Viscoelasticity represents an important component of respiratory mechanics, being responsible, in some cases, for most of the pressure dissipated during breathing. Hitherto, the methods available for determining the viscoelastic properties have been simplified, but are still time-consuming and depend on a great deal of calculation. In this study, a simple means of determining respiratory viscoelastic properties during mechanical ventilation was introduced.

The viscoelastic constants of the respiratory system, modelled as a Maxwell body, were studied in 17 normal subjects and seven patients with acute lung injury (ALI) using two end-inspiratory occlusions; one with a short inspiratory time (t) to determine the elastic component of viscoelasticity and the other with a long t to assess the resistive component of viscoelasticity.

The results were reproducible and similar to those provided by the previously described multiple-breath method (MB). The mean±SD viscoelastic resistance was 5.31±1.50 cm H2O L−1 s with the proposed method and 5.71±1.87 cm H2O L−1 s with the MB method in normal subjects, and 8.93±2.82 cm H2O L−1 s and 10.36±3.13 cm H2O L−1 s, respectively, in ALI patients. The mean±SD viscoelastic elastance was 3.92±0.84 cm H2O L−1 and 4.94±1.01 cm H2O L−1 in normal subjects and 7.08±2.01 cm H2O L−1 and 8.21±1.16 cm H2O L−1 in ALI patients, respectively. The mean±SD viscoelastic time constant was 1.36±0.24 s and 1.17±0.34 s in normal subjects and 1.26±0.35 s and 1.24±0.23 s in ALI patients, respectively.

The method was easy to perform and applicable at the bedside in clinical routine.

Viscoelasticity represents an important component of respiratory mechanics, being responsible, in some cases, for most of the pressure dissipated during breathing. In order to study the viscoelastic behaviour of the respiratory system, it is necessary to assess the pressure developed by the viscoelastic components. Direct measurement of the viscoelastic inspiratory pressure can be performed by the technique of rapid end-inspiratory airway occlusion and viscoelastic inspiratory pressure can be performed by the technique of rapid end-inspiratory airway occlusion and viscoelastic behaviour interpreted according to a Maxwell body. This linear viscoelastic model has been shown to provide an accurate description of the time-dependency of resistance and elastance of the respiratory system observed in normal animal [1] and human lungs [2].

In this model, the viscoelastic properties which impact such time-dependency can be characterized by two parameters, the theoretical maximal viscoelastic resistance (R2) and elastance (E2). A third useful variable, the viscoelastic time constant (τ2), can also be obtained from R2/E2 [3]. Using the technique of rapid airway occlusion (RAO) during constant-flow (V') inflation, it has been possible to determine the values of these viscoelastic constants for the lung, chest wall and total respiratory system in normal mechanically ventilated humans [3–5] and experimental animals [1, 6], based on either of the following functions:

\[ P_{visc}(t) = R2V'(1 - e^{-t/\tau2}) \]  

\[ \Delta R_{visc}(t) = P_{visc}(t)/V' = R2(1 - e^{-t/\tau2}) \]  

where \( P_{visc}(t) \) is the viscoelastic pressure (\( P_{visc} \)) dissipated within the lung, chest wall or both during constant- \( V' \) inflation started from the relaxed volume of the respiratory system, \( t \) is time during lung inflation and \( \Delta R_{visc} \) is viscoelastic resistance, obtained by dividing both sides of Equation 1 by \( V' \) [3]. This analysis, however, is time-consuming and technically complex because it requires either a series of isovolumic inflations with different inspiratory \( V' \) or multiple iso- \( V' \) inflations with different volumes [3, 6]. As a result, the above analysis has been used only in a limited number of studies on normal subjects [3–5] and patients [7–9].

Recently, a single-breath method was proposed for assessing the viscoelastic properties of the respiratory system, which was applied to normal subjects and acute lung injury (ALI) patients [10]. This method is based on the exponential analysis of the time course of tracheal pressure (\( P_{tr} \)), after RAO at baseline inflation volume. After occlusion, \( P_{tr} \) shows an immediate fast fall from the peak value down to an inflection point (\( P_{i} \)), followed by a
slow decay to an apparent plateau, from which the elastic recoil pressure of the respiratory system ($P_{rs,sl}$) can be determined.

