Variations in airways impedance during respiratory cycle derived from combined measurements of input and transfer impedances

W. Tomalak*, R. Peslin**, C. Duvivier**


ABSTRACT: Simultaneous measurement of input (Ze) and transfer impedances (Ztr) allows separation of airway and tissue properties at a single frequency, without making assumptions concerning the structure of the two compartments. This approach offers the possibility of studying the variation in airway impedance (Ze) during the respiratory cycle.

Ze and Ztr were measured at frequencies of 10, 20, 30 and 40 Hz in eight healthy subjects to study the variations in Ze according to a modification of the Rohrer’s equation: X = K1 + K2(V’ao), where V is volume and V’ao the flow at the airway opening.

The results showed that Ze could be modelled as a simple resistance-inertance pathway. Variations in airway resistance (Rze) with flow were greater during expiration than during inspiration with K1 values varying from 0.76–0.90 hPa s2 L–2 during inspiration and 0.84–1.47 hPa s2 L–2 during expiration, independently of frequency. Rze was negative volume dependent; it decreased more with increasing volume during inspiration than during expiration. Airways inertance calculated from the imaginary part of Ze also underwent systematic variations during the respiratory cycle, but, in contrast to Rze, flow dependence was negative during both phases.

In conclusion, the approach used in this study allows flow and volume dependencies of airways mechanical properties to be studied and can also provide indices of airway patency independently of flow, which is of great potential interest for studying variations in airway resistance during bronchomotor tests.

where $j$ is the unit imaginary number, $P_{H2O}$ the barometric pressure, $P_{H2O}$ is a water vapour pressure and $\omega = 2\pi f$ with $f$ the oscillation frequency; equations 1 and 2 provide a convenient means of obtaining $Z_t$ and $Z_{aw}$. This approach offers two specific advantages: 1) the separation is achieved without making assumptions about the nature of the mechanical properties of the airways and of the tissues, i.e. without modelling $Z_{aw}$ and $Z_t$; and 2) the separation may be done from the data obtained at a single frequency, which provides the best possible time resolution, and, should the system be nonlinear, avoids the interference between frequencies which may happen with signals containing several components. Equation 2 was used recently to measure $Z_t$ in normal subjects and it was shown that tissue elastance exhibits large variations during the respiratory cycle [8]. The aim of the present investigation was to assess the potential of that approach to study airway properties and their variations during quiet breathing.

**Material and methods**

The study was performed on a group of eight healthy adults (five males and three females) recruited from the laboratory staff. They were all trained in respiratory manoeuvres. Their biometric characteristics and lung volumes are shown in table 1.

**Equipment**

The experimental set-up has been described in detail previously [8]. In brief, the subject was seated in a 350-L flow-type body plethysmograph (Emerson, Cambridge, MA, USA) and breathed from the outside through a Fleisch 2 pneumotachograph (METABO, Switzerland). The flow element of the box was made of two layers of metal screen (area 144 cm$^2$, resistance 0.083 hPa·s·L$^{-1}$) and its time constant (screen resistance times gas compressibility) was about 18 ms. $V_{bs}$ was derived from box pressure measured with a Validyne MP45 ±2 cmH$_2$O differential transducer (Validyne, Northridge, CA, USA). The pressure drop across the pneumotachograph, which provided $V_{ao}$, as well as $P_{ao}$, were measured with similar transducers. Us-ing appropriate connecting tubes, all three transducers were matched within 2° of phase and 1% of amplitude up to 40 Hz. The flow channels were calibrated before each series of measurements by the integral method using a 1-L syringe and $P_{ao}$ was calibrated using a slanted fluid manometer.

Pressure oscillations with a peak-to-peak amplitude of about 2 hPa were applied at the airway opening by a 100 W loudspeaker connected to the distal end of the pneumotachograph; the loudspeaker was supplied with computer-generated sinusoidal signals through a power amplifier. The subjects breathed through a low resistance–high-inertance side-tube, branched in parallel with the loudspeaker and connected to a small reservoir where the inspired gas was conditioned to body temperature and ambient pressure, and saturated with water vapour (BTPS) to eliminate any difference between $Z_{in}$ and $Z_{tr}$ related to differences in temperature and $P_{H2O}$ of the gas.

During the measurements $V_{ao}$, $V_{bs}$ and $P_{ao}$ were digitized at 360 Hz by a 12-bit analogue/digital conversion board (Digimetriq-PCLab, Perpignan, France) and stored on the disk of a 486-type computer for later analysis.

