RESPIRATORY MECHANICS IN ANAESTHESIA

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The present knowledge of respiratory mechanics in anaesthetized humans is astonishingly scanty. This probably reflects the notion that measurement of the mechanics of ventilation during anaesthesia is difficult to perform. In fact, in mechanically ventilated anaesthetized-paralysed humans, a detailed analysis of respiratory mechanics can be performed readily with simple and commonly available equipment, namely a pneumotachograph to measure flow ($\dot{V}$), an integrator to obtain volume changes ($\Delta V$) from the flow signal, and a pressure transducer to measure the pressure at the airway opening (Pao) or, preferably, in the trachea (Ptr) some distance beyond the distal end of the tracheal tube [12, 33]. Several commercial ventilators allow direct measurement of these variables (e.g. Siemens 900C, Puritan-Bennett 7200). With this equipment it is possible to determine, non-invasively, the static and dynamic elastance of the total respiratory system, the flow-resistances of the total respiratory system, airways and thoracic tissues and intrinsic PEEP [11, 34]. By adding an oesophageal balloon catheter system, overall respiratory system mechanics data can be partitioned into lung and chest wall components. In fact, contrary to previous belief, the oesophageal balloon technique is valid in the supine position in both awake [6] and anaesthetized subjects [21].

The present review is not intended to provide a comprehensive account of the literature, but rather to focus on new methodological and conceptual advances which stem from recent studies of the mechanics of ventilation in anaesthetized-paralysed animals [35] and man [11]. Several earlier accounts of respiratory mechanics during anaesthesia [17, 32, 33] deal with aspects not considered in this article because of limitation of space.

KEY WORDS
Lung: mechanics, general anaesthesia.

FORCES INVOLVED IN BREATHING

Breathing movements require work involving several mechanisms: (1) elastic forces; (2) resistive forces resulting from flow of gas through the airways; (3) viscoelastic forces attributable to stress adaptation units within the thoracic tissues (lung and chest wall) [22]; (4) plastoelastic forces within the thoracic tissues which cause "quasistatic hysteresis", as reflected by differences in static elastic recoil pressure of the lung and chest wall between lung inflation and deflation [22]; (5) inertial forces which depend on the mass of gases and tissues; (6) gravitational forces that may be considered as part of inertial forces but in practice are included in the measurement of elastic forces; (7) compressibility of intrathoracic gas; and (8) distortion of the chest wall from its passive (relaxed) configuration [18].

Inertial forces are normally negligible [25], and the same is true for compressibility of gas [23]. Similarly, in normal subjects with relaxed respiratory muscles, the thoracoabdominal configuration during lung inflation and deflation is close to that obtaining under static conditions [1]. Accordingly, in anaesthetized-paralysed subjects the pressure losses resulting from distortion of the chest wall during lung inflation and deflation should be negligible.

Resistance

Theoretical estimation

Based on the above premises, the conventional equation for describing the relationship between the flow-resistance of the total respiratory system (Rrs) and flow at a fixed lung volume is given by [26]:

$$Rrs = Rt + K_1 + K_2 \dot{V}$$

(1)

where Rt is flow-resistance of thoracic tissues, and
$K_1$ and $K_2$ are empirical constants which describe the relationship between airway resistance (Raw) and flow:

$$\text{Raw} = K_1 + K_2 \dot{V} \tag{2}$$

Equation (1) is the basis for one of the tenets of respiratory mechanics, namely that, at a given lung volume, $R_{rs}$ should increase with $\dot{V}$. Another basic tenet is that, at a given flow, $R_{rs}$ should decrease with increasing lung volume because of a decrease in both Raw [8] and $R_t$ [20], the former reflecting airway dilatation while the latter results because the linear velocity of thoracic tissues decreases with increasing lung volume, and hence the flow-dependent pressure losses within the thoracic tissues are reduced.

