Mean airway pressure and mean alveolar pressure during high-frequency jet ventilation in rabbits

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PÉREZ FONTÁN, J. J., G. P. HELDT, AND G. A. GREGORY. Mean airway pressure and mean alveolar pressure during high-frequency jet ventilation in rabbits. J. Appl. Physiol. 61(2): 456-463, 1986.—Mean airway pressure underestimates mean alveolar pressure during high-frequency oscillatory ventilation. We hypothesized that high inspiratory flows characteristic of high-frequency jet ventilation may generate greater inspiratory than expiratory pressure losses in the airways, thereby causing mean airway pressure to overestimate, rather than underestimate, mean alveolar pressure. To test this hypothesis, we ventilated anesthetized paralyzed rabbits with a jet ventilator at frequencies of 5, 10, and 15 Hz, constant inspiratory-to-expiratory time ratio of 0.5, and mean airway pressures of 5 and 10 cmH₂O. We measured mean total airway pressure in the trachea with a modified Pitot probe, and we estimated mean alveolar pressure as the mean pressure corresponding in the static pressure-volume relationship to the mean volume of the respiratory system measured with a jacket plethysmograph. We found that mean airway pressure was similar to mean alveolar pressure at frequencies of 5 and 10 Hz but overestimated it by 1.1 and 1.4 cmH₂O at mean airway pressures of 5 and 10 cmH₂O, respectively, when frequency was increased to 15 Hz. We attribute this finding primarily to the combined effect of nonlinear pressure frictional losses in the airways and higher inspiratory than expiratory flows. Despite the nonlinearity of the pressure-flow relationship, inspiratory and expiratory net pressure losses decreased with respect to mean inspiratory and expiratory flows at the higher rates, suggesting rate dependence of flow distribution. Redistribution of tidal volume to a shunt airway compliance is thought to occur at high frequencies. If redistribution is present, our method may have overestimated mean alveolar pressure, and the difference between mean airway and alveolar pressure may have actually been greater.

Airway impedance; lung volume; mean airway pressure; mechanical ventilation

MEAN AIRWAY PRESSURE is a predictor of arterial oxygenation during both conventional (2, 5) and high-frequency mechanical ventilation (9, 11, 15). The correlation between mean airway pressure and oxygenation is often interpreted as meaning that the mean pressure in the proximal airway is identical to the mean alveolar pressure and that it therefore bears a direct link to the mean volume of the lungs. The validity of this interpretation has been challenged during high-frequency ventilation. Kolton et al. (9) have shown that the mean volume of the lungs of rabbits with injured lungs is higher during high-frequency oscillatory ventilation than during conventional mechanical ventilation at matched mean airway pressures. More recently, using the occlusion technique (6) to estimate mean alveolar pressure, Simon et al. (16) have demonstrated that mean airway pressure underestimates mean alveolar pressure during high-frequency oscillation in healthy dogs. They attributed their findings to greater airway impedance during expiration than during inspiration and speculated that similar results would be expected during high-frequency jet ventilation.

In their analysis, Simon et al. (16) assumed a linear pressure-flow relationship of the airways. During high-frequency oscillatory ventilation, a nonlinear pressure-flow relationship of the airways may not affect the mean pressure transmitted to the alveoli because inspiratory and expiratory flows are similar. During high-frequency jet ventilation, however, inspiration is kept shorter than expiration to avoid gas trapping within the lungs. Inspiratory flows are thus higher than expiratory flows. If the pressure-flow relationship of the airways is nonlinear, then resistive pressure losses during inspiration will be greater than during expiration, and mean airway pressure will overestimate mean alveolar pressure. We tested this hypothesis by examining the relationship between mean airway pressure and mean alveolar pressure in anesthetized paralyzed rabbits ventilated with a jet ventilator at three frequencies and at two levels of mean airway pressure. High-frequency jet ventilators deliver their stroke volume directly into the airway as a pulse of compressed gas. Therefore airway pressures must be measured distally in the airway (usually in the trachea), where the characteristics of gas flow may induce errors in pressure measurement (4, 14, 16). For the same reason, mean alveolar pressure cannot be measured practically with the occlusion technique during high-frequency jet ventilation. To overcome these difficulties, we devised a method based on the Pitot probe to measure tracheal pressure and a plethysmographic method to estimate mean alveolar pressure.

