Eur Respir J
1991, 4, 458–464

A single-compartment model cannot describe passive expiration in intubated, paralysed humans

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ABSTRACT: The time-course of thoracic volume changes (respiratory inductive plethysmograph) during relaxed expiration was studied in 11 intubated, paralysed, mechanically ventilated subjects. The semilog volume-time curves show that expiration is governed by two apparently separate mechanisms: one causes emptying of most of the expired volume (~80%) with a time constant of 0.50±0.22 s for a baseline tidal volume of 0.44±0.12 *l* (mean±SD) and 0.37±0.14 s when the tidal volume is reduced (V_{TP}); the other contributes a relatively small amount to the expired volume over a significantly longer time, the time constant amounting to 3.27±1.54 s for baseline V_T and 2.95±1.65 s for V_{TP}. The first mechanism probably reflects the standard elastic and flow resistive properties of the respiratory system, while the second, slower compartment, is probably an expression of the viscoelastic properties of the pulmonary and chest wall tissues.
Eur Respir J., 1991, 4, 458–464.

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Keywords: Mechanical ventilation; static compliance of the respiratory system; viscoelasticity.

Received: November 30, 1989; accepted after revision November 7, 1990.

G.L.C. was supported by a grant from the National Council for Scientific Research, Italy.

During passive expiration, the time-course of the fall in thoracic gas volume is determined by the mechanical properties of the respiratory system. The driving pressure is then provided entirely by the elastic recoil pressure of the total respiratory system, which is used to overcome the total expiratory flow resistance. Theoretical calculations, first proposed by BRODY [1] in 1954 for a single-compartment model, indicate that if the compliance (Crs) and resistance (Rrs) of the respiratory system were fixed, the time-course of volume during passive expiration should follow a single exponential function:

$$V(t) = Vo \cdot e^{-t/\tau rs} \quad (1)$$

where V is the volume of gas exhaled at any time t, Vo is the initial volume above the relaxation volume of the respiratory system, and τrs is time constant of the respiratory system, equal to the product of resistance and compliance (τrs = Rrs·Crs).

BATES *et al.* [2] have recently shown that in anaesthetized-paralysed dogs the time-course of volume during passive expiration can better be described in terms of a double-exponential function:

$$V(t) = A \cdot e^{-t/\tau' rs} + B \cdot e^{-t/\tau'' rs} \quad (2)$$

the relatively rapid first component reflecting pure resistive behaviour, while the second slower component is probably mainly due to the viscoelastic properties of the pulmonary and chest wall tissues. In previous studies on humans, no mention has been made of a slow component during relaxed expiration [3–7].

The aim of this study was to determine which of the two models (single- or bi-exponential) better describes the volume-time profile during passive expiration in mechanically ventilated, paralysed humans.

Subjects and methods

The studies were carried out on eleven comatose subjects, mean age (±SD) 30±10 yrs, who had been admitted into the medical intensive care unit for self-poisoning. All studies were made within 24 h after admission. Their physical characteristics and nature of poisoning are listed in table 1. None had a history of lung disease. All had normal chest radiography and the arterial oxygen tension/fractional inspiratory oxygen ratio (Pao_2/Fio_2) was greater than 300, indicating normal gas exchange within the lung [8], and completely recovered within two days of the study without evidence of residual lung damage. The investigation was approved by the institutional Ethics Committee, and informed consent was given by the next of kin.

Table 1. – Anthropometric data, clinical findings and drugs ingested by subjects

Subject no.	Sex	Age yrs	Weight kg	Height m	Pao_2 kPa	Fio_2	ID mm	Drugs
1	F	26	55.6	1.60	29.9	0.50	7.5	BZ + AL
2	F	36	53.5	1.60	28.7	0.50	7.0	BZ + AT
3	F	49	58.5	1.50	25.4	0.50	8.0	BZ + AT
4	F	35	54.5	1.65	24.5	0.45	7.5	BZ
5	F	22	54.6	1.62	43.2	0.60	7.5	BZ + BA
6	F	39	54.8	1.60	25.4	0.35	7.0	BZ + BA
7	F	20	50.8	1.62	27.7	0.30	7.5	BZ + AT
8	F	24	59.0	1.73	29.5	0.40	8.0	BZ + AT
9	F	43	56.6	1.75	24.3	0.60	7.5	BZ + AL
10	F	20	58.7	1.68	28.6	0.50	7.5	BZ
11	M	21	65.0	1.80	41.1	0.50	7.5	BZ + OP

BZ: benzodiazepines; BA: barbiturates; AL: alcohol; AT: antidepressant; OP: opiates; ID: inner diameter of endotracheal tube; Pao_2: arterial oxygen tension; Fio_2: fractional inspiratory oxygen.

