RELATIONSHIP BETWEEN RESONANCE AND GAS EXCHANGE DURING HIGH FREQUENCY JET VENTILATION

E. S. LIN, M. J. JONES, S. D. MOTTRAM, B. E. SMITH AND G. SMITH

SUMMARY
We have studied the relationship between gas exchange and mechanical frequency response during high frequency jet ventilation (HFJV) at 0.5–5.0 Hz in anaesthetized pigs. The mechanical gain curve showed a minimal “anti-resonant” response at 0.8 Hz (f1) and a maximal “resonant” response at 5.0 Hz (f2). This finding may be explained by modelling the thorax and abdomen as a system of coupled masses and compliances which undergo two different modes of forced oscillation in the frequency range studied. Gas exchange was optimal in the frequency range between the minimal and maximal responses. The tidal volumes produced were greater than anatomical deadspace, suggesting that gas transport was mainly convective in this range.

KEY WORDS
Ventilation: high frequency, jet. Gas exchange.

Although high frequency jet ventilation (HFJV) has been used for more than a decade, widespread application has been inhibited by a lack of understanding of the mechanisms involved in gas exchange [1]. Consequently, ventilator settings such as frequency, driving pressure and duty cycle (inspiratory : expiratory time, I : E ratio) have been chosen empirically.

Ventilation frequency can influence gas exchange by affecting tidal volume, gas transport mechanisms and gas mixing within the lungs. Gas exchange is dependent also on the mechanical properties of the respiratory system and its frequency response. At resonance, the mechanical response is maximal for a given energy input and might enhance gas exchange by facilitating bulk gas mixing in the lungs or by increasing tidal volume [2]. The existence of resonance has been demonstrated [3] within the respiratory system during HFJV. Knowledge of the mechanical frequency response of the respiratory system during HFJV may be useful, therefore, in determining which ventilation frequency is associated with optimal gas exchange.

We have investigated the relationship between gas exchange and mechanical frequency response of the respiratory system at ventilation frequencies between 0.5 and 5.0 Hz in anaesthetized pigs. Tidal volumes have been measured in order to study the mechanisms of gas transport.

MATERIALS AND METHODS
We studied 10 healthy Large White pigs of body weight 19–34 kg. Anaesthesia was induced with etorphine (Immobilon) 25 μg kg⁻¹, and an alphaxalone/alphadolone mixture (Saffan) identical to Althesin 0.05 ml kg⁻¹. After intubation of the trachea with a Mallinkrodt Hi-Lo jet tube, HFJV was applied to the lungs using a prototype Penlon Bromsgrove jet ventilator and an entrainment system (5 litre min⁻¹), both supplying an air in oxygen mixture (Bird Microblender) with an FiO₂ of 0.4 (fig. 1). The jet drive pressure
was maintained constant at 20 lbf in⁻² (approx, 138 kPa) with a duty cycle of 50% (I:E = 1:1).

Anaesthesia was maintained using an infusion of Saffan 0.5 ml kg⁻¹ h⁻¹. Body temperature was kept constant by covering the animal in warm gamgee and using a heated mattress. ECG, intra-arterial pressure and rectal temperature were monitored continuously.

**Determination of mechanical frequency response**

The mechanical frequency response of the respiratory system was studied by measuring chest and abdominal wall displacement and relating these to the amplitude of the airway pressure signal. Airway pressure was measured using a piezoresistive transducer (Honeywell 162PC01D) calibrated against a water manometer. Displacements of the chest wall at the level of the xiphisternum and abdominal wall at the umbilicus were measured using strain gauges (Lectromed type 4320). Transducer signals were recorded on a multi-channel digital storage oscilloscope (Gould 1604).

Mechanical gain was calculated using the peak-to-trough amplitudes of the strain gauge and airway pressure transducer signals, averaged over eight consecutive breaths, as follows:

\[ |G| = \frac{|S|_{	ext{CGG}} + |S|_{	ext{ASG}}}{|S|_{	ext{AWP}}} \]

where |G| is the modulus of the gain (i.e. does not contain phase information), |S|_{	ext{CGG}} and |S|_{	ext{ASG}} are the chest and abdominal strain gauge signal amplitudes, respectively, and |S|_{	ext{AWP}} is the airway pressure transducer signal amplitude. All amplitudes are expressed in volts. |G| is dimensionless because of the use of voltages rather than directly measured units in order to simplify calibration. This approach was used as it was necessary only to detect changes in gain, rather than measure absolute values.