**Protocol**

The measurements were made in triplicate at oscillation frequencies of 10, 20, 30 and 40 Hz in random order. The subjects supported their cheeks firmly with their palms. $V_{ao}$ and lung volume ($V$ obtained on-line by digital integration of $V_{ao}$) were displayed on the computer screen to help the subject perform the manoeuvres. The measurements consisted of recording 4–8 tidal breaths followed by a slow vital capacity (VC) manoeuvre serving to provide a volume reference. At the beginning and at the end of each measurement, zero flow offsets were recorded to correct the flow signal for any drift. The flows were also measured while the subject was off the mouthpiece for several seconds; the corresponding relationship between $V_{ao}$ and $V_{bs}$ ($V_{ao}/V_{bs}$) represented the equipment flow transfer function (FTFeq) and was used to correct the data for the relative frequency responses of the pneumotachograph and of the plethysmograph.

Since the vibrations of upper airway walls may be responsible for errors in both $Z_{in}$ and $Z_{tr}$, upper airway shunt impedance ($Z_{aw}$) was also measured in all subjects during Valsalva manoeuvres [9] at the same four frequencies; this was performed using the same equipment as above to record $P_{ao}$ and $V_{ao}$. Total lung capacity was also measured in each subject using a constant-volume body plethysmograph which, in conjunction with the above-mentioned VC manoeuvres, permitted the calculation of TGV and $Z_{g}$ (equation 3) at any time during breathing.

**Data analysis**

To correct for any departure from BTPS conditions of inspired air or for any small difference in gain between the flow channels, the slope of the relationship between $V_{ao}$ and $V_{bs}$ during the slow VC manoeuvres was computed by linear regression and the $V_{ao}$ data were divided by this value. The correction factor ranged 0.996–1.040. After elimination of their low-frequency component [10], the Fourier coefficients of the signals at the frequency of interest were computed on a cycle-by-cycle basis and combined to obtain $Z_{in}$ and $Z_{tr}$. $Z_{in}$ was corrected for the 2.1 ms time constant of the pneumotachograph [10] and

### Table 1

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Sex</th>
<th>Age yrs</th>
<th>Height cm</th>
<th>Weight kg</th>
<th>FRC L</th>
<th>VC L</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>50</td>
<td>168</td>
<td>92</td>
<td>1.96</td>
<td>5.13</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>34</td>
<td>171</td>
<td>70</td>
<td>3.28</td>
<td>4.27</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>58</td>
<td>168</td>
<td>66</td>
<td>2.82</td>
<td>4.33</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>65</td>
<td>168</td>
<td>71</td>
<td>2.69</td>
<td>4.84</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>40</td>
<td>155</td>
<td>48</td>
<td>3.12</td>
<td>3.54</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>40</td>
<td>160</td>
<td>60</td>
<td>2.60</td>
<td>3.80</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>58</td>
<td>178</td>
<td>55</td>
<td>5.39</td>
<td>5.09</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>34</td>
<td>187</td>
<td>84</td>
<td>3.45</td>
<td>6.10</td>
</tr>
</tbody>
</table>

M: male; F: female; FRC: functional residual capacity; VC: vital capacity.
Zaw/Za for FTFeq. Both were also corrected for the flow shunted through upper airway walls according to:

\[ Z_{in,c} = Z_{in}X_{aw} / (Z_{aw} - Z_{in}) \]  
\[ (Zu/Zu)_c = (Zu/Zu - H) / (1-H) \]

where \( H = Zaw/Zaw \) and \( c \) refers to corrected values. Finally, \( Zaw \) was computed according to equation 2, using values of \( Zg \) computed from the instantaneous TGV (equation 3).

**Results**

A time plot of \( V_ao \), \( V \), the real (\( R_{aw} \)) and the imaginary (\( I_{aw} \)) parts of \( Zaw \) obtained with an oscillation frequency of 20 Hz in a representative subject is shown in figure 2. The four curves have been low-pass filtered at 1 Hz to eliminate the high frequency components of \( V_ao \) and \( V \) and smooth the impedance data. It can be seen that both \( R_{aw} \) and \( I_{aw} \) undergo systematic variations during the respiratory cycle. \( R_{aw} \) increases during the early part of both inspiration and expiration and decreases during the latter part. \( R_{aw} \) was usually slightly greater at end-expiration than at end-inspiration. That pattern was similar in all subjects. The variations of \( I_{aw} \) almost mirrored those in \( R_{aw} \), exhibiting a decrease in the early part of both phases and an increase in the latter part.