The subjects were intubated, paralysed and mechanically ventilated with a gas mixture with Fio_2 range 0.35–0.6 (table 1). They were curarized to make sure that all respiratory muscle activity was abolished. After adequate sedation, if needed, with flunitrazepam (Narcozep®) the subjects were paralysed with 4 mg of vecuronium bromide (Norcuron®) followed by aliquots of 2 mg every 10 min thereafter. Respiratory muscle relaxation was evidenced by the fact that there was no breath-by-breath variation in the airway pressure waveform, tidal volume and end-expiratory level. The subjects all exhibited a plateau in airway pressure after end-inspiratory airway occlusion.

Volume changes were measured by respiratory inductive plethysmography (RIP) placed around the rib cage. Since our subjects were paralysed, we considered that the respiratory system behaved to a good degree of approximation with a single degree of freedom [9, 10] and, hence, a single RIP coil (with a direct current (DC) circuit) was used [11]. The coil was fixed on the skin of the abdomen at mid-distance from the iliac crest and the axilla. The DC mode was used in order to monitor the end-expiratory level. Since in DC mode the oscillator is sensitive to temperature [12], we waited for 30 min before taking any measurements to allow for thermal equilibrium. Non-cumulative calibration was performed by incremental inflation with a hand-driven 2 *l* syringe, as described previously [10, 13]. Airway pressure (Paw) was measured at the proximal end of the endotracheal tube with a differential pressure transducer (Validyne MP15±60 cmH_2O, Northridge, CA) with reference to barometric pressure. Paw and RIP outputs were recorded as a function of time on a two-pen (2 YT Sefram, Valizy, France) potentiometric recorder.

All subjects were intubated with Portex cuffed endotracheal tubes ranging in internal diameter from 7–8 mm. A two-way stopcock was used to occlude the airways at end-inflation. During passive deflation the subjects were disconnected from the ventilator and expired at barometric pressure (so excluding any interference of the equipment resistance, except for the endotracheal tube (ET). The inflation volume was initially set at 10 $ml \cdot kg^{-1}$ but was subsequently adjusted to keep arterial blood gases within normal limits. As a result, the baseline tidal volume ranged from 5 to12 $ml \cdot kg^{-1}$. The respiratory frequency ranged between 15–18 $cycles \cdot min^{-1}$, and the end-expiratory ventilator pressure was always zero (ZEEP).

Procedure

After a period of stable mechanical ventilation, end-inspiratory airway occlusions were performed with the manual tap. During the ensuing period of apnoea (5–6 s), relaxation of the respiratory muscles was shown by the rapid appearance of a plateau on the tracheal pressure tracing. The occlusion was then rapidly released, and the subjects allowed to expire freely at barometric pressure until full expiration was achieved, *i.e.* until the RIP signal was both steady for at least 1.5 s and indistinguishable from baseline. Although opening the airway to atmospheric pressure took a short but finite time, we did not measure it. Its effects, however, on the early changes in volume with time should have been small. End-inspiratory occlusions were performed at baseline tidal volume (V_T) and at a lesser degree of lung inflation. Our aim was to use an inflation volume of about one-half of baseline V_T. However, it turned out, retrospectively, that the inflation achieved was not always precisely $V_T/2$ (table 2). Henceforth, this lower inflation volume will be labelled V_{Tp}, where p stands for partial inflation. Two end-inspiratory occlusions were performed at each V_T setting. Each occlusion was preceded by ten regular mechanical inflations with baseline V_T. Prior to each study the lungs were inflated with three cumulative baseline V_Ts by occluding the expiratory line of the ventilator for three breaths in order to produce a constant previous lung volume history.

