Effects of Recruitment Maneuver and Positive End-expiratory Pressure on Respiratory Mechanics and Transpulmonary Pressure during Laparoscopic Surgery

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ABSTRACT

Background: The authors tested the hypothesis that during laparoscopic surgery, Trendelenburg position and pneumoperitoneum may worsen chest wall elastance, concomitantly decreasing transpulmonary pressure, and that a protective ventilator strategy applied after pneumoperitoneum induction, by increasing transpulmonary pressure, would result in alveolar recruitment and improvement in respiratory mechanics and gas exchange.

Methods: In 29 consecutive patients, a recruiting maneuver followed by positive end-expiratory pressure 5 cm H_2O maintained until the end of surgery was applied after pneumoperitoneum induction. Respiratory mechanics, gas exchange, blood pressure, and cardiac index were measured before (T_{BSL}) and after pneumoperitoneum with zero positive end-expiratory pressure (T_{preOLS}), after recruitment with positive end-expiratory pressure ($T_{postOLS}$), and after peritoneum desufflation with positive end-expiratory pressure (T_{end}).

Results: Esophageal pressure was used for partitioning respiratory mechanics between lung and chest wall (data are mean ± SD): on T_{preOLS}, chest wall elastance (E_{cw}) and elastance of the lung (E_L) increased (8.2 ± 0.9 *vs.* 6.2 ± 1.2 cm H_2O/L, respectively, on T_{BSL}; P = 0.00016; and 11.69 ± 1.68 *vs.* 9.61 ± 1.52 cm H_2O/L on T_{BSL}; P = 0.0007). On $T_{postOLS}$, both chest wall elastance and E_L decreased (5.2 ± 1.2 and 8.62 ± 1.03 cm H_2O/L, respectively; P = 0.00015 *vs.* T_{preOLS}), and PaO_2/inspiratory oxygen fraction improved (491 ± 107 *vs.* 425 ± 97 on $T_{preOLS;}$ P = 0.008) remaining stable thereafter. Recruited volume (the difference in lung volume for the same static airway pressure) was 194 ± 80 ml. $Pplat_{RS}$ remained stable while inspiratory transpulmonary pressure increased (11.65 + 1.37 cm H_2O *vs.* 9.21 + 2.03 on T_{preOLS}; P = 0.007). All respiratory mechanics parameters remained stable after abdominal desufflation. Hemodynamic parameters remained stable throughout the study.

Conclusions: In patients submitted to laparoscopic surgery in Trendelenburg position, an open lung strategy applied after pneumoperitoneum induction increased transpulmonary pressure and led to alveolar recruitment and improvement of E_{cw} and gas exchange.

What We Already Know about This Topic

- Trendelenburg position and pneumoperitoneum worsen chest wall elastance and gas exchange during laparoscopic pelvic surgeries

What This Article Tells Us That Is New

- Open lung strategy, consisting of a recruitment maneuver followed by the application of positive end-expiratory pressure, in 29 consecutive American Society of Anesthesiologists 1 and 2 nonobese patients, led to significant alveolar recruitment and improved chest wall and lung elastance in all the patients

LAPAROSCOPY is a well-established procedure for pelvic gynecologic surgery often performed in Trendelenburg position.[1,2] To facilitate laparoscopic surgical manipulation, a pneumoperitoneum is usually induced through carbon dioxide inflation. Both the increase in abdominal pressure as a result of carbon dioxide inflation and the head down body position have been shown to impair the respiratory function during the procedure, mainly inducing atelectasis formation in the dependent lung regions.[1,3–6] The resulting decrease in functional residual capacity poses

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patients at risk of perioperative complications, particularly if they are obese and/or submitted to intricate surgical procedures.[4,7] In fact, ventilation at low lung volumes may generate tidal alveolar recruiting (*i.e.*, some alveolar units that are collapsed at end-expiration are cyclically reopened during tidal inflation), a mechanism generating an alveolar shear stress known as "atelectrauma".[8] Furthermore, in presence of atelectasis, delivering the tidal volume to the limited amount of patient lung parenchyma may induce alveolar stress and strain.[9] Both stress, strain and atelectrauma are main mechanisms underlying ventilator-induced lung injury.[9,10] Despite definitive evidences that ventilator-induced lung injury may play a role when ventilating normal lungs (as generally done during anesthesia) are lacking,[11] the hypothesis that a lung-protective ventilatory strategy should be applied during general anesthesia is sound and widely debated.[1,6,7,12–14]

Several ventilatory strategies aiming at improving arterial oxygenation and respiratory mechanics during laparoscopic surgery have been investigated: the application of positive end-expiratory pressure (PEEP) has been shown to counterbalance the diaphragm cranial shift increasing functional residual capacity and decreasing respiratory system elastance (E_{RS}).[12,13,15–17] Recently, translating the concept of lung-protective ventilatory strategy from the adult respiratory distress syndrome context,[9,14,18–20] the application of an "open lung" strategy consisting in a recruiting maneuver (RM) followed by the subsequent application of PEEP has been suggested to effectively reexpand pneumoperitoneum-induced atelectasis and improve oxygenation during laparoscopic surgery.[21–25] However, to our knowledge, the effects of the open lung strategy on respiratory mechanics partitioned between its chest wall and lung components (*i.e.*, the relative effect exerted by the open lung strategy on lung and chest wall mechanics) have not been thoroughly investigated.[26–28] This is of particular interest because classical physiologic concepts[29] and recent experimental and clinical data clearly show that chest wall mechanical impairment has a deep impact on the response to any open lung strategy.[9,16,20,30] In fact, during controlled positive pressure ventilation, the real lung-distending pressure is the transpulmonary pressure (P_L), that is, the difference between airways opening pressure (P_{AO}) and the pressure required to expand the chest wall,[28,29,31] clinically estimated by measuring esophageal pressure (P_{ES}) as a surrogate of pleural pressure.[32]

