

Respiratory and haemodynamic effects of volume-controlled vs pressure-controlled ventilation during laparoscopy: a cross-over study with echocardiographic assessment

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Background. The effects of pressure-controlled (PC) ventilation on the ventilatory and haemodynamic parameters during laparoscopy procedures had not been carefully assessed. This prospective cross-over study was undertaken to compare how volume-controlled (VC) and PC modes could affect pulmonary mechanics, gas exchange, and cardiac function in patients undergoing laparoscopy.

Methods. Twenty-one patients undergoing laparoscopic urological procedures had their lungs ventilated at the beginning with VC ventilation. PC ventilation was instituted at the end of the VC sequence. Ventilator settings were adjusted to keep tidal volume, respiratory rate, and $F_{I_{O_2}}$ constant in every mode. A complete set of ventilatory, haemodynamic, and gas exchange parameters was obtained under VC after 40 min of pneumoperitoneum and 20 min after switching for PC. Transoesophageal echocardiography was performed in order to evaluate systolic and diastolic function of the heart.

Results. When VC was switched to PC, peak airway pressure decreased [mean (SD) 32 (6) vs 27 (6) cm H₂O; $P < 0.0001$], peak inspiratory flow increased [17 (3) vs 48 (8) litre min⁻¹; $P < 0.0001$], and dynamic compliance improved [+15 (8)%]. No difference was noted for static airway pressure, static compliance, and arterial oxygenation. No significant change could be demonstrated in the systolic [left ventricular end-systolic wall stress 66 (16) vs 63 (14) $\cdot 10^3$ dyn cm⁻² m⁻²] or diastolic function [early diastolic velocity 10.3 (2.5) vs 10.5 (2.7) cm s⁻¹].

Conclusions. In this study, no short-term beneficial effect of PC ventilation could be demonstrated over conventional VC ventilation in patients with pneumoperitoneum.

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Laparoscopic surgery has increasingly been used during the last decade in many surgical procedures.^{1–3} Cardiopulmonary physiology and pathophysiology of pneumoperitoneum is now well understood.⁴ Besides cardiovascular effects, one of the most obvious ventilatory consequences is the increased peak airway pressure (P_{peak}). In order to limit this increase in P_{peak} , the anaesthetist could change the respiratory rate (RR), tidal volume, or change from volume-controlled (VC) to pressure-controlled (PC) ventilation; PC ventilation is now frequently used in the operating room in the management of patients with elevated

P_{peak} , despite an incomplete understanding of its ventilatory and haemodynamic effects, or its potential complications.^{1,5}

The PC ventilation is a time-cycled mode in which square waves of pressure are applied and released by means of a decelerating flow.⁶ The decelerating flow often results in a higher mean inflation pressure when compared with constant flow.^{7,8} Therefore, with the concomitant presence of pneumoperitoneum, a complex cardiopulmonary response can occur.⁹ In acute lung injury, PC vs VC ventilation has been well studied.^{10–13} Although a decrease in P_{peak} has been reported by all the studies, no difference

has been found for plateau pressure (P_{plat}), or for respiratory system compliance, when the tidal volumes are kept constant.^{10–12} Likewise, there was only minimal differences in gas exchange and haemodynamics studied by right heart catheterization.^{10–12}

As far as we know, the effects of PC ventilation on the ventilatory and haemodynamic parameters during laparoscopy procedures have not been assessed carefully by controlled studies. The aim of this study was, therefore, to compare how VC and PC modes could affect pulmonary mechanics, gas exchange, and cardiac function in patients undergoing laparoscopy. We used transoesophageal echocardiography (TOE) for non-invasive evaluation of systolic and diastolic function.

Methods

This study was approved by our hospital ethics committee (Comité de Protection des Personnes se Prêtant à la Recherche Biomédicale, CHU de Pointe-à-Pitre, Pointe-à-Pitre, France; reference no. 06-04), and informed consent was obtained from the patients. All data used in subsequent analyses were anonymized.

