA) was used. Respiratory rate was set at 10Hz, I/E ratio at 1:2, bias flow 15–20 L/min, and mean airway pressure at 2 cm H2O above the mean airway pressure applied during CMV. Theretore the mean airway pressure was adjusted according to arterial and mixed venous oxygen saturation values. Oscillatory pressure was set at 35 cm H2O and adjusted according to the Paco2 levels.

Protocol

The experimental protocol consisted of 3 consecutive stable periods: when ventricular and pulmonary function was normal (control), and after the induction of CF and RF. The control period began 30 min after completing the surgical procedure. We induced CF by a 10 min intravenous infusion of metoprolol (2 mg/kg) followed by a continuous infusion of 2 mg/kg/hr; RF was induced by repeated lung lavages using 0.9% saline (10 mL/kg/lavage). During this period FiO2 was increased to 1.0. Within each period the 3 different modes of MV were randomly and sequentially applied to each animal with assessment of respiratory function at the end of each mode of ventilatory support.

All animals tolerated the experimental protocol. After the end of the trial the animals were sacrificed with an overdose of potassium chloride.

Data Analysis

Values are given as mean ± SEM. Initially, multifactorial analysis of variance (ANOVA) was applied using the Statview 4.0® software for Macintosh. When significant effects were seen, more detailed analyses using the
TABLE 1—Blood Gas Tensions, Oxygen Saturations, Tracheal Airway Pressure, and Cardiac Index During Different Modes of Mechanical Ventilation Under Control and During Cardiac and Respiratory Failure

<table>
<thead>
<tr>
<th></th>
<th>$P_{co2}/F_{co2}$ (kPa)</th>
<th>$P_{co2}$ (kPa)</th>
<th>pH</th>
<th>$HCO_3^-$ (mmol/L)</th>
<th>$S_{O2}$ (%)</th>
<th>$S_{O2}$ (%)</th>
<th>$P_{aw}$ (kPa)</th>
<th>CI (ml/kg/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>70.4 ± 7.3</td>
<td>5.1 ± 0.3</td>
<td>7.43 ± 0.03</td>
<td>25.4 ± 0.8</td>
<td>96.2 ± 0.9</td>
<td>76.1 ± 2.1</td>
<td>0.58 ± 0.1</td>
<td>151.5 ± 18.1</td>
</tr>
<tr>
<td>CMV</td>
<td>56.1 ± 4.6</td>
<td>5.1 ± 0.3</td>
<td>7.40 ± 0.04</td>
<td>24.3 ± 0.8</td>
<td>96.7 ± 0.4</td>
<td>78.9 ± 3.1</td>
<td>0.43 ± 0.1</td>
<td>151.3 ± 12.1</td>
</tr>
<tr>
<td>HFJ</td>
<td>62.1 ± 9.9</td>
<td>3.8 ± 0.2**</td>
<td>7.54 ± 0.04*</td>
<td>23.8 ± 0.9</td>
<td>97.1 ± 0.3</td>
<td>82.7 ± 2.3</td>
<td>0.22 ± 0.1*</td>
<td>193.3 ± 19.3*</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>CMV</td>
<td>48.1 ± 5.2</td>
<td>4.7 ± 0.3</td>
<td>7.41 ± 0.04</td>
<td>24.1 ± 1.8</td>
<td>95.5 ± 1.5</td>
<td>71.1 ± 3.1</td>
<td>0.57 ± 0.06</td>
</tr>
<tr>
<td>HFO</td>
<td>37.2 ± 4.6</td>
<td>4.9 ± 0.4</td>
<td>7.43 ± 0.04</td>
<td>22.1 ± 1.1</td>
<td>94.7 ± 1.4</td>
<td>71.4 ± 2.8</td>
<td>0.49 ± 0.08</td>
<td>106.7 ± 9.3</td>
</tr>
<tr>
<td>HFJ</td>
<td>47.2 ± 6.2</td>
<td>3.8 ± 0.2*</td>
<td>7.42 ± 0.01</td>
<td>21.1 ± 1.0</td>
<td>95.2 ± 0.9</td>
<td>70.1 ± 3.8</td>
<td>0.57 ± 0.12</td>
<td>103.7 ± 10.7</td>
</tr>
<tr>
<td>Respiratory failure</td>
<td>CMV</td>
<td>25.1 ± 5.2</td>
<td>5.8 ± 0.3</td>
<td>7.36 ± 0.02</td>
<td>23.3 ± 0.7</td>
<td>95.6 ± 2.6</td>
<td>59.1 ± 8.5*</td>
<td>1.18 ± 0.08</td>
</tr>
<tr>
<td>HFO</td>
<td>20.4 ± 6.1</td>
<td>5.3 ± 0.6</td>
<td>7.38 ± 0.05</td>
<td>24.1 ± 1.7</td>
<td>91.6 ± 4.1</td>
<td>65.0 ± 6.6</td>
<td>1.38 ± 0.24</td>
<td>125.1 ± 15.9</td>
</tr>
<tr>
<td>HFJ</td>
<td>27.7 ± 9.1</td>
<td>4.7 ± 0.3</td>
<td>7.42 ± 0.04</td>
<td>23.2 ± 0.7</td>
<td>87.6 ± 8.9</td>
<td>69.6 ± 6.2</td>
<td>0.93 ± 0.08*</td>
<td>129.8 ± 12.6</td>
</tr>
</tbody>
</table>

