Eur Respir J, 1994, 7: 286–291
DOI: 10.1183/09031936.94.07020286
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European Respiratory Journal
ISSN 0903 - 1936

The effect of positive end-expiratory pressure on respiratory resistive properties in anaesthetized paralysed humans

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The effect of positive end-expiratory pressure on respiratory resistive properties in anaesthetized paralysed humans. R. Cohendy, J. Ripart, J-J. Eledjam. ©ERS Journals Ltd 1994.

ABSTRACT: The respiratory resistive properties of the normal human respiratory system are volume-dependent. The overall flow resistance (Rmax,rs) can be partitioned into airway resistance (Raw) and the additional resistance (ΔRrs) which may result from the viscoelastic properties of the respiratory system, from inequality of time constants (pendelluft), or from both. Because positive end-expiratory pressure (PEEP) increases end-expiratory lung volume and may equalize ventilation within the lungs, the effect of PEEP on Raw, ΔRrs, and their sum (Rmax,rs) was assessed in anaesthetized surgical patients without evidence of lung disease.

Fifteen men were studied during paralysis and isoflow isovolume mechanical ventilation, using the end-inflation occlusion method. Ten men were studied with incremental levels of PEEP, up to 16 cmH_2O (Group A). Five men were studied without PEEP (Group B).

In Group A, Rmax,rs did not change with PEEP. In contrast, Raw decreased and ΔRrs increased significantly. Moreover, there was a linear relationship between PEEP and the contribution of ΔRrs to Rmax,rs. In Group B, Rmax,rs, Raw and ΔRrs, and the contribution of ΔRrs to Rmax,rs did not change. In both groups, atropine elicited a decrease in Rmax,rs, linked to a decrease in Raw, without any notable effect on the static elastance of the respiratory system (Est,rs) or on ΔRrs.

We conclude that the overall flow resistance was not affected by PEEP. In contrast, PEEP clearly modified the contribution of its two components. The decrease in Raw with PEEP could have resulted, at least in part, from modification in the basal vagal tone.

Eur Respir J., 1994, 7, 286–291.

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Keywords: Mechanical ventilation
positive end-expiratory pressure
respiratory mechanics

Received: April 5 1993
Accepted after revision August 24 1993

Supported by a grant (No. 7–88) from the "Commission de Recherche Clinique" of the University Hospital of Nîmes, France.

The overall respiratory system resistance Rmax,rs can be partitioned into a resistance due to the effect of the conducting airway (Raw) and a non-Newtonian additional resistance (ΔRrs) that may result from the viscoelastic properties of the respiratory system, from inequality of regional time constants (pendelluft), or from both [1, 2]. The magnitude of ΔRrs is predicted to increase when the tidal volume (VT) increases. Indeed, D'ANGELO and coworkers [3] have shown in man that, in isoflow condition, ΔRrs increased progressively with inflation volume. On the other hand, Raw is known to decrease when the lung volume increases [4]. Therefore, positive end-expiratory pressure (PEEP) may elicit some effect on the respiratory resistive properties, since it increases the end-expiratory lung volume [4]. Moreover, as PEEP is known to equalize the distribution of the ventilation within the lungs, it may decrease the role of a possible pendelluft phenomenon.

Therefore, the aim of this study was to describe the effects of PEEP on respiratory mechanics, and was focused on resistive properties and their partition. For this purpose, we used the end-inflation occlusion method (EIOM) and incremental levels of PEEP in anaesthetized, paralysed and mechanically-ventilated humans. As the respiratory mechanics might have been affected by the duration of the general anaesthesia or some variation in its depth, a group of patients without PEEP was also studied. Finally, since atropine decreases the overall resistance of the respiratory system (Rmax,rs) during general anaesthesia in man [5], its effect on the partitioned resistances were also described.