During a 3-month period, 23 consecutive patients undergoing radical prostatectomy or lymphadenectomy by laparoscopy were prospectively enrolled in the study. Patients with left atrial dilation (>4.0 cm), left ventricular (LV) dilation [LV end-diastolic (ED) internal dimensions >5.7 cm], decreased shortening fraction ($<31\%$), regional wall motion abnormalities, valvular heart disease, dilated cardiomyopathy, and pericardial disease were excluded from the study. Patients who were ASA III or IV, or had oesophageal disease or dysphagia, were also excluded. Patients with intraoperative bleeding defined as the need for rapid volume expansion or blood transfusion before completion of the protocol and with haemodynamic instability defined as mean arterial pressure variation $>10\%$ before completion of the protocol were excluded.

Anaesthetic management and intraoperative care were standardized. Premedication, hydroxyzine, was given orally 2 h before induction of anaesthesia, and all cardiovascular drugs, except angiotensin receptor blockers, were taken with the premedication. Propofol was infused using a target-controlled infusions system (Diprifusor-TCI[®], ZenecaTM). Target plasma concentration for induction and maintenance of anaesthesia was $4\text{--}6 \mu\text{g ml}^{-1}$. Analgesia was provided by sufentanil $0.3 \mu\text{g kg}^{-1}$ i.v. bolus followed by a continuous infusion of $0.3 \mu\text{g kg}^{-1} \text{h}^{-1}$. Orotracheal intubation was facilitated by cisatracurium besylate 0.15 mg kg^{-1} , and the muscle relaxation was maintained with continuous infusion of $0.15 \text{ mg kg}^{-1} \text{h}^{-1}$. All patients received 500 ml of colloids before induction of pneumoperitoneum; crystalloids were then infused during operation at a basal rate of $10 \text{ ml kg}^{-1} \text{h}^{-1}$. Inspired oxygen fraction ($F_{\text{I}_{\text{O}_2}}$) was set at 60% in air and

minute ventilation adjusted to maintain end-tidal CO_2 ($P_{\text{E}'\text{CO}_2}$) between 3.7 and 4.2 kPa. Carbon dioxide pneumoperitoneum was induced with 14 mm Hg intra-abdominal pressure in the supine position after a 20° Trendelenburg position. This position was maintained throughout the surgical procedure.

The patients were first studied during VC with a constant square waveform and an inspiratory/expiratory ($I:E$) ratio of 1:2 without inspiratory plateau. Respiratory and echocardiographic data were collected 10 min after induction of anaesthesia ($\text{VC}_{\text{baseline}}$, T1), 15 min after pneumoperitoneum (VC_{pno} , T2), and 40 min after pneumoperitoneum which was the period retained for comparison with PC ($\text{VC}_{\text{protocol}}$, T3). PC ventilation with a decelerating flow waveform and an $I:E$ ratio 1:2 was started at the end of the VC sequences ($\text{PC}_{\text{protocol}}$, T4). Peak pressure was set to match expired V_t measured during $\text{VC}_{\text{protocol}}$. Because PC allows some variability in V_t with changes in impedance, a value of $\pm 5\%$ was tolerated. After a stabilization period of 20 min, respiratory and echocardiographic data were collected.

All patients were ventilated with a Horus 4 ventilator (Taema, Antony, France). During the study period, $F_{\text{I}_{\text{O}_2}}$, RR, $I:E$, and tidal volume (V_t) were held constant. External positive end-expiratory pressure (PEEP) was not added.