METHODS

Six New Zealand White rabbits weighing 3.00-3.95 kg (3.39 ± 0.33, mean ± SD) were anesthetized with halothane and placed supine. After cannulating the marginal vein of the ear for fluid and drug administration, we exposed the extrathoracic trachea through a midline
incision. We resected a 3- to 4-cm tracheal segment starting 1 cm below the larynx and introduced a cuffed endotracheal tube into the distal tracheal stump. While the rabbit breathed through this tube, a jet-ventilation endotracheal tube (0.3 cm ID, Mallinckrodt, Glens Falls, NY) was inserted into the trachea over a flexible introducer that was passed from the trachea through the mouth. The jet-ventilation endotracheal tube was secured by suturing it to the laryngeal cartilage. A tie was placed around the trachea and tube to avoid gas leaks.

We then resected the resected segment of trachea with an insert consisting of a piece of laminated silicone tubing 2.5 cm long attached to two brass rings 0.5 cm long (Fig. 1). The compliance of the silicone tubing was similar to that of the rabbit's trachea. The brass rings connected the silicone tubing to the tracheal stumps. The internal diameter of the rings and silicone tubing was 0.58 cm. Since the external diameter of the rings was slightly larger than the internal diameter of the rabbit's trachea (0.62 vs. 0.59 ± 0.02 cm), we had to gently dilate the trachea to accommodate the rings. This method of attachment permitted smooth alignment of the internal surfaces of the insert and trachea, thereby preventing sudden changes in the internal diameter of the airway. Once continuity of the airway had been restored with the insert, we switched the halothane to the jet-ventilation tube. A tie secured the trachea to the brass rings and prevented gas leaks. We tested for leaks by pouring warm saline into the tracheostomy wound and searching for gas bubbles while applying a continuous pressure of 30 cmH₂O at the endotracheal tube connector.

To measure changes in the volume of the respiratory system, we wrapped a jacket plethysmograph around the chest and abdomen of the rabbit (3). This jacket is made of nonstretchable canvas and is closed in front by a mercury-in-rubber strain gauge. We calibrated the jacket at the beginning and end of the experiment by injecting 10, 20, and 30 ml of air into the trachea with a calibrated glass syringe while recording the output of the mercury-in-rubber strain gauge. The appropriate length of the strain gauge was chosen for each rabbit so that the strain gauge produced a linear output to tidal volumes greater than those used during the experiment. We placed a catheter in the urinary bladder to prevent bladder distension from causing changes in the base line of the jacket.

Following these preparations, halothane was discontinued and 20 mg/kg of pentobarbital sodium and 0.1 mg/kg of pancuronium bromide were injected intravenously as needed for sedation and paralysis. Mechanical ventilation with a time-cycled, pressure-limited infant ventilator (Bear Cub BP 2001, Bear Medical Systems, Riverside, CA) was then started via the main lumen of the jet endotracheal tube. The rabbit received a continuous infusion of 6 ml·kg⁻¹·h⁻¹ of normal saline. Body temperature was maintained at 38.5 ± 1°C with a servo-controlled radiant warmer.

We used a high-frequency jet ventilator (Bunnell, Salt Lake City, UT) based on a solenoid-activated pinch valve that periodically interrupts the gas flow generated by a high pressure source (40-50 psi). A timing mechanism regulates the duration of the jet pulse (inspiratory time). A small-bore cannula built into the wall of the endotracheal tube delivers the pulse into the trachea through an opening located ~0.5 cm from the tip of the endotracheal tube. A second cannula, also built into the wall of the tube, transmits the tracheal pressure to an electronically controlled feedback system, which regulates the peak pressure in the trachea by modifying the supply pressure in the ventilator. Positive end-expiratory pressure (PEEP) in the trachea is determined by the resistance of endotracheal tube and ventilatory circuit and by the expiratory flow. To regulate PEEP, we left the endotracheal tube connected to the exhalation circuit of the Bear Cub infant ventilator and adjusted the internal flow rate and resistance of this ventilator.

**Mean airway pressure.** During high-frequency jet ventilation, pulses of gas are delivered via a jet cannula placed into the airway. In most clinically used jet ventilators, the cannula is inserted into the endotracheal tube or directly into the trachea. The small diameter of the trachea and the high instantaneous flow rates generated by the jet make accurate measurement of airway pressure difficult, especially in small animals or human infants. For instance, flow separation in the vicinity of the jet and the resultant alteration in the velocity profile of the flow downstream from the jet may unpredictably affect the measurement of tracheal pressure, particularly if a lateral port is used (4, 14). When comparing tracheal and alveolar pressures at high flow rates, it is also necessary to account for the convective pressure losses caused by

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1. Schematic representation of tracheal insert used to measure inspiratory and expiratory airway pressures (Ptot₁ and Ptot₂) and lateral pressure (P₂₄₃) and to perform airway occlusions.
the increase of the effective diameter of the airways from the trachea to the alveoli (16). Convective pressure losses are often predicted with the Bernoulli equation. This equation assumes, however, that flow is steady and that pressure is homogeneous within the flow stream, two conditions that may not be fulfilled during high-frequency jet ventilation.

