Respiratory mechanics in anaesthesia

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RESPIRATORY MECHANICS IN ANAESTHESIA

J. MILIC-EMILI, F. M. ROBATTO AND J. H. T. BATES

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 (ΔV) from the flow signal, and a pressure transducer to measure the pressure at the airway opening (Pao) or, preferably, in the tracheà (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 "quasi-static 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]:

$$R\text{rs} = R\text{t} + K_1 + K_2\dot{V} \qquad (1)$$

where Rt is flow-resistance of thoracic tissues, and

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K_1 and K_2 are empirical constants which describe the relationship between airway resistance (*R*aw) and flow:

$$Raw = K_1 + K_2\dot{V} \quad (2)$$

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 *R*aw [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 *R*aw 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 *R*aw 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]:

$$Rt = R_2(1 - e^{-T_I/\tau_2}) \quad (3)$$

where τ_2 is the time constant of the viscoelastic component of the thoracic tissues ($\tau_2 = R_2/E_2$). 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 $T_I > 3\tau_2$ it approximates to R_2.

During constant-flow inflation there is a fixed relationship between inflation volume (ΔV) 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:

$$Rt = R_2(1 - e^{-\Delta V/\dot{V}\tau_2}) \quad (4)$$

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

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

$$Rrs = R_2(1 - e^{-T_I/\tau_2}) + K_1 + K_2\dot{V} \quad (5)$$

or

$$Rrs = R_2(1 - e^{-\Delta V/\dot{V}\tau_2}) + K_1 + K_2\dot{V} \quad (6)$$

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

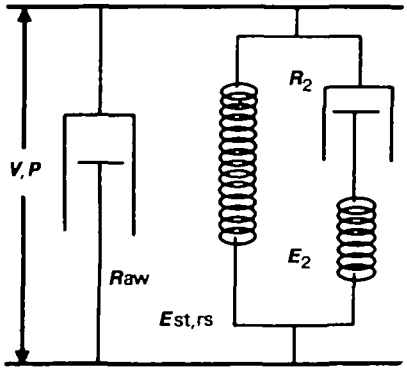

FIG. 1. Scheme of spring-and-dashpot model for interpretation of respiratory mechanics during flow interruption. The respiratory system consists of standard airway resistance (*R*aw) in parallel with a standard static elastance (*E*st,rs), and a series spring-and-dashpot body (E_2 and R_2, respectively) which represents the stress adaptation units. The distance between the two horizontal bars is the analogue of lung volume (V) and the tension between these bars is the analogue of pressure at the airway opening (P).

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 *P*ao from a maximal value (*P*max) to P_1. Dividing $P\text{max} - P_1$ by the constant-flow immediately preceding the occlusion yields the interrupter resistance (*R*int). (Other symbols, such as *R*init [35] and *R*min [11] have also been used to describe the interrupter resistance.) Thus:

$$R\text{int} = (P\text{max} - P_1)/\dot{V} \qquad (7)$$

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 $P\text{max} - P_1$ does not include pressure dissipations within the tissues of the chest wall. Therefore, in man, *R*int represents *R*aw, as originally proposed by von Neergaard and Wirz [29].

As shown in figure 2, the immediate decrease in *P*ao after occlusion is followed by a gradual decay in pressure from P_1 to an apparent plateau value (P_2) that represents the static end-inspiratory elastic recoil pressure of the respiratory system (*P*st,rs). This plateau pressure is usually reached

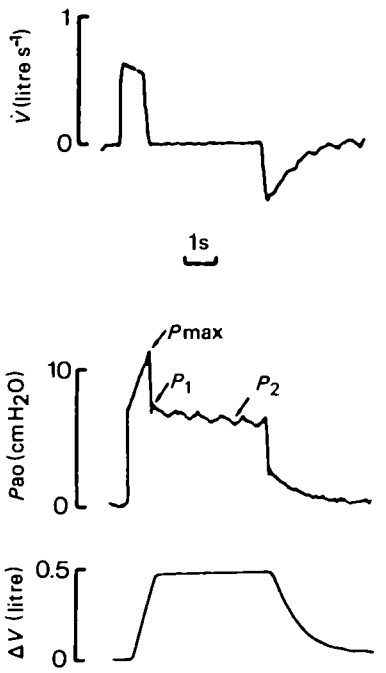

FIG. 2. Records of flow ($\dot{V}$), pressure at the airway opening (*P*ao), and changes in lung volume (ΔV) from an anaesthetized–paralysed subject which illustrate the technique of rapid airway occlusion during constant-flow inflation. After end-inspiratory airway occlusion there is an immediate decrease in pressure from *P*max to P_1, followed by a slow decay to a plateau value (P_2) that represents static elastic recoil pressure at end-inspiratory lung volume. The decrease in pressure from *P*max to P_1 includes resistive pressure attributable to the tracheal tube. For further explanations see text.

in approximately 3 s. Dividing $P_1 - P_2$ by the preceding flow gives an additional resistance (ΔRrs) [11]:

$$\Delta R\text{rs} = (P_1 - P_2)/\dot{V} \qquad (8)$$

The slow pressure decrease, $P_1 - P_2$, 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 ΔRrs should essentially reflect the effective thoracic tissue resistance resulting from the viscoelastic behaviour of the thoracic tissues ($\Delta R\text{rs} \simeq R\text{t}$) [11, 35].