The magnitude of P_{peak} and mean airway pressure (P_{mean}) was obtained directly from the ventilator. Static lung compliance was measured during a 2 s inspiratory and expiratory hold for calculation of P_{plat} and intrinsic PEEP. Total respiratory compliance (C_{st}) was calculated with the following equation: $C_{\text{st}} = V_t / (P_{\text{plat}} - \text{total PEEP})$. Total respiratory dynamic compliance (C_{dyn}) was calculated with the following equation: $C_{\text{dyn}} = V_t / (P_{\text{peak}} - \text{total PEEP})$. Arterial blood was sampled for measurement of $P_{\text{a}_{\text{O}_2}}$, $P_{\text{a}_{\text{CO}_2}}$, and pH and lactates only at $\text{VC}_{\text{protocol}}$ T3 and $\text{PC}_{\text{protocol}}$ T4 time intervals. Physiologic dead space (V_d/V_t) was estimated according to the Hardman and Aitkenhead equation: $V_d/V_t = 1.14 \times (P_{\text{a}_{\text{CO}_2}} - P_{\text{E}'\text{CO}_2}) / P_{\text{a}_{\text{CO}_2}} - 0.005$.¹⁴

TOE was performed with an HDI[®] 5000 imaging system model (ATL ultrasound, Bothell, WA, USA) equipped with a multiplane 4.0–7.0 MHz probe. The probe was inserted into the oesophagus, after the induction of anaesthesia and before surgery. To minimize the risk of gastro-oesophageal mucosal injury from pressure of the transducer probe, flexion of the probe was intermittently released and the transducer probe inactivated.

Standard transgastric and upper and lower oesophageal views were obtained. LV diameters were measured from the M-mode echocardiogram according to the standards of the European Society of Cardiology.¹⁵ All echocardiographic measurements were performed at the end of expiration and averaged over three consecutive cardiac cycles. LVED and LV end-systolic (ES) diameters (D) were measured from M-mode recording, permitting

calculation of LVED and LVES volumes (V) using the formula of Teicholz: $V=7D^3/(2.4+D)$. LV wall thickness at end-systole was also measured from M-mode recording. LV end-systolic wall stress (LVESWS) was calculated according to the formula proposed by Grossman and colleagues:¹⁶ $[0.334 \text{ cuff arterial systolic pressure (LVESD)}]/[LVPWT (1+LVPWT/LVESD)]$, where PWT is the posterior wall thickness. Velocity–time integral of pulmonary flow (VTIP) was recorded at the level of the right ventricular (RV) outflow tract, together with pulmonary artery diameter (D_p), permitting calculation of RV stroke volume (SV), as $RVSV=VTIP \times [\pi]D_p^2/4$. The mean acceleration of the flow in the pulmonary artery (Ac_{mean}) was calculated as peak velocity/acceleration time.¹⁷ All echocardiographic measurements were indexed to body-surface area. Pulsed-Doppler at the mitral annulus was performed; peak early (E) and peak late (A) velocities were measured and expressed as the E/A ratio.¹⁸ A Doppler tissue imaging (DTI) at the corner of the mitral annulus was also performed and the early diastolic velocity (E_a) was recorded (Fig. 1).^{19 20}

Recordings were stored on a videotape recorder. Videotapes were reviewed on an off-line analyser system that allowed slow-down or stop of the picture. A random sample of 10 patients was submitted twice for analysis in order to test the reproducibility of the measurements.

Statistical analysis

Data were computerized and analysed using the Statview 5.0 statistical packages (SAS Institute Inc., Cary, NC, USA). Normality of the distribution of data was assessed by the Kolmogorov–Smirnov test. We expressed continuous variables as the mean (SD). Changes in haemodynamic and respiratory parameters during VC sequences and PC_{protocol} were analysed using repeated measures analysis of variance (ANOVA) followed by the Scheffe f -test, as appropriate. Otherwise, normally distributed continuous

variables were compared using the Student's t -test. When PC was applied instead of VC ventilation, airway pressure was expected to change.^{7 8} The cardiovascular effects were related to the degree to which applied airway pressure was transmitted to the pleural space. Thus, we chose to focus on the LVESWS, which reflected systolic function. Taking into account the preliminary results for the first 10 patients and on the basis of a relevant 20% change in wall stress, we calculated that 18 patients could test the null hypothesis at 0.05 significance with a power of 0.90. A P -value of <0.05 was considered significant.