Table 2. – Inflation volume (V_T), static elastic recoil pressure (Pst,rs) and compliance (Cst,rs) of the respiratory system

Deflation from V_T				Deflation from V_{TP}			
Subject no.	V_T ml	Pst,rs kPa	Cst,rs $l \cdot kPa^{-1}$	Subject no.	V_{TP} ml	Pst,rs kPa	Cst,rs $l \cdot kPa^{-1}$
1	400	0.7	0.56	1	202	0.4	0.55
2	545	0.8	0.67	2	340	0.6	0.57
3	285	0.7	0.39	3	202	0.6	0.33
4	374	1.1	0.32	4	255	0.9	0.29
5	505	0.9	0.57	5	218	0.5	0.44
6	702	0.9	0.79	6	472	0.7	0.69
7	385	0.8	0.48	7	222	0.6	0.36
8	288	0.8	0.36	8	133	0.4	0.29
9	550	0.8	0.70	9	282	0.4	0.64
10	416	0.7	0.58	10	187	0.4	0.48
11	400	0.7	0.57	11	230	0.5	0.47
Mean	441	0.8	0.54	Mean	249	0.5	0.46
SD	124	0.1	0.15	SD	91	0.1	0.14

Each individual point is the mean of two determinations, which did not differ significantly (Student's paired t-test). V_T: tidal volume; V_{TP}: partial tidal volume.

Data analysis

The static compliance of the total respiratory system (Cst,rs) was computed by dividing the volume expired to atmospheric pressure by the plateau in Paw (representative of static elastic recoil pressure of the total respiratory system, Pst,rs) obtained during the preceding end-inspiratory airway occlusion. Individual curves (two curves for each V_T setting) relating RIP volume and time were digitized manually at intervals of 0.5 s (2 Hz) until end-expiration and the last three points were deleted.

In order to assess whether our results best fitted Eq. 1 or 2, the experimental data were analysed in terms of "goodness of fit". This was performed by parameter estimation using an optimized computer algorithm based on a Gauss-Newton procedure using the least-squares methods (Ph. D'Atis., Programme Triomphe, Laboratoire d'Informatique Médicale, Faculté de Médecine, CHU Dijon, 211000, France).

Since, in many cases, the fast exponential had to be evaluated on few points, we fitted exponentials simultaneously using the two digitized curves, instead of their mean values (on the basis of their almost perfect overlapping), thus in practice duplicating the number of points. Moreover, fittings performed on a few volume-time curves digitized at 10 Hz gave the same parameter values, supporting our analytical procedure.

Statistical analysis

The single exponential model was compared with the bi-exponential one by analysis of variance based on the least-squares sum calculated from each curve fitting: the bi-exponential model was preferred to the single exponential one when the Fisher test indicated a significant result ($p<0.05$) [14].

Decay slopes (τ' and τ'') obtained from the V_T and V_{TP} experiments were compared by an analysis of variance (ANOVA) taking into account the matched data. For comparison of the data, the measured values were expressed as means ±SD. Statistical analysis was performed using Student's t-test for paired data (significance was accepted at $p<0.05$). Regression analysis was done with the least-squares method.

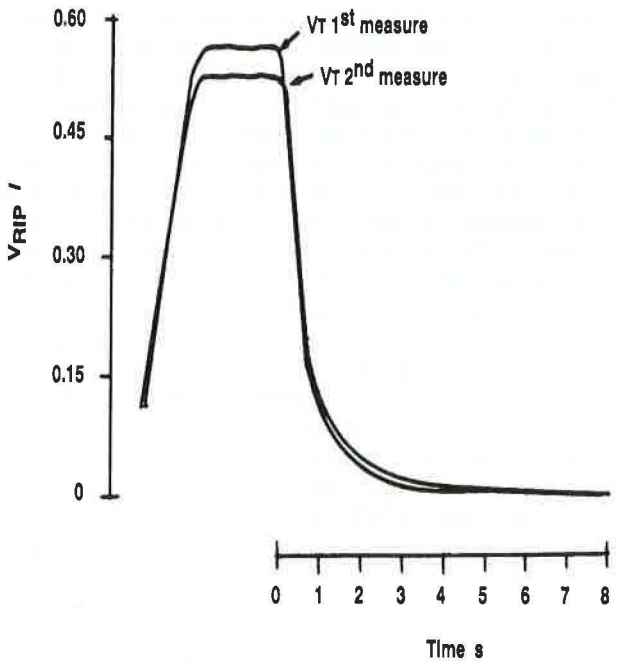

Fig. 1. – Illustrative example of changes in thoracic volume measured by respiratory inductance plethysmograph as a function of time. Duplicate curves 1 and 2 are identified. In this example, passive deflation starts from baseline inflation volume (V_T) measured at plateau of volume (RIP)-time curves. RIP: respiratory inductive plethysmograph.