The hypothesis of the current study is that during laparoscopic gynecologic surgery, both the Trendelenburg position and pneumoperitoneum worsen chest wall elastance (E_{CW}), concomitantly decreasing P_L, and that an open lung strategy consisting of an RM followed by ventilation with PEEP applied after pneumoperitoneum induction, by increasing P_L, would result in alveolar recruitment and improvement in respiratory mechanics and gas exchange.

Materials and Methods

After approval of the Policlinico Riuniti, Foggia, Italy, ethics committee and written informed consent from each patient, the study was performed in consecutive patients scheduled to undergo elective gynecologic laparoscopic surgery from January to July 2011. Inclusion criteria were age more than 18 yr and American Society of Anesthesiology physical status I and II. Patients with preexisting lung or cardiac disease, pathologic lung function, or obesity (body mass index ≥ 30 Kg/m^2) were excluded from the study.

On their arrival in the operating room, patients were premedicated with midazolam 0.03–0.04 mg/kg. After applying standard monitoring device (electrocardiogram and pulse oximeter [Intellivue MP40 monitor, Philips, Boeblingen, Germany]), the radial artery was cannulated (Radial Artery Catheterization Set, Arrow International, Reading, PA) for continuous monitoring of blood pressure. The arterial line was connected to the FloTrac sensor and the Vigileo monitor (Edwards Life Sciences LLC, Irwine, CA, software version 03.10), which allows cardiac output and stroke volume estimation from the arterial pressure waveform and computes the stroke volume variation (SVV) in response to positive pressure mechanical ventilation as an index of cardiac preload and fluid responsiveness.[33]

Patients were given 8 ml/kg of normal saline intravenously before the induction of anesthesia and were then maintained with 5 $ml \cdot kg^{-1} \cdot h^{-1}$ of normal saline solution. Anesthesia was induced with propofol 2 mg/kg, fentanyl 3 ng/kg, and succinylcholine 1 mg/kg. After induction, the trachea was intubated with an endotracheal tube of appropriate size (Rushelit Rush AG Lab, Waiblingen, Germany). Anesthesia was maintained with an infusion of propofol 150–200 $\gamma \cdot kg^{-1} \cdot min^{-1}$, remifentanil 0.1–0.2 $\gamma \cdot kg^{-1} \cdot min^{-1}$, and cisatracurium 1.5 $\gamma \cdot kg^{-1} \cdot min^{-1}$. The level of anesthesia was assessed through bispectral index monitoring (Aspect A-2000®; Aspect Medical System, Newton, MA). The infusion rate of propofol was varied to target a bispectral index value between 50 and 60. The lungs were ventilated through a Servo Ventilator 900C (Siemens-Elema AB, Berlin, Germany) with a square flow waveform with a tidal volume (V_t) of 8 ml/kg ideal body weight, respiratory rate of 12 breath/min, inspiratory time of 33%, and an inspiratory pause of 20%. Patients were ventilated using oxygen in air with an inspiratory oxygen fraction set at 40% as needed to maintain the SaO_2 ≥ 95%. No PEEP was initially added.

An esophageal thin latex balloon-tipped catheter (Compliance catheter, Microtek Medical B.V. Zutphen, The Netherlands) was inserted through the mouth, advanced into the esophagus and connected by means of a polyethylene catheter to a pressure transducer (Digima-Clic, Nordlingen, Germany), to measure P_{ES}. The esophageal balloon was filled with 1–1.5 ml of air, and its correct positioning in the lower third of the esophagus was verified according to literature, by allowing a brief period of spontaneous breathing after the induction of anesthesia and comparing the esophageal and the airway opening pressure traces.[32] In addition, the correct balloon position in the lower third of the esophagus was confirmed by the presence of appropriate esophageal

pressure deflections induced by mechanical ventilation and moderate push on the abdomen. All the data were controlled on the computer software of recording and analysis (ICU Lab, KleisTEK Engineering, Bari, Italy) through an optimal waveform.