The significance of ΔRrs may be further interpreted as follows. When the model in figure

1 is elongated at constant speed (v), the charge of the spring E_2 increases with T_I until, at $T_I > 3\tau_2$, 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, ΔRrs is not a pure (Newtonian) resistance, and hence the term effective tissue resistance appears preferable.

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

$$R\text{rs} = (P\text{max} - P_2)/\dot{V} \qquad (9)$$

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 Pmax) 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 Raw (and hence Rint), 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 Rint was corrected for resistance of the tracheal tube, as described by Behrakis and co-workers [7]. Figure 3A depicts the average relationship between Rint and inflation volume during constant-flow inflation ($\dot{V} = 0.56$ litre s^{-1}) in the 16 subjects. In agreement with previous results on awake humans [8], Raw (as reflected by Rint) decreased slightly with increasing ΔV. The relationship between Rint and flow obtained in the 16 subjects at a fixed inflation volume ($\Delta V =$ 0.47 litre) is depicted in figure 4A. Rint 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_2O litre^{-1} s and 0.5 (0.1) cm H_2O litre^{-2} s^2. These values are close to those observed in normal awake humans breathing through the mouth [26]. There are no previous studies of Rint in anaesthetized man, except for

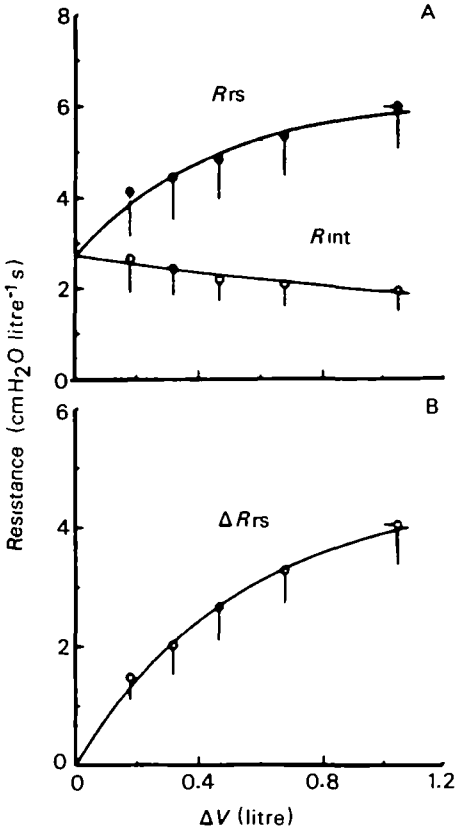

FIG. 3. A: Average relationship of Rrs and Rint with volume obtained at constant inflation flow (0.56 litre s^{-1}) in 16 anaesthetized–paralysed subjects. Bars: 1 SD. Rrs was obtained by adding ΔRrs (computed according to equation (4)) to Rint. B: Similar relationship in terms of ΔRrs ($\simeq R$t). Curves computed according to equation (4). (Reproduced with permission, from D'Angelo and colleagues [11].)

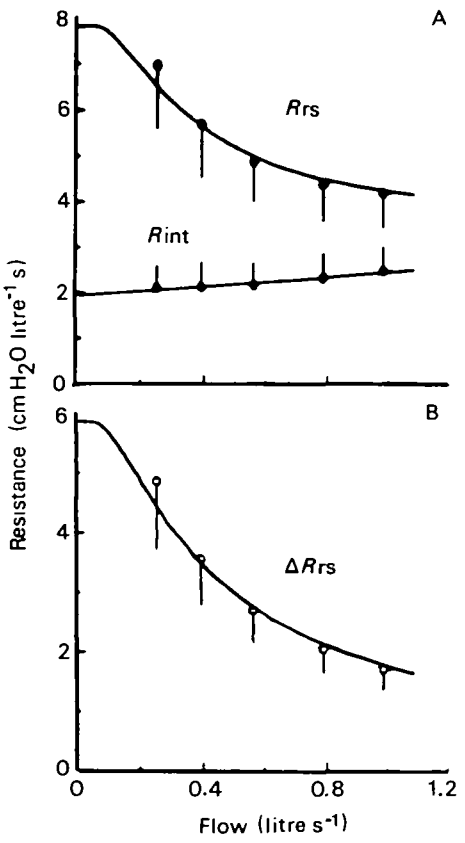

FIG. 4. A: Average relationship of *R*rs and *R*int with inflation flow obtained at constant inflation volume (0.47 litre) in same subjects as figure 3. Bars: 1 SD. *R*rs was obtained by adding Δ*R*rs (computed according to equation (4)) to *R*int. B: Similar relationship in terms of Δ*R*rs. Curves computed according to equation (4). (Reproduced with permission from D'Angelo and colleagues [11].)

that of Gottfried and colleagues [19], who made measurements during passive lung deflation in six anaesthetized subjects (halothane–nitrous oxide) both before and after paralysis. In this study, *R*int was essentially constant during most of lung deflation, amounting to 2.0 (0.8) cm H_2O litre^{-1} s during anaesthesia and 1.9 (0.6) cm H_2O litre^{-1} s during anaesthesia–paralysis. These values are similar to those in figure 3. It should be noted that, during lung deflation, volume (fig. 3A) and flow (fig. 4A) dependence of airway resistance tend to cancel out [36], and this may explain the constant values of *R*int found by Gottfried's group [19].