Using similar time plots, ensemble-averaged cycles were built for all variables and for all subjects with 32 points per respiratory cycle (16 for each phase). The mean values of \( R_{aw} \) and \( I_{aw} \) over the whole cycle in the eight subjects at the four oscillation frequencies are shown in figure 3. \( R_{aw} \) tended to increase slightly with increasing frequency, rising from a mean value of 0.81±0.13 hPa·s·L⁻¹ at 10 Hz to 3.59±0.57 hPa·s·L⁻¹ at 40 Hz (p<0.001). This increase is consistent with the usual assumption that the airways may be modeled as a resistance–inertance pathway [1], so that \( Zaw = Iaw \times R_{aw} \), with \( Iaw \) the airway inertance. \( Iaw \) as obtained from that relationship, did not vary significantly with increasing frequency; it averaged 1.29±0.21 Pa·s²·L⁻¹ at 10 Hz and 1.40±0.23 Pa·s²·L⁻¹ at 40 Hz.

The time dependence of \( R_{aw} \) and \( I_{aw} \) was analyzed by studying their relationship to \( V_ao \) and \( V \). This was done by fitting the data from the block-averaged cycles to the following equation by multiple linear regression:

\[ X = K_1 + K_2 \times \text{abs}(V_ao) + K_3 \times V \]

where \( X \) stands for either \( R_{aw} \) or \( I_{aw} \); \( \text{abs}(V_ao) \) is the absolute value of \( V_ao \); \( K_1 \) and \( K_2 \) express the dependence of \( X \) over the absolute flow and lung volume, respectively, and \( K_3 \) is the value of \( X \) at zero flow and zero volume (arbitrarily taken at midtidal volume). The analysis was made separately for the inspiratory and expiratory phases and the results are presented in tables 2 and 3.

Equation 6 usually gave a good fit to \( R_{aw} \) data; the residuals averaged 0.03–0.07 hPa·s·L⁻¹ and the multiple \( r^2 \) 0.94–0.98. \( R_{aw} \) was slightly, but not significantly, larger during expiration than during inspiration at 10–30 Hz; this was not due to any difference in \( K_1 \), but to a substantially
larger flow dependence during the expiratory phase (p < 0.05 at 20 Hz). \( K_2 \) did not vary significantly with the oscillation frequency; it was statistically significant in all subjects and at all frequencies, except in one subject at 10 Hz during expiration. \( K_3 \) was also significant in most instances; on average, it was negative at all frequencies, i.e. \( R_w \) tended to decrease with increasing lung volume, and this trend was stronger during the inspiratory phase.

Equation 6 also gave a good fit to \( I_w \) data; the residuals averaged 0.01–0.04 Pa·s²·L⁻¹ and the multiple \( r^2 \) 0.96–0.99. The mean \( I_w \) and \( K_1 \) were similar during inspiration and expiration. As for \( R_w \), the flow dependence was stronger during the expiratory phase but, in contrast to \( R_w \), \( I_w \) decreased with increasing absolute flow; \( K_2 \) was significant in all instances during both phases. Although statistically significant in most instances, the volume dependence was weak except at 10 Hz during inspiration.

**Discussion**

The data presented in this study show that airway impedance varies during the respiratory cycle; thus, it is possible to distinguish between its flow-dependent and flow-independent portions, which is of great potential interest.

