Respiratory Impedance Estimated from Airway and Intrapleural Pressure Curves

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At the cessation point of inspiratory flow on an airway pressure curve of constant-flow CMV (controlled mechanical ventilation), the pressure falls rapidly at first and then gradually tapers down to an endinspiratory plateau. By fitting these two pressure changes to the voltage changes in the respiratory circuit model, the resistance and compliances in the lung periphery and the resistance in the large airway can be estimated. We designed an algorithm estimating the values of circuit elements using a circuit simulator MicroCap III. The validity of this method was examined on the mechanical lung model. The sensitivity came to within ±15%. Further validation was carried out on five Beagles treated with methacholine. The large airway resistance, the small airway resistance and compliance, and alveolar compliance demonstrated a two- to three-fold increase in resistances and a two thirds to one-half decrease in compliances, when compared with the base-line values before administration of methacholine. This method can be applied not only to patients during CMV, but also during surgery.

(Key Words: respiratory impedance, constant flow, end-inspiratory plateau, airway pressure, methacholine)

INTRODUCTION

Many ventilators incorporate end-inspiratory plateau pressure, a mechanism by which airway pressure is kept positive during a period beginning at inspiratory flow shut-off and lasting until opening of the expiratory valve (end-inspiratory pause with no-flow). The airway pressure remains above zero and at a level below the peak pressure (end-inspiratory plateau). The rationale is to help allow that portion of inspired air which has been trapped in the more pliable, faster-distending airspaces to be redistributed to the more rigid, slower-filling areas. Participation of a greater number of gas exchange units, a more even distribution of inspired air and, ultimately, improved oxygenation are expected. The time course of airway pressure immediately after cessation of the inspiratory flow is of particular interest because it reflects the interactions of various factors involved in the filling of airspaces (peripheral, distensible airways, and alveoli), e.g. inspiratory airflow velocity, inspiratory time, flow resistance of the conduits, distensibility of the airspaces themselves, and recoil pressures of the lung and thoracic cage. Conversely, analyses of airway pressure curves allow the estimation of mechanical properties (resistance and compliance) of peripheral small airways and lung tissues.

We present a theoretical (mathematical) analysis of this phenomenon on an equivalent circuit mode, then report the supportive results of experiments on a mechanical lung model. Finally we present a clinical model of the feasibility of our method in an animal study using methacholine-induced bronchoconstriction in Beagles.

THEORY

Figure 1 shows a schematic airway pressure curve (Paw) with end-inspiratory plateau using constant inspiratory flow. The total respiratory resistance (Rrs) and compliance (Crs) can be expressed by the following equations:
Fig. 1 Schematic airway pressure curves during constant-flow CMV with end-inspirator plateau $I_c$ denotes the magnitude of the constant flow and is delivered during a period of $T_I$. The airway pressure drops by $P_1$ after the constant flow stops and remains close to $P_2$ for a predetermined period. When the respiratory system is represented by a circuit model as in the figure, $R_{ss}$ and $C_{rs}$ can be calculated as $R_{ss}=P_1/I_c$ and $C_{rs}=(I_c \times T_I)/P_2$. Also, it can be seen from the figure that $P_1$ is composed of two components; a vertical pressure drop ($E_1$) and an ensuing, more gentle exponential drop ($E_2$). $E_2$ represents the pressure drop due to slow redistribution of the delivered tidal volume in the lung periphery. See text for further details.

$R_{ss}=P_1/I_c$, $C_{rs}=(I_c T_I)/P_2$  \hspace{1cm} (1)

where $I_c$ denotes the magnitude of the constant flow, $T_I$ denotes the inspiration time, $P_1$ denotes the pressure drop after the end of inspiration, and $P_2$ denotes the plateau pressure. In this case, the respiratory system is represented by the circuit model in the figure.

In addition, it should be noted that the pressure drop ($P_1$) consists of two components; a vertical pressure drop ($E_1$) immediately after the end of inspiration and an ensuing gentle exponential decrease ($E_2$). The mechanisms forming these two pressure changes are explained as follows.

