

Respiratory mechanics and morphometric changes during pneumoperitoneum in normal rats

L.F.P. Moreira, C.F. Gobbi, M. Feijóo, P.R.M. Rocco, W.A. Zin

Respiratory mechanics and morphometric changes during pneumoperitoneum in normal rats. L.F.P. Moreira, C.F. Gobbi, M. Feijóo, P.R.M. Rocco, W.A. Zin. ©ERS Journals Ltd 1997.

ABSTRACT: Pneumoperitoneum may give rise to several respiratory changes; nevertheless, no comprehensive analysis of respiratory mechanics has been performed under this condition.

Respiratory mechanics and thoracoabdominal morphometry were evaluated in six sedated, anaesthetized, paralysed, and mechanically-ventilated rats before (control) and during pneumoperitoneum. After airway occlusion at end-inspiration, respiratory system, pulmonary, and chest wall resistive pressures ($\Delta P_{1,rs}$, $\Delta P_{1,L}$ and $\Delta P_{1,cw}$, respectively) and viscoelastic/inhomogeneous pressures ($\Delta P_{2,rs}$, $\Delta P_{2,L}$ and $\Delta P_{2,cw}$, respectively) were determined. Total pressure changes (ΔP_{tot}) were calculated as the sum of ΔP_1 and ΔP_2 , yielding the values of $\Delta P_{tot,rs}$, $\Delta P_{tot,L}$ and $\Delta P_{tot,cw}$, respectively. Respiratory system, lung, and chest wall static ($E_{st,rs}$, $E_{st,L}$ and $E_{st,cw}$, respectively), and dynamic elastances ($E_{dyn,rs}$, $E_{dyn,L}$ and $E_{dyn,cw}$, respectively), and the corresponding changes in elastance (ΔE) (calculated as $E_{dyn}-E_{st}$) were also obtained. Chest wall configuration both at functional residual capacity (FRC) and end-inspiration (FRC + tidal volume (VT)) was also evaluated in another four rats.

Pneumoperitoneum significantly increased $\Delta P_{tot,rs}$, $\Delta P_{tot,cw}$, $\Delta P_{2,rs}$, $\Delta P_{2,cw}$, ΔE_{rs} , ΔE_{cw} , $E_{st,rs}$, $E_{st,L}$ and $E_{st,cw}$. Lateral and anteroposterior diameters increased significantly, with the exception of lateral diameters at the level of crista iliaca. Cephalocaudal diameter and FRC decreased.

In conclusion, pneumoperitoneum augments elastances and increases the pressure dissipated against viscoelasticity/inhomogeneity of the respiratory system and chest wall. These changes are related to a cephalad displacement of the diaphragm plus changes in thoracoabdominal configuration.

Eur Respir J 1997; 10: 1321–1326.

Laboratory of Respiration Physiology, Carlos Chagas Filho Biophysics Institute, Federal University of Rio de Janeiro, Ilha do Fundão, Rio de Janeiro, Brazil.

Correspondence: W.A. Zin
Universidade Federal do Rio de Janeiro
Centro de Ciências da Saúde
Instituto de Biofísica Carlos Chagas Filho
Ilha do Fundão
21949-900 Rio de Janeiro
Brazil

Keywords: Constant flow inflation elastance
mechanical inhomogeneities
morphometry
pneumoperitoneum
viscoelasticity

Received: August 22 1996
Accepted after revision January 20 1997

Supported by the Brazilian Council for Scientific and Technological Development (CNPq), Financing for Studies and Projects (FINEP), and Graduated Teaching and Research Council of the Federal University of Rio de Janeiro (CEPG-UFRJ).

The use of laparoscopic techniques in surgery is increasing in popularity. During laparoscopy, a pneumoperitoneum is generated, which may yield several respiratory changes, e.g. pulmonary atelectasis [1], decreased functional residual capacity (FRC) and vital capacity (VC) [1–3], reduced pulmonary compliance [4, 5], and increased peak airway pressure [2, 6]. Although these alterations are important, no comprehensive analysis of respiratory mechanics has previously been reported during pneumoperitoneum. Thus, the present investigation was designed to evaluate the effects of pneumoperitoneum on respiratory system, pulmonary, and chest wall elastic, viscous, and viscoelastic mechanical properties, thoracoabdominal morphometry, and changes in FRC in anaesthetized paralysed rats.