Equation (1) assumes that the thoracic tissues exhibit ohmic (Newtonian) behaviour. However, recent studies in anaesthetized-paralysed animals [35] and man [11] have shown that this is not the case. Bates, Brown and Kochi [2] demonstrated that a spring-and-dashpot model explains adequately the viscoelastic behaviour of thoracic tissues. In its simplest form, this model consists of two compartments in parallel: a dashpot representing Raw and a Kelvin body (fig. 1). The latter consists of a spring representing the static elastance of the respiratory system ($E_{st,rs}$) in parallel with a Maxwell body—that is, a spring ($E_2$) and a dashpot ($R_2$) arranged serially. $E_2$ and $R_2$ represent viscoelastic properties of the thoracic tissues (lung and chest wall), while $E_{st,rs}$ and Raw are standard elastic and resistive elements. According to this model, $R_t$ measured during constant-flow inflation from relaxed FRC (see below) should increase with the duration of inspiration ($T_i$) according to the following exponential function [11, 35]:

$$R_t = R_2 (1 - e^{-T_i/T_2}) \tag{3}$$

where $T_2$ is the time constant of the viscoelastic component of the thoracic tissues ($T_2 = R_2 / E_2$). This equation implies that $R_t$ is not constant but increases with $T_i$. At the onset of lung inflation, $R_t$ is zero; at $T_i > 3T_2$ it approximates to $R_2$.

During constant-flow inflation there is a fixed relationship between inflation volume ($\Delta V$) and $T_i$:

$$\Delta V = \dot{V} T_i$$

and accordingly:

$$T_i = \Delta V / \dot{V}$$

If the latter is substituted into equation (3), it follows that:

$$R_t = R_2 (1 - e^{-\Delta V / \dot{V} T_2}) \tag{4}$$

This equation implies that, during constant-flow inflation ($\dot{V} = \text{const}$) $R_t$ should increase towards $R_2$ with inflation volume, and that, at fixed inflation volume ($\Delta V = \text{const}$), $R_t$ should decrease progressively with increasing flow.

From equations (1), (3) and (4), it follows that, during constant-flow inflation:

$$R_{rs} = R_2 (1 - e^{-\Delta V / \dot{V} T_2}) + K_1 + K_2 \dot{V} \tag{5}$$

or

$$R_{rs} = R_2 (1 - e^{-\Delta V / \dot{V} T_2}) + K_1 + K_2 \dot{V} \tag{6}$$

The significance of equations (3)–(6) will become apparent below.

**Technique of rapid airway occlusion during constant-flow inflation**

There are four approaches available for measuring flow-resistance: (1) the elastic subtraction method [29]; (2) the interrupter method [29]; (3) the forced oscillation method [15]; and (4) the plethysmographic method [14]. For obvious reasons, the last of these cannot be applied during anaesthesia. In the past, the forced oscillation technique could not be applied to anaesthetized subjects because of technical problems caused by the tracheal tube; however, a possible solution has been proposed recently [28]. The technique of rapid airway occlusion during
constant-flow inflation is essentially a combination of two of the basic approaches for measuring flow-resistance described in 1927 by von Neergaard and Wirz [29]: the interrupter and the elastic subtraction methods. This approach was originally proposed by Rattenborg in 1956 [31]. A virtue of the technique is that flow-resistance can be measured at a fixed inflation flow but different inflation volumes, or at fixed inflation volume but different inflation flows. Furthermore, with this approach the measurements can be carried out with any preselected previous lung volume history.

**Principles of measurement**

Figure 2 illustrates a representative record obtained in an anaesthetized—paralysed human ventilated with a Siemens Servo 900C ventilator. Sudden end-inspiratory airway occlusion during constant-flow inflation resulted in an immediate decrease in Pao from a maximal value (Pmax) to P1. Dividing Pmax — P1 by the constant-flow immediately preceding the occlusion yields the interrupter resistance (Rint). (Other symbols, such as Rinit [35] and Rmin [11] have also been used to describe the interrupter resistance.) Thus:

\[
R_{\text{int}} = \frac{(P_{\text{max}} - P_1)}{V}
\]  

(7)

Its significance has been clarified both in theory [5] and experimentally; essentially, it reflects airway resistance. Indeed, in elegant experiments in open-chested dogs in which alveolar pressure was measured directly using the alveolar capsule technique described by Fredberg and his colleagues [16], Bates' group [4] found that the immediate change in transpulmonary pressure following rapid airway occlusion during expiration was virtually identical to the pre-interruption pressure decrease between trachea and alveolus. Furthermore, in anaesthetized—paralysed man there is no appreciable change in oesophageal pressure immediately following airway occlusion [unpublished observations], indicating that Pmax — P1 does not include pressure dissipations within the tissues of the chest wall. Therefore, in man, Rint represents Raw, as originally proposed by von Neergaard and Wirz [29].