**Measurement of gas flow and tidal volume**

Gas flow was measured using a differential pressure transducer (Honeywell 162PC01D) connected to a pair of rigid catheters. The catheters were inserted in the tracheal tube via a suction swivel adaptor, with their tips separated by a distance of 5 cm proximal to the jet orifice, so that the lumen of the tracheal tube acted as a pneumotachograph. This was validated for gas flow in both directions, using a bench procedure in which pulses from the jet ventilator were passed through the tracheal tube and catheter mount, collected in a Douglas bag over a known time period, and the gas volume measured using a dry gas meter (S.R.I.). Measured volumes were plotted against the integrated pressure values, confirming the linearity of the system within the flow ranges used (fig. 2).

Entrained and tidal gas volumes were derived by partial integration of the flow curves (fig. 3). As the flow measurement was taken proximally to the jet it did not register injected gas flow during inspiration, but only entrained flow. Flow into the
respiratory system was defined as positive. The volume of gas entrained was represented by the area $V_e$ above the zero line, occurring during inspiration. Area $V_{bkb}$ which represented "blow-back" or spill-over volume during inspiration, was calculated as the integrated flow between the negative-sloped crossing of the zero flow line and the end of the jet pulse.

Area $V_T$, the outward flow during expiration corresponding to expired tidal volume, appeared below the zero flow line during the time when the jet was not active.

**Measurement of gas exchange**

Arterial blood samples were taken after each change in ventilator frequency (after allowing a 15-min period for stabilization) for measurement of gas tensions corrected to body temperature (Radiometer, Copenhagen). Total exhaust minute volume (jet plus bias) was measured in a 1-min period using a Wright's respirometer. Total elimination of carbon dioxide was calculated by measuring the average concentration of carbon dioxide in the expired volume as follows: gas was sampled over a 3-min period into a 1-litre gas syringe, and the concentration determined using an infra-red analyser (P. K. Morgan) calibrated using two known gas standards, accurate to 0.01%. Arterial blood samples were obtained midway through the minute volume measurement for determination of $P_{a\text{CO}_2}$, which enabled a value for the impedance to elimination of carbon dioxide to be determined:

$$\text{CO}_2 \text{ impedance (kPa litre}^{-1}\text{min)} = \frac{P_{a\text{CO}_2} \text{(kPa)}}{\text{CO}_2 \text{ elimination (litre min}^{-1})}$$

**Measurement of chest compliance**

Static chest compliance was measured in each animal by inflating the lungs to a given airway pressure and measuring the resulting expired tidal volume using a Wright's respirometer. This was repeated for 15 points over the tidal volume range used experimentally (50–500 ml), the slope of the volume–pressure line obtained by least-squares linear regression being taken as an index of static compliance.
FIG. 4. Mechanical gain–frequency response curve, where gain = (\(S_{\text{Cgo}} + S_{\text{Ago}}\)) / \(S_{\text{AWP}}\). Minimum gain at 0.8 Hz (f1) and maximum at 5.0 Hz (f2).

FIG. 5. Abdominal gain response curve (mean, SEM) as a fraction of the mean over-all frequencies. Non-normalized data.

FIG. 6. Thoracic gain response curve (mean, SEM) as a fraction of the mean over-all frequencies. Non-normalized data.