Materials and methods

Ten male adult Wistar rats were initially sedated with diazepam (5 mg *i.p.*), anaesthetized with pentobarbital sodium (20 mg/kg *i.p.*), and a snugly-fitting cannula (1.5

mm internal diameter (ID)) was introduced into the trachea. They were then placed in the supine position on a surgical table. An Insyte I.V. 18-gauge catheter (Becton Dickinson Vascular Access, Sandy, UT, USA) was inserted into the peritoneal cavity in the mid-line, about 2.0 cm cephalad to the pubic bone. An indwelling catheter segment of about 1 cm was positioned, and the needle was then removed. The catheter was secured in place and the airtightness assured by stitching the skin around the catheter. Pneumoperitoneum was produced by injecting room air through the catheter until an intra-abdominal pressure of 12 ± 1.2 (\pm SD) cmH₂O was reached.

In order to measure respiratory mechanics, six rats weighing 190 ± 10 (\pm SD) g (range 180–210 g) were used. An adequate pneumotachograph, constructed according to MORTOLA and NOWORAJ [7], was connected to the tracheal cannula for the measurements of airflow (V'), and, by electronic integration, changes in tidal volume (VT). The flow resistance of the equipment (R_{eq}), tracheal cannula included, was constant at flow rates of up to 26 mL·s⁻¹ and amounted to 0.125 cmH₂O·mL⁻¹·s. Equipment resistive pressure ($R_{eq} \cdot V'$) was subtracted from respiratory system and pulmonary viscous pressure, so that

the present results represent intrinsic values. Because abrupt changes of diameter were not present in the circuit, errors in measurement of flow resistance were probably avoided [8, 9]. The equipment dead space was 0.4 mL.

Tracheal pressure (P_{tr}) was measured with a Hewlett-Packard 270 differential pressure transducer (Waltham, MA, USA). Changes in oesophageal pressure (P_{oes}), which reflects chest wall pressure (P_{cw}), were measured with a 30 cm long water-filled catheter (PE-240) with side holes at the tip connected to a PR23-2D-300 Statham differential pressure transducer (Hato Rey, Puerto Rico). The catheter was passed into the stomach and then slowly returned into the oesophagus; its proper positioning was assessed using the "occlusion test" [10]. The frequency responses of the pressure measurement systems (P_{tr} and P_{oes}) were flat up to 20 Hz, without appreciable phase shift between the signals. All signals were conditioned and amplified in a Beckman type R Dynograph (Schiller Park, IL, USA) and recorded on paper at a speed of 5 mm·s⁻¹. Flow and pressure signals were also passed through 8-pole Bessel filters (902LPF; Frequency Devices, Haverhill, MA, USA) with the corner frequency set at 100 Hz, sampled at 200 Hz with a 12-bit analogue-to-digital converter (DT2801A; Data Translation, Marlboro, MA, USA), and stored on a computer (PC-AT; IBM, Armonk, NY, USA). All data were collected using LABDAT software (RHT-InfoData Inc., Montreal, Quebec, Canada).

Muscle relaxation was achieved with gallamine triethyl iodide (2 mg·kg⁻¹ *i.p.*), and artificial ventilation was provided by a Salziner constant flow ventilator (Instituto do Coração-USP, São Paulo, Brazil). During the test breaths, a 5 s end-inspiratory pause could be generated by adjusting the ventilator settings, whereas during the baseline ventilation no pause was used. In order to avoid the effects of different flows and volumes [11, 12] and inspiratory duration [13] on the variables measured, special care was taken to keep \dot{V}_T (=2 mL) and V' (=10 mL·s⁻¹) constant in all animals.

The measurements were performed before and immediately after the injection of room air into the peritoneal space. The experiments did not last more than 40 min. Respiratory mechanics were measured from end-inspiratory occlusions after constant flow inflations [14]. Although this method has been used for a long time, the significance of the variables measured has only recently been clarified [13, 15–18]. After end-inspiratory occlusion, there is an initial fast drop in tracheal pressure ($\Delta P_{1,rs}$) from the preocclusion value down to an inflection point ($P_{i,rs}$), followed by a slow pressure decay ($\Delta P_{2,rs}$), until a plateau is reached. This plateau corresponds to the elastic recoil pressure of the respiratory system. $\Delta P_{1,rs}$ selectively reflects the combination of airways, pulmonary, and chest wall Newtonian resistances in normal animals [11, 13, 16–18] and humans [12]. $\Delta P_{2,rs}$ reflects stress relaxation, or viscoelastic properties, of the lung and chest wall tissues, together with a small contribution of pendelluft in normal situations [12, 17, 18].

The same procedures apply to the chest wall pressure (P_{cw}) yielding the values of $\Delta P_{1,cw}$, $P_{i,cw}$, $\Delta P_{2,cw}$ and $P_{el,cw}$, respectively. Transpulmonary pressures ($\Delta P_{1,L}$, $P_{i,L}$, $\Delta P_{2,L}$ and $P_{el,L}$) were calculated by subtracting the

chest wall pressure from the corresponding values pertaining to the respiratory system. Total pressure drop (ΔP_{tot}) is equal to the sum of ΔP_1 and ΔP_2 , yielding the values of $\Delta P_{tot,rs}$, $\Delta P_{tot,L}$, and $\Delta P_{tot,cw}$.