The large airway, in which volume change is negligible under normal airway pressure, is considered a rigid conduit and expressed as a resistor ($R_c$), and the constant flow produces a pressure difference across it, expressed by $R_c \times I_c$. At the end of inspiration, the pressure difference becomes zero immediately and, as a result, the airway pressure drops precipitously by $R_c \times I_c$. This pressure drop corresponds to $E_1$. Next, the small airway is distensible and capable of accumulating a small portion of the delivered gas as mentioned above. After cessation of the constant flow, the volume of gas accumulated in the small airway flows down to the alveoli. As a result, the small airway pressure decreases gradually until the pressure between the small airway and the alveoli reaches equilibrium. The pressure change caused by this process corresponds to $E_2$, and its time course depends on the peripheral impedance distribution, i.e., the small airway resistance ($R_p$) and compliance ($C_p$) and the alveolar compliance ($C_{at}$).

Of course, the airway pressure curve is affected by the thorax. When the intrapleural pressure is measured together with the airway pressure, the difference between the two gives the transpulmonary pressure acting on the lung alone.

The present study is based on the above introduction. By fitting the airway pressure curve to a respiratory circuit model, the resistance and compliances in the lung periphery and also the resistance of the large airway may be estimated. Algorithms estimating the values are presented below.

Figure 2 shows a circuit model which divides the respiratory system into a large airway, small airway, alveolus, and thorax. The resistance in the large airway is represented by $R_c$; the resistance and compliance in the small airway by $R_p$ and $C_p$; the alveolar compliance by $C_{at}$; and the thoracic resistance and compliance by $R_w$ and $C_w$. Although the boundary between the large and small airways is not clearly defined, the portion of the bronchiole
from its entrance (the 12th bifurcation) to the alveolar sac (the 23rd bifurcation) is defined as the small airway, and the portion from the tranchea to the 11th bifurcation as the large airway (10, 11).

In Figures 3 (1) and 3 (2), Paw is the airway pressure and Ppl is the intrapleural pressure. The pressure curve obtained by subtracting Ppl from Paw represents the transpulmonary pressure acting on the lung alone, excluding the effect of the thorax, and is represented by PL. Ic is the constant flow, Ia represents the flow into the alveoli through the small airway resistance, and Ir shown by the broken line represents the redistribution flow from the small airway into the alveoli after the constant flow stops. When the inspiration time is represented by T1 and the gradient of PL by Sl, then, \( \frac{Ic \times T1}{(Cp + Ca)} = Sl \times T1 \). Therefore,
\[
S_l = \frac{Ic}{(Cp + Ca)}
\]
(2)
When the vertical pressure drop in PL, immediately after the end of inspiration, is represented by e1,
\[
e_1 = RcIc
\]
(3)

### Fig. 2 Respiratory circuit model
The circuit comprises the large airway, small airway, alveolus, and thorax. The resistance in the large airway is represented by Rc, the resistance and compliance in the small airway by Rp and Cp, the alveolar compliance by Ca, and the thoracic resistance and compliance by Rw and Cw.

### Fig. 3 (1) Relationship of transpulmonary pressure with airway and intrapulmonary pressure
The airway pressure is denoted by Paw, and the intrapulmonary pressure by Ppl. The transpulmonary pressure is obtained by subtracting Pp from Pa and represented as Pt. A vertical drop in Pt (e1) is calculated by subtracting e3 from E1, and a gentle exponential drop (e2) is equal to E2.
When \( I_r \) flows from \( C_p \) to \( C_a \) via \( R_p \), its time constant is equal to the product of \( R_p \) and \( C_p \) and \( C_a \) in series. Therefore,

\[
T = R_p \left( \frac{C_p C_a}{C_p + C_a} \right)
\]

The magnitude of \( e_2 \) is equal to the product of the pressure difference across \( R_p \) and the ratio of \( C_p \) to \( C_a \). Since the pressure difference across \( R_p \) is equal to \( R_p \times I_a \), and the ratio of \( C_p \) to \( C_a \) is expressed as \( \frac{C_a}{C_p + C_a} \), then,

\[
e_2 = R_p I_a \left( \frac{C_p}{C_p + C_a} \right)
\]

As \( I_a \) is the fraction of \( I_c \), and is expressed as \( I_c \times \frac{C_a}{C_p + C_a} \), equation 5 becomes:

\[
e_2 = R_p I_c \left( \frac{C_a}{C_p + C_a} \right)^2
\]