The approach used in this study to separate airway and tissue properties does not imply any assumption concerning the mechanical properties of the two compartments. Thus, in contrast to the analysis of \( Z \) data using the six-component model of Dr. Bos et al. [1], it is not assumed that the chest wall behaves as a resistance–elastance–inertance system and that the airways are a resistance–inertance pathway; it is only after the separation is made that modelling may be performed if one wishes to interpret the compartmental impedances in terms of specific mechanical properties. Moreover, provided alveolar pressure is homogeneous, equations 1 and 2 are still valid if the tissues are made of several parallel compartments with different properties. This is clearly of importance since the chest wall has been shown to behave inhomogeneously above a few Hertz [6, 11]. When the alveolar pressure is inhomogeneous, that is when the respiratory system is made of several T-networks (fig. 1), the distribution of flow between the compartments depends on both their individual tissue and airway properties. It follows, for instance, that an increase in a local elastance will modify the apparent local and global resistance. In that situation, airways and tissue effective properties are interdependent and, except by making local measurements, it is not possible to separate them, whatever the approach. To evaluate the potential influence of mechanical inhomogeneity on \( Z_w \), as measured by equation 2, a system made of two T-networks in parallel, with inhomogeneous properties, was simulated numerically. Their overall \( Z_w \) and \( Z_t \) at 20 Hz were derived and their apparent \( Z_w \) computed according to equation 2. In the homogeneous case, both networks had a \( Z_w \) of 2 hPa·s⁻²·L⁻¹ and an \( I_w \) of 2 Pa·s⁻¹·L⁻¹, a TGV of 2 L, a tissue compliance (C) of 0.05 L·hPa⁻¹ and a tissue resistance (Rt) 1 hPa·s⁻²·L⁻¹. The data are shown in table 4. Decreasing one of the TGV by a factor of 10 increased the computed \( R_w \) by 1.4% and \( I_w \) by 3.1%. Varying one of the local \( C \) or \( R_t \) by any amount, however, did not influence at all the estimated \( R_w \) and \( I_w \). Increasing one of the \( R_w \) by a factor of 10 increased, as expected, the global \( R_w \), but also increased the global \( I_w \). In this situation, additional inhomogeneities of the TGV or the tissues changed little the apparent \( R_w \), but could modify the apparent \( I_w \). It was concluded that: 1) mechanical inhomogeneity of the tissues and gas distribution in the lung has little influence on the estimation of airway properties, the fact that \( R_w \) and \( I_w \) varied little with the oscillation frequency (tables 2 and 3) supports this conclusion; and 2) inhomogeneity of \( R_w \) may interfere with the estimation of \( I_w \).

The accuracy of \( Z_w \), as obtained by this approach, also depends on methodological factors. In practice, the relative frequency response of the body box and the pneumotachograph (PTTFeq), which was recorded after each measurement, appeared extremely reproducible in a given individual. A problem common to all forced oscillation measurements with the pressure input applied at the airway opening is the upper airway artefact [12], that is the flow shunted by

<table>
<thead>
<tr>
<th>( f_o ) Hz</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>40</th>
</tr>
</thead>
<tbody>
<tr>
<td>( R_w ) hPa·s·L⁻¹</td>
<td>1.42±0.65</td>
<td>1.44±0.63</td>
<td>1.60±0.99</td>
<td>1.80±1.07</td>
</tr>
<tr>
<td>( K_1 ) Pa·s⁻²·L⁻¹</td>
<td>1.10±0.52</td>
<td>1.06±0.52</td>
<td>1.27±0.90</td>
<td>1.48±1.05</td>
</tr>
<tr>
<td>( K_2 ) Pa·s⁻²·L⁻¹</td>
<td>0.82±0.38</td>
<td>0.90±0.40</td>
<td>0.83±0.44</td>
<td>0.76±0.36</td>
</tr>
<tr>
<td>( K_3 ) Pa·s⁻²·L⁻¹</td>
<td>-0.35±0.22</td>
<td>-0.34±0.24</td>
<td>-0.39±0.32</td>
<td>-0.35±0.30</td>
</tr>
</tbody>
</table>

Values are means±SD. \( K_1 \): value of \( R_w \) at zero flow and zero volume; \( K_2 \) and \( K_3 \): dependence of \( R_w \) over the absolute flow and lung volume, respectively. *: p<0.05 for difference between inspiration and expiration (paired t-test).

**Values of airway inertance (\( I_w \)), \( K_1 \), \( K_2 \) and \( K_3 \) for inspiration and expiration at four oscillating frequencies (\( f_o \))**

<table>
<thead>
<tr>
<th>( f_o ) Hz</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>40</th>
</tr>
</thead>
<tbody>
<tr>
<td>( I_w ) Pa·s⁻¹·L⁻¹</td>
<td>1.34±0.22</td>
<td>1.51±0.27</td>
<td>1.52±0.28</td>
<td>1.49±0.25</td>
</tr>
<tr>
<td>( K_1 ) Pa·s⁻¹·L⁻¹</td>
<td>1.46±0.25</td>
<td>1.62±0.34</td>
<td>1.53±0.28</td>
<td>1.51±0.31</td>
</tr>
<tr>
<td>( K_2 ) Pa·s⁻¹·L⁻¹</td>
<td>-0.33±0.16</td>
<td>-0.25±0.19</td>
<td>-0.20±0.26</td>
<td>-0.12±0.17</td>
</tr>
<tr>
<td>( K_3 ) Pa·s⁻¹·L⁻¹</td>
<td>0.30±0.17</td>
<td>0.16±0.30</td>
<td>0.05±0.10</td>
<td>0.04±0.10</td>
</tr>
</tbody>
</table>