As shown in figure 2, the immediate decrease in Pao after occlusion is followed by a gradual decay in pressure from P1 to an apparent plateau value (P2) that represents the static end-inspiratory elastic recoil pressure of the respiratory system (Pst,rs). This plateau pressure is usually reached in approximately 3 s. Dividing P1 — P2 by the preceding flow gives an additional resistance (ΔRsrs) [11]:

\[
\Delta R_{\text{rs}} = \frac{(P_1 - P_2)}{V}
\]  

(8)

The slow pressure decrease, P1 — P2, reflects two phenomena: "pendelluft", which may be brought out by time constant inequalities within the lung or chest wall, and stress relaxation as a result of viscoelastic properties of the thoracic tissues. In normal humans, however, the time constant inequalities within the lung [30] and chest wall [1] appear to play a negligible role, and hence ΔRsrs should essentially reflect the effective thoracic tissue resistance resulting from the viscoelastic behaviour of the thoracic tissues (ΔRsrs ≈ Rt) [11, 35].

The significance of ΔRsrs may be further interpreted as follows. When the model in figure
1 is elongated at constant speed \( \dot{v} \), the charge of the spring \( E_2 \) increases with \( T_1 \) until, at \( T_1 > \pi \), the speed in the dashpot \( R_2 \) approaches \( v \), and hence the force exerted by the spring \( E_2 \) asymptotes to \( R_2 \cdot v \). If a “flow interrupter” manoeuvre is performed by suddenly halting the relative movement of the two horizontal bars in figure 1, the length of spring \( E_2 \) will decay exponentially to its equilibrium length. In terms of this model, the post-interruption pressure decay \( (P_1 - P_2) \) is thus interpreted as the relaxation of the spring \( E_2 \), resulting in resistive energy dissipation in the dashpot \( R_2 \). The amount of relaxation of tension (stress relaxation) thus depends on the degree of stretch of spring \( E_2 \) at the time of interruption of flow. Clearly, \( \Delta R_{rs} \) is not a pure (Newtonian) resistance, and hence the term effective tissue resistance appears preferable.

Since \( R_{rs} = R_{int} + \Delta R_{rs} \), it follows from equations (7) and (8) that the total effective resistance of the respiratory system is given by:

\[
R_{rs} = (P_{max} - P_2)/\dot{V} \quad (9)
\]

This equation can be viewed as an application of the elastic subtraction principle of von Neergaard and Wirz [29] because \( P_2 \) (which is subtracted from total pressure \( P_{max} \)) is the static end-inspiratory elastic recoil pressure of the total respiratory system.

The technical aspects of the present technique have been discussed in detail elsewhere [3, 11]. Two points are of particular importance, namely the speed of valve occlusion and the effect of the compliance of the upper airways on the occlusion pressure. During anaesthesia the upper airways are bypassed by the tracheal tube, and hence artefacts related to the compliance of these structures are negligible. It should be noted also that determination of \( P_1 \) and \( P_2 \) is best made by curve fitting using a computer [3]. In assessment of \( R_{raw} \) (and hence \( R_{int} \)), the nature of the gas breathed should be taken into account [33].