A standardized protocol for hemodynamic management was applied to ensure fluid volume optimization. In brief, if SVV was lower than 13%, no additional fluids were given, whereas if SVV was higher than 13%, additional boluses of 250 ml of artificial colloid were infused over 15–20 min. After each bolus, SVV was re-evaluated, and a further bolus was administered if stroke volume increased by more than 10%, until reaching an SVV lower than 13%.[33]

Study Protocol

About 15 min after pneumoperitoneum induction, if the patients were hemodynamically stable, that is, with mean blood pressure ≥ 80 mmHg, heart rate ≥ 60 beats/min, and SVV < 13%, the open lung strategy was applied as already described.[34] In brief, the ventilator was switched to pressure-control ventilation, inspiratory time was increased to 50%, the peak inspiratory pressure gradient (above PEEP) was set at 20 cm H_2O, and PEEP was progressively increased to obtain a stepwise increase of peak inspiratory to 30, 35, and 40 cm H_2O every three breaths. The final recruiting pressure of 40 cm H_2O was applied for six breaths. After ward, the ventilator switched again to the volume-control ventilation with baseline settings but with a PEEP level of 5 cm H_2O that was maintained after abdominal deflation until the end of surgery. Overall, the RM procedure lasted approximately 1 min. Measurements were obtained (1) 15 min after anesthesia induction in supine position before inducing the pneumoperitoneum (T_{BSL}); (2) 15 min after pneumoperitoneum induction (abdominal carbon dioxide inflation to obtain and intra-abdominal pressure of 10 mmHg) with the patient positioned at 20° head down (T_{preOLS}); (3) 20 min after the application of the open lung strategy ($T_{postOLS}$); and (4) at the end of surgery, after abdominal deflation and in supine position with a PEEP level of 5 cm H_2O.

Measurements

Hemodynamic and respiratory mechanics parameters were recorded, digitized, and collected on a personal computer through a 12-bit analog-to digital converter board (DAQ-Card 700; National Instrument, Austin, TX) at a sample rate of 200 Hz (ICU Lab, KleisTEK Engineering).

Blood pressure was measured through a radial catheter connected to the pressure transducer of the MP40 monitor. Hemodynamic parameters obtained through the Vigileo monitor included stroke volume, cardiac output, and SVV, whereas stroke volume index and cardiac index were calculated using standard formulae. All intravascular pressure measurements were zeroed to the mid-axillary line. Analysis of arterial blood gases was performed (ABL 330; Radiometer, Copenhagen, Denmark).

Flow was measured with a heated pneumotachograph (Fleisch no. 2; Fleisch, Lausanne, Switzerland), connected to a differential pressure transducer (Diff-Cap, ±1 cm H_2O; Special Instruments, Nordlingen, Germany) inserted between the Y-piece of the ventilator circuit and the endotracheal tube. The pneumotachograph was linear over the experimental range of flow. Volume was obtained by numerical integration of the flow signal. P_{AO} was measured proximal to the endotracheal tube with a pressure transducer (Special Instruments Digima-Clic ± 100 cm H_2O; Nordlingen, Germany). The difference between the level of the PEEP set on the ventilator (read as the P_{AO} value at the end of a regular breath) (PEEPexternal) and the pressure in P_{AO} during a 3- to 5-s end-expiratory occlusion ($PEEPtot_{RS}$) was measured and regarded as the static intrinsic PEEP of the respiratory system according to Pepe.[35] The end-expiratory occlusion was performed through the expiratory hold on the Servo 900C ventilator.

Static E_{RS} was calculated using:

$$E_{RS} = Pplat_{RS} - PEEPtot_{RS}/V_t \quad (1)$$

where $Pplat_{RS}$ is the value of P_{AO} obtained by adding an end-inspiratory pause of 2–3 s through the inspiratory hold of the ventilator.

Static E_{CW} was calculated as:

$$E_{CW} = (Pplat_{CW} - PEEPtot_{CW})/V_t, \quad (2)$$

where $Pplat_{CW}$ is the value of P_{ES} obtained contemporaneously to $Pplat_{RS}$ during the end-inspiratory pause. Lung static elastance (E_L) was calculated as:

$$E_L = E_{RS} - E_{CW} \quad (3)$$

Transpulmonary end-inspiratory pressure ($Pplat_L$) was computed, according to Gattinoni *et al.*[9,29] as:

$$Pplat_L = Pplat_{RS} \times E_L/(E_L + E_{CW}) \quad (4)$$

Transpulmonary end-expiratory pressure ($PEEPtot_L$) was computed during the end-expiratory pause as:

$$PEEPtot_L = PEEPtot_{RS} \times E_L/(E_L + E_{CW}) \quad (5)$$

Alveolar recruitment was measured though a physiologic method originally described by Ranieri *et al.*,[36] subsequently applied in several physiologic clinical studies[18,28,34,37–39] and recently validated by Dellamonica *et al.*[40] In brief, for each experimental condition, we first measured the quasistatic volume–pressure curves of the respiratory system through the low-flow inflation technique as originally described by Lu *et al.*[41] and subsequently measured the delta end-expiratory