Values are means±SD. \( K_1 \): value of \( I_w \) at zero flow and zero volume; \( K_2 \) and \( K_3 \): dependence of \( I_w \) over the absolute flow and lung volume, respectively.
Table 4. – Influence of mechanical inhomogeneity on estimates of airway resistance ($R_{aw}$) and inertance ($I_{aw}$) (computer simulation)

<table>
<thead>
<tr>
<th>Condition</th>
<th>$R_{aw}$ hPa·s·L$^{-1}$</th>
<th>$I_{aw}$ Pa·s$^2$·L$^{-1}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Homogeneous</td>
<td>1.000</td>
<td>1.000</td>
</tr>
<tr>
<td>TGV/10</td>
<td>1.014</td>
<td>1.031</td>
</tr>
<tr>
<td>C/t/10</td>
<td>1.000</td>
<td>1.000</td>
</tr>
<tr>
<td>$R_{aw}$×10</td>
<td>1.000</td>
<td>1.000</td>
</tr>
<tr>
<td>$R_{aw}$×10 and TGV/10</td>
<td>2.193</td>
<td>1.474</td>
</tr>
<tr>
<td>$R_{aw}$×10 and C/t/10</td>
<td>1.952</td>
<td>1.602</td>
</tr>
<tr>
<td>$R_{aw}$×10 and C/t/10</td>
<td>2.533</td>
<td>1.435</td>
</tr>
<tr>
<td>$R_{aw}$×10 and C/t/10</td>
<td>2.199</td>
<td>1.515</td>
</tr>
<tr>
<td>$R_{aw}$×10 and C/t/10</td>
<td>2.006</td>
<td>1.264</td>
</tr>
<tr>
<td>$R_{aw}$×10 and C/t/10</td>
<td>2.114</td>
<td>1.584</td>
</tr>
<tr>
<td>$R_{aw}$×10 and C/t/10</td>
<td>2.173</td>
<td>0.729</td>
</tr>
</tbody>
</table>

TGV: thoracic gas volume; C: tissue compliance; R: tissue resistance. Homogeneous case: $R_{aw}$: 2 hPa·s·L$^{-1}$; $I_{aw}$: 2 Pa·s$^2$·L$^{-1}$; TGV: 2 L; C: 0.05 L·hPa$^{-1}$; $R_c$: 1 hPa·s·L$^{-1}$ in both compartments. Subscripts 1 and 2 identify the compartments in which the properties are altered compared to the homogeneous case.

The motion of upper airway walls (cheeks, mouth floor, pharynx). With the present set-up, this flow could modify the motion of upper airway walls (cheeks, mouth floor, pharynx). With the present set-up, this flow could modify.

The advantages of separating airway and tissue properties from impedance data obtained at a single frequency are many. Firstly, it avoids the crosstalk between frequencies observed with nonlinear systems when the input signal does not meet a number of very stringent criteria [16]. Secondly, it provides the best possible time resolution; indeed, while, in theory, the time resolution of data from multiple frequency inputs may be as good as the period of the lowest frequency component, the minimal duration of a data block containing an integer number of cycles of all frequency components may be much longer with inputs meeting the above-mentioned criteria. Thirdly, the signal/noise ratio is better when all of the energy is concentrated in a single frequency than when it is distributed among several components. Finally, it avoids assuming that the mechanical properties of the respiratory system are independent of frequency, which is implicit when analysing Zn data with the model of DuBois. The results shown in figure 3 suggest that $R_{aw}$ may vary substantially with frequency in some individuals; also, as mentioned above, the effective resistance and elastance of the tissues may vary with frequency as a result of the mechanical inhomogeneity of the chest wall.

This study showed substantial variations in $R_{aw}$ during the respiratory cycle. These variations were analysed with a descriptive model (equation 6) inspired from Rohrer’s equation [17], which expresses the nonlinear pressure–flow relationship along the airways:

$$P_{ao}-P_{av}=K_{ao} \times V_{av0} + K \times V_{av}^2$$

where $P_a$ is the alveolar pressure. From that relationship, $R_{aw}$, as measured by forced oscillation from small variations of pressure and flow, is expected to be proportional to flow:

$$R_{aw} = \frac{d(P_{ao}-P_{av})/dV_{av0}}{K_{ao}+2K \times V_{av0}}$$

From this equation, the coefficient $K_1$ in equation 6 would correspond to twice Rohrer’s $K_1$ coefficient when the pressure oscillations are infinitely small. This is not the case in practice and the expected ratio between Rohrer’s $K_1$ and our $K_1$ coefficient may vary between 1 and 2, depending on the relative amplitudes of the oscillatory and respiratory flows [18]. In the present model, a third term was included to account for any variations in $R_{aw}$ in relation to lung volume. The analysis showed a good fit of the data to the model with $K_1$ values around 0.8 hPa·s$^{-2}$·L$^{-1}$ during the inspiratory phase and 1.3 hPa·s$^{-2}$·L$^{-2}$ during the expiratory phase.