Methods

Patients

After approval from the local Ethics Committee, 15 men scheduled for elective surgery under general anaesthesia were included in this study. The surgical procedure was in each case a testicular exploration under

Table 1. – Physical characteristics of the two study groups, nature of anaesthetics and doses given for induction of intravenous general anaesthesia

	n	Age yrs	Height cm	Weight kg	Methohexitone $mg \cdot kg^{-1}$	Fentanyl $\mu g \cdot kg^{-1}$	Vecuronium $mg \cdot kg^{-1}$
Group A	10	35±2.5	176±5	78±13	3.15±0.39	4.94±0.12	0.20±0.01
Group B	5	31±5.5	175±4	74±7	3.27±0.63	5.00±0.13	0.18±0.02

Data are presented as mean±SD.

operative microscope. The exclusion criteria were: any clinical or radiological abnormality of the respiratory system, smoking (more than 5 $packs \cdot yr^{-1}$), suspected (history of atopy [6]) or overt (history of wheezes) bronchial hypersensitivity, treatment with a β-blocker. With respect to the experimental protocol, the subjects were divided in two groups: 10 patients were studied with incremental levels of PEEP (Group A), five men were studied without application of external PEEP (Group B). The physical characteristics of the men are shown in the table 1.

Material

Mechanical ventilation was performed with a Servo Ventilator 900 D (Siemens Elema, Solna, Sweden), which provided PEEP. This ventilator gives a constant inflation flow ($\dot{V}_I$), and an accurate V_T [7], and allows end-expiratory and end-inspiratory occlusions [8] (valve closing time 40 ms). The respiratory circuit was made with short tubings (60 cm) without humidifier, in order to reduce the compressible volume [9]. Each disposable endotracheal tube (internal diameter 8 mm) was fitted with a lateral port at its tracheal end ("Blue Line", ref. 100/196/080 Portex, UK), that allowed measurement of the tracheal pressure [10, 11]. The port was connected to a pressure transducer (model SK range ±40 cmH_2O, EFFA, Le Pré St-Gervais, France) and a linear amplifier (model MRC 4411, LEIM, Aix en Provence, France). The pressure signal and the flow signal fed by the ventilator were recorded on a Gould TA 550 polygraph, at a paper speed of 15 $mm \cdot s^{-1}$. This apparatus allowed the detection of inflection in airway pressure of ±0.05 cmH_2O.

Parameters

Respiratory mechanics of anaesthetized, paralysed and mechanically-ventilated subjects were studied using the EIOM [2]. Briefly, at the end of the inflation of a preset V_T, an occlusion of about 6 s was performed at the ventilator valves (when pressing on the "inspiratory hold" button) to detect any air leak by visual inspection of the pressure tracing. After airway occlusion, there was an abrupt drop in the tracheal pressure from a maximum pressure (Pmax) to an inflection point (P_1), and a more gradual decay to a plateau pressure, which is the elastic recoil pressure of the respiratory system (Pel). Gas exchange during occlusion has a negligible effect on Pel [12], which was measured at the end of the third second of end-inspiratory occlusion. The initial drop in pressure ($Pmax-P_1$) reflects the resistive pressure attributable to the conducting airway resistance (Raw) during the constant flow preceding the occlusion ($\dot{V}_I$) [13, 14]. Thus, Raw equals $(Pmax-P_1)/\dot{V}_I$. This initial drop in pressure can be underestimated because there is residual gas flowing through the closing valve. This error was minimized by using correction of the closure time of the valve with a linear extrapolation on the paper chart of the pre- and post-occlusion pressure signals to the point in time when the valve was half-closed [15]. Similarly, overall resistance Rmax,rs, was calculated as $Rmax,rs = (Pmax-Pel)/\dot{V}_I$; additional resistance (ΔRrs) = Rmax,rs - Raw. In order to take the weight and the height of the subjects into account, the contribution of ΔRrs to Rmax,rs was calculated as ΔRrs/Rmax,rs. The tracheal pressure at end-expiration, due to the applied PEEP, was measured by an end-expiratory occlusion performed at the ventilator valves (while pressing the "expiratory hold" button). The static elastance of the respiratory system (Est,rs) was calculated as $(Pel-PEEP)/V_T$ [12]. It would have been necessary to take account of any intrinsic PEEP in calculating elastance, but this was never seen in our patients. To avoid absorption atelectasis, the lungs were ventilated with 30% O_2 in nitrogen [16]. At each experimental time, at least one end-expiratory occlusion followed by at least three end-inspiratory occlusions were performed. All of the occlusions were separated by five tidal breaths. As respiratory mechanics are flow and frequency-dependent [3], all the patients underwent mechanical ventilation with the same pattern: frequency = 10 $breaths \cdot min^{-1}$, V_T=0.9 *l*, T_I=1.5 s, $\dot{V}_I$=0.6 $l \cdot s^{-1}$.