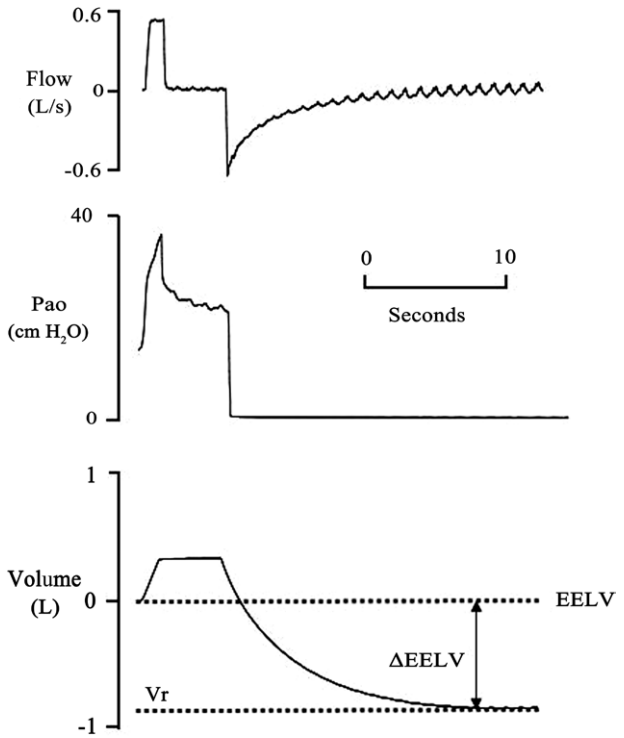

Fig. 1. Measurement of the difference (ΔEELV) between EELV during mechanical ventilation and elastic equilibrium V_r. Records of Flow, Pao, and lung volume are shown. To allow for complete lung emptying to V_r, the patient was disconnected from the ventilator circuit distally from the pneumotachograph after a prolonged end-inspiratory pause and a prolonged (15–20 s) expiration to atmospheric pressure was allowed. EELV = end-expiratory lung volume; ΔEELV = delta end-expiratory lung volume; Pao = airway opening pressure; V_r = volume of the respiratory system.

lung volume as the difference between the end-expiratory lung volume during mechanical ventilation and the elastic equilibrium volume of the respiratory system at zero end-expiratory pressure, or relaxation volume (V_r) or functional residual capacity. The delta end-expiratory lung volume was measured by disconnecting the patient from the ventilator circuit distally from the pneumotachograph and allowing a prolonged expiration (15–20 s; fig. 1). Of note, this method does not measure V_r *per se* but assumes that any alveolar recruitment obtained by applying RM or PEEP does not modify V_r and that therefore V_r remains the same in all the experimental conditions.[36] Knowing the respective delta end-expiratory lung volume, the quasistatic volume–pressure curves obtained before and after the RM were plotted on the same volume–pressure axis and referred to V_r (fig. 2). To do so, each volume–pressure curve started at a point corresponding to $PEEP_{TOT}$ (referred to the X axis) and to the end-expiratory lung volume (referred to the Y axis) (fig. 2). The recruited volume (the gas volume of collapsed or fluid-filled alveolar units eventually reaerated by the open lung strategy) was computed as the difference in lung volume at the same static P_{AO} read on the two pressure–volume curves (fig. 2).

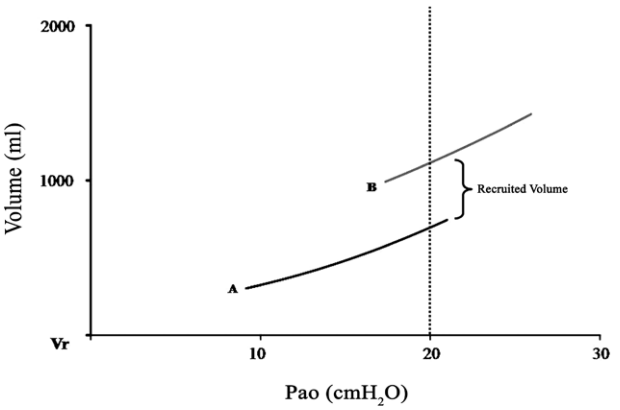

Fig. 2. Illustration of the method used to measure recruited volume. Quasistatic volume–pressure relationship of the respiratory system during low-flow tidal inflation, obtained during the lower positive end-expiratory pressure (PEEP) strategy (*black* curve) and the higher PEEP strategy (*gray* curve) were referred to the static relaxation V_r and plotted on the same volume–pressure axis. The volume at the starting point of each curve is the end-expiratory lung volume during lower and higher PEEP strategy (points A and B, respectively). The *dotted line* indicates the amount of alveolar recruitment measured as the volume difference at the same pressure (20 cm H2O) between the two curves. V_r = volume of the respiratory system; Pao = airway opening pressure.

Total airways resistances were calculated as the difference between $Ppeak_{RS}$ and $Pplat_{RS}$ divided by the inspiratory airflow.[37,41]

Statistical Analysis

A sample size calculation was performed using data from the study by Grasso *et al.*[18] on the effects of RMs in patients with adult respiratory distress syndrome ventilated with protective ventilatory strategy. On the basis of these data, the significant recruitment was designated as a 100-ml increase in end-expiratory lung volume with an SD of 116.7. By using a one-sample, one-sided test, the sample size calculated was of 23 patients; this number was increased to 31 to allow for an expected dropout of around one third of patients and was used for patient enrolment. The α and β errors for the sample size were chosen as 0.05 and 90%, respectively. Because only two patients dropped out from the analysis (fig. 3), the study power was 99%.