In the present investigation, a practical method for assessing the viscoelastic constants of the respiratory system, which can be performed without cumbersome exponential analysis is described. For such purpose, $P_I$ and $P_{rs,sl}$ are measured in only two different breaths, i.e. with short and prolonged inspiratory time ($t$). This method was used in normal anaesthetised paralysed subjects and in patients with ALI. The results were compared with those obtained on the same subjects using the multiple iso-$V'$ inflations method.

**Theory**

As previously reported [10], after end-inspiratory airway occlusion, the difference between $P_I$ and $P_{rs,sl}$ reflects $P_{visc}$. At the time of occlusion, $t$, $P_{visc}$ is maximal and can be denoted as $P_{visc,max}$.

From Equation 1, $P_{visc,max}$ is given by:

$$P_{visc,max} = R_{rs} V'(1 - e^{-t/t_2})$$

(3)

Dividing both sides of Equation 3 by tidal volume ($Vr$), the value of the additional elastance of the respiratory system ($\Delta E_{rs}$) at $t$ can be obtained [4]:

$$\Delta E_{rs} = P_{visc,max}/Vr = R_{rs} V'(1 - e^{-t/t_2})/Vr$$

(4)

For very small $t$, the exponential term of Equation 4 can be considered equivalent to $1-t/t_2$ and $\Delta E_{rs}$ can be approximated to $E_2$ [11]. Conversely, for large $t$ the exponential term of Equation 2 becomes negligible and $\Delta E_{rs}$ can be approximated to $R_2$. $t_2$ can then be obtained from $t_2 = R_2/E_2$.

Thus, using two single constant-$V'$ inflations with one short and one long $t$ each followed by an end-inspiratory pause with occluded airway, the viscoelastic constants of the respiratory system can be obtained.

**Materials and methods**

Seventeen patients (12 male) undergoing general anaesthesia (premedication: diazepam, 7.5 mg p.o.; anaesthesia: propofol, 2 mg kg$^{-1}$ i.v.; muscle paralysis: succinylcholine, 1 mg kg$^{-1}$ i.v.; maintenance: continuous infusion of propofol, 8–12 mg kg$^{-1}$ h$^{-1}$, and vecuronium, 0.15 mg kg$^{-1}$ h$^{-1}$) for minor lower abdominal or limb surgery were studied before skin incision. An inspiratory oxygen fraction ($F_{I,O_2}$) of 0.4 and no positive end-expiratory pressure (PEEP) were used. None had a history or clinical evidence of either restrictive or chronic lung disease, nor cardiogenic pulmonary oedema or active cardiac disease. The patients were studied at zero end-expiratory airway pressure. PEEP was removed 30 min before the study, and patients were judged to have reached a steady state by stability of respiratory mechanics and pulse oximetry records.