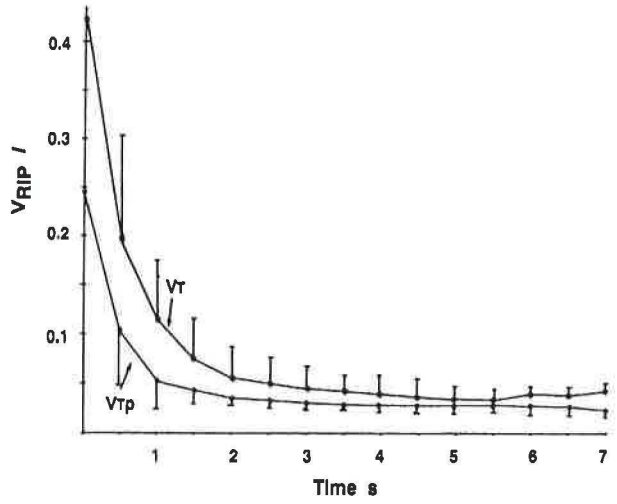

Fig. 2. – Average (±SD) volume-time profile during passive expiration from baseline tidal volume (V_T) and partial tidal volume (V_{Tp}) of 11 subjects. Volume was measured with a respiratory inductive plethysmograph (V_{RIP}).

Results

Table 2 provides the values of Pst,rs and Cst,rs obtained with baseline V_T and V_{Tp} in the 11 subjects. Static compliance was significantly lower at V_{Tp} ($p<0.001$). The shape of the volume-time curve (fig. 1) obtained in each subject was highly reproducible with both V_T and V_{Tp}. Pst,rs was 0.8±0.1 kPa and 0.8±0.1 kPa on duplicate determinations at V_T and 0.5±0.1 kPa and 0.5±0.1 kPa at V_{Tp}. Similarly, Cst,rs did not differ between duplicate determinations at both V_T (0.54±0.15 *vs* 0.54±0.14 $l \cdot kPa^{-1}$) and V_{Tp} (0.46±0.14 *vs* 0.46±0.14 $l \cdot kPa^{-1}$). Therefore, the mean value of the duplicate measurements was used throughout this study.

The group average volume-time curves during passive expiration in the 11 subjects are shown in figure 2. These were clearly not mono- but rather bi-exponential, as indicated in figure 3. In all individuals, the biexponential model (Eq. 2) better fitted the data than the mono-exponential one (Eq. 1) for both V_T and V_{Tp}, as indicated by a significant difference by Fisher's test

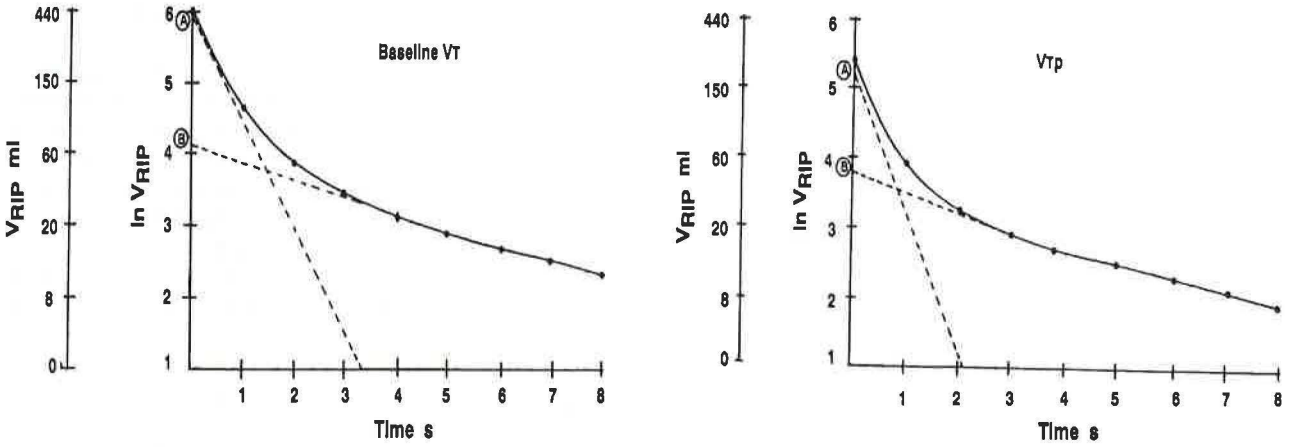

Fig. 3. – Semilog plots of group-average volume-time curves for baseline tidal volume (V_T, left panel) and partial tidal volume (V_{Tp}, right panel). Curves were drawn through use of group-average parameters of the bi-exponential model provided in table 3. Dotted lines represent the two exponential functions.