Respiratory system, lung and chest wall static elastances ($E_{st,rs}$, $E_{st,L}$ and $E_{st,cw}$, respectively) were calculated by dividing $P_{el,rs}$, $P_{el,L}$, and $P_{el,cw}$, respectively, by V_T . Dynamic elastances of the respiratory system, lung, and chest wall ($E_{dyn,rs}$, $E_{dyn,L}$ and $E_{dyn,cw}$, respectively) were obtained by dividing $P_{i,rs}$, $P_{i,L}$ and $P_{i,cw}$, respectively, by V_T . ΔE was calculated as the difference $E_{dyn} - E_{st}$, yielding the values of ΔE_{rs} , ΔE_L , and ΔE_{cw} . The data concerning respiratory system, lung, and chest wall elastances were presented in terms of static elastance and ΔE instead of dynamic elastances, since they represent, respectively, the elastic and viscoelastic properties of the respiratory system [19]. Measurements of respiratory mechanics were performed 8–10 times in each animal in all instances. Immediately before each data collection period, the airways were aspirated to remove possible collection of mucus, and the respiratory system was inflated three times to total lung capacity (TLC) to maintain constant volume history.

The delay between the beginning and the end of the valve closure (10 ms) was allowed for by back extrapolation of the pressure records to the actual time of occlusion, and the corrections in pressure, although very small, were performed as described previously [11].

All data were analysed using ANADAT data analysis software (RHT-InfoData Inc., Montreal, Quebec, Canada).

Chest wall configuration and FRC changes were determined in four male Wistar rats (195±10 (±SD) g, range 185–210 g) prepared as described previously [20], and ventilated in the same manner as the other six rats in the study. Thoracoabdominal circumferences (C), antero-posterior (D_{ap}), and lateral (D_l) diameters at the third intercostal space (ic), xiphoid (x), and crista iliaca (ci) levels were measured at control and during pneumoperitoneum, both at FRC and FRC+ \dot{V}_T . Circumferences were measured with an inextensible thread, and diameters were obtained directly with calipers. The measurements were performed three times in each animal by the same investigator, under the same circumstances as described for the mechanical analysis. Special care was taken to perform the measurements at the same reference points and to avoid errors due to the soft tissue compressibility [20].

Pulmonary cephalocaudal diameter (D_{cc}), distance from the lung apex to the diaphragmatic dome, was also determined in these four Wistar rats [21] before (control) and during pneumoperitoneum at FRC. For this purpose, two needle shafts were introduced transversally through the rat skin at 90° relative to its body length, at the 3rd intercostal space and xiphoid levels. Under radioscopic examination, two lengths were measured on the monitor: 1) between the two needle shafts; and 2) the lung apex-diaphragmatic dome distance. Since the space between the two needles was measured in the rats with calipers and the display was linear, D_{cc} could be easily calculated. The measurements were performed three times in each animal by the same investigator, under the same circumstances as described for the mechanical analysis.

In addition, the possible changes in functional residual capacity (Δ FRC) induced by the experimental protocol were also measured in all animals [20]. For this purpose, the same method as used in gas chromatography for flow and volume quantification was applied. A 1 mL graduated pipette with a soap meniscus in its lumen lay horizontally, and its tip was connected *via* a three-way stopcock to the breathing circuit. Just before abdominal inflation, the ventilator was disconnected and at FRC the stopcock was positioned to communicate the airways to the pipette. The abdomen was then rapidly inflated and the displacement of the soap meniscus, *i.e.* lung volume change, was determined. The manoeuvre did not last more than 20 s. The experiments lasted less than 40 min.

Statistical analysis was performed by means of Student's paired t-test, and the significance level was established at 5%. Values are presented as mean \pm SEM unless otherwise specified. A p-value less than 0.05 was considered statistically significant.

Results

Table 1 presents the mean values (\pm SEM) of inspiratory flow, V_T , respiratory system, lung, and chest wall pressure changes, static elastances, and change in elastance obtained before (Control) and during pneumoperitoneum.

Pneumoperitoneum modified the respiratory mechanical profile. It can be seen in figure 1a that $E_{st,rs}$, $E_{st,L}$ and $E_{st,cw}$ increased significantly during pneumoperitoneum (89, 54, and 217%, respectively). In addition, ΔE_{rs} and ΔE_{cw} increased by 53 and 446%, respectively, whereas ΔE_L remained unaltered (fig. 1b). Pneumoperitoneum also led to statistically significant increases in $\Delta P_{tot,rs}$ (27%), secondary to a rise in $\Delta P_{2,rs}$ (49%). These were consequent to changes in chest wall parameters, since only $\Delta P_{tot,cw}$ (188%) and $\Delta P_{2,cw}$ (428%) increased significantly. These results are depicted in figure 2.