All subjects lay in the supine position, were intubated with a Rush cuffed endotracheal tube (inside diameter (ID) 7.5–8.0 mm) and ventilated with constant inflation $V'$-controlled ventilation by means of a Servo Ventilator 900C (Siemens-Elema AB, Solna, Sweden). Rapid airway occlusions were performed using a solenoid valve (Airmatic SV, Airmatic-Allied, Wilmington, OH, USA) placed next to the oral end of the endotracheal tube. The solenoid valve had a closing time of 11 ms. The closing time was measured in the Electronics Laboratory, Dept of Energetics, Faculty of Engineering, Trieste, Italy, with an accelerometer (Brüel & Kjær 4332; Brüel & Kjær Italiana, Milan, Italy) and a current probe (AC-DC Fluke Y8100; Fluke Corporation, Everett, WA, USA) connected to a rapid recorder (Hioki 8830; Hioki E.E. Corporation, Nagano, Japan). $V'$ was measured using a heated Jäger Baby pneumotachograph (Jäger, Würzburg, Germany) with a $\pm 3$ L s$^{-1}$ linearity range. The pneumotachograph was inserted between the end of the tracheal tube and the solenoid valve, and connected to a Validyne pressure transducer ($\pm 2$ cmH$_2$O, MP-45, Validyne; Northridge, CA, USA) and to a carrier amplifier (13-4615-35, Gould, Inc.; Cleveland, OH, USA). Tracheal pressure was measured via a polyethylene catheter protruding 2–3 cm beyond the tracheal end of the endotracheal tube with a piezoresistive differential pressure transducer (Microswitch 142PC6SD, Honeywell Ltd.; Scarborough, Ontario, Canada). The tracheal catheter (ID 1.5 mm) had six side holes around its distal end and an occluded tip. The system used to measure $P_r$ showed no appreciable phase shift and the response was flat up to 20 Hz. The overall dead space of the measuring equipment (excluding the endotracheal tube) was 35 mL. The resistance offered by this equipment was 8 cmH$_2$O L$^{-1}$s at a $V'$ of 1 L s$^{-1}$. $V'$ and $P_r$ signals were fed through a 12-bit analogue-to-digital converter (Data Translation DT2801A, Data Translation, Inc.; Marlboro, MA, USA) into an IBM-compatible personal desk computer. The sampling frequency was established at 200 Hz. Volume was obtained by numerical integration of the $V'$ signal. All data were analysed using ANADAT data analysis software (RHT-InfoData, Inc., Montreal, Quebec, Canada).

Great care was taken to avoid leaks around the tracheal cuff and within the equipment. In all patients, the electrocardiogram and arterial blood pressure were continuously monitored as well as peripheral arterial oxygen saturation and end-tidal arterial carbon dioxide tension (Ohmeda 5250 RGM, Ohmeda, Louisville, CO, USA). An anaesthetist not involved in the experiment was continuously present to provide patient care.

**Experimental procedure and data analysis**

Normal subjects were ventilated as follows: $F_{I,O_2}$=0.4, $V'$=0.53±0.02 L s$^{-1}$, tidal volume ($Vr$)=7.5±0.8 mL kg$^{-1}$. Their anthropometric characteristics are shown in Table 1. None had a history or clinical evidence of either restrictive or chronic lung disease, nor cardiogenic pulmonary oedema or active cardiac disease.
inspiratory time ($t I$) = 0.99±0.09 s, and respiratory frequency ($f R$) = 14–15 breaths min$^{-1}$. With these settings, there was normocapnia in all normal subjects and intrinsic PEEP (PEEPi) was absent, as indicated by each inflation being preceded by a period of zero $V R$ (end-expiratory pause) and $P E$ being zero during end-expiratory airway occlusions. The ALI group was ventilated as follows: $F I O_{2} = 0.5$, $V T = 0.52±0.09 L\cdot s^{-1}$, $P C O_{2} = 6.3±0.7 mL\cdot kg^{-1}\cdot s^{-1}$, $f R = 0.93±0.26 s^{-1}$, $f t$ of 16–18 breaths min$^{-1}$. The mean±SD PEEPi was 3.1±2.2 cmH$_2$O.

Data collection started during an end-expiratory occlusion for assessment of PEEP. The end-expiratory occlusion was followed by five recovery breaths at baseline ventilatory settings, followed by an end-inspiratory occlusion. All measurements were repeated three times under the same experimental conditions. Between each test, $V R$, $P E$, and $P E$ were allowed to return to baseline levels.

End-tidal inspiratory occlusions, which lasted for 5 s, were achieved using the solenoid valve triggered by the Servo Ventilator. The $V R$ loss due to the continuous gas exchange had a negligible impact on $P E$. After the occlusion, an initial rapid drop in $P E$ to $P I$ (maximum pressure ($P_{\text{max}}$) pressure after first drop ($P I$)) was followed by a subsequent gradual decrease to an apparent plateau pressure ($P_{\text{RST}}$). $P E$ measured 5 s after the onset of occlusion, was taken as the end-inspiratory $P_{\text{RST}}$. By dividing maximum $P E$ ($P_{\text{RST, max}}$, $P_{\text{RST, max}}$, and $P_{\text{RST, max}}$) by the $V R$ immediately preceding the occlusion, the total resistance of the respiratory system ($R_{T}$) and the ohmic resistance of the respiratory system ($R_{\text{int}}$) were obtained. $R_{T}$ was calculated as the difference between $R_{\text{ST}}$ and $R_{\text{int}}$ [14, 15]. In computing $R_{\text{int}}$ the errors caused by the closing time of the valve, although minute, were corrected as previously described [16]. In this analysis, the onset of the occluded inspiration corresponded to the end of the preceding constant $V R$ inflation obtained with a given $t I$. Accordingly, the difference $P I- P_{\text{RST}}$ should correspond to the $P_{\text{RST}}$ at $t I$ ($P_{\text{RST, max}}$).