We therefore chose to measure the total or stagnation pressure (the sum of lateral pressure and convective pressure losses) directly in the trachea with a straight-bore cylindrical Pitot probe. Cylindrical Pitot probes are less sensitive to misalignment than probes having conical or ogival sections (1). Our probe was made of two 23-gauge needles with their ends occluded. A beveled orifice of ~0.08 cm was made on the side of each needle within 0.1 cm of its end. The needles were glued back-to-back and their surfaces were polished to smoothness. The orifices of the needles faced opposite directions: one needle measured inspiratory total pressure, the other measured expiratory total pressure. The total pressures measured by the needles were symmetrical to flows >1 L/s. The needles were mounted inside a 0.23-cm OD brass tube (Fig. 1), which fit snugly into a perpendicular sidearm of the distal brass ring of the tracheal insert. The mounting tube and needles easily slid in and out of the sidearm so that total pressures could be measured at different points along the diameter of the airway. The sampling orifices were maintained in the centerline of the airway during the experiment. This was facilitated by an external indicator, which was also used to align the orifices with the direction of flow. The connection between the mounting tube and the sidearm on the brass ring was made leak-proof with silicone grease.

We connected each needle to a pressure transducer (model LCVR, linear within ±50 cmH₂O, Celeco, Canoga Park, CA) with noncompliant plastic tubing (0.12 cm ID). The transducers were mounted so that the length of this tubing did not exceed 5 cm to maximize the frequency response of the pressure measuring system. Both pressure measuring systems were matched by placing them inside a sealed box connected to the jet ventilator: identical pressure readings and no phase differences were found in the range of frequencies studied in the experiment.

We computed mean airway pressure (Paw) as

\[ \text{Paw} = \left( \int_{0}^{Tl} \text{Ptot}_i \, dt + \int_{Tl}^{Tc} \text{Ptot}_e \, dt \right) / (Tl + Te) \]  

(1)

where Ptot_i and Ptot_e represent the total pressures measured by the needles facing inspiratory and expiratory flow, and Tl and Te the inspiratory (positive flow) and expiratory (negative flow) time, respectively.

Airway pressures reported during mechanical ventilation are usually lateral pressures. For comparison with the total pressure measured by the Pitot probe, we measured lateral pressure (Plat) using an additional sidearm on the distal brass ring of the insert, opposite to the Pitot probe. This sidearm was connected to a pressure transducer similar to those used to measure total pressures. Mean lateral pressure (Plat) was computed as

\[ \text{Plat} = \int_{0}^{Tc} \text{Ptot} \, dt / (Tl + Te) \]  

(2)

Mean alveolar pressure. We calculated mean alveolar pressure from the mean volume of the respiratory system. We first established a correlation between alveolar pressure and respiratory system volume by occluding the airway and measuring the equilibration pressure in the trachea and the jacket volume at different points within tidal volume range. The mean alveolar pressure was calculated as the alveolar pressure corresponding to the mean respiratory system volume in the pressure-volume correlation. Mean volume (V) was defined as

\[ \text{V} = \int_{0}^{Tc} \text{V} \, dt / (Tl + Te) \]  

(3)

where V is the volume measured by the jacket. Airway occlusions were performed with a solenoid-operated pinch valve applied on the silicone tubing of the tracheal insert (Fig. 1). The valve was activated by the discharge of a large capacitor and closed completely in <0.004 s.