Table 1. – Respiratory data before (Control) and during pneumoperitoneum (PNE)

	Control	PNE
V' mL·s ⁻¹	10.05 \pm 0.09	10.01 \pm 0.19
V_T mL	2.00 \pm 0.02	1.95 \pm 0.04
$\Delta P_{tot,rs}$ cmH ₂ O	4.12 \pm 0.24	5.25 \pm 0.29*
$\Delta P_{1,rs}$ cmH ₂ O	2.18 \pm 0.12	2.36 \pm 0.12
$\Delta P_{2,rs}$ cmH ₂ O	1.94 \pm 0.17	2.89 \pm 0.24*
$\Delta P_{tot,L}$ cmH ₂ O	3.56 \pm 0.28	3.63 \pm 0.36
$\Delta P_{1,L}$ cmH ₂ O	1.84 \pm 0.16	1.94 \pm 0.12
$\Delta P_{2,L}$ cmH ₂ O	1.72 \pm 0.17	1.69 \pm 0.31
$\Delta P_{tot,cw}$ cmH ₂ O	0.56 \pm 0.08	1.62 \pm 0.23*
$\Delta P_{1,cw}$ cmH ₂ O	0.34 \pm 0.05	0.42 \pm 0.05
$\Delta P_{2,cw}$ cmH ₂ O	0.23 \pm 0.02	1.20 \pm 0.21*
$E_{st,rs}$ cmH ₂ O·mL ⁻¹	3.46 \pm 0.11	6.54 \pm 0.48*
$E_{st,L}$ cmH ₂ O·mL ⁻¹	2.71 \pm 0.18	4.17 \pm 0.58*
$E_{st,cw}$ cmH ₂ O·mL ⁻¹	0.75 \pm 0.10	2.38 \pm 0.53*
ΔE_{rs} cmH ₂ O·mL ⁻¹	0.97 \pm 0.08	1.49 \pm 0.14*
ΔE_L cmH ₂ O·mL ⁻¹	0.86 \pm 0.09	0.87 \pm 0.16
ΔE_{cw} cmH ₂ O·mL ⁻¹	0.11 \pm 0.01	0.62 \pm 0.12

Values are presented as mean \pm SEM of six animals (8–10 determinations·rat⁻¹). V' : flow; V_T : tidal volume; rs: respiratory system; L: lung; cw: chest wall; ΔP_{tot} , ΔP_1 and ΔP_2 : total, initial and slow pressure drops after end-inspiratory airway occlusion, respectively; E_{st} : static elastance; ΔE : difference between dynamic and static elastance. *: p<0.05, compared to respective control value.

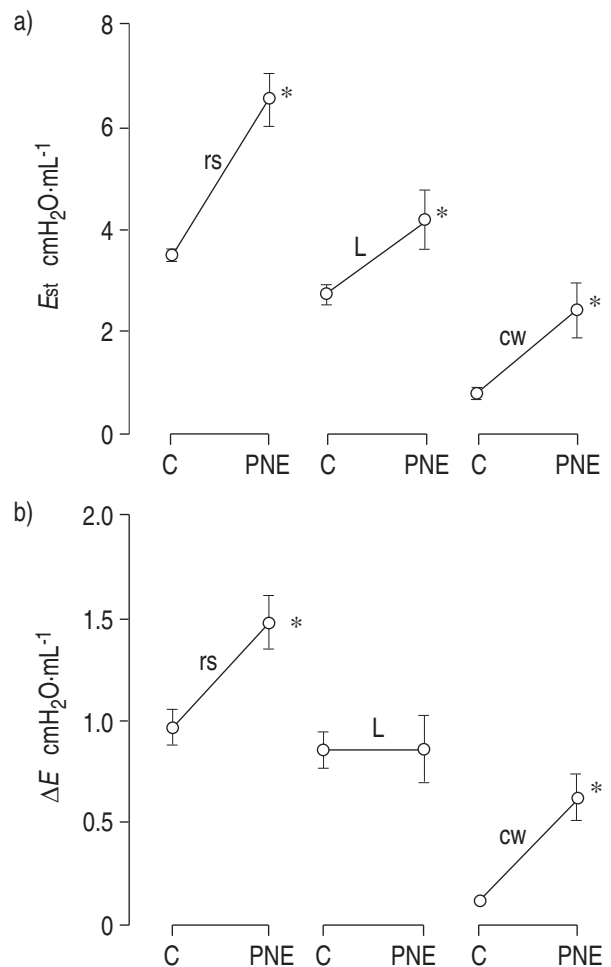


Fig. 1 – a) Respiratory system (rs), lung (L), and chest wall (cw) static elastance (E_{st}); and b) differences between dynamic and static elastance (ΔE) before (Control (C)) and during pneumoperitoneum (PNE). Values are presented as mean \pm SEM of 6 rats (8–10 determinations·rat⁻¹). When not depicted, error bars are smaller than symbols. *: p<0.01, compared to control value.