Substituting equation 2 into equation 6 gives:

\[
e_2 = R_p S_l C_a \left( \frac{C_p}{C_p + C_a} \right)
\]

The values on the left side of equations 2 to 7 can be measured from \( P_L \). Therefore, \( R_c \) can be obtained from equation 3, and \( R_p \), \( C_p \), and \( C_a \) can be determined from equations 2, 4, and 7. Next, when \( S_p \) is the gradient of \( P_p \), then, \( \frac{I_c \times T_l}{C_w} = S_p \times T_l \). Therefore,

\[
S_p = I_c / C_w
\]

When the pressure drop of \( P_p \) after the end of inspiration is represented by \( e_3 \), then,

\[
e_3 = R_w I_c
\]

The values on the left side of equations 8 and 9 can be measured from \( P_p \), and so \( C_w \) and \( R_w \) can be determined.

The process to measure \( e_1 \), \( e_2 \), \( e_3 \), \( S_l \), \( S_p \), \( C_w \) and \( T \) from \( P_L \) and \( P_p \) will be described. At 250 milliseconds before the end of inspiration, using a least square method during 200 milliseconds (equivalent to 100 points at a sampling rate of 500 Hz), \( P_L \) and \( P_p \) can be assigned as follows:

\[
\begin{align*}
P_L &= S_L \times t + B_1 \\
P_p &= S_p \times t + B_2
\end{align*}
\]

From equations 10 and 11, \( S_L \) and \( S_p \) are determined. \( B_1 \) and \( B_2 \) are dummy numbers. Next, the mean plateau pressure (\( P_{L_m} \)) and mean intrapleural pressure (\( P_{p_m} \)) are calculated as the average values during 200 milliseconds (100 points) at 400 milliseconds after the end of inspiration. Next, to avoid the oscillation immediately after the stop of constant flow (to be described later),

\[
P_L = P_L \times e^{(-t/T)} + P_{L_m}
\]

is applied to the gentle exponential decrease 5 to 10 milliseconds after the end of inspiration. In actual calculation, the logarithms of both sides of equation 12 are applied to a least square method. That is,

\[
\ln \left( \frac{P_L - P_{L_m}}{P_{L_m}} \right) = \frac{1}{T} \ln \left( P_{L_S} \right)
\]

From equation 13, the pressure \( P_{L_S} \) at \( t=0 \) and \( T \) are obtained. Then, \( e_1 \) is calculated by subtracting \( P_{L_S} \) from the peak airway pressure, and \( e_2 \) is equal to \( P_{L_S} \); \( e_3 \) is calculated by subtracting \( P_{p_m} \) from the peak intrapleural pressure.
METHODS

(1) Evaluation of the theory by using a lung model

Figure 4 shows a mechanical lung model using a test lung (TTL Model 1600; Michigan Instruments, USA) in which two Fleisch type resistor tubes (corresponding to Rc and Rp) were connected to two compliances (Cp and Ca) of the test lung. Although this model does not include a thorax, it enables verification of equations 2 to 7. The values of resistances including connecting tubes, and compliances of the test lung, were calibrated by a Calibration Analyzer (RT-200; Tymeter Instrument, USA) and a super syringe (TJ08C; Minato Medical, Japan). A ventilator (E200; Newport Medical Instruments, USA) capable of facilitating the function for constant-flow CMV with an end-inspiratory plateau was connected to the model. The constant flow was set at 0.5L/sec, the inspiration time at 1 second and the duration of the plateau at approximately 1 second. The pressure curve at the connecting port between Rc and the ventilator was taken as the airway pressure (equivalent to PL) and read into a computer (PC9801; NEC, Japan) through an AD converter (AnalogPro II; Canopus Electronics, Japan). The AD converter had a sampling rate of 500 Herz (Hz) and a resolution of 12 bits. The values of Rc, Rp, Cp and Ca were calculated following the process described in the previous section and evaluated by a comparison with the preset values.