We estimated that the isolated amplitude-frequency response of the jacket was flat to a frequency of 100 Hz from its transient response to the explosion of a rubber balloon. Unlike the balloon, however, the rabbit's respiratory system has two degrees of freedom (10). Volume shifts between the rib cage and abdomen produced by inertia of the abdominal contents may cause a phase lag between alveolar volume and jacket volume if either the rib cage or abdomen are overrepresented in the jacket output. As a preliminary check for this error, we tested the symmetry of the jacket output by ventilating the rabbit's lungs at a frequency of 2 Hz, with a tidal volume of 10 ml/kg and a Tl of 0.2 s. During this test, gas flow was measured with a Fleisch no. 00 pneumotachograph inserted between the Bear Cub infant ventilator and the endotracheal tube and attached to a differential pressure transducer (Celeco model LCVR, ±2 cmH₂O, amplitude-frequency response of the system was flat to 25 Hz). The position of the jacket was adjusted so that no phase lag existed between the volume measured by the jacket and the volume obtained by digital integration of the flow. As an additional check, we occluded the airway at various points throughout the ventilatory cycle while recording pressure, jacket volume, and gas flow (Fig. 2).

The oscillations generated volume accelerations that exceeded those seen during high-frequency jet ventilation. The oscillations produced by the occlusions on jacket volume were, however, <5% of the tidal volume; their frequency was 9–11 Hz, which is similar to the predicted natural frequency of the rabbit respiratory system (17). We were confident, therefore, that the jacket behaved symmetrically, and that its amplitude-frequency response was adequate to measure tidal volumes. The airway occlusions also provided an estimate of the in situ amplitude-frequency response of the pressure measuring systems. This response was flat to frequencies >80 Hz in all rabbits.

The pressure, volume, and (when pertinent) flow sig-
During high-frequency jet ventilation.

The lungs were then ventilated with the jet ventilator at 5, 10, and 15 Hz in a random order. $T1/TE$ and the fractional concentration of $O_2$ in the inspired gas were kept constant at 0.5 and 1.0, respectively. Tidal volume and FRC (as shown by the jacket) were also maintained constant at all ventilatory frequencies by adjusting the peak and end-expiratory pressures in the trachea. At each ventilatory frequency, the lungs were inflated three times with a pressure of 25 cm H$_2$O to provide a consistent volume history. After obtaining a stable jacket volume tracing for 5 min, we recorded P_{tot1} and P_{tot2} and jacket volume using a digitization rate of 500 Hz. We then performed 15–25 airway occlusions to establish a correlation between jacket volume and alveolar pressure within the range of tidal volume at each ventilatory frequency. After the last frequency was tested, the rabbit’s lungs were ventilated again with the Bear Cub infant ventilator, and the FRC was determined by helium equilibration. The high-frequency measurements were repeated if the FRC measured by helium equilibration and that estimated from the base line of the jacket tracing differed by >1.5 ml/kg.

We expected that the velocity profile of the flow would be blunt in the trachea during high-frequency jet ventilation and that the total pressure measured in the center line would therefore be representative of the energy content of the flow in the corresponding cross section of the airway. To validate this expectation, we mapped the velocity profile of the flow at each ventilatory frequency by measuring the P_{tot1} and P_{tot2} at ~0.1-cm increments across the diameter of the tracheal implant. These measurements were performed first without changing the radial angle of the Pitot probe and then rotating the ring to sample different sectors of the airway section.

To compare mean total and lateral pressures, at the conclusion of the experiment, we recorded total pressures (inspiratory and expiratory) while the lungs were ventilated at frequencies of 5 and 15 Hz, with a mean airway pressure of 5 cm H$_2$O. At each frequency, we also recorded lateral pressure by switching one of the pressure transducers to the sidearm of the distal ring of the insert.

**Data analysis.** The temporal relationships between airway pressure and flow found in this study were very complex. This results in part from the nonsinusoidal waveform of the pressure created by the jet. We found it convenient to characterize the pressure-flow relationship by an average impedance, which is different from the impedance defined in linear oscillatory theory. We defined average inspiratory ($ZI$) and expiratory ($ZE$) impedances of the respiratory system as

$$ZI = \int_0^{T1} P_{tot1} \cdot dt/VT$$  \hspace{1cm} (4)

$$ZE = \int_{T1}^{TE} P_{tot2} \cdot dt/VT$$  \hspace{1cm} (5)

where $VT$ represents the tidal volume.

Statistical differences caused by ventilatory frequency or mean airway pressure were tested by two-factor anal-
RESULTS

Mean airway pressure was similar to mean alveolar pressure at ventilatory frequencies of 5 and 10 Hz, but overestimated mean alveolar pressure at 15 Hz (Fig. 3). The magnitude of this overestimation was independent of mean airway pressure.