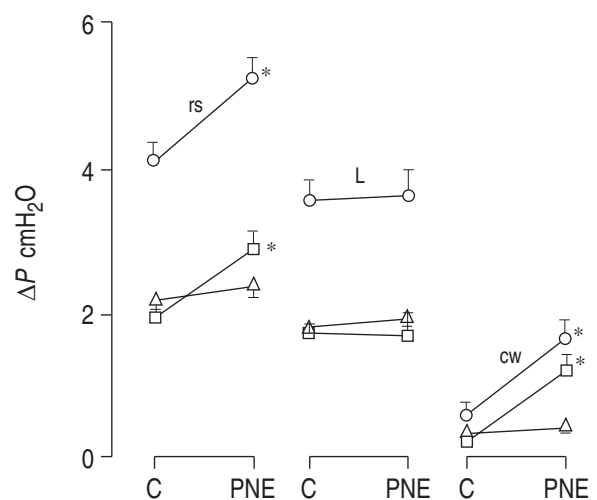


Fig. 2. – Respiratory system (rs), lung (L), and chest wall (cw) total pressure drop (ΔP_{tot}) its viscous (ΔP_1) and viscoelastic/inhomogeneous (ΔP_2) components after end-inspiratory airway occlusion in rats, before (Control (C)) and during pneumoperitoneum (PNE). Values are presented as mean \pm SEM of 6 rats (8–10 determinations·rat⁻¹). *: p<0.05, compared to control value. ○—○: ΔP_{tot} ; Δ—Δ: ΔP_1 ; □—□: ΔP_2 .

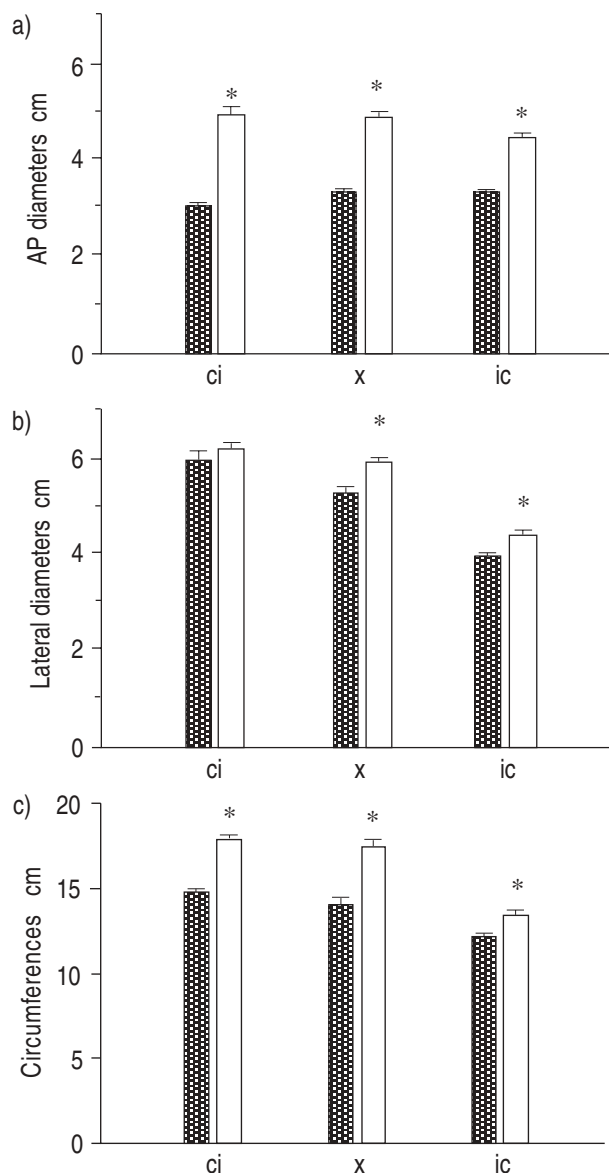




Fig. 3. — Thoracoabdominal dimensions measured at functional residual capacity (FRC) before (Control (C)) and during pneumoperitoneum (PNE). a) Anteroposterior (AP) diameters; b) lateral (LAT) diameters; and c) circumferences at crista iliaca (ci), xiphoid (x), and 3rd intercostal space (ic) levels. Values are presented as mean \pm SEM of 6 rats (8–10 determinations \cdot rat $^{-1}$). *: $p < 0.05$ compared to control value.  : Control;  : PNE.