Statistical comparison of respiratory mechanics, hemodynamic, and gas exchange data was performed between the four study steps: data were tested for normal distribution by the Kolmogorov–Smirnov goodness-of-fit test and are presented as mean ± SD. Data analysis was performed using repeated-measures one-way ANOVA; if significant, the test of Tukey was applied for *post hoc* comparison between the different experimental conditions. A *P* value of less than 0.05 was considered statistically significant. Statistical analysis was performed using Statistica 8.0 (Statsoft Italia srl 2008, Vigonza Padova, Italy).

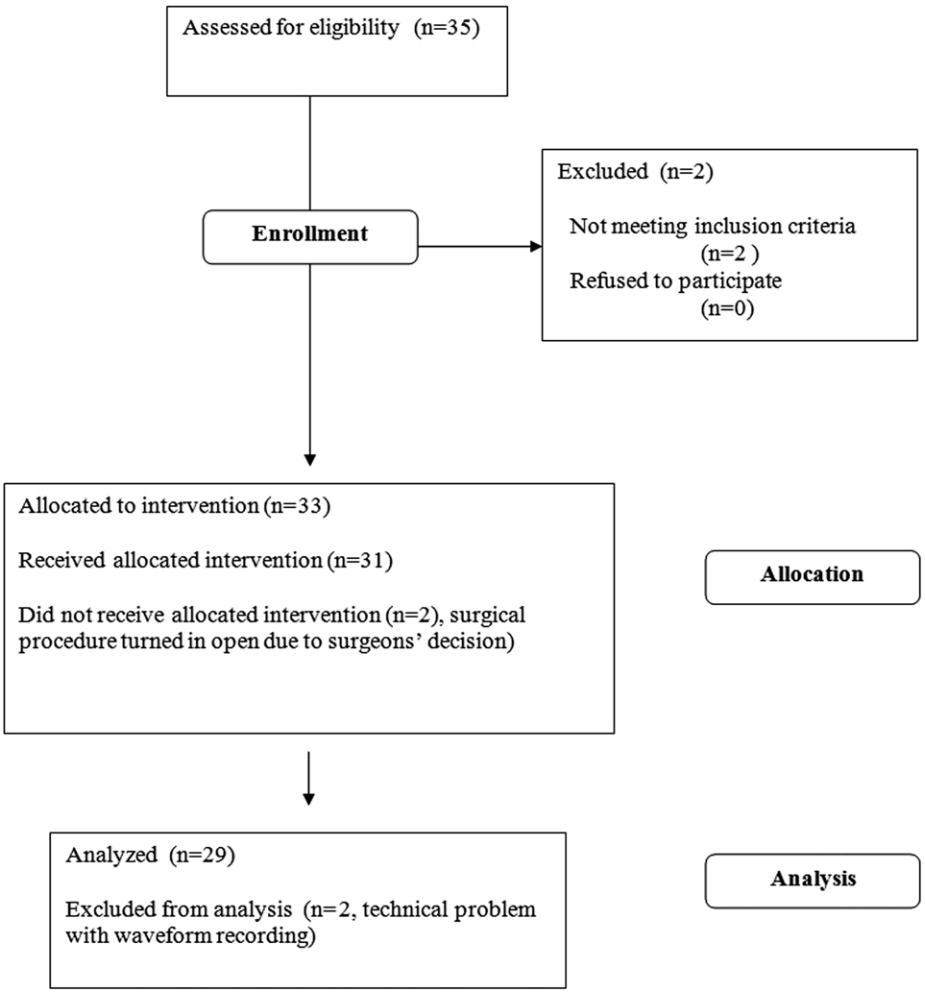

Fig. 3. Flow diagram of the progress through the phases of the trial.

Results

Twenty-nine of 35 patients initially candidate for enrolment were included in the study. The enrolment flow diagram is reported in figure 3. Demographic characteristics of the population studied are presented in table 1. The study was completed successfully in every patient without complications or adverse events as a result of the study protocol. Data are presented as mean ± SD).

Table 1. Patients Demographic Characteristics

Age, yr	39 ± 13
ASA I n(%)	27 (95%)
II n(%)	2 (5%)
BMI, Kg/m^2	23 ± 2.41
Duration of surgery, min	
Surgery n(%)	90 + 15
Ovarian cyst excision	23 (80%)
Hysterectomy	6 (20%)

Data are shown as mean ± SD and as percentage as appropriate. ASA = American Society of Anesthesiologists; BMI = body mass index.

Respiratory Mechanics

Compared with the baseline (table 2), the induction of pneumoperitoneum and the Trendelenburg position worsened E_{CW} and E_L resulting in an overall increase of E_{RS} (P = 0.00015 on T_{preOLS} *vs.* T_{BSL}, respectively). The open lung strategy decreased both E_{CW} and E_L (P = 0.0007 on $T_{postOLS}$ *vs.* T_{preOLS}, for both parameters). Both end-inspiratory and end-expiratory transpulmonary pressure ($Pplat_L$ and $PEEPtot_L$, respectively) decreased at T_{preOLS} compared with T_{BSL} (P = 0.008) and both increased on $T_{postOLS}$ (P = 0.008 *vs.* T_{preOLS}).