Table 3. – Results obtained during relaxed expirations in 11 subjects

	Deflation from V_T							Deflation from V_{Tp}						
Subject no.	Analysis time s	Fisher	r	A ml	τ'rs fast s	B ml	τ"rs slow s	Analysis time s	Fisher	r	A ml	τ'rs fast s	B ml	τ"rs slow s
1	7.5	98.7	0.999	357	0.31	43	4.17	8.0	54.5	0.983	157	0.34	45	5.56
2	4.5	57.8	0.987	573	0.42	74	1.67	3.0	55.5	0.998	188	0.27	150	0.88
3	5.5	64.9	0.998	231	0.22	53	2.27	7.0	141.7	0.998	152	0.25	50	2.70
4	3.0	18.0	0.999	280	0.27	94	1.18	1.5	9.9	0.999	233	0.27	22	2.00
5	8.5	167.0	0.998	411	0.41	93	4.17	5.5	102.2	0.998	169	0.35	49	3.45
6	7.5	8.8	0.988	612	0.79	94	5.56	4.0	6.1	0.999	451	0.68	20	5.56
7	5.5	68.2	0.996	299	0.53	89	2.22	3.0	45.1	0.972	175	0.33	53	1.39
8	5.5	48.0	0.995	197	0.34	90	2.56	4.0	86.6	0.997	95	0.29	37	2.86
9	5.0	8.6	0.998	495	0.81	56	3.70	3.0	16.3	0.999	198	0.48	84	1.56
10	7.5	48.4	0.998	377	0.81	41	5.88	2.0	12.5	0.998	149	0.27	38	1.92
11	4.5	47.4	0.995	318	0.56	40	2.63	4.5	23.3	0.998	201	0.59	28	4.54
Mean	5.9		0.995	377	0.50	70	3.27	4.1		0.994	197	0.37	52	2.95
SD	1.7		0.004	135	0.22	23	1.54	2.0		0.009	91	0.14	37	1.65

Analysis time, expiratory time over which data were analysed; Fisher: Fisher's coefficient of the comparison analysis of variance (ANOVA) between mono- and bi-exponential models was statistically significant in all instances; r: correlation coefficient of fit to Eq. 2; A and B: τ'rs fast and τ"rs slow, coefficients in Eq. 2.

(table 3). The correlation coefficients ranged between 0.972–0.999 for the bi-exponential model and 0.953–0.998 for the mono-exponential one. Comparison of the time constants of Eq. 2 obtained for V_T and V_{TP} reveals a significant difference for $\tau''rs$ ($p<0.05$) but not for $\tau'rs$. The difference in $\tau'rs$ probably reflects the lower Cst,rs with V_{TP} (table 2). Similarly, while the constant A of Eq. 2 decreased significantly as V_T was reduced to V_{TP} ($p<0.002$), the reduction in B was not significant. The rapid component (Eq. 2) represented 84±10% and 79±11% of the total volume change at V_T and V_{TP}, respectively. Since our results were best fitted by a bi-exponential model, we only present the time constants calculated with Eq. 2 (table 3).

Table 4. – Total respiratory system resistance, including ET tube, (Total R) corresponding to the fast phase of passive expiration

	Deflation from	
Subject no.	V_T Total R kPa·l^{-1}·s	V_{TP} Total R kPa·l^{-1}·s
1	0.56	0.62
2	0.63	0.47
3	0.58	0.75
4	0.85	0.93
5	0.73	0.78
6	1.01	1.00
7	1.13	0.91
8	0.97	1.01
9	1.18	0.74
10	1.41	0.57
11	1.00	1.25
Mean	0.91	0.82
SD	0.27	0.23

ET: endotracheal.