(2) Measurement of respiratory impedance distribution in an anesthetized Beagles given methacholine

This part of the study was approved by the Animal Experimentation Committee of Tokai University school of Medicine. Five non-premedicated Beagles weighing 7.0 to 9.4 kg were anesthetized in the lateral position. After induction of anesthesia by 0.1 mg/kg of intravenous sodium pentobarbital, the dogs were paralyzed with 0.1 mg/kg of intravenous pancuronium bromide, intubated with an 8 mm (internal diameter) endotracheal tube, and ventilated at a frequency of 20 minute−1 and a tidal volume (TV) of 20 ml/kg. An oxygen concentration of 50% was used throughout the experiment. A femoral artery and vein were cannulated for arterial blood pressure monitoring and supplemental anesthetic agent infusion, respectively. A pressure transducer with a cuff at the tip (PT-147; Goodtec, USA) was inserted into the thorax at the fifth intercostal space. The cuff was inflated and the intrapleural pressure (equivalent to Ppl) was monitored. During measurements, the constant flow was 0.2 to 0.35L/sec, the inspiratory time was 750 milliseconds, duration of the plateau was 750 milliseconds, and the respiratory rate was 14 minute−1. The pressure derived from the mouth was taken as the airway pressure (equivalent to Paw). Paw and Ppl were fed into the computer as described above and the values of Rc, Rp, Cp, Ca, Rw, and Cw calculated. Control values were measured after the respiratory and circulatory states reached a steady state. Methacholine bromide, which provokes airway
constriction directly by stimulating cholinergic receptors in airway smooth muscle, was administered intravenously at 100 μg/kg and the impedance values at the highest peak airway pressure point were defined as the values after methacholine administration.

RESULTS

In both experiments, some trials brought about a great oscillation in the airway pressure immediately after the end of constant flow, presenting difficulties in identifying the vertical pressure drop (See Figure 5). To prevent the oscillation, a resistor tube of 5 to 10 cmH2O/L/sec was inserted between the endotracheal tube and the ventilator. Theoretically, the inserted resistor is added to Rc, but does not affect the values of other impedance elements.

(1) Evaluation of the theory

Figure 5 shows an example of waveforms in the experiment using the test lung. The upper trace shows the airway pressure (corresponding to Pt) when Rc and Rp were set at 3.8 and 7.3 cmH2O/L/sec, and Cp and Ca were set at 0.015 and 0.15 L/cmH2O, respectively. All were calibrated values including the connecting tubes. In this figure, because a damper resistor was not used, a great oscillation occurred immediately after the end of inspiration. When the impedance elements were calculated from PL, Rc=4.0 and Rp=7.5 cmH2O/L/sec; and Cp=0.012, and Ca=0.13 L/cm H2O. All elements were estimated to be sensitive to within 15% of the preset values. The lower trace shows the constant-flow waveform. Oscillations did not occur at the end of the flow. The preset and estimated values using various combinations of the elements are summarized in Table 1. The sensitivity of estimation was within 15% in all cases.

(2) Respiratory impedance distribution in Beagles

Figure 6 shows an example of the airway and intrapleural pressure curves before and after methacholine administration in an 8.2 kg Beagle. The control values before methacholine were as follows: Rc=4.5 and Rp=3.7 cmH2O/L/sec; Cp=0.006, Ca=0.014, and Cw=0.08 L/cmH2O; and Rw=0 (undetectable). After methacholine administration, resistances increased approximately three-fold, compliances decreased by two-thirds to one-half, but the thoracic resistance and compliance were not changed. In one of the Beagles, impedance

![Fig. 5 Airway pressure curve in the lung model experiments](image)

The upper trace denoted as airway pressure shows the waveform at the connecting port between Rc and the ventilator, and is equivalent to Pt. The lower trace shows the waveform of constant flow. Oscillation did not occur either at the start or end of the flow.
Table 1 Evaluation of sensitivity