Anaesthesia

The supine subjects were studied during intravenous general anaesthesia and paralysis. The study was performed in the operating room, before the beginning of the surgical procedure. Midazolam 5 mg *i.m.*, was given as premedication. Anaesthesia was induced with methohexitone and fentanyl (induction doses, see table 1) and maintained with a continuous infusion of these drugs (methohexitone 0.1 $mg \cdot kg^{-1} \cdot min^{-1}$; fentanyl 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$) [17, 18]. Paralysis was produced with vecuronium bromide, and monitored with train-of-four stimulation of the adductor pollicis muscle. A surgical level of paralysis was observed in every subject during the duration of the study. The trachea was intubated with the orotracheal tube described above, the lungs ventilated manually with several large breaths in order to equalize the volume history, and the patient was connected to the ventilator.

Protocol

Group A. Baseline values were recorded 6 min after connection to the ventilator. Four consecutive measurements were performed during the last minute of 4 min steps of incremental levels of PEEP (4, 8, 12 and 16 cmH_2O) without any other change in the respiratory pattern. To avoid change in the volume history, the lungs were not allowed to return to ventilatory resting level between each step of incremental PEEP. PEEP was released after the 16 cmH_2O step, and respiratory mechanics parameters were recorded 3 min later. Atropine, 1 mg *i.v.*, was given immediately after the release of PEEP, 3 min before the last measurement.

Group B. The protocol was identical in all aspects to that for Group A, but PEEP was not applied.

Statistical analysis

Data are given as mean±standard deviation. The means were compared with two-way analysis of variance for repeated measures (ANOVA) and paired Student's t-test. The value of $p<0.05$ was considered to be significant. When paired t-tests were used to compare the effect of incremental PEEP (*i.e.* five comparisons) a Bonferroni correction was applied to the critical p-value for significance ($p<0.01$).

Results

Elastance and resistances in the two study groups are shown in tables 2 and 3.

Effect of PEEP

Group A was studied to determine the effect of PEEP. The values measured at baseline, and with incremental levels of PEEP were considered. Est,rs significantly (ANOVA p=0.034) decreased from baseline as PEEP was set to 4 cmH_2O (p=0.007) and to 8 cmH_2O (p=0.01). Est,rs reached a plateau as PEEP was further increased to 12 cmH_2O and 16 cmH_2O. Rmax,rs remained stable with PEEP (ANOVA p=0.58). By contrast, Raw decreased (ANOVA p=0.0001). Raw with 12 cmH_2O PEEP (-36%, p=0.0001) and with 16 cmH_2O PEEP (-48%, p=0.0001) was significantly lower than at baseline. As Rmax,rs remained stable and Raw decreased, ΔRrs increased significantly (ANOVA p=0.0021). ΔRrs was significantly higher than baseline with 12 cmH_2O PEEP (+10%, p=0.007) and with 16 cmH_2O PEEP (+23%, p=0.004). The contribution of ΔRrs to Rmax,rs (ΔRrs/Rmax,rs) increased significantly (ANOVA p=0.0001) with the magnitude of PEEP. There was a significant relationship between the level of PEEP and ΔRrs/Rmax,rs with the linear regression: ΔRrs/Rmax,rs (%) = 63.57 + 0.99 PEEP (cmH_2O)

Table 2. – Effect of PEEP and atropine on respiratory mechanics in Goup A (n=10)

	Baseline	PEEP cmH_2O					Atropine
		4	8	12	16	0	
Est,rs $cmH_2O \cdot l^{-1}$	11.3±1.94	10.6±1.88*	10.3±1.62*	10.5±1.54	10.8±1.27	11.3±1.66	11.7±2.09
Rmax,rs $cmH_2O \cdot l^{-1} \cdot s$	5.6±1.04	5.6±1.07	5.4±0.86	5.2±1.14	5.3±1.11	5.5±0.96	4.4±0.85+
Raw $cmH_2O \cdot l^{-1} \cdot s$	2.1±0.70	1.9±0.68	1.7±0.50*	1.4±0.62**	1.1±0.53**	1.8±0.52	1.1±0.33++
ΔRrs $cmH_2O \cdot l^{-1} \cdot s$	3.5±0.46	3.7±0.55	3.8±0.65	3.8±0.67*	4.2±0.73*	3.7±0.53	3.2±0.56
ΔRrs/Rmax,rs %	62.8±6.40	67.1±6.34*	69.6±6.88*	74.5±7.01**	80.4±6.14**	67.5±5.26	73.3±7.78