Figure 3B depicts the average relationship between Δ*R*rs and inflation volume during constant-flow inflation. The data fit equation (4) in all instances ($P < 0.001$), the average (SD) values of the coefficients R_2 and τ_2 amounting to 4.6 (0.8) cm H_2O litre^{-1} s and 1.0 (0.3) s. The increase in Δ*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 Δ*R*rs depends on the rate of constant-flow inflation: for any given ΔV, an increase in $\dot{V}$ will reduce the magnitude of Δ*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 Δ*R*rs to flow at a fixed inflation volume: Δ*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 Δ*R*rs) should have a fixed value. The decrease in Δ*R*rs with increasing $\dot{V}$ 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_2 and τ_2 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 ΔV of 0.7–1.0 litre, they found *R*rs values of 3.4 (1.4) cm H_2O 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 ΔV 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_2 and τ_2 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 Δ*R*rs [D'Angelo and colleagues, unpublished observations].

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:

$$Et = (P_1 - P_2)/\Delta V \qquad (10)$$

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:

$$Et = (P_1 - P_2)/\Delta V = R_2(1 - e^{-TI/\tau_2})/TI \qquad (11)$$

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 $TI = 3\tau_2$, *E*t equals $0.22\,E_2$.

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

$$Et = R_2(1 - e^{-\Delta V/\dot{V}\tau_2})\dot{V}/\Delta V \qquad (12)$$

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*st,rs) and E_2 are arranged in parallel (fig. 1), the effective or *dynamic* elastance is the sum of *E*st,rs 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*st,rs is obtained by dividing the difference between end-inspiratory and end-expiratory *P*st,rs by ΔV. The end-expiratory *P*st,rs is commonly defined as intrinsic PEEP (PEEPi) [34]. Thus:

$$Est,rs = (\text{end-inspiratory } Pst,rs - PEEPi)/\Delta V \qquad (13)$$

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*st,rs.

Dynamic *E*rs (*E*dyn,rs) is obtained by dividing the difference in *P*ao measured at points of zero flow by ΔV. In the subject in figure 2, who had no PEEPi, *E*dyn,rs is given by:

$$Edyn,rs = P_1/\Delta V \qquad (14)$$

where P_1 is *P*ao measured at the point where inspiratory flow becomes 0.

The difference between *E*dyn,rs and *E*st,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, *E*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 *E*st,rs is caused primarily by an increase in static pulmonary elastance, probably reflecting an alteration in lung surfactant function. Dynamic *E*rs has also been measured during anaesthesia. The results, however, are difficult to interpret because *E*dyn,rs depends on the experimental conditions [11]. In 16 anaesthetized–paralysed subjects, *E*st,rs and *E*dyn,rs were measured at fixed inflation volume ($\Delta V = 0.47$ litre) but at different inflation flows. *E*st,rs was greater than normal but did not change with flow (fig. 5) [11]. In contrast, *E*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 *E*dyn,rs decreased with increasing volume. It should be stressed that the apparent volume and flow-dependence of *R*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)).

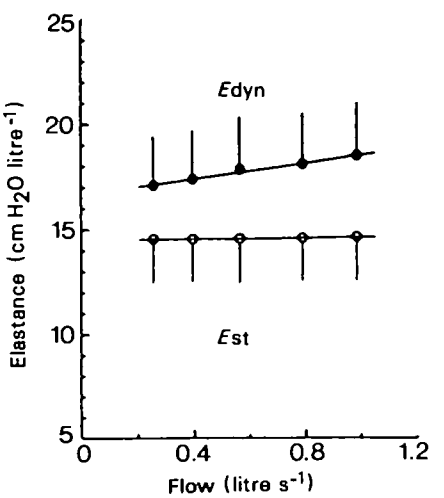

FIG. 5. Average relationship of static and dynamic elastance of the total respiratory system with flow obtained at constant inflation volume in same subjects as figure 4. Bars: 1 SD. (Reproduced with permission, from D'Angelo and colleagues [11].)

The above results indicate that *E*dyn,rs varies markedly with the experimental conditions. Unless $\dot{V}$ and ΔV are standardized, interpretation of *E*dyn,rs is difficult. In contrast, *E*st,rs together with the constants E_2 and τ_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_2O $litre^{-1}$, 4.5 (0.9) cm H_2O $litre^{-1}$ 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_2 (fig. 1) that increases the impedance of the respiratory system, it could be argued that analysis should be limited to dynamic *E*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*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*aw and *E*st,rs) and the impedance of the thoracic tissues. Determination of R_2, E_2 and τ_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.

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