The static elastance of the respiratory system ($E_{R, S T}$) was computed by dividing the corresponding $V R$ by $P E$. The dynamic elastance of the respiratory system ($E_{S D, s t}$) was computed by dividing the corresponding $P E$ by $P E$.

**Short-and-long-breath method**

In each patient, $E_{2}$ was approximated to by $E_{R S}$ obtained from inflations with small $t I$ and identical $V R$. Conversely, $E_{2}$ was approximated to by $E_{R S}$ obtained during inspirations with large $t I$. From the ratio between $P I- P_{\text{RST}}$ and $P T$ pertaining to the corresponding short constant-$V R$ lung inflation, $\Delta E_{R S}$ was computed.

The mean values from three pairs of short-and-long breaths were used to measure $R_{2}$, $E_{2}$, and $t_{2}$.

### Multiple-breath method

The iso-$V R$ occlusion multiple breath (MB) method previously described in detail was used [2, 4]. Under the same baseline conditions, 5-s end-inspiratory occlusions were performed at four or five different inflation $V R$ by intermittently changing $t I$ while keeping the basal inflation $V R$ constant. The corresponding occluded inflation $V R$ ranged 0.19–1.55 L. These tests were repeated three times. At each occlusion $V R$, $\Delta R S$ was obtained and, together with the corresponding $V R$ and $t I$, fitted to Equation 2 to obtain $R_{2}$ and $E_{2}$.

The investigation was approved by the local Ethics Committee, and informed consent was obtained from each individual or their next of kin.

### Statistical analysis

Regression analysis was performed using the least-squares method. The paired t-test was used to compare the data from the first test with the corresponding mean values obtained from the three repeat tests. Comparison of the results provided by the short- and long-breath and MB methods was performed by means of the limit of agreement [17], as modified for small sample sizes [18]. Correlations of the ratios $\Delta E_{R S} E_{2}$ and $\Delta R S R_{2}$ with $t I / t_{2}$ were evaluated using the Pearson test. The significance level was established at $p<0.05$.

### Results

Table 2 depicts the mean±SD of $R_{\text{int}}$, $\Delta R S E_{R S}$, and $E_{S D, s t}$ in normal subjects and ALI patients under baseline ventilatory settings obtained from three end-inspiratory occlusion tests.

Table 3 depicts the mean $t_{2}$, $R_{2}$, and $E_{2}$ obtained from three short and long $t I$ breaths in normal subjects and ALI patients. The mean±SD intrasubject coefficients of variation of $t_{2}$, $R_{2}$, and $E_{2}$ were $8±4\%$, $5±3\%$, and $7±2\%$, respectively, in normal subjects, and $11±5\%$, $6±3\%$, and $10±3\%$ respectively, in ALI patients. Table 3 also depicts the mean±SD differences between the results of the short-and-long-breath and MB methods in normal subjects were: $t_{2}$, $-0.17±0.26 s$; $R_{2}$, $0.43±0.77 cmH$_2$O$L^{-1}\cdot s^{-1}$; and, $E_{2}$, $1±0.57$.