Data are presented as mean±SD. Comparison of incremental levels of PEEP to baseline (paired Student's t-test, Bonferroni correction): *:p<0.01; **: p<0.001. Effect of atropine (*versus* PEEP 0 cmH_2O) (paired Student's t-test): +: p<0.01; ++: p<0.001. PEEP: positive end-expiratory pressure; Est,rs: static elastance of the respiratory system; Rmax,rs: overall flow resistance of the respiratory system; Raw: airway resistance; ΔRrs: additional resistance which may result from viscoelastic properties of the respiratory system from inequality of time constants (pendelluft) or from both.

Table 3. – Effects of time (in minutes elapsed after baseline measurement) and of atropine on the respiratory mechanics of Group B (n=5)

	Baseline	4th min	8th min	12th min	16th min	20th min	Atropine
Est,rs $cmH_2O \cdot l^{-1}$	12.8±3.52	13.3±3.42	13.5±3.34	13.5±3.45	13.5±3.78	13.9±3.92	14.0±3.48
Rmax,rs $cmH_2O \cdot l^{-1} \cdot s$	6.2±2.17	6.4±2.03	6.3±1.76	6.2±1.92	6.1±1.61	6.1±1.39	4.2±0.63
Raw $cmH_2O \cdot l^{-1} \cdot s$	2.8±1.89	2.9±1.61	2.7±1.43	2.8±1.71	2.6±1.22	2.5±1.14	1.2±0.34
ΔRrs $cmH_2O \cdot l^{-1} \cdot s$	3.4±0.91	3.3±0.42	3.5±0.83	3.5±0.81	3.5±0.73	3.6±0.54	2.9±0.73
ΔRrs/Rmax,rs %	57.4±17.40	55.4±15.66	57.8±14.04	57.8±15.27	59.2±11.43	60.2±11.28	70±9.30

Data are presented as mean±SD. For abbreviations see legend to table 2.

Table 4. – Individual relationships between the contribution of ΔRrs to Rmax,rs (ΔRrs/Rmax,rs) (%) and expiratory pressure (cmH_2O) assessed by linear regression in Group A (n=10)

Pt No.	Intercept	Slope	r	p
1	57.58	1.26	0.81	0.09
2	70.72	0.92	0.96	0.01
3	57.19	1.64	0.98	0.004
4	68.07	0.85	0.90	0.03
5	52.53	1.04	0.96	0.01
6	69.41	0.64	0.92	0.02
7	64.9	1.31	0.89	0.04
8	63.12	1.42	0.90	0.03
9	65.31	0.45	0.64	0.24
10	64.96	1.09	0.86	0.06

For abbreviations see legend to table 2.

r0.63, p=0.0001, with a difference between the mean observed value of ΔRrs/Rmax and its predicted value at the intercept of -0.79. The individual relationships of ΔRrs/Rmax,rs and PEEP are summarized in table 4. Among the 10 subjects studied, seven (patients Nos 2–8) showed a significant and close relationship.

Effect of the release of PEEP

Group A was studied to determine the effect of release of PEEP (table 2). Est,rs returned to its baseline value when PEEP was released (baseline 11.25±1.94 $cmH_2O \cdot l^{-1}$; PEEP0 11.27±1.66 $cmH_2O \cdot l^{-1}$). Rmax,rs, Raw, ΔRrs and ΔRrs/Rmax,rs were very close to their baseline values when PEEP was released.

Effect of time

Group B was studied to determine the effect of time (table 3). All experimental times, except "atropine" time, were considered. Est,rs, Rmax,rs, Raw, ΔRrs and ΔRrs/Rmax,rs remained stable.

Effect of atropine

The parameters recorded when PEEP was released (Group A, table 2) and at the 20th minute for Group B were compared to the parameters measured under atropine in each group. These parameters were not pooled. The mean values of the Est,rs after the administration of atropine were very close to their values when PEEP was released in Group A and at the 20th minute in Group B. The decrease in Rmax,rs with atropine was significant in the two groups: -20% in Group A (p=0.01), -31% in Group B (p=0.015). Raw decreased significantly in Group A: -40%, (p=0.001), but the decrease in Raw in Group B (-51%) did not reach statistical significance (p=0.07).