**TABLE 1. Static respiratory compliance and body weight of the 10 pigs studied**

<table>
<thead>
<tr>
<th>Pig No.</th>
<th>Static respiratory compliance (ml/cm H₂O)</th>
<th>Body weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>28.8</td>
<td>19</td>
</tr>
<tr>
<td>2</td>
<td>27.5</td>
<td>23</td>
</tr>
<tr>
<td>3</td>
<td>46.0</td>
<td>25</td>
</tr>
<tr>
<td>4</td>
<td>34.4</td>
<td>25</td>
</tr>
<tr>
<td>5</td>
<td>37.1</td>
<td>26</td>
</tr>
<tr>
<td>6</td>
<td>26.3</td>
<td>29</td>
</tr>
<tr>
<td>7</td>
<td>33.6</td>
<td>29</td>
</tr>
<tr>
<td>8</td>
<td>35.3</td>
<td>29</td>
</tr>
<tr>
<td>9</td>
<td>42.9</td>
<td>33</td>
</tr>
<tr>
<td>10</td>
<td>38.3</td>
<td>34</td>
</tr>
</tbody>
</table>

**RESULTS**

**Mechanical frequency response**

Using the airway pressure changes and the chest and abdominal wall displacements, a gain–frequency response curve was plotted for each animal studied (fig. 4). As the animals differed in body weight and compliance (table 1), the frequency axis was normalized for these factors. Previous theoretical work [2] has shown that the high gain resonance frequency of the respiratory system may be derived from the formula:

\[ f_0 = \frac{1}{2\pi} \sqrt{\frac{S}{LC\rho}} \]

where \(f_0\) = resonant frequency, \(S\) = effective cross sectional area of the upper airway, \(L\) = effective length of the upper airway, \(C\) = thoracic compliance, \(\rho\) = density of gas.

It has been assumed that \(S\) is relatively independent of body weight, whilst \(L\) is proportional to body weight (\(W\)) and gas density is constant. Thus

\[ f_0 = \alpha \sqrt{\frac{1}{WC}} \]

Normalization of each frequency was therefore performed using the following equation:

\[ f_n = f_x \cdot \sqrt{\frac{\bar{W}C}{WC}} \]
where \( f_n \) is the normalized frequency, \( f_s \) is the measured frequency, \( \bar{W} \) is the mean body weight and \( C \) the mean static compliance of the group. In effect, this equation corrects for the value expected if the animal had average body weight and compliance.

In an adult population it would be expected that \( S \) would be proportional to \( \bar{W}^{2/3} \) and \( L \) proportional to \( \bar{W}^{1/3} \). However as the animals studied were juveniles, possibly with non-linear growth patterns, this assumption cannot be made. The relationship used was found empirically to produce an acceptable reduction in the scatter of resonant frequencies, and so was used to simplify calculation.

There was a gradual increase in total gain (fig. 4) with ventilation frequency at 1–2.5 Hz, as would be anticipated for a simple resistance–compliance model of the respiratory system.

However, two modes of oscillation were identified which cannot be explained by such a model, with a minimum gain at 0.8 Hz (\( f_1 \)) and a maximal gain at 5 Hz (\( f_2 \)).

Abdominal gain (fig. 5) was obtained by plotting the amplitude ratio of abdominal strain gauge and airway pressure transducer signals against frequency. There was a “cut off” at approximately 4 Hz as abdominal wall movement decreased to undetectable amounts at higher frequencies. There was a sharp peak in thoracic gain (fig. 6) at 5 Hz. Figure 4, showing total gain, is not the sum of figures 5 and 6, as the data in figures 5 and 6 have not been normalized for frequency. This normalization was not applied to the separate abdominal and thoracic data because, although it reduced the population scatter of resonant frequencies, the scatter of “cut off” frequencies was increased, hiding this feature.

**Tidal volume**

\( V_T \) was greater than 300 ml at all frequencies less than 3 Hz, except at \( f_1 \), where the tidal volume decreased to less than 200 ml (fig. 7). At frequencies greater than 3 Hz, tidal volumes decreased significantly and no change occurred at the maximum gain frequency, \( f_2 \).

**Gas exchange**

\( P_{a\text{O}_2} \) initially decreased at 0.5–0.8 Hz, but recovered at frequencies greater than 1 Hz and remained relatively constant at frequencies up to 3 Hz (fig. 8). The optimal frequency range for oxygenation appeared to lie between the low and high mechanical gain responses. Similarly, the
changes in $P_{a\text{CO}_2}$ and derived carbon dioxide impedance plotted against frequency (fig. 9) also appear to be associated with the mechanical gain frequency response, with minimal impedance to elimination of carbon dioxide between the two oscillation modes.