Figure 4 shows the quasistatic volume–pressure curves of the respiratory system measured immediately before and 20 min after the application of open lung strategy and plotted on the same volume–pressure axis (see Materials and Methods), in a representative patient. Of note, the curve obtained during the open lung strategy is shifted upward on the volume axis, clearly indicating the occurrence of alveolar recruitment. Overall, the open lung strategy resulted in a

Table 2. Breathing Pattern and Gas Exchange Parameters during the Different Experimental Conditions

	Baseline (ZEEP)	Pneumo Pre-OLS (ZEEP)	Pneumo Post-OLS (PEEP 5)	End Surgery (PEEP 5)	Repeated-Measures One-Way ANOVA (*P*)
V_t, ml/kg	8.96 ± 2.10	8.62 ± 1.51	8.73 ± 2.43	9.16 ± 2.20	0.141
RR, beats/min	11 ± 1	12 ± 1	12 ± 1	12 ± 1	0.625
$Pplat_{RS}$, cm H_2O	13 ± 1.71	14.5 ± 2.13 *	15.3 ± 1.67*	14.7 ± 1.83 *	0.008
$Pplat_{CW}$, cm H_2O	2.4 +1.38 ‡	5.31+2.33	3.7+1.53 ‡	3.1+1.34 ‡	0.007
$Pplat_{L}$, cm H_2O	10.59 ± 1.35	9.21 ± 2.03 *	11.65 ± 1.37 *†	11.57 ± 1.64*†	0.008
Raw_{TOT}, cm $H_2O{\cdot}L^{-1}{\cdot}s^{-1}$	7.66 ± 0.64	11.25 ± 0.91 *	8.52 ± 0.86 *†	7.11 ± 0.73 *†	0.00015
$PEEPi_{RS}$, cm H_2O	3.19 ± 1.59	3.59 ± 1.30	2.06 ± 1.06 *†	1.91 ± 0.99 * †	0.0005
$PEEPtot_{RS}$, cm H_2O	3.19 ± 1.59	3.59 ± 1.30	7.06 ± 1.06 *†	6.91 ± 0.99 *†	0.00015
$PEEPtot_{L}$, cm H_2O	4.6 ± 1.5	2.8 ± 1.9 *	6.51 ± 1.31*†	6.78 ± 1.22*†	0.008
E_{RS}, cm H_2O/L	15.8 ± 1.5	19.9 ± 2.1*	13.9 ± 1.9 *†	12.5 ± 1.9 *†	0.00015
E_{CW}, cm H_2O/L	6.2 ± 1.2	8.2 ± 0.9 *	5.2 ± 1.2 *†	4.8 ± 1.1 *†	0.007
E_{L}, cm H_2O/L	9.61 ± 1.52	11.69 ± 1.68 *	8.62 ± 1.03 *†	7.72 ± 1.11*†	0.007
E_{CW}/E_{RS}	0.39 + 0.07 ‡	0.41+ 0.04	0.37 + 0.05 ‡	0.38 + 0.05 ‡	0.008
pH	7.37 ± 0.08	7.33 ± 0.06	7.32 ± 0.05	7.34 ± 0.07	0.625
$PaCO_2$, mmHg	36.2 ± 5.8 ‡	41.1 ± 5.3	35.4 ± 5.3 ‡	38.9 ± 6.2 ‡	0.008
$EtCO_2$, mmHg	32.9 ± 4.43	34.36 ± 4.36	36.68 ± 4.86 *	37.13 ± 4.43	0.981
PaO_2/FIO_2	493 ± 127‡	425 ± 97	465 ± 133‡	492 ± 108‡	0.008

Repeated-measures one-way ANOVA between the four steps: Test of Tukey: * $P = 0.008$ *vs.* baseline; † $P = 0.00018$ *vs.* pneumo pre-OLS; ‡ $P = 0.007$ *vs.* pneumo pre-OLS.

E_{CW} = chest wall elastance; E_L = lung elastance; E_{RS} = respiratory system elastance; FiO_2 = inspiratory oxygen fraction; PaO_2 = arterial oxygen tension; PEEP = positive end-expiratory pressure; $PEEPi_{RS}$ = static intrinsic PEEP; $PEEPtot_L$ = transpulmonary expiratory pressure; $PEEPtot_{RS}$ = total PEEP; $Pplat_{CW}$ = chest wall plateau pressure; $Pplat_L$ = inspiratory transpulmonary pressure; $Pplat_{RS}$ = respiratory system plateau airways pressure; Raw_{TOT} = inspiratory airway resistances; RR = respiratory rate; V_t = tidal volume; OLS = open lung strategy; ZEEP = zero end-expiratory pressure.

significant alveolar recruitment in all patients (194 + 80 ml, range 65–323 ml).

Gas Exchanges

The PaO_2/inspiratory oxygen fraction ratio (table 2) worsened at T_{preOLS} compared with T_{BSL} ($P = 0.008$) and returned to baseline values at $T_{postOLS}$ ($P = 0.008$ *vs.* T_{preOLS}). Despite alveolar minute ventilation remained unchanged, $PaCO_2$ increased at T_{preOLS} compared with T_{BSL} ($P = 0.008$) and returned to baseline value after the application of the open lung strategy.

Hemodynamics

During the RM (table 3), cardiac index decreased by approximately 20% compared with T_{preOLS} ($P = 0.0007$), returned to T_{preOLS} values immediately after the RM and remained stable thereafter. SVV was 8.10 ± 2.37% on T_{BSL} and remained stable throughout all the experimental conditions. Mean blood pressure and heart rate remained stable throughout the study.