*P < 0.05; **P < 0.01.

CMV, conventional mechanical ventilation; HFO, high-frequency oscillation; HFJ, high-frequency jet ventilation; $S_{CO2}$, arterial oxygen saturation; $S_{O2}$, mixed venous oxygen saturation; $P_{aw}$, mean tracheal airway pressure; CI, cardiac index.

Fig. 1. Mean proximal and tracheal airway pressures during different modes of mechanical ventilation in the control period. CMV, conventional mechanical ventilation; HFO, high-frequency oscillation; HFJ, high-frequency jet ventilation; $P_{aw}$, mean tracheal airway pressure; $P_{aw}$, mean proximal pressure.

Fig. 2. Mean proximal and tracheal airway pressures during different modes of mechanical ventilation in the cardiac failure period. CMV, conventional mechanical ventilation; HFO, high-frequency oscillation; HFJ, high-frequency jet ventilation; $P_{aw}$, mean tracheal airway pressure; $P_{aw}$, mean proximal pressure.

Fisher PLSD test were performed. A $P$-value < 0.05 was considered statistically significant.

RESULTS

Mean values for the measured blood gas tensions, arterial and mixed venous oxygen saturations, mean tracheal $P_{aw}$, and cardiac index during the different modes of MV in each of the 3 experimental conditions are summarized in Table 1. The mean $P_{aw}$ increased significantly from 0.41 ± 0.14 kPa during the control period to 1.15 ± 0.17 kPa ($P < 0.0001$) during the RF period. The proximal and tracheal $P_{aw}$ during the different modes of MV in the control, CF and RF conditions are depicted in Figures 1 to 3.

Control Period

The proximal $P_{aw}$ was significantly lower during HFJ (0.63 ± 0.06 kPa) than during either CMV (0.94 ± 0.05 kPa) or HFO (1.23 ± 0.05 kPa) ($P < 0.01$) (Fig. 1). On the other hand, there was no significant difference between the proximal and tracheal mean $P_{aw}$ during the three modes of MV. During HFJ $P_{CO2}$ was significantly lower and cardiac output significantly higher than during CMV and HFO.