These observations are similar to those of Cunha-Brock and van De Wouw-Arntz [19], who found response of the respiratory system ($R_{is}$) values to be lower during peak tidal inspiratory flow and greater during maximal inspiratory flow. They have also shown that $R_{is}$ increased with increasing values of expiratory flows. The values of $K_1$ coefficients are similar to those obtained by Pelle et al. [20], who studied Zn variations during the respiratory cycle. Mean $K_1$ values in seven healthy subjects at 4–6 Hz were 0.95±0.47 and 1.49±0.55 hPa·s$^{-2}$·L$^{-2}$ for inspiration and expiration, respectively. In another work from the same laboratory [18] the analysis of Zn variations measured with a head pressure generator gave values of $K_1$ varying from 0.65 and 1.18 at 10 Hz to 1.2 and 1.7 hPa·s$^{-2}$·L$^{-2}$ at 30 Hz for inspiration and expiration, respectively.

The analysis revealed some negative volume dependence of $R_{aw}$, which is consistent with previous data. Oster-Veen et al. [21] reported a decrease with volume of airway resistance derived from transfer impedance during inspiratory manoeuvres. Pellic et al. [18] reported values of $K_1$ ranging from about -0.3 to -0.7 for inspiration and -0.8 to -1.4 hPa·s$^{-2}$·L$^{-2}$ for expiration. These values are larger than those found in the present study, but they were calculated from Zn variations during normal breathing and not from $Zaw$, as in this study.
The study also revealed systematic variations in $I_{aw}$ during the respiratory cycle (fig. 2) which, analysed with equation 6, corresponded mostly to negative flow dependence (table 3). This finding is in agreement with the observations of Ostven et al. [21] who derived $I_{aw}$ from 4–30 Hz $Z_{e}$ data obtained during constant-flow inspiratory and expiratory manoeuvres. During inspiration $I_{aw}$ decreased from 2.12 Pa·s²·L⁻¹ at 0.1 L·s⁻¹ to 1.79 hPa·s²·L⁻¹ at 0.4 L·s⁻¹; the changes, however, were smaller during the expiratory phase (2.15 and 1.91 Pa·s²·L⁻¹ at 0.1 and 0.4 L·s⁻¹, respectively), which is at variance with the present observations. Such a negative flow dependence is consistent with a blunter velocity profile of air in the airways when the flow is larger [22].

While the possibility of separating airway and tissue mechanical properties is obviously of interest for a number of clinical and physiological applications, one may wonder about the practical usefulness of studying the variations in $I_{aw}$ during the respiratory cycle. The flow dependence, and to a lesser extent, the volume dependence of $I_{aw}$, make it frequently difficult to interpret its value in patients and its variations during bronchomotor challenge. Indeed, changes in ventilation or in the respiratory pattern may modify $I_{aw}$ and obscure the actual changes in airway patency. Moreover, most of the flow dependence of $I_{aw}$ has been shown to be located in extra-thoracic airways [23] and it has also been observed that induced bronchoconstriction may reflexly increase extra-thoracic airway resistance [24]. Then, the flow-dependent component of $I_{aw}$ may be largely irrelevant when studying bronchial responsiveness. When $I_{aw}$ is measured by body plethysmography, the problem may be solved to some extent by standardizing the flow range over which the pressure is measured [25]; the data, however, still include nonlinear components. The advantage of studying the variations in $I_{aw}$ during the cycle and analysing the data with a descriptive model, such as equation 6, is that it makes it possible to isolate its linear component, $K$. The latter should represent an index of airway patency that is much more independent of the ventilation and of the extrathoracic airways than the mean resistance or the resistance and relative displacement of relaxed chest wall up to 4 Hz. $I_{aw}$ may be largely irrelevant when studying bronchial responsiveness.

Further studies are needed to verify that prediction and the value of this type of analysis in pulmonary function testing, especially in the evaluation of the results of bronchomotor tests.

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References