Discussion

Our data show that in patients submitted to pelvic laparoscopic surgery in Trendelenburg position under general anesthesia, the application of an RM followed by PEEP after pneumoperitoneum induction leads to alveolar recruitment and improvement of chest wall and lung elastance.

Because the diaphragm is mechanically coupled to the abdominal wall, any increase in abdominal pressure may decrease functional residual capacity.[28,42] During laparoscopy, the raised abdominal pressure distends the abdominal wall, increases its elastance, shifts cranially the diaphragm, and moves a large part of the ventilation-related volume changes through the rib cage.[7,26,27] The Trendelenburg position often needed to perform pelvic surgery facilitates the transmission of the abdominal weight to the lung parenchyma. Our data confirm the pneumoperitoneum and Trendelenburg position–related worsening of respiratory mechanics: E_{CW} increased by 30%, whereas E_L increased by 20%, and furthermore, static intrinsic PEEP of the respiratory system and total airways resistances increased compared with those recorded at baseline, after the induction of anesthesia in supine position. On the other hand, we did record a worsening in PaO_2/inspiratory oxygen fraction ratio that although significant was never clinically relevant (table 2). However, severe hypoxemia is not an hallmark of pneumoperitoneum-induced worsening of respiratory function during laparoscopy in healthy subjects, and moreover, oxygenation has been shown to be a poor indicator of the extent of pneumoperitoneum-induced lung collapse.

Several strategies have been proposed to counterbalance the derangements in respiratory mechanics induced by pneumoperitoneum during laparoscopic surgery. External PEEP

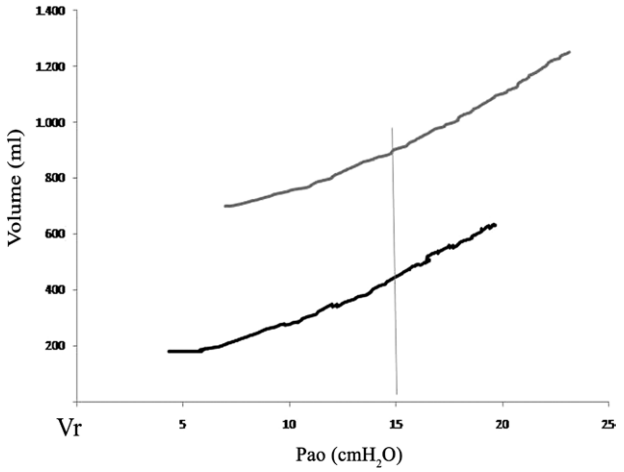

Fig. 4. Quasistatic volume–pressure relationship of the respiratory system during low-flow inflation, obtained before (*black* curve) and after (*gray* curve) the recruiting maneuver (RM) in a representative patient. The elastic equilibrium volume of the respiratory system was used as a reference for the volume–pressure curves. The volume–pressure curve measured after the RM is shifted upward along the volume axis, suggesting alveolar recruitment. Recruited volume, measured as the difference in lung volume for the same static Pao (15 cm H_2O), is indicated by the dotted line. Please note that, on the curve pre-OLS, PEEPtot was higher than zero (4.34 cm H_2O) indicating the presence of air trapping. Because in the RM external PEEP was applied, in this condition, PEEPtot is equal to PEEPi. On the other hand, the curve obtained after the RM (with a PEEP of 5 cm H_2O) shows a PEEPtot of 6.79 cm H_2O, with a PEEPi of 1.79 cm H_2O. OLS = open lung strategy; Pao = airway opening pressure; PEEP = positive end-expiratory pressure; V_r = volume of the respiratory system.

alone has been repeatedly shown to have beneficial but transient effects, whereas recently Valenza *et al.*[22] showed that the combination of beach chair position and PEEP in morbidly obese patients during pneumoperitoneum improves oxygenation. This strategy, however, is not feasible in surgical settings in which other positions are needed. Futier *et al.*[24] demonstrated that an RM applied after pneumoperitoneum induction and followed by the application of PEEP provides significant improvements in E_{RS} and oxygenation both in healthy and obese patients. We confirm the ability of a recruiting strategy to induce alveolar recruitment accompanied by a substantial improvement of respiratory mechanics in patients undergoing laparoscopic surgery in Trendelenburg position. Our focus in partitioning respiratory mechanics between lung and chest wall revealed that the amount of pressure applied to the airway opening that was dissipated to distend the chest wall increased after the induction of pneumoperitoneum in Trendelenburg position, resulting in a decrease in both end-inspiratory and end-expiratory P_L. As expected, the RM reopened collapsed lung units and subsequently PEEP kept them opened by increasing P_L. However, we must point out that the physiologic method used in the current study to estimate alveolar recruitment[36,37] has been shown to underestimate alveolar recruitment in some instances (see below for the limitations).[43]

Interestingly, we document that the open lung strategy decreased E_{CW}. To explain this result, we speculate that PEEP, applied after the RM, at least partially counterbalanced the diaphragm cranial shift induced by pneumoperitoneum and Trendelenburg position and that the corresponding lung expansion lead to a more physiologic chest wall shape leading to the observed reduction in E_{CW}.