Discussion

PEEP elicited significant changes in the respiratory mechanics of anaesthetized, surgical patients with normal lungs. Est,rs decreased up to a PEEP of 8 cmH_2O. Since Rmax,rs remained stable with PEEP, PEEP clearly modified the contribution of Raw and of ΔRrs, as Raw decreased. Therefore, the contribution of ΔRrs to Rmax,rs increased with PEEP, up to 80% of the value of Rmax,rs. Moreover, as PEEP was released, respiratory mechanics returned to baseline, and the duration of the study was not a confounding factor. Finally, atropine induced a substantial bronchodilation, without notable effect on Est,rs.

Intravenous general anaesthesia avoided the bronchodilatory effect of halogenated anaesthetics, and their effect on pulmonary tissue resistance [19]. If the opioid fentanyl increases Rmax,rs, and induces a parallel increase in both Raw and ΔRrs when it is given as a 5 $\mu g \cdot kg^{-1}$ bolus during barbiturate general anaesthesia [10], its effect on the pulmonary resistance is stable when it is given as a continuous infusion [20]. As the continuous infusion of anaesthetics gives a predictible plasma concentration [17, 18], a stable effect on resistances was expected and was confirmed by the control study of Group B.

The EIOM allows Rmax,rs to be partitioned into Raw and ΔRrs [2]. As respiratory mechanics vary with the ventilatory pattern [3], we maintained the same pattern with the exception of PEEP in all subjects, to allow comparison. Moreover, as the technique of airway occlusion during constant flow inflation can be performed at any inflation volume, the EIOM is a convenient means to standardize it. Any occlusion has a finite time and allows a residual flow which depends on T_I and V_T [15]. The volume resulting from this residual flow should have been small, since it can be estimated as about 2.5% of the V_T applied to our patients, according to the calculation of KOCHI *et al*. [21], who have studied the respiratory mechanics in cats with the EIOM and a very similar ventilator (Servo Ventilator 900 C). However, in spite of correction for the valve closing time, the residual flow leads to underestimation of the initial pressure drop and, thus, of the values of Rmax,rs and Raw. Because the inspiratory pattern was the same in each patient at each experimental time, and because each subject acted as his own control, this was not a confounding factor. At least, a qualitative comparison was allowed. It can be argued, additionally, that incremental PEEP could have modified the residual flow, impeding the validity of the intrasubject comparisons. In fact, the working pressure (60 cmH_2O) of the Servo Ventilator was sufficient to largely overcome the increased effort the ventilator had to produce after applying PEEP.

Rmax,rs, Raw and ΔRrs have previously been measured in humans with the EIOM by D'ANGELO and coworkers [3], with differing inspiratory volumes and flows, that allowed the calculation of the regression equations corresponding to isovolume and isoflow inflations. For the sake of comparison to the baseline resistance of Group A, we have derived from the data reported by this group the predicted values of resistance with the inspiratory

volume (0.9 *l*) applied to our subjects. The inflation flow that we used (0.6 $l \cdot s^{-1}$) was close to the mean flow used by D'ANGELO and co-workers [3] (0.557 $l \cdot s^{-1}$). Rmax,rs was estimated to be 5.53 $cmH_2O \cdot l^{-1} \cdot s$ as compared to the measured baseline value of 5.57 $cmH_2O \cdot l^{-1} \cdot s$ (table 2). Similarly, Raw was predicted as 1.89 $cmH_2O \cdot l^{-1} \cdot s$ and was measured at 2.11 $cmH_2O \cdot l^{-1} \cdot s$, and ΔRrs was predicted as 3.63 $cmH_2O \cdot l^{-1} \cdot s$ and was measured at 3.45 $cmH_2O \cdot l^{-1} \cdot s$. ΔRrs/Rmax,rs was estimated to 66%, as compared to the baseline value in our study (63%): the large contribution of ΔRrs to Rmax,rs was probably due to the high VT. Therefore, the baseline resistances measured in our study were close to those reported by D'ANGELO and co-workers [3], who had to correct Raw for the resistive pressure drop due to the endotracheal tube. Moreover, *in vitro* measurement may not accurately predict the effect of the tube *in vivo* [22]. In our study, the airway pressure was measured at the tracheal tip of the endotracheal tube, in order to avoid this subtraction [11]. The Bernouilli effect could have affected the measurement of pressure, leading to underestimation of actual tracheal pressure. However, the magnitude of this effect varies with the flow; because the inspiratory flow was the same in each case, the error resulting from this effect was not a confounding factor. The close similarity of our values with those derived from the work of D'ANGELO and co-workers [3] suggests that the procedure we had chosen, although simplified, was valid.