In line with previous reports [15–18] we assumed that the fast exponential compartment of the volume-time decay reflects the time constant due to Cst,rs and the pure (Newtonian) resistance offered by the respiratory system plus that due to the ET tube. Henceforth, the total resistance offered by the respiratory system plus ET tube will be labelled total R. Total resistance was computed as follows: total $R=\tau'rs/Cst,rs$. The individual and group average values (±SD) of total resistance of the fast component are given in table 4.

Discussion

The main result of this study is that passive deflation at barometric pressure of intubated, paralysed subjects with normal lungs is best described by a bi-exponential decrease of thoracic volume as a function of time. Such behaviour is in line with recent studies on anaesthetized-paralysed humans [17] and dogs [18] which showed that the respiratory system consists of two compartments: 1) a fast compartment reflecting standard respiratory compliance (Cst,rs) and resistance; and 2) a slow compartment reflecting the viscoelastic properties of the thoracic tissues. These findings are consistent with a model of the respiratory system originally proposed by Mount [19].

The time-course of volume during relaxed expirations has previously been studied in anaesthetized and paralysed subjects by BERGMAN [4] and BEHRAKIS *et al.* [3]. Volume was obtained by electronic integration of the pneumotachograph signal. The late slow changes in volume during expiration were considered as integrator drift and, hence, were neglected. In anaesthetized-paralysed dogs, BATES *et al.* [2] made computerized numerical integration of the pneumotachograph flow signal, thus avoiding problems due to drift. They were able to show that in dogs the volume-time profile includes a slow compartment, as described by Eq. 2. Our results indicated that in humans also the time-course of volume during relaxed expiration is better described by a bi-exponential than by a mono-exponential function.

Before probing the nature of this finding, it is necessary to discuss the limitations of the present analysis. Both Eqs 1 and 2 are based on the assumption that the relevant time constants are the product of a fixed compliance and expiratory resistance. Our results, however, suggest that the system deviates from linearity. Firstly, Cst,rs was slightly but significantly ($p<0.001$) higher at the lower inflation volume (V_{TP}) in line with the results obtained by D'ANGELO *et al.* [17] on anaesthetized-paralysed normal humans. In this connection it should be noted that in anaesthetized-paralysed humans with normal lungs Cst,rs decreases very slightly while approaching the functional residual capacity [17]. This, however, cannot explain the slow deflation compartment which occurs late in expiration. In fact, the lowering of Cst,rs with decreasing lung volume would be expected to result in relatively faster lung emptying. Secondly, when the resistance of the endotracheal tube is taken into account, the flow resistance of the respiratory system cannot be constant. Indeed the endotracheal tubes used in the present study (ID ranging from 7–8 mm) offer a variable flow resistance that follows Rohrer's equation: $R = K_1 + K_2 \cdot \dot{V}$, where K_1 and K_2 are constants [3]. This should result in proportionately greater diminution of expiratory flow rates during early passive expiration rather than later on, and hence cannot explain the slow deflation component which was found in the present study. Because our subjects were young and non-obese (table 1), small airway closure was probably nonsignificant during lung deflation, and hence could not have significantly affected the slow deflation component. Similarly, it is unlikely that changes in thoracic volume due to continuing gas exchange [13] would appreciably affect the volume-time profile during the relatively short duration of expiration. Thirdly, changes in thoracic blood volume could have an effect on the V_{RIP} signals

[20]. However, since positive end-expiratory pressure reduces both right and left ventricular volumes [21], it is likely that intrathoracic blood volume increased during passive expiration when intrathoracic pressure fell to barometric pressure. Therefore, changes in thoracic blood volume cannot account for the slow compartment that we observed in our subjects during passive expiration at barometric pressure. Lastly, small airway closure and gas trapping or delayed gas emptying *via* collateral ventilation could have contributed to the slow expiratory compartment in our subjects. However, it is highly likely that the slow compartment reflects the viscoelastic properties of the pulmonary and chest wall tissues, as originally suggested by BATES *et al.* [2]. In our subjects, the rapid deflation component caused emptying of 84±10% of the expired volume for inflation volume of 441±124 ml, whereas the slow component contributed only 16±7% of the expired volume with a time-constant about six times longer than that of the fast compartment. When the inflation volume was reduced to 249±91 ml, the time constant of the slow compartment did not change significantly while that of the rapid compartment decreased by 26% ($p<0.05$). This was probably due to both the concomitant decrease (15%) in Cst,rs (table 2) and the curvilinear pressure-flow relationships of the endotracheal tubes [3].