**Measurements**

Using the above technique, D'Angelo and his colleagues [11] studied the resistive properties of the ventilatory system in 16 anaesthetized-paralysed supine humans (enflurane-pancuronium; 50% nitrous oxide) undergoing ventilation of the lungs via a Siemens Servo 900C ventilator. All inflations started from relaxed FRC, and \( R_{int} \) was corrected for resistance of the tracheal tube, as described by Behrakis and co-workers [7]. Figure 3A depicts the average relationship between \( R_{int} \) and inflation volume during constant-flow inflation \( (\dot{V} = 0.56 \text{ litre s}^{-1}) \) in the 16 subjects. In agreement with previous results on awake humans [8], \( R_{raw} \) (as reflected by \( R_{int} \)) decreased slightly with increasing \( \Delta V \). The relationship between \( R_{int} \) and flow obtained in the 16 subjects at a fixed inflation volume \( (\Delta V = 0.47 \text{ litre}) \) is depicted in figure 4A. \( R_{int} \) increased linearly with flow according to equation (2), the average (SD) values of \( K_1 \) and \( K_2 \) amounting, respectively, to 1.9 (0.5) cm H\(_2\)O litre\(^{-1}\) s and 0.5 (0.1) cm H\(_2\)O litre\(^{-1}\) s\(^2\). These values are close to those observed in normal awake humans breathing through the mouth [26]. There are no previous studies of \( R_{int} \) in anaesthetized man, except for...
constant-flow inflation. The data fit equation (4) in all instances ($P < 0.001$), the average (sd) values of the coefficients $R_z$ and $\tau_z$ amounting to 4.6 (0.8) cm H$_2$O litre$^{-1}$ s and 1.0 (0.3) s. The increase in $\Delta R_{rs}$ with inflation volume was greater than the concomitant reduction in $R_{int}$ (fig. 3); as a result, $R_{rs}$ increased markedly with inflation volume. Thus, contrary to previous belief, $R_{rs}$ does not decrease with increasing lung volume, but increases in line with our model predictions. It should be noted that equation (4) predicts that the volume-related increase in $\Delta R_{rs}$ depends on the rate of constant-flow inflation: for any given $\Delta V$, an increase in $V$ will reduce the magnitude of $\Delta R_{rs}$ because that volume will be reached with a shorter $T_i$ (equation (3)). This is shown in figure 4B, which illustrates the relationship of $\Delta R_{rs}$ to flow at a fixed inflation volume: $\Delta R_{rs}$ decreases with increasing flow according to equation (4) ($P < 0.001$). This is in contrast to equation (1), which indicates that $R_t$ (and hence $\Delta R_{rs}$) should have a fixed value. The decrease in $\Delta R_{rs}$ with increasing $V$ is greater than the concomitant increase in $R_{int}$, and consequently $R_{rs}$ decreases markedly with increasing flow (fig. 4). The values of the constants $R_z$ and $\tau_z$ in these isovolume experiments were similar to those found in the isoflow studies.

Don and Robson [12] made similar measurements of $R_{rs}$ in anaesthetized-paralysed man (thiopentone; 75% nitrous oxide). Under normocapnic conditions, at constant inflation flow of 1 litre s$^{-1}$ and $\Delta V$ of 0.7-1.0 litre, they found $R_{rs}$ values of 3.4 (1.4) cm H$_2$O litre$^{-1}$ s. These are within the range of those found in the present study at corresponding flow (fig. 4A), but at a lower inflation volume (0.47 litre). Figures 3 and 4 show that $R_{rs}$ varies markedly according to the experimental conditions. Unless $V$ and $\Delta V$ are standardized, comparisons of $R_{rs}$ appear to be meaningless. In contrast, the results of D'Angelo and colleagues [11] show that it is possible to describe adequately the resistive properties of the respiratory system in terms of $R_{int}$, which represents airway resistance, and the constants $R_z$ and $\tau_z$ which characterize the viscoelastic properties of the thoracic tissues. By measuring oesophageal pressure, it is possible to partition thoracic tissue resistance into lung and chest wall components. Preliminary results indicate that the lung tissues account for about 60% of $\Delta R_{rs}$ [D'Angelo and colleagues, unpublished observations].

Fig. 4. A: Average relationship of $R_{rs}$ and $R_{int}$ with inflation flow obtained at constant inflation volume (0.47 litre) in same subjects as figure 3. Bars: 1 SD. $R_{rs}$ was obtained by adding $\Delta R_{rs}$ (computed according to equation (4)) to $R_{int}$. B: Similar relationship in terms of $\Delta R_{rs}$. Curves computed according to equation (4). (Reproduced with permission from D'Angelo and colleagues [11].)
Respiratory Mechanics in Anaesthesia

Elastance

Theoretical estimation

The viscoelastic properties of the thoracic tissues not only contribute to $R_{rs}$, but also affect the elastance of the respiratory system. In fact, since $P_1-P_2$ may be interpreted as a charge on the spring $E_2$ (fig. 1), it follows that the effective elastance of the viscoelastic units within the thorax ($E_t$) is given by:

$$E_t = (P_1 - P_2)/A\Delta V$$

Based on the model in figure 1, during constant-flow inflation from relaxed FRC, $E_t$ should decrease with inspiratory time according to the following function:

$$E_t = (P_1 - P_2)/A\Delta V = R_s(1-e^{-7/t_i})/T_i$$

This equation shows that $E_t$ is time-dependent, as is $R_t$ (equation (3)). In contrast to $R_t$, however, $E_t$ decreases progressively during lung inflation: at onset of inflation $E_t$ equals $E_2$, while at $T_i = 3e_2$, $E_t$ equals 0.22 $E_2$.