<table>
<thead>
<tr>
<th>No</th>
<th>Rc</th>
<th>Rp</th>
<th>Cp</th>
<th>Ca</th>
<th>Measured Rc</th>
<th>Rp</th>
<th>Cp</th>
<th>Ca</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.0</td>
<td>3.8</td>
<td>0.029</td>
<td>0.10</td>
<td>4.6</td>
<td>3.5</td>
<td>0.024</td>
<td>0.11</td>
</tr>
<tr>
<td>2</td>
<td>4.0</td>
<td>7.3</td>
<td>0.029</td>
<td>0.15</td>
<td>4.5</td>
<td>7.1</td>
<td>0.025</td>
<td>0.15</td>
</tr>
<tr>
<td>3</td>
<td>7.3</td>
<td>7.3</td>
<td>0.029</td>
<td>0.15</td>
<td>7.9</td>
<td>6.4</td>
<td>0.024</td>
<td>0.16</td>
</tr>
<tr>
<td>4</td>
<td>7.3</td>
<td>3.8</td>
<td>0.029</td>
<td>0.075</td>
<td>7.8</td>
<td>3.5</td>
<td>0.025</td>
<td>0.09</td>
</tr>
<tr>
<td>5</td>
<td>3.8</td>
<td>7.3</td>
<td>0.015</td>
<td>0.15</td>
<td>4.0</td>
<td>7.5</td>
<td>0.012</td>
<td>0.13</td>
</tr>
</tbody>
</table>

Table shows the preset and estimated values at various combinations of the elements. The sensitivity was within 15% in all instances.

CONTROL

AIRWAY PRESSURE

<table>
<thead>
<tr>
<th>30</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
</tr>
<tr>
<td>10</td>
</tr>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

AFTER METHACHOLINE

<table>
<thead>
<tr>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
</tr>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

1sec/div

Fig. 6 Airway and intrapleural pressure curves in the animal experiments. After methacholine administration, the peak pressure and plateau pressure remarkably increased, and also the slope of the airway pressure rose in steepness during inspiration. These observations suggest increases in resistance and decreases in compliance in the airway. The intrapleural pressure waveform did not change, indicating that the thoracic resistance and compliance did not change.

DISCUSSION

The gentle exponential pressure drop observed in the airway pressure curve obtained during constant flow CMV with an end-inspiratory plateau has not previously been studied. We concluded that this pressure change was due to the redistribution of gas flow in the lung periphery. This effect was examined using a circuit simulator (MicroCap III; Spectrum Software, USA). In Figure 7, the circuit diagram corresponds to Figure 2. In the circuit, resistors (in meg-ohm) correspond to resistances (in cmH2O/L/sec), capacitors (in micro-farads) to compliances (in L/cmH2O), and the current source of IX (in micro-amperes) to the constant flow (in L/sec), hence node voltages (in Volts) to pressures (in cmH2O). The upper trace is the voltage change at node 1 (equivalent to Pa), and the lower trace shows the current change flowing into Cp. The current changes to the negative direction immediately after the end of constant flow, indicating that the charge accumulated in Cp flows out to Ca through Rp. Even under the premise that a respiratory system can be modeled as shown in Figure 2, the above simulation proves that the decrease in gentle exponential pressure is caused by the accumulated gas flow in the lung periphery. The model in Figure 2 is the same model used by Mead (11), Pimmel et al (12),
Table 2 Estimated values obtained in the animal experiments

<table>
<thead>
<tr>
<th>Dog No</th>
<th>Wt</th>
<th>Rc</th>
<th>Rp</th>
<th>Control Cp (× 10^2)</th>
<th>Ca (× 10^2)</th>
<th>Rw</th>
<th>Cw</th>
<th>After Methacholine Cp (× 10^2)</th>
<th>Ca (× 10^2)</th>
<th>Rw</th>
<th>Cw</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8.2</td>
<td>4.5</td>
<td>3.7</td>
<td>0.6</td>
<td>1.4</td>
<td>0</td>
<td>0.08</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>9.4</td>
<td>1.5</td>
<td>3.1</td>
<td>0.6</td>
<td>1.9</td>
<td>0.6</td>
<td>0.07</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>8.8</td>
<td>3.3</td>
<td>2.9</td>
<td>0.5</td>
<td>1.5</td>
<td>0</td>
<td>0.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>7.0</td>
<td>7.9</td>
<td>18.2</td>
<td>0.5</td>
<td>1.4</td>
<td>1.5</td>
<td>0.05</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>8.8</td>
<td>3.1</td>
<td>3.2</td>
<td>0.6</td>
<td>1.6</td>
<td>0.2</td>
<td>0.08</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>±SD</td>
<td>0.60</td>
<td>1.51</td>
<td>0.42</td>
<td>0.06</td>
<td>0.3</td>
<td>0.3</td>
<td>0.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In the table, the values of Beagle 4 deviated greatly from those of the other dogs and were excluded from the average values.

and Hantos *et al* (4) to demonstrate the frequency dependence of the respiratory system impedance. When various voltage changes at node 1 are read and applied to estimate the impedance elements according to the algorithm, the estimated values agree with the values of the circuit elements, confirming the validity of the algorithm.