**DISCUSSION**

**Gain–frequency response curve**

Thoracic gain (fig. 6) exhibited a maximum (i.e. resonance) at a frequency of 5 Hz ($f_2$), which is within the range predicted if the thorax behaves as a simple mechanical oscillator with lumped elements defined solely by thoracic properties [1]. This is likely to be the case when the thorax becomes mechanically isolated from the abdomen at frequencies greater than the cut off threshold demonstrated in the abdominal gain curve (fig. 5). At lesser frequencies, the abdomen and thorax interact mechanically as a system of coupled damped resonators, in response to the excitation of the jet pulses. At 0.8 Hz ($f_1$) the total mechanical gain reached a minimum, lower than predicted for a simple resistance-compliance model; that is, the system exhibited anti-resonance.

The mechanical response of this so-called “coupled oscillation” model may be understood best by reference to electrical analogues, although strict equivalence between components of the electrical model and the anatomical components of the respiratory system cannot be assumed. In this model, inductance is represented as inductance and compliance as capacitance. At the lower frequency ($f_1$) at which mechanical anti-resonance is occurring, the reactance of the airway gas inductance is negligible, so that the main interaction in the system is between thoracic compliance and the inductance of the abdominal contents. This system behaves as an electrical parallel network and so has a high impedance and low gain as its resonant frequency (gain = 1/impedance within the confines of the definition of gain used in this paper). At higher frequencies, as the reactance of the abdominal inductance increases and the fractional gain of the abdomen is reduced, the interaction is governed almost exclusively by airway inductance and thoracic compliance. These components are analogous to an electrical series circuit with minimal impedance and high mechanical gain at resonant frequency ($f_2$).

At greater frequencies (> 5 Hz) the thorax may behave as if isolated mechanically from the abdomen, as the abdominal response appears to be negligible at these frequencies. However, at low frequencies the thorax and abdomen interact to produce an anti-resonant response.

**Tidal volume**

We found that tidal volume was low where mechanical anti-resonance was occurring ($f_1$), as would be expected with increasing respiratory system impedance at this frequency. After recovering at frequencies just greater than $f_1$, tidal volume decreased as ventilation frequency increased, and no significant change was noted at the resonant frequency $f_2$. This may indicate that, at greater frequencies, increased chest and abdominal wall movements resulted in enhanced intrapulmonary gas movement, analogous to the phenomenon of “pendelluft” at lesser frequencies, rather than increasing gas flows in the trachea.

**Gas exchange and mechanical response**

The frequencies of jet ventilation selected for this study ranged from the upper end of conventional IPPV (0.5 Hz) to 5 Hz; at greater frequencies, gas exchange has been found to deteriorate [4, 5]. Oxygenation was found to deteriorate at $f_1$, as may be anticipated from the reduction in tidal volume that occurred at this frequency. There was a corresponding increase in $P_{a\text{CO}_2}$ and increase in impedance to elimination of carbon dioxide. Between the oscillation modes $f_1$ and $f_2$, arterial oxygenation and carbon dioxide elimination were maintained well, even though tidal volume decreased. Over this range, tidal volumes approached the expected deadspace volume. As ventilation frequency approached $f_2$, tidal volume decreased significantly and both oxygenation and elimination of carbon dioxide deteriorated.

In summary, with frequencies used commonly in clinical practice, and constant jet drive pressure and $I:E$ ratio (1:1), it appeared that gas exchange was maintained within a range of approximately 4 Hz, but a sharp deterioration occurred at the anti-resonant frequency. Over the range of frequencies studied, tidal volumes were generally larger than anatomical deadspace and comparable, at low frequencies, to those used in IPPV. At the high frequency end of the range, tidal volume approached the expected deadspace and gas exchange became less efficient.
We conclude that, during HFJV with these settings and in the given frequency range, gas exchange is dependent mainly on convective transport, rather than the other mechanisms which are involved at greater frequencies and small tidal volumes. At low frequencies, the presence of a mechanical anti-resonance may impair the efficiency of gas exchange during HFJV.

ACKNOWLEDGEMENT

This work was supported by a grant from the Medical Research Council of Great Britain.

REFERENCES