### Table 1. – Acute lung injury patient characteristics and diagnosis

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sex</th>
<th>Height cm</th>
<th>Weight kg</th>
<th>Age yrs</th>
<th>Diagnosis</th>
<th>$P_{a,O2}/F I O_{2}$ mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>177</td>
<td>86</td>
<td>44</td>
<td>Sepsis</td>
<td>160</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>165</td>
<td>68</td>
<td>61</td>
<td>Sepsis</td>
<td>155</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>173</td>
<td>75</td>
<td>67</td>
<td>Multiple injury</td>
<td>120</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>161</td>
<td>66</td>
<td>71</td>
<td>Sepsis</td>
<td>185</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>176</td>
<td>71</td>
<td>73</td>
<td>Sepsis</td>
<td>190</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>187</td>
<td>89</td>
<td>49</td>
<td>Sepsis</td>
<td>180</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>168</td>
<td>73</td>
<td>43</td>
<td>Sepsis</td>
<td>160</td>
</tr>
</tbody>
</table>

$P_{a,O2}$: arterial oxygen tension; $F I O_{2}$: inspiratory oxygen fraction; M: male; F: female. (1 mmHg = 0.133 kPa.)
cmH₂O·L⁻¹. The mean±SD differences between τ₂, R₂, and E₂ given by the short-and-long-breath and MB methods in ALI patients were: -0.01±0.13 s, 1.24±0.68 cmH₂O·L⁻¹·s and 1.06±0.47 cmH₂O·L⁻¹, respectively. No lack of agreement could be detected between the two methods for all variables in normal subjects and ALI patients.

The mean±SD short-breath τF and τI pertaining to occlusions of the present method were, in normal subjects, 0.345±0.04 L and 0.65±0.08 s and, in ALI patients, 0.358±0.06 L and 0.68±0.14 s, respectively. The mean±SD ratio of short-breath τI and τ₂ (ΔtI)/τ₂, was 0.60±0.19 in normal subjects, and 0.56±0.13 in ALI patients. Figure 1 depicts the discrepancy between the ΔtI obtained with the short-and-long-breath and MB methods (ΔtI/Δt₂) as a function of the discrepancy between the τI pertaining to ΔtI computation and the corresponding viscoelastic time constant, tI(ΔtI)/t₂, for normal subjects and ALI patients. There was a significant inverse relationship between these two parameters for normal subjects (y=1.08-0.464x, r=-0.851, p<0.0001). The overall regression data for both normal subjects and ALI patients (y=1.09-0.458x, r=-0.809, p<0.0001), are very similar to those gathered from normal subjects alone.

The mean±SD long-breath τF and τI were, in normal subjects, 1.146±0.18 L and 2.22±0.36 s and, in ALI patients, 1.124±0.33 L and 2.11±0.49 s, respectively. The mean±SD ratio tI(ΔtI)/t₂ was 2.07±0.72 in normal subjects, and 1.71±0.39 in ALI patients. The relationship between tI(ΔtI)/t₂ and the ratio ΔtI/Δt₂ for normal subjects is illustrated in fig. 2. There was a significant relationship between these two parameters for normal subjects: (y=0.637x+0.148x, r=0.768, p<0.0003). The overall regression data for both normal subjects and ALI patients (y=0.628±0.151x, r=0.779, p<0.0001), are very similar to those obtained from normal subjects alone.

The viscoelastic constants were also obtained from the baseline end-inspiratory occlusion manoeuvre using the single-breath (SB) method [10]. The mean±SD differences between the results of the short-and-long-breath and SB methods in normal subjects were: τ₂, 0.14±0.20 s; R₂, -0.11±0.68 cmH₂O·L⁻¹·s; and E₂, -0.55±0.88 cmH₂O·L⁻¹. The mean±SD differences between τ₂, R₂, and E₂ given by the short-and-long-breath and SB methods in ALI patients were: 0.06±0.36 s, -0.37±1.37 cmH₂O·L⁻¹·s, and -0.28±1.76 cmH₂O·L⁻¹, respectively. No lack of agreement could be detected between the two methods for all variables in normal subjects and ALI patients.

**Discussion**

As in previous studies [1, 4, 5, 10, 14], standard mechanical variables were obtained after end-inspiratory occlusions performed at baseline ventilation in the present study. The values of Eₘₙ, Eₘₙ,dyn, Rₛ, and ΔRₛ obtained in normal subjects are within the range reported in the literature [1, 4, 5, 10, 14]. The values obtained in ALI patients reflect marked inter-individual variation in respiratory mechanics (table 2).