The experiments using the mechanical lung model also helped prove the validity of the algorithm. The resistance in the tubing used to connect the resistor to the test lung was very scattered, therefore, the preset values themselves were subjected to errors. The estimated sensitivity may be better than 15%.

In the animal experiments, methacholine administration increased resistances and reduced compliances. Methacholine provoked constriction of the bronchi and bronchioles, which also might have reduced the compliances. Although there are many reports on the effect of methacholine on airway dynamics, few permit direct comparison with our data, due to the technical difficulties in measuring the impedance of the large and small airways separately.
Hirshman et al (5, 6) measured pulmonary resistance and dynamic compliance in anesthetized greyhound dogs challenged with methacholine. They reported a two- to six-fold increase in pulmonary resistance and a 30 to 75% decrease of dynamic pulmonary compliance after methacholine administration. Kaplan et al (7) inserted a fine fiberoptic bronchoscope into a peripheral bronchus in mongrel dogs and measured the pressure and flow at the tip of the bronchoscope in order to calculate peripheral resistance. They reported up to a ten-fold increase in resistance following methacholine administration. Lutchen et al (9) also reported similar changes in the peripheral resistance and compliance after methacholine administration by using a forced random noise oscillation technique. The resistance and compliance values of these studies were very scattered due to variations in the functional residual capacity (FRC) and/or experimental conditions, but it is obvious that peripheral or total respiratory resistance increases remarkably and compliance decreases after methacholine administration. Our method also detected these changes.

Early detection of pathologic changes in the peripheral part of the airway is very difficult, and so this part is called a silent zone (15). To aid in early detection, a few tests are being employed, such as measurements of frequency dependence of dynamic compliance, closing volume, or flow volume curve. These tests require great operational skills, specially designed instruments, and the active cooperation of the individuals being examined. Obviously, intra-operative or continuous measurement is impossible. A test using a forced oscillation technique also has been reported (3, 8, 12, 14). This technique is based on a measurement of the frequency characteristics of the respiratory system and is the only method capable of estimating impedance distribution during surgery. However, this technique also requires specially designed instruments, such as an oscillatory air-flow source generator, and complicated mathematical processes to estimate impedance distribution from the measured frequency characteristics (1). The method we propose makes use, so to speak, of a by-product of the monitoring of airway pressure curves during CMV, and it does not require any supplemental instruments. It is applicable not only to patients during surgery, but also to patients during CMV. Another advantage is that measurements can be made breath by breath, theoretically.

However, there are several problems in the method described in this study. The first is the oscillation which occurs after the end of inspiration, presenting difficulties in determining $e_1$ and/or $e_2$. The constant flow has a clear rectangular waveform free from oscillation, as shown in Figure 5; therefore, the oscillation might be caused by resonance produced by the inertia and elasticity of the gas when the constant flow stops. In this study, the oscillation could be dampened by inserting a resistor into the respiratory circuit. As a result, the sensitivity might be considerably reduced. Another problem arises along with the oscillation. Mead (11) reported $C_p=0.01$ L/cm H$_2$O, $R_p=0.5$ cm H$_2$O/L/sec, and $C_a=0.15$ L/cm H$_2$O in an adult, with the time constant of gas redistribution then becoming 50 milliseconds or less. Other reports in the literature (2, 13) exhibited data similar to that of Mead. The period of 50 milliseconds is not longer than the duration of the oscillation. If the time constant is shortened to the same order as the duration of the oscillation, estimation then becomes more difficult. This will be the subject of a future study.

REFERENCES

156-H. FUKUYAMA et al.