Cardiac Failure Period

In comparison to the control period, both the proximal and tracheal mean $P_{aw}$ was slightly higher during all modes of MV. There was no difference between the proximal and tracheal mean $P_{aw}$ during CMV and HFJ.
In recent years HFO and HFJV have been introduced as an alternative form of respiratory support for infants and children with severe acute respiratory failure whether or not they had suffered pulmonary barotrauma.6,7,8,10,11 By these techniques the patients can be ventilated at lower airway pressures than with conventional mechanical ventilation. In 1983 Frantz and co-workers showed that in infants with severe RDS, tracheal pressure was lower than proximal airway pressure during HFOV although these pressures were equal during conventional mechanical ventilation.18 In 1984 Boynton et al. confirmed that proximal $P_{aw}$ was higher than tracheal $P_{aw}$, using a special pressure-measuring system and a Hi-Lo jet tracheal tube.19 In addition, they found that the phasic pressure swings at the tracheal site were less than those at the airway opening. These findings varied from patient to patient and changes in individual patients were observed during the course of their illness. Therefore, these authors concluded that pressure measurements at the airway opening do not predict pressures in the trachea and consequently both pressures should be measured. Gerstman et al. found that airway pressures varied considerably with change in inspiratory time fraction. However, the changes in tracheal and alveolar $P_{aw}$ were not reflected in proximal pressure measurement. Proximal $P_{aw}$ was higher than tracheal and alveolar $P_{aw}$ when inspiratory time fraction was 0.3. Tracheal $P_{aw}$ exceeded proximal $P_{aw}$ when inspiratory time fraction was increased to 0.5. Alveolar $P_{aw}$ was consistently higher in the middle than in the upper lobe. We observed no significant difference between the proximal and tracheal $P_{aw}$ during CMV and HFJV throughout the study. However, during HFO the proximal to tracheal $P_{aw}$ difference increased significantly when cardiopulmonary function decreased. HFJV provided adequate respiratory support at lower $P_{aw}$ than CMV and HFO. In addition, HFJV produced lower $P_{aco2}$ and higher pH values throughout the study periods. On the other hand, no difference was observed in the oxygenation index between the three modes of MV. The combination of lower transpulmonary airway pressure and lower $P_{aco2}$ values might result in an improved hemodynamic situation. The same result was observed by Boros et al. in an experimental study comparing HFO and HFJV in cats with normal lungs.20

Gerstman et al. found a 97% decrement in oscillatory amplitude between the proximal airway and trachea and an additional decrease between trachea and alveolus.15 In our series we found an 88% decrease in oscillatory amplitude between the proximal and trachea sites. 

In conclusion, HFJV provides adequate respiratory support at lower $P_{aw}$ than CMV and HFO. Proximal $P_{aw}$ closely reflects tracheal $P_{aw}$ during CMV and HFJV, therefore, additional intratracheal $P_{aw}$ monitoring seems to offer no advantage in clinical practice. However, during HFJV great pressure differences between the proximal

However, during HFO the mean proximal $P_{aw}$ (1.45 ± 0.08 kPa) was significantly higher than the mean tracheal $P_{aw}$ (1.04 ± 0.09 kPa, $P < 0.001$). Again, $P_{aco2}$ was significantly lower during HFJV.

**Respiratory Failure**

Four to six lung lavages resulted in a significant decrease in arterial (96.5 ± 0.5 vs 90.2 ± 2.6%) and mixed venous (81.1 ± 1.2 vs 65.7 ± 2.6%) oxygen saturations ($P < 0.01$). Adequate oxygenation was achieved by significantly increasing mean proximal $P_{aw}$. The proximal mean $P_{aw}$ was significantly higher during HFO (3.13 ± 0.27 kPa) than during CMV (1.87 ± 0.09 kPa) and HFJV (1.6 ± 0.14 kPa) ($P < 0.01$). On the other hand, there was no significant difference between the mean tracheal $P_{aw}$ during the 3 modes of MV. In this period venous oxygen saturation ($S_{V02}$) was significantly lower and cardiac output slightly lower during CMV than during HFO and HFJV. During HFO the mean external oscillatory amplitude was 4.3 ± 0.14 kPa and the intratracheal measured oscillatory amplitude was only 0.49 ± 0.06 kPa.

**DISCUSSION**

The use of $P_{aw}$ has been advocated as the major determinant of gas exchange during both conventional mechanical ventilation and high frequency ventilation.1,2,17 This value changes with any change in peak inspiratory pressure, positive end-expiratory pressure, or inspiratory-to-expiratory ratio, and provides a clinically useful composite measure of all pressures transmitted to the airways by the ventilator.
and tracheal airways are evident. Therefore, additional intratracheal airway pressure monitoring should be useful for optimizing ventilator settings.

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