Figure 3 shows the data on thoracoabdominal configuration measured before and during pneumoperitoneum at FRC. All diameters and circumferences, except the lateral diameter at the level of the crista iliaca, increased significantly. Pulmonary cephalocaudal diameter decreased significantly after injection of air into the peritoneal space, from 2.77 ± 0.08 to 2.54 ± 0.06 cm (9%). FRC was also reduced during pneumoperitoneum (Δ FRC 0.33 ± 0.06 mL).

Discussion

Pneumoperitoneum reduces pulmonary volumes [1–3], and may affect respiratory mechanics [4, 5], as observed

previously. However, no data have previously been reported concerning respiratory system, lung, and chest wall viscous, elastic, and viscoelastic mechanical properties during pneumoperitoneum.

Laparoscopy requires the generation of a pneumoperitoneum, most commonly achieved with the insufflation of CO_2 into the peritoneal cavity. Pulmonary atelectasis, decreased FRC, high peak inspiratory pressures, and raised arterial CO_2 ($\Delta\text{CO}_2 < 10$ mmHg) and alveolar CO_2 ($\Delta\text{CO}_2 < 8$ mmHg) may result, as a consequence of the greater intra-abdominal pressure, and absorption of CO_2 [2].

In the present investigation, peritoneal insufflation yielded an increase of 12.2 ± 1.2 cmH $_2$ O in abdominal pressure, which is similar to the values reported during laparoscopic cholecystectomy [22] or tubal ligation [3] (12–18 cmH $_2$ O). However, pressure values up to 40 cmH $_2$ O can be used in gynecological surgery [22].

In the present study, in order to avoid the effects of CO_2 , room air was injected intra-abdominally. The procedure is supported by the finding that when nitrous oxide was substituted for CO_2 no increase in the levels of the latter could be detected in dogs [23]. Thus, only the mechanical changes subsequent to increased intra-abdominal pressure are dealt with here.

Pneumoperitoneum yielded higher $E_{st,L}$ and $E_{st,cw}$, which led to increased $E_{st,rs}$ (fig. 1), thus indicating that the elastic component of the respiratory impedance was augmented under the present experimental conditions. These results are in accordance with those achieved during abdominal effusions [21] and pneumoperitoneum [2, 4, 5]. The increase in $E_{st,L}$ could be attributed to atelectasis at the lung base secondary to the cephalad movement of the diaphragmatic dome (see below), as also found previously [4, 20]. A higher $E_{st,L}$ could also be due to the smaller FRC found during pneumoperitoneum, which moved the pulmonary resting volume to lower values along the pressure-volume (P - V) curve of the lung, *i.e.* towards a less compliant region, and yielded airway closure. The significantly higher $E_{st,cw}$ (fig. 1) could be ascribed to conformational changes of the chest wall (fig. 3), and also to the increased stiffness of the diaphragm as a result of its cephalad displacement.

ΔE_{rs} and ΔE_{cw} increased significantly during pneumoperitoneum, whereas ΔE_L remained unaltered (fig. 1), suggesting that chest wall (and, thus, respiratory system) viscoelasticity/inhomogeneity became more prominent after pneumoperitoneum. This finding is confirmed by the increase in $\Delta P_{2,cw}$ (fig. 2), as discussed below.

When airflow and tidal volume remain constant, changes in $\Delta P_{tot,rs}$ reflect modification of respiratory system resistance, viscoelasticity and/or inhomogeneity. Pneumoperitoneum raised $\Delta P_{tot,rs}$. There was no significant change in $\Delta P_{tot,L}$, but $\Delta P_{tot,cw}$ was found to be significantly higher during pneumoperitoneum (table 1 and fig. 2).

It has been demonstrated in cats [11], dogs [13, 16, 17], and rats [24] that changes in $\Delta P_{1,L}$, when airflow and V_T remain constant, reflect pressure losses against Newtonian pulmonary resistances, whereas $\Delta P_{1,cw}$ also contributes to the chest wall component of $\Delta P_{1,rs}$ [13, 18]. Pneumoperitoneum did not induce significant changes in lung and chest wall Newtonian flow resistances.