As postulated by BATES *et al.* [2], the time constant of the fast compartment probably reflects the product of standard Rrs and Cst,rs, where Rrs is the true (Newtonian) component of total expiratory flow resistance (including the endotracheal tube) whereas the time constant of the slow compartment reflects the viscoelastic properties of the pulmonary and chest wall tissues [15–18].

The time constant of the rapid compartment in our subjects was substantially longer than that reported for conscious normal subjects by SHEE *et al.* [22], namely 0.27±0.06 s. Similarly, the values of resistance (Rrs = τ'rs/Cst,rs) of our fast component were considerably higher than those reported by SHEE *et al.* [22]. This difference is probably mainly due to the high flow resistance offered by the endotracheal tubes in the present study and the small size of our subjects. The values of static compliances of our subjects were lower than normal (~100ml·cmH_2O^{-1}), probably reflecting the small size of our subjects. As noted above, due to the flow-dependent nature of the resistance offered by the endotracheal tubes, the size of which varied among subjects, our estimates of the time constant of the fast compartment are probably of limited value. Indeed, the time constant obtained with the mono-exponential model in our subjects was 0.63±0.22 s at VT. This is close to the time-constant of the fast compartment of the bi-exponential model and is largely due to the flow-resistive properties of the endotracheal tube.

In contrast, the values of time constant of the slow compartment should be less affected by such factors. In fact, it is this slow compartment that may well explain the frequency- dependence of compliance and flow resistance of the lung and chest wall found both in animals [18, 23, 24] and humans [9, 17].

In conclusion, the present results show that in intubated humans as in anesthetized-paralysed dogs [2], the time course of volume during passive expiration is characterized by a fast and a slow exponential compartment and suggest that the mono-exponential model is not suitable to determine the mechanical properties of the respiratory system in such patients.

Acknowledgements: The authors express their gratitude to their colleagues, Drs A.L. Coates and C. Giuntini for many stimulating discussions and helpful suggestions while this work was in progress.

References

1. Brody AW. – Mechanical compliance and resistance of the lung-thorax calculated from the flow recorded during passive expiration. *Am J Physiol*, 1954, 178, 189–196.
2. Bates JHT, Decramer M, Chartrand D, Zin WA, Boddener A, Milic-Emili J. – Volume-time profile during relaxed expiration in the normal dog. *J Appl Physiol*, 1985, 59, 732–737.
3. Behrakis PK, Higgs BD, Baydur A, Zin WA, Milic-Emili J. – Respiratory mechanics during halothane anesthesia and anesthesia-paralysis in humans. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1983, 55, 1085–1092.
4. Bergman NA. – Measurement of respiratory resistance in anesthetized subjects. *J Appl Physiol*, 1966, 21, 1913–1917.
5. England SJ, Bartlett D Jr, Daubenspeck JA. – Influence of human vocal cord movements on airflow and resistance during eupnea. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1982, 52, 773–779.
6. McIlroy MB, Tierney DF, Nadel JA. – A new method for measurement of compliance and resistance of lungs and thorax. *J Appl Physiol*, 1963, 17, 424–427.
7. Mortola JP, Fisher JT, Smith B, Fox G, Weeks S. – Dynamics of breathing in infants. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1982, 52, 1209–1215.
8. Murray JF, Matthay MA, Luce JM, Flick MR . – An expanded definition of the adult respiratory distress syndrome. *Am Rev Respir Dis*, 1988, 138, 720–723.
9. Barnas GM, Yoshino K, Loring SH, Mead J . – Impedance and relative displacements of relaxed chest wall up to 4 Hz. *J Appl Physiol*, 1987, 62, 71–81.
10. Dall'Ava-Santucci J, Armaganidis A, Morisseau D, Dhainaut JF, Brunet F, Nouira S, Monsallier JF, Lockhart A. – Stability of thoraco-abdominal partitioning simplifies indirect spirometry calibration in paralyzed patients. *Am Rev Respir Dis*, 1988, 137 (Abstract), 468.
11. Cohn MA, Rao ASV, Broudy M, Birch S, Watson H, Atkins N, Davis B, Stott FD, Sackner MA. – The respiratory inductive plethysmograph: a new non-invasive monitor of respiration. *Bull Eur Physiopathol Respir*, 1982, 18, 643–658.
12. Hudgel DW, Capehart, M, Johnson B, Hill P, Robertson D. – Accuracy of tidal volume, lung volume, and flow measurements by inductance vest in COPD patients. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1984, 56, 1659–1665.
13. Dall'Ava-Santucci J, Armaganidis A, Brunet F, Dhainaut JF, Chelucci GL, Monsallier JF, Lockhart A. – Causes of error of respiratory pressure-volume curves in paralyzed subjects. *J Appl Physiol*, 1988, 64, 42–49.
14. Wallenstein S, Zucker CL, Fleiss JL. – Some statistical methods useful in circulation research. *Circ Res*, 1980, 47, 1–9.