Study of the viscoelastic behaviour of the respiratory system implies assessment of the pressure developed by the viscoelastic components. RAO permits the direct measurement of viscoelastic pressure and the present investigation shows that, from constant-Vᵐₙ lung inflation manoeuvres performed at short and prolonged periods of time, the viscoelastic constants of the respiratory system can be derived.

The results provided by the short-and-long breath and MB methods were in agreement. The mean differences between the two methods were nonsignificant, but E₂ determined by the MB method in normal subjects and ALI patients were ~1 cmH₂O·L⁻¹ higher than those measured by the short-and-long-breath method. However, for the short tI inflations, the normal subjects were partitioned into two subpopulations, using as a cut-off the mean τ₂/2, namely, 0.58 s. When t<0.58 s and >0.58 s, the mean±SD differences in E₂ between short-and-long-breath and MB methods were, respectively: 0.53±0.26 cmH₂O·L⁻¹ and 1.33±0.49 cmH₂O·L⁻¹. In other words, when using shorter inspirations (adequate for the determination of E₂) the difference

### Table 2. Respiratory mechanical variables of 17 normal subjects and 7 acute lung injury (ALI) patients obtained at baseline tidal volume

<table>
<thead>
<tr>
<th></th>
<th>Normal subjects</th>
<th>ALI patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Eₘₙ,st cmH₂O·L⁻¹</td>
<td>Eₘₙ,dyn cmH₂O·L⁻¹</td>
</tr>
<tr>
<td>Mean</td>
<td>18.1</td>
<td>21.2</td>
</tr>
<tr>
<td>SD</td>
<td>4.8</td>
<td>4.5</td>
</tr>
<tr>
<td></td>
<td>21.9</td>
<td>31.7</td>
</tr>
<tr>
<td></td>
<td>7.1</td>
<td>12.2</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD. Eₘₙ,st: static elastance of the respiratory system; Eₘₙ,dyn: dynamic elastance of the respiratory system; Rₛ,st: static elastance of the respiratory system; ΔRₛ: viscoelastic resistance of the respiratory system.

### Table 3. Viscoelastic time constant (τ₂), resistance (Rₑ), and elastance (Eₑ) of the respiratory system in 17 normal subjects and 7 acute lung injury (ALI) patients

<table>
<thead>
<tr>
<th></th>
<th>Normal subjects</th>
<th>ALI patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Method</td>
<td>τ₂ s</td>
<td>Rₑ cmH₂O·L⁻¹·s</td>
</tr>
<tr>
<td>SLB</td>
<td>1.36±0.24</td>
<td>5.31±1.50</td>
</tr>
<tr>
<td>MB</td>
<td>1.17±0.34</td>
<td>5.71±1.87</td>
</tr>
<tr>
<td></td>
<td>1.26±0.35</td>
<td>8.93±2.82</td>
</tr>
<tr>
<td></td>
<td>1.24±0.23</td>
<td>10.36±3.13</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD. SLB: short-and-long-breath (mean of three tests); MB: multiple-breath.
Equation 3, using \( P \) and \( f \) [17, 18]. In ALI patients, the difference in the agreement between the results was statistically confirmed, showing a systematic bias between the methods, although short-and-long-breath and MB methods were not zero, short-and-long-breath method was similar to the MB method. In order to obtain an \( R \) as close as possible to the predicted value, a \( t \) determining a high ratio between \( t \) and \( t \) should be chosen. As can be seen, at \( \pi(\Delta R E) = \pi(\Delta R E) \) the values of \( R \) obtained with the present method are similar to the corresponding predicted values. Conversely, the linearity of the system can be compromised using a very prolonged inflation. In figure 2, the