Variations in $\Delta P_{2,rs}$ can be closely related to stress relaxation (or stress recovery) properties of lung and chest wall tissues (modelled by Kelvin bodies, each composed of an elastic element in parallel with a series elastance-resistance), together with a tiny contribution of pendelluft and asynchrony of movement within and between the chest wall components [12, 17, 24]. In other words, ΔP_2 can reflect pressure losses due to viscoelastic properties and/or mechanical inhomogeneities of lung and chest wall. During pneumoperitoneum, $\Delta P_{2,cw}$ increased significantly, augmenting $\Delta P_{2,rs}$. In a recent study AUGUSTO *et al.* [21] showed that after intraperitoneal injection of 50 mL of saline solution (0.9% NaCl) there were no statistically significant alterations in respiratory ΔP_s . However, in the present study, chest wall viscoelastic/inhomogeneity pressure losses were substantial. This apparent discrepancy could be explained by the different conformational changes of the chest wall between the two studies (see below). Interestingly, $\Delta P_{1,L}$ and $\Delta P_{2,L}$ did not change in other models of disease (pleural effusion, pneumothorax and intraperitoneal effusion) leading to a decrease in FRC [21, 25], under experimental conditions similar to the present one.

Although the methods used to measure configuration parameters could lead to error in the measurements, great care was taken to minimize this. Thus, the highest intranimal value of SD/mean ($n=3$) in the determination of D_{ap} , D_i and circumferences amounted to only 4.8%. When D_{cc} was measured the greatest ratio was 6.3%.

The conformational changes were similar at FRC and FRC+VT, therefore only changes in FRC are shown (fig. 3). Circumferences and D_{ap} values increased in the three levels studied. The variations in D_i were significant only at the xiphoid and intercostal levels. The most evident increase in D_i was found at the xiphoid level, confirming the effect of the larger abdominal volume on the inferior rib cage. The same applies to the measurement of circumferences. D_{ap} increased mostly at the level of the crista iliaca (fig. 3), where the distensibility of the abdominal wall is possibly larger due to the absence of bones in the anterior abdominal wall.

D_{cc} decreased during pneumoperitoneum because the diaphragm was pushed upwards. According to SHA *et al.* [1], using chest radiographs, during pneumoperitoneum there is a marked cephalad shift of the inspiratory and the expiratory position of the diaphragm. FRC was also reduced. Taking into account body weight (BW), $\Delta FRC/BW$ (after pneumoperitoneum) equals $2.3 \text{ mL}\cdot\text{kg}^{-1}$. Furthermore, according to CROSFILL and WIDDICOMBE [26], in nonparalysed rats FRC/BW equals $6 \text{ mL}\cdot\text{kg}^{-1}$. Thus, under the present experimental conditions, and assuming that FRC did not change with muscle paralysis, the total exhaled amount of gas was approximately 38% of FRC.

Interestingly, the injection of 50 mL of saline solution (0.9% NaCl) into the abdominal cavity yielded a $\Delta FRC/BW$ value of $2.4 \text{ mL}\cdot\text{kg}^{-1}$ [21], a value similar to that found in the present study. However, D_{ap} and C during pneumoperitoneum (expressed as percentage of control values) were greater than in the face of peritoneal effusion [21], namely: $D_{ap,ci}$ 62 vs 26%, respectively; $D_{ap,x}$ 47 vs 11%; $D_{ap,ic}$ 36 vs 3%; C_{ci} 21 vs 14%; C_x 26 vs 8%; and C_{ic} 12 vs 1%. On the other hand, $D_{i,ci}$ (13 vs 4%), and $D_{i,x}$ (17 vs 13%) were greater during

peritoneal effusion, but $D_{i,ic}$ was larger during pneumoperitoneum (10 vs 6%). Finally, D_{cc} change was greater during peritoneal effusion (16 vs 9%). Thus, it seems that the conformational changes induced by peritoneal effusion and pneumoperitoneum are different: because of the higher density of the saline solution, during abdominal effusion the fluid tends to accumulate at the dependent parts of the cavity, thus allowing greater D_i and D_{cc} than during pneumoperitoneum. The different conformational changes could also be responsible for the apparent discrepancy between the values of ΔP_2 and ΔP_{tot} found in the two studies. Finally, the conformational changes of the chest wall (diaphragm included) in the face of pneumoperitoneum could account for the increased $\Delta P_{2,w}$ reported in the present study.

In conclusion, it seems that not only are elastic changes present during pneumoperitoneum but there is also a substantial loss of pressure dissipated against viscoelastic/inhomogeneous segments of the chest wall. The mechanical findings were supported by measurements of chest wall morphometry, functional residual capacity and diaphragmatic displacement. Thus, during laparoscopic surgery the ventilator settings should be readjusted after the introduction of gas into the abdominal cavity because of the higher respiratory impedance found under these circumstances.

Acknowledgement: The authors thank A. Carlos de Souza Quaresma for skilful technical assistance.