The static pressure-volume relationships of the respiratory system (occlusions) were linear, even with high lung volumes at a mean airway pressure of 10 cmH₂O (Fig. 4). The correlation coefficient of the linear regression characterizing this relationship exceeded 0.975 in all cases. The dynamic pressure-volume relationships, however, became progressively nonlinear at the higher ventilatory frequencies (Fig. 5). The major component of this nonlinearity was in phase with gas flow.

Ptot and Ptotg were uniform in the section of the airway sampled by the Pitot probe at all ventilatory frequencies (Fig. 6), indicating a blunt velocity profile of the flow at the point of pressure measurement.

Z₁ and Zₑ decreased with ventilatory frequency (Table 1). Their ratio remained relatively constant, however, and slightly >2 in most cases. The higher FRC at a mean airway pressure of 10 cmH₂O (Table 2) had no measurable effect on average impedance.

Pplat measured at the sidearm of the tracheal insert was up to 0.4 cmH₂O lower than the mean total pressure at a ventilatory frequency of 5 Hz, and up to 2 cmH₂O lower at a frequency of 15 Hz. The difference between mean total and lateral pressures exceeded the values estimated with the Bernoulli equation (16) by 13–68% at a ventilatory frequency of 15 Hz.

DISCUSSION

Our results show that mean tracheal pressure adequately estimates mean alveolar pressure at ventilatory frequencies <10 Hz during high-frequency jet ventilation in rabbits. At higher ventilatory frequencies, mean airway pressure overestimates mean alveolar pressure by a magnitude that is independent of mean airway pressure. These results differ from those obtained during high-frequency oscillatory ventilation, in which mean airway pressure underestimates mean alveolar pressure in a magnitude that depends not only on frequency but also on mean airway pressure and body position (16). We attribute our findings to the combined effect of the nonlinear pressure-flow behavior of the airways and the higher inspiratory than expiratory flows characteristic of high-frequency jet ventilation.

Several mechanisms may explain the nonlinear pressure-flow behavior of the airways observed at high frequencies. These include nonlinearity of frictional pressure losses in the airways, inertial effects, volume dependence of airway resistance, and gas compression. We did not assess the pressure-flow relationship of the airways directly. We found, however, that the phase lag between pressure and respiratory system volume was greatest at points of maximal inspiratory flow (Fig. 5). This finding suggests that nonlinear frictional losses were dominant in the overall pressure-flow behavior of the airways at high frequencies, and that they were primarily responsible for mean alveolar pressure being lower than mean airway pressure. The remaining mechanisms may have a variable contribution. For instance, inertial pressure losses should decrease the mean pressure transmitted to the alveoli in a frequency-dependent manner because the volume acceleration generated by the jet is higher than that generated by the recoil forces of the respiratory system. The magnitude of this effect is probably small, nonetheless, because of the small inertia of the gas contained in the airways (17). Volume dependence of airway resistance should also decrease net pressure transmission to the alveoli at high frequencies because higher inspiratory flows are forced into the airways at lower lung volumes, when airway resistance is not only greater, but also more sensitive to flow. The constancy of the difference between mean airway and alveolar pressures and the similar average impedances measured at different lung volumes suggest that this mechanism did not contribute to our results. It may be more important, however, when lung volumes are smaller and the airways are on a steeper portion of their pressure-volume curve. Expiratory flow limitation may play a major role in the higher mean alveolar pressures observed during high-frequency oscillation, particularly at low mean airway pressures (16). The low expiratory flows and positive expiratory airway pressures, however, make

![Figure 3](image-url)  
**FIG. 3.** Difference between mean alveolar (Pål) and mean airway pressure (Paw) in 6 rabbits studied (mean ± SD). Pål - Paw was significantly smaller (*P < 0.05*) than 0 at 15 Hz, and independent of Paw at all frequencies.
flow limitation unlikely during high-frequency jet ventilation.

Nonlinear frictional pressure losses were predictable in our study conditions, since the average Reynolds number of the tracheal flow was ~3,000 at a frequency of 5 Hz and exceeded 7,500 at 15 Hz. Also included in these pressure losses is a portion of the local losses caused by energy dissipation downstream from the expansion of the jet flow. If, for simplicity, we ignore nonfrictional pressure losses and represent the linear and nonlinear components of airway resistance with the two Rohrer's constants, $K_1$ and $K_2$, we can express the difference between airway and alveolar pressures as

$$P_{alv} - P_{aw} = K_1 \cdot \frac{dV}{dt} + K_2 \cdot (\frac{dV}{dt})^2 \quad (6)$$