During constant-flow inflation $T_i = A\Delta V/\dot{V}$, equation (11) can be transformed into:

$$E_t = R_s(1-e^{-A\Delta V/\dot{V}})\dot{V}/A\Delta V$$

This equation shows that, at fixed inflation flow, $E_t$ should decrease progressively with increasing inflation volume. By contrast, at fixed inflation volume, $E_t$ should increase progressively with increasing flow, reflecting the shorter $T_i$ needed to reach that volume. $T_i$-dependence of $E_t$ (equation (11)) implies $T_i$-dependence of $E_{rs}$. In fact, since standard elastance ($E_{rs},E_t$) and $E_2$ are arranged in parallel (fig. 1), the effective or dynamic elastance is the sum of $E_{rs},E_t$ and $E_t$. Time-dependence of $E_t$, and hence of $E_{rs}$, implies frequency-dependence of these variables. Otis and colleagues [30] have shown that frequency-dependence of $E_{rs}$ can be brought about by time-constant inequalities within the lung and (if any) chest wall. In normal humans, however, such inequalities are probably too small to play any significant role [30]. By contrast, the viscoelastic properties of the thoracic tissues play a significant role in determining dynamic $E_{rs}$, as shown below.

When occlusion at end-inflation is maintained until a plateau in $P_{ao}$ is achieved (fig. 2), all elastic charge stored in the spring $E_2$ results in resistive energy dissipation in the dashpot $E_2$ (fig. 1). No external work is done. In contrast, if expiration does not entail an end-inspiratory hold, some of the stored elastic energy will be available for expiration, resulting in increased expiratory flow. In other words, the rate of lung emptying during passive lung deflation is greater the shorter the end-inspiratory hold, as described by Mortola, Magnante and Saetta [27].

Principles of measurement

$E_{rs}$ is obtained by dividing the difference between end-inspiratory and end-expiratory $P_{st,rs}$ by $A\Delta V$. The end-expiratory $P_{st,rs}$ is commonly defined as intrinsic PEEP (PEEPi) [34]. Thus:

$$E_{rs} = (\text{end-inspiratory } P_{st,rs} - \text{PEEPi})/A\Delta V$$

In anaesthetized-paralysed subjects with normal lungs, PEEPi is usually 0, provided that: (1) the expiratory duration is 3 s or more; (2) the size of the tracheal tube is not very small; and (3) the expiratory impedance offered by the ventilator is not too large. In patients with severe airway obstruction, PEEPi is almost invariably present [9]. If present, PEEPi has to be taken into account for correct measurement of $E_{rs}$. Dynamic $E_{rs}$ ($Edyn,rs$) is obtained by dividing the difference in $P_{ao}$ measured at points of zero flow by $A\Delta V$. In the subject in figure 2, who had no PEEPi, $Edyn,rs$ is given by:

$$Edyn,rs = P_1/A\Delta V$$

where $P_1$ is $P_{ao}$ measured at the point where inspiratory flow becomes 0.

The difference between $Edyn,rs$ and $E_{rs}$ represents the tissue viscoelastic component of elastance, as defined by equation (10).

The technique of rapid airway occlusion during constant-flow inflation allows not only the measurement of static and dynamic $E_{rs}$ for a given volume change (equations (13) and (14)), but also determination of the static volume-pressure relationship of the total respiratory system, and definition of the function relating $E_t$ to inspiratory duration (equation (11)) or to inflation flow and volume (equation (12)). Using this technique it is also possible to determine the "quasi-static hysteresis" of the system without artefacts attributable to continuing gas exchange and shift of blood from the thorax [24] which are present when using the "super-syringe" method [10].
Measurements