As in normal subjects, in ALI patients, the viscoelastic constants could be obtained by the short-and-long-breath method, and the mean differences in \( \Delta E \) and \( \Delta P \) between the short-and-long-breath and MB methods were not zero, showing a systematic bias between the methods, although the agreement between the results was statistically confirmed [17, 18]. In ALI patients, the difference in \( R \) between the two methods was, on average, 13.8%. One patient showed a difference of 25% (see fig. 2). If this patient is not taken into account, the patients showed a mean difference of 10%. Indeed, figures 1 and 2 show that the results for patients and normal subjects overlap. The systematic differences in \( R \) and \( E \) (on average of 10%) in normal subjects and ALI patients are probably related to different computation of the viscoelastic constants, by direct measurements of parameters pertaining to the short-and-long-breath method in the present study and by exponential analysis of measurements of parameters pertaining to different inflation volumes. Thus, using the short-and-long-breath method, a small sacrifice in accuracy allows the easy determination of viscoelastic constants at the bedside.

Recently, an SB method for obtaining viscoelastic constants from a baseline end-inspiratory occlusion manoeuvre was presented [10]. Although better agreement was found between the results of the SB and MB methods [10] with respect to those of the present study, the viscoelastic constants obtained in the present investigation were not significantly different from those obtained using the SB method in the present subjects. The SB method requires exponential analysis of the time course of \( P \) after baseline end-inspiratory occlusion in order to obtain \( \Delta E \) and \( \Delta P \) and computation of \( R \) from Equation 3, using \( \Delta E \) and \( \Delta P \), together with the \( \Delta E \) and \( \Delta P \) pertaining to the corresponding constant-\( \Delta E \) lung inflation. The present variant of the method requires only measure-ment of \( P \) and \( P \) and \( V \) at short \( t \) inflation and \( P \), \( P \), and \( V \) at long \( t \) inflation.

Figure 3 depicts the relationships of the mean \( E \) and \( R \) to \( t/\Delta \) obtained with the present method in the 17 normal subjects and in seven ALI patients, together with those predicted according to Equations 2 and 4, using the mean values of the viscoelastic constants obtained with the MB method. In order to obtain an \( R \) as close as possible to the predicted value, a \( t \) determining a high ratio between \( t \) and \( t \) should be chosen. As can be seen, at \( t(\Delta R E) = t(\Delta R E) \) the values of \( R \) obtained with the present method are similar to the corresponding predicted values. Conversely, the linearity of the system can be compromised using a very prolonged inflation. In figure 2, the

Fig. 1. – Relationship between \( \pi(\Delta E) \) and \( \pi(\Delta E) \) in the short-breath lung inflation occlusion test in normal subjects (●) and acute lung injury patients (○), where \( \pi(\Delta E) \) inspiratory time pertaining to the lung inflation occlusion test, \( \pi(\Delta E) \) the viscoelastic time constant, \( \pi(\Delta E) \) the viscoelastic elastance of the respiratory system and \( \pi(\Delta E) \) viscoelastic elastance. •••• regression line for normal subjects (r = 0.851, p < 0.0001).

Fig. 2. – Relationship between \( \pi(\Delta R E) \) and \( \pi(\Delta R E) \) in the short-breath lung inflation occlusion test in normal subjects (●) and acute lung injury patients (○), \( \pi(\Delta R E) \) inspiratory time pertaining to the respiratory system, \( \pi(\Delta R E) \) viscoelastic time constant, \( \pi(\Delta R E) \) the viscoelastic elastance of the respiratory system and \( \pi(\Delta R E) \) viscoelastic resistance. •••• regression line for normal subjects (r = 0.768, p < 0.0003).

Fig. 3. – Relationship of the mean viscoelastic elastance (\( E \)) and viscoelastic resistance (\( R \)) to the ratio inspiratory time (\( t \))/viscoelastic time constant (\( t \)) in normal subjects and acute lung injury (ALI) patients obtained with the short-and-long-breath method (— normal subjects; •••• ALI patients) together with those predicted according to Equations 2 and 4, using the mean viscoelastic constants obtained with the multiple-breath method (— normal subjects; •••• ALI patients).
subject with \( t(\Delta R_0)\tau_2 > 3 \) and \( \Delta R_0/\tau_2 > 1.3 \) may represent a case of loss of linearity. In the same figure, it can be observed that good agreement for \( R_2 \) \( (0.9 < \Delta R_0/\tau_2 < 1) \) can be obtained with a wide range of \( t/\tau_2 \) \( (1.2–2.6) \). At the extremity of this range, there were subjects with \( \Delta R_0/\tau_2 > 1 \) (possible expression of overdistension) and subjects and patients showing a significant difference between the two methods. Thus the mean value of the range (approximately \( t/\tau_2 = 2 \)) should be a good approximation for choosing the value of prolonged \( t \). In the present experimental conditions, it means a \( \tau_2 = 2.4 \) s.