Flow resistances returned to control values when PEEP was released in Group A. In Group B there were no sizeable variations in resistance with time or with unexpected variation in the depth of the anaesthesia. Therefore, the variations in the flow resistances observed in our study can be assigned to the effect of incremental PEEP.

Rmax,rs was insensitive to the effect of PEEP. This has also been described in anaesthetized and mechanically-ventilated normal subjects by PESENTI *et al*. [23], who measured this resistance for PEEP up to 10 cmH_2O. In our study, Rmax,rs decreased with atropine in both groups, mainly because of a consistent decrease in Raw, which demonstrated the presence of a vagal tone at baseline. On the other hand, Raw decreased with PEEP, up to -48% of its baseline value at 16 cmH_2O. The significant decrease in Est,rs with lower levels of PEEP (up to 8 cmH_2O) (table 2) was consistent with opening of lung units; the decrease in Raw should have been related to effective increase in lung volume. Indeed, it is generally held that Raw is inversely related to lung volume [4]. By contrast, some overdistension (signalled by increase in Est,rs) occurred with higher levels of PEEP, but Raw still decreased almost linearly. Thus, the bronchodilating effect of PEEP may not be purely mechanical in nature. Indeed, it has been suggested that PEEP could have a vagally-mediated inhibitory influence, resulting from the stimulation of pulmonary stretch receptors [24]. It should be noted that the initial pressure drop after occlusion has been shown to reflect Raw, despite introduction of mechanical heterogeneities [25].

As Raw decreased, ΔRrs/Rmax,rs increased, up to 80% when PEEP was set to 16 cmH_2O, as compared to 62% at baseline (table 2). ΔRrs may result from the peripheral additional impedance caused by stress relaxation of the respiratory tissues (viscoelasticity) and/or time constant inhomogeneities between lung units (pendelluft) [3, 14, 26, 27]. The pendelluft phenomenon is generally held as negligible in normal conscious subjects [1]. However, it has been shown in surgical patients [28] that at the very beginning of a general anaesthesia with paralysis, dependent area of consolidation develop within the lung. These so-called compression atelectatic areas are no longer detectable when PEEP is applied [29]. Because in our patients Est,rs decreased with the lower levels of PEEP, suggesting opening of closed lung units, ΔRrs at baseline could have resulted, at least in part, from heterogeneities related to such atelectases. Nevertheless, if baseline ΔRrs actually reflected a substantial pendelluft phenomenon, it should have decreased (and not increased) with PEEP in our patients. Moreover, the viscoelastic model has been shown to be the most appropriate two compartment model for describing normal canine pulmonary mechanics with EIOM [13] within the tidal volume range. PESENTI *et al*. [23], in a study that did not specifically address the variations of ΔRrs or of ΔRrs/Rmax,rs with PEEP, reported a nonsignificant trend of ΔRrs to increase with PEEP (up to 10 cmH_2O) in normal subjects. More recently, D'ANGELO and co-workers [26] found that external PEEP (7.76±1.83 cmH_2O) during mechanical ventilation did not change ΔRrs. This is in line with the present results, because ΔRrs increased significantly only with a PEEP of 12 cmH_2O (table 2). Therefore, we speculate that pendelluft had only a small role, if any, in the creation of ΔRrs at baseline in our patients. The increase in ΔRrs with the higher levels of PEEP should have signalled the development of mechanical inhomogeneities within the respiratory system.

In conclusion, in surgical patients without evidence of respiratory disease, PEEP did not affect the overall flow resistance of the respiratory system, but clearly modified the contribution of ΔRrs and Raw. The decrease in Raw with PEEP could have resulted, at least in part, from modification of the basal vagal tone. It seemed that the pendelluft phenomenon had a only small role, if any, in the creation of ΔRrs at baseline.

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