The effects of anaesthesia on static elastance of the total respiratory system, lung and chest wall have been extensively studied. The results have been discussed in review articles [32, 33]. In man, £st,rs is usually found to increase after induction of anaesthesia; no further change occurs when either depth of anaesthesia is increased or muscle paralysis is added. The increase in £st,rs is caused primarily by an increase in static pulmonary elastance, probably reflecting an alteration in lung surfactant function. Dynamic £rs has also been measured during anaesthesia. The results, however, are difficult to interpret because £dyn,rs depends on the experimental conditions [11]. In 16 anaesthetized-paralysed subjects, £st,rs and £dyn,rs were measured at fixed inflation volume ($\Delta V = 0.47$ litre) but at different inflation flows. £st,rs was greater than normal but did not change with flow (fig. 5) [11]. In contrast, £dyn,rs increased with increasing flow, as predicted by equation (12). D’Angelo and colleagues also made similar measurements at fixed inflation flow ($0.56$ litre s$^{-1}$), but at different inflation volumes. In agreement with equation (12), under these conditions £dyn,rs decreased with increasing volume. It should be stressed that the apparent volume and flow-dependence of £dyn,rs merely reflects time-dependent behaviour of the viscoelastic compartment, as indicated by equation (11). The same is true in terms of the effective resistance offered by the thoracic tissues (equation (3)).

The above results indicate that £dyn,rs varies markedly with the experimental conditions. Unless $V$ and $\Delta V$ are standardized, interpretation of £dyn,rs is difficult. In contrast, £st,rs together with the constants $E_2$ and $\tau_2$ provide a full characterization of the elastic behaviour of the respiratory system. In the 16 subjects, the average (SD) values of these three variables were $14.5 (2.1)$ cm H$_2$O litre$^{-1}$, $4.5 (0.9)$ cm H$_2$O litre$^{-1}$ and $1.0 (0.3)$ s, respectively.

Critique

The four-element model in figure 1 is not intended to be a complete and perfect representation of respiratory mechanics. Rather, it is what we consider to be a useful representation of the behaviour of the normal respiratory system during flow interruption. Obviously, one could invoke more viscoelastic elements in series and parallel, with a commensurate increase in the number of model parameters, in order to describe more accurately a particular set of data. However, D’Angelo and colleagues’ results [11] closely fit the model predictions, indicating that such an analysis may be adequate. The same was true for experiments in anaesthetized-paralysed dogs [35]. Our model is superior to those described previously. For example, equation (1) predicts that $R_{rs}$ should increase with increasing flow, but this clearly is not the case (fig. 4). Our model predicts a non-linear relationship which is similar to observed data.

Our model contains no inertive elements such as are required to account for the behaviour of the respiratory system when very high frequency pressure oscillations are applied at the airway opening [13]. This is because the only evidence of inertia that can be detected during flow interruption is rapid and highly damped oscillation in $P_{ao}$ immediately after interruption. These oscillations invariably subside within 50 ms and can be discounted by back-extrapolation of the subsequent pressure signal.

Since, during lung inflation it is actually the charge on spring $E_t$ (fig. 1) that increases the impedance of the respiratory system, it could be argued that analysis should be limited to dynamic £rs and that assessment of effective tissue resistance ($R_t$) is irrelevant. In theory, this argument has merit. In practice, however, all
measurements of chest wall resistance, pulmonary flow resistance and \( R_{\text{rs}} \) axiomatically include a variable component reflecting viscoelastic behaviour of the tissues of the chest wall, lung, or both. Therefore, analysis in terms of \( R_t \) seems to be justified. It should be stressed, however, that assessment of \( R_t \) represents an "as if" analysis: it characterizes respiratory impedance "as if" the viscoelastic compartment behaved as a pure resistance. As if analyses are common in respiratory mechanics, as exemplified by the classic paper of Otis and colleagues [30] on the frequency-dependence of pulmonary compliance and resistance.

CONCLUSION

The technique of rapid airway occlusion during constant-flow inflation allows non-invasive determination of airway resistance, static compliance of the respiratory system, and thoracic tissue impedance. The effects of anaesthetic agents and of various drugs used during anaesthesia can thus be quantified in terms of both the standard mechanical properties of the respiratory system (\( R_{\text{aw}} \) and \( E_{\text{aw}} \)) and the impedance of the thoracic tissues. Determination of \( R_{\text{aw}}, E_{\text{aw}} \), and \( \tau_2 \) opens a new field for research. What are the effects of different anaesthetics on the viscoelastic properties of the thoracic tissues? What are the effects of age, lung disease, etc? These questions can now be answered quantitatively.

The technique of rapid airway occlusion during constant-flow inflation was first used by anaesthetists [12, 31]. Now they must apply it systematically in order to gain better insight to the effects of anaesthesia on respiratory mechanics.

REFERENCES