Integrating both terms of this equation between 0 and $T_f$ and dividing them by ($T_i + T_e$), we obtain

$$P_{alv} - P_{aw} = K_2 \cdot f \left[ \int_0^{T_i} (\frac{dV}{dt})^2 \cdot dt + \int_{T_i}^{T_e} (\frac{dV}{dt})^2 \cdot dt \right] \quad (7)$$

where $f$ represents ventilatory frequency in Hz. According to this expression, the magnitude by which mean airway pressure overestimates mean alveolar pressure depends not only on the nonlinearity of airway resistance but also on the difference between the absolute values of inspiratory and expiratory flows. This differ-
TABLE 1. Average impedances of respiratory system of rabbits during high-frequency jet ventilation

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Paw = 5</th>
<th>Paw = 10</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Z_i</td>
<td>Z_e</td>
</tr>
<tr>
<td>6 Hz</td>
<td>0.043±0.015*</td>
<td>0.039±0.008*</td>
</tr>
<tr>
<td>10 Hz</td>
<td>0.030±0.021</td>
<td>0.011±0.003</td>
</tr>
<tr>
<td>15 Hz</td>
<td>0.024±0.015</td>
<td>0.012±0.003</td>
</tr>
</tbody>
</table>

Values are means ± SD. Paw, mean airway pressure (cmH_2O); Z_i and Z_e, inspiratory and expiratory average impedances (cmH_2O ml⁻¹ s⁻¹). *P < 0.001 for differences between 5 and 10 and 5 and 15 Hz.

TABLE 2. Functional residual capacity and tidal volume during high-frequency jet ventilation

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Paw = 5</th>
<th>Paw = 10</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FRC</td>
<td>VT</td>
</tr>
<tr>
<td>5 Hz</td>
<td>13.8±1.3</td>
<td>3.7±0.4</td>
</tr>
<tr>
<td>10 Hz</td>
<td>13.2±1.5</td>
<td>3.8±0.8</td>
</tr>
<tr>
<td>15 Hz</td>
<td>14.2±2.0</td>
<td>3.3±0.7</td>
</tr>
</tbody>
</table>

Values are means ± SD. Paw, mean airway pressure (in cmH_2O); FRC, functional residual capacity; VT, tidal volume (both in ml/kg).

cence is a direct function of VT and an inverse function of the T1/Te. Therefore overestimation of mean alveolar pressure by mean airway pressure would have been greater if we had used a lower T1/Te (or simply a shorter inspiratory time), as it is common in clinical practice.

If the airways behave as a pure nonlinear resistor, as implicit in Eqs. 6 and 7, we would expect that the average impedance of the respiratory system increased with f (VT being constant). We found, however, that both Z_i and Z_e decreased (although proportionally) at the higher frequencies. This finding introduces rate dependence of flow distribution as a new element in the analysis of pressure transmission to the alveoli.

At the ventilatory frequencies used in our experiment, airway resistance is the primary component of respiratory system impedance. Rate-dependent decreases in airway resistance are usually explained either by parallel heterogeneities within the lung parenchyma (13) or by the airways acting as a parallel shunt compliance (12). According to the first explanation, as f increases, flow is redistributed to areas of the lung with the shortest time constant; according to the second explanation, flow is redistributed to the airways. Recent experimental evidence indicates that a significant portion of VT is redistributed to the conducting airways of rabbits during high-frequency oscillatory ventilation at frequencies similar to those of our study (18). If, as this evidence and our measurements suggest, a portion of the tidal volume was redistributed to the airways, then mean alveolar pressure would have been further underestimated by the mean airway pressure measured proximally to those airways. This is so because our method included airway volume changes as part of the volume used to estimate mean alveolar pressure under dynamic conditions.

Our results have important clinical implications. They indicate that the efficacy of high-frequency oscillatory and jet ventilation cannot be compared on the basis of mean airway pressure. In addition, our results demonstrate that mean airway pressure accurately estimates mean alveolar pressure during high-frequency jet ventilation at frequencies commonly used in clinical practice (<10 Hz). At those frequencies, mean lateral pressure measured in the trachea also reasonably estimates mean alveolar pressure because convective and nonlinear frictional pressure losses between the trachea and the alveoli are relatively small and negate one another. At higher ventilatory frequencies (or with very low T1/Te), however, mean airway pressure tends to underestimate mean alveolar pressure substantially, and convective pressure losses become greater and more difficult to predict. Plethysmographic devices such as the jacket used in this study may then be of greater help than mean airway pressure in monitoring patients ventilated at high frequencies.

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