Results

Among the 23 patients initially enrolled, only 21 could be studied: in one patient, a severe bronchospasm occurred after induction of anaesthesia and the quality of the echocardiographic records was inadequate in the other. Intraobserver and interobserver reproducibility were 9.1 (4.8)% and 8.9 (6.1)%, respectively, for wall thickness, and 3.2 (1)% and 5.0 (4.0)%, respectively, for E_a . Demographic characteristics, duration of surgery, and blood loss are summarized in Table 1. Eleven patients (52%) had BMI of $>25 \text{ kg m}^{-2}$ and 11 patients suffered from arterial hypertension. Lactate values at T3 and T4 periods were $1.0 (0.2) \text{ mmol litre}^{-1}$ (range 0.3–1.5). The normal lactate values ranged from 0.5 to $2.0 \text{ mmol litre}^{-1}$.

Ventilatory parameters measured at each ventilation period are shown in Table 2. During all the study periods, RR and V_t were comparable, but the peak inspiratory flow was very different between the two modes of ventilation. When compared with VC, P_{peak} was lower in PC whereas P_{mean} was slightly increased. Dynamic compliance significantly improved after switching VC to PC [+15 (8)%] (Fig. 2). However, static airway pressures (P_{plat} and intrinsic-PEEP) and static compliance were not different between the two modes. No patient presented an intrinsic PEEP $>5 \text{ cm H}_2\text{O}$. Effects on oxygenation also did not

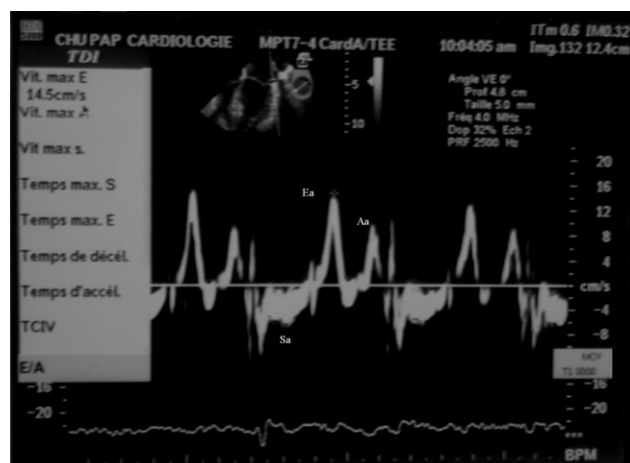


Fig 1 Representative tracing of DTI mitral annular velocity patterns obtained by TOE. The following measurements were made from the DTI recordings: peak systolic velocity (S_a) and early (E_a) and late (A_a) diastolic velocities.

Table 1 Patient characteristics. Data are given as mean (range), mean (SD) or absolute numbers. COPD: chronic obstructive pulmonary disease

Characteristic	
Age (yr)	64 (48–76)
BMI (kg m^{-2})	25.4 (3.7)
Body surface area (m^2)	1.90 (0.21)
ASA physical status (I/II)	1/20
COPD (n)	1
Arterial hypertension (n)	11
Cardiovascular drugs (n)	
Angiotensin receptor inhibitor	8
Ca^{2+} channel antagonists	7
β -blockers	1
Surgical procedure (n)	
Radical prostatectomy	16
Lymphadenectomy	5
Total blood loss (ml)	588 (558)
Mean operative time (min)	215 (74)

Table 2 Comparisons of variables of ventilatory and respiratory mechanics, and blood gas analysis during VC and PC ventilation. V_t , tidal volume; RR, respiratory rate; P_{peak} , peak airway pressure; P_{plat} , plateau pressure; P_{mean} , mean airway pressure; $P_{E'CO_2}$, end-tidal CO_2 ; V