15. Bates JHT, Decramer M, Chartrand D, Zin WA, Boddener A, Milic-Emili J. – Respiratory resistance with histamine challenge by single-breath and forced oscillation methods. *J Appl Physiol*, 1986, 61, 873–880.
16. Bates JHT, Baconnier P, Milic-Emili J. – A theoretical analysis of interrupter technique for measuring respiratory mechanics. *J Appl Physiol*, 1988, 64, 2204–2214.
17. D'Angelo E, Calderini E, Torri G, Robatto FM, Bono D, Milic-Emili J. – Respiratory mechanics in anesthetized paralyzed humans: effects of flow, volume, and time. *J Appl Physiol*, 1989, 67, 2556–2564.
18. Similowski T, Levy P, Corbeil C, Albala M, Pariente R, Derenne JP, Bates JHT, Jonson B, Milic-Emili J. – Viscoelastic behavior of lung and chest wall in dogs determined by flow interruption. *J Appl Physiol*, 1989, 67, 2219–2229.
19. Mount LE. – The ventilation flow-resistance and compliance of rat lungs. *J Physiol*, 1955, 127, 157–167.
20. Chadha TS, Lopez F, Jenouri G, Birch S, Sackner MA. – Effects of partial anti-G suit inflation on thoracic volume and breathing pattern. *Aviat Space Environ Med*, 1983, 54, 324–327.
21. Fewell JE, Abendschein DR, Carlson CJ, Rappaport E, Murray JF. – Mechanism of decreased right and left ventricular end-diastolic volumes during continuous positive-ventilation in dogs. *Circ Res*, 1980, 47, 467–472.
22. Shee CD, Ploy-Song-Sang Y, Milic-Emili J. – Decay of inspiratory muscle pressure during expiration in conscious humans. *J Appl Physiol*, 1985, 58, 1859–1865.
23. Kochi T, Okubo S, Zin WA, Milic-Emili J. – Flow and volume dependence of pulmonary mechanics in anesthetized cats. *J Appl Physiol*, 1988, 64, 441–450.
24. Kochi T, Okubo S, Zin WA, Milic-Emili J. – Chest wall and respiratory system mechanics in cats: effect of flow and volume. *J Appl Physiol*, 1988, 64, 2636–2646.

Un model à simplie compartément ne peut décribe l'exporation passive chez les humains intubés et paralysés. G.L. Chelucci, F. Brunet, J. Dall'Ava-Santucci, J.F. Dhainaut, D. Paccaly, A. Armaganidis, J. Milic-Emili, A. Lockhart.
RÉSUMÉ: La décroissance du volume thoracique en fonction du temps (pléthysmographe à variation d' inductance) pendant une expiration passive par une modèle cinétique bi-exponentiel a été évaluée chez 11 sujets à poumons normaux à partir de 2 volumes d'insufflation (V_T et V_{Tp}). Ils étaient tous intubés, paralysés et ventilés. Nos résultats montrent que la première partie de l'expiration passive était responsable de 84±10% (V_T) contre 79±15% (V_{Tp}) de la variation de volume thoracique et montrait une constante de temps égale à 0.50±0.22 s (V_T) contre 0.37±14 s (V_{Tp}) ($p<0.05$). La deuxième exponentielle contribuait moins que la première à la variation de volume: 16±7% (V_T) contre 21±11% (V_{Tp}) et sa constante de temps était 3.27±1.54 s (V_T) contre 2.95±1.65 s (V_{Tp}) (p: NS). Nos résultats suggèrent que la première exponentielle est liée aux résistances des voies aériennes pendant la première phase de l'expiration tandis que la deuxième exponentielle plus lente représente l'impédance du système respiratoire liée aux propriétés viscoélastiques du poumon.
Eur Respir J., 1991, 4, 458–464.