It has recently been demonstrated in patients with adult respiratory distress syndrome[19,20,30,31] that customizing to PEEP and RM accommodate for individual variations in lung and chest wall mechanics, to target an "optimal" end-expiratory[20,31] or end-expiratory P_L[9,30,38], improves lung aeration and oxygenation. In patients with adult respiratory distress syndrome, targeting either a $PEEPtot_L$ between 0 and 10 cm H_2O and or a $Pplat_L$ threshold of 24 cm H_2O has been shown to maximize oxygenation without inducing barotrauma.[9,20,30] Data on P_L in healthy patients submitted to general anesthesia for laparoscopic surgery are scanty. We show that monitoring P_L over time would be important to better understand the complex interplay between respiratory mechanics, anesthesia, pneumoperitoneum, and Trendelenburg position. An intriguing hypothesis is that optimizing P_L on individual basis during laparoscopic surgery would be a physiologically sound strategy to obtain maximal alveolar recruitment and respiratory mechanics improvement. Nevertheless, we must point out that our study was not designed to test this hypothesis, and instead, we applied a standardized open lung strategy successfully tested in a different context.[34] Further studies are required to define the "optimal" end-expiratory and end-inspiratory P_L targets and to test a P_L-based ventilatory strategy in this setting.

Table 3. Hemodynamic Parameters during the Different Experimental Conditions

	Baseline	Pneumo Pre-OLS	OLS	Pneumo Post-OLS	End Surgery	ANOVA (*P*)
HR, beats/min	71 ± 12	67 ± 7	66 ± 15	68 ± 9	69 ± 14	0.625
Mean blood pressure, mmHg	87 ± 18	82 ± 14	89 ± 12	81 ± 11	109 ± 13	0.141
CI, l/m²	2.8 ± 0.3	3.04 ± 0.7	2.4 ± 0.6 *†	3.1 ± 0.7	3.2 ± 0.2	0.008
SVV, %	8.10 ± 2.37	9.08 ± 3.94	9.7 ± 2.75	10.41 ± 3.5	9.81 ± 2.42	0.141

Repeated-measures one-way ANOVA between the time points: Test of Tukey: * $P = 0.008$ *vs.* baseline; † $P = 0.008$ *vs.* end surgery.
CI = cardiac index; HR = heart rate; OLS = open lung strategy; SVV = stroke volume variation.

Despite the increase in intrathoracic pressure induced by the open lung strategy, patients' hemodynamic status remained stable. Cardiac output was only transiently affected by the RM itself (it decreased by about 20% during the maneuver and returned to its baseline values immediately after, despite the application of PEEP; table 3). An important issue to explain our finding is that, by protocol, in our patients cardiac preload was optimized before the open lung strategy (see Materials and Methods).

The current study has some limitations: (1) our patients were otherwise healthy women undergoing elective surgery and therefore more studies are required to study the effects of the open lung strategy on partitioned respiratory mechanics in patients with coexisting cardiopulmonary diseases; (2) some inhomogeneity in the surgical procedures among our patients must be taken into account when interpreting our data; (3) we cannot prove that, after the RM and the subsequent PEEP application, the position of the esophageal balloon in the esophagus remained the same. Nevertheless, at least to our knowledge, in all the previous clinical studies, after the correct balloon position was checked at baseline, the esophageal balloon position was not changed thereafter because it was assumed that the application of RM and/or different PEEP levels did not influence P_{ES} measurements.[9,20,27–31,38] Furthermore, in our study, according to the "Gattinoni method,"[9,29] we did not trust on the absolute P_{ES} values, but instead, we used the positive tidal P_{ES} excursions to calculate E_{CW} and subsequently calculated P_L based on the ration between E_{CW} and E_L.[29] Furthermore, for the same reason, our P_L measurement is likely less prone than the absolute P_{ES} to be influenced by different degrees of lung atelectasis; (4) in our study, we applied a standardized open lung strategy consisting in an RM followed by PEEP 5 cm H_2O. Although our data show that such strategy resulted in significant alveolar recruitment and improvement in lung mechanics, we must point out that the PEEP level applied in our study was likely inadequate to maximize alveolar recruitment. As we discussed above, tailoring PEEP to optimize alveolar recruitment is an important issue that deserves further studies; (5) to estimate open lung strategy–induced alveolar recruitment, we used the method originally proposed by Ranieri *et al.*[36] We point out that although this method is useful to indicate that recruitment occurred, it may fail to precisely estimate the amount of alveolar recruitment in some instances, as recently shown by Patroniti *et al.*[43] We acknowledge this as a limitation of our study; and (6) the FloTrac/Vigileo has not been thoroughly validated in the surgical context and furthermore is validated to monitor the trend of cardiac output rather than its absolute values.[33,44]

In conclusion, we document the physiologic effects of a ventilatory strategy aiming at reversing pneumoperitoneum-induced alveolar collapse in patients with normal lung function undergoing laparoscopic surgery in Trendelenburg position. It should be emphasized that this was a physiologic study not designed to evaluate the impact of the tested open lung strategy on meaningful outcome parameters and therefore any extrapolation of our results to the clinical scenario must be conducted with caution.

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