In order to obtain an \( E_2 \) as close as possible to the predicted value, the ratio \( t/\tau_2 \) should be very low. At \( t = 0.6 \) s, a good agreement was obtained for \( E_2 \). Because the mean \( \tau_2 \) are similar in normal subjects and ALI patients \( (1.2 \) s \( \) \[1, 6\], \( t = 0.6 \) s corresponds to \( t/\tau_2 < 0.5 \) in normal subjects and patients. Conversely, in relation to inflation \( V' \), \( \tau_2 \) must be long enough to obtain a \( V' \) that inflates the lungs. At an inspiratory \( V' \) of \( 0.5 \) L\(\text{s}^{-1} \), it is not possible to reduce \( \tau_2 \) below 0.3 s. Also, under the present experimental conditions, a short \( \tau_2 \) is one of \( 0.3–0.6 \) s.

In the present investigation, the effect of inhomogeneity has not been taken into consideration, which may affect measurements of viscoelastic properties. Even though inhomogeneity is expected to be greater in ALI patients, it did not jeopardize the results.

Using the iso-\( \tau_1' \) MB method and a modified MB method, D’ANGELO et al. \[4\] and JONSON et al. \[19\], respectively, determined the viscoelastic constants of the total respiratory system in normal subjects under similar experimental conditions to those of the present study. The values obtained were not significantly different to those presently reported. The present analysis was based on the assumption that the respiratory system behaves as a linear viscoelastic model, which clearly should not be regarded as a complete and perfect representation of respiratory mechanics. More complex nonlinear viscoelastic \[20\] and viscoplastic models \[21\] have been used to explain the volume and time dependency of energy dissipation within the respiratory system. Nevertheless, the linear viscoelastic model has been shown to provide an accurate description of the time dependency of resistance and elastance over the lung volume range used in the present study \[1, 4, 5\]. The present results show that the \( t/\tau_2 \)-dependency of resistance and the elastance of the total respiratory system can be predicted with reasonable accuracy from the viscoelastic constants obtained using the present method.

The short-and-long-breath method is reproducible and requires only two occlusion tests compared to many repeated occlusion manoeuvres, as is the case for the iso-\( \tau_1' \) and iso-\( \tau_2' \) MB methods used previously \[1, 4, 5, 10, 14\]. In the present investigation, a rapid closure valve was used to close the airway. To ascertain the applicability of the proposed method in the clinical arena, the occluding valve of the ventilator was used to perform the end-inspiratory occlusion manoeuvre. Under the same experimental conditions, similar values of \( \tau_2, \tau_3 \) and \( E_2 \) were obtained in normal subjects and patients. As the short-and-long-breath method is applicable under the most common conditions in clinical routine and requires only measurement of \( P_i, P_{fss}, V \) and \( V' \), avoiding complicated mathematical approaches, e.g. exponential analysis of the pressure curve or multiple end-inspiratory occlusions, it can be useful at the bedside, and may prove a powerful tool for the rapid evaluation of the effectiveness of therapeutic manoeuvres, e.g. sigh and removal of secretions by oscillatory ventilation.

Conclusions

The time-dependency of the resistance and elastance of the respiratory system can be explained using a linear viscoelastic model \[1, 2\]. In order to evaluate this dependency, it is necessary to know the values of the parameters in this model \[2–4, 7, 9\]. The clinical utility of the present method lies in the possibility of performing only a few bedside measurements in order to obtain viscoelastic constants. This utility can justify the use of a simplified but perhaps less reliable method.

References