References

1. Sha M, Ohmura A, Yamada M. Diaphragm function and pulmonary complications after laparoscopic cholecystectomy. *Anesthesiology* 1991; 75: A254.
2. Alexander GD, Noe FE, Brown EM. Anaesthesia for pelvic laparoscopy. *Anesth Anal* 1969; 48: 14–18.
3. Brown DR, Fishburne JI, Roberson VO, Hulka JF. Ventilatory and blood gas changes during laparoscopy with local anesthesia. *Am J Obstet Gynecol* 1976; 124: 741–745.
4. Puri GD, Singh H. Ventilatory effects of laparoscopy under general anesthesia. *Br J Anaesth* 1992; 68: 211–213.
5. Johannsen G, Nidersen M, Juhl B. The effect of general anesthesia on the haemodynamic events during laparoscopy with CO₂ insufflation. *Acta Anaesthesiol Scand* 1989; 33: 132–136.
6. Smith I, Benzie RJ, Gordon NLM, Kelman GR, Swapp GH. Cardiovascular effects of pneumoperitoneal insufflation of carbon dioxide for laparoscopy. *Br Med J* 1971; 3: 410–411.
7. Mortola JP, Noworaj A. Two-sidearm tracheal cannula for respiratory airflow measurements in small animals. *J Appl Physiol: Respirat Environ Exercise Physiol* 1983; 55: 250–253.
8. Chang HK, Mortola JP. Fluid dynamic factors in tracheal pressure measurements. *J Appl Physiol: Respirat Environ Exercise Physiol* 1981; 51: 218–225.
9. Loring SH, Elliot EA, Drazen JM. Kinetic energy loss and convective acceleration in respiratory resistance measurements. *Lung* 1979; 156: 33–42.
10. Baydur A, Behrakis PK, Zin WA, Jaeger M, Milic-Emili J. A simple method for assessing the validity of the

- oesophageal balloon technique. *Am Rev Respir Dis* 1982; 126: 788–791.
11. Kochi T, Okubo S, Zin WA, Milic-Emili J. Chest wall and respiratory system mechanics in cats: effects of flow and volume. *J Appl Physiol* 1988; 64: 2636–2646.
 12. D'Angelo E, Calderini E, Torri G, Robbato 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.
 13. Similowski T, Levy P, Corbeil C, *et al.* Viscoelastic behavior of lung and chest wall in dogs determined by flow interruption. *J Appl Physiol* 1989; 67: 2219–2229.
 14. Bates JHT, Rossi A, Milic-Emili J. Analysis of the behavior of the respiratory system with constant inspiratory flow. *J Appl Physiol* 1985; 58: 1840–1848.
 15. Bates JHT, Baconnier P, Milic-Emili J. A theoretical analysis of the interrupter technique for measuring respiratory mechanics. *J Appl Physiol* 1988; 64: 2204–2214.
 16. Bates JHT, Brown KA, Kochi T. Respiratory mechanics in the normal dog determined by expiratory flow interruption. *J Appl Physiol* 1989; 67: 2276–2285.
 17. Bates JHT, Ludwig MS, Sly PD, Brown KA, Martin JG, Fredberg JJ. Interrupter resistance elucidated by alveolar pressure measurements in open-chest normal dogs. *J Appl Physiol* 1988; 65: 408–414.
 18. 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.
 19. Eissa NT, Ranieri VM, Corbeil C, Chassé M, Braidy J, Milic-Emili J. Effect of PEEP on the mechanics of the respiratory system in ARDS patients. *J Appl Physiol* 1992; 73: 1728–1735.
 20. Zin WA, Martins MA, Silva PRM, Sakae RS, Carvalho ALI, Saldiva PHN. Effects of abdominal opening on respiratory system mechanics in ventilated rats. *J Appl Physiol* 1989; 66: 2496–2501.
 21. Augusto VM, Sousa AS, Moll RJ, Duarte JGC, Zin WA. Respiratory mechanics and morphometry after progressive intraperitoneal effusion. *Respir Physiol* 1995; 102: 217–224.
 22. Hanley ES. Anesthesia for laparoscopic surgery. *Surg Clin North Am* 1992; 72: 1013–1019.
 23. Ivankovich AD, Miletich DJ, Albrecht RF, Heyman HJ, Bonnet RF. Cardiovascular effects of intraperitoneal insufflation with carbon dioxide and nitrous oxide in the dog. *Anesthesiology* 1975; 42: 281–287.
 24. Saldiva PHN, Zin WA, Santos RLB, Eidelman DH, Milic-Emili J. Alveolar pressure measurement in open-chest rats. *J Appl Physiol* 1992; 72: 302–306.
 25. Sousa AS, Moll RJ, Pontes CF, Saldiva PHN, Zin WA. Mechanical and morphometrical changes in progressive bilateral pneumothorax and pleural effusion in normal rats. *Eur Respir J* 1995; 8: 99–104.
 26. Crosfill ML, Widdicombe JG. Physical characteristics of the chest and lungs and the work of breathing in different mammalian species. *J Physiol Lond* 1961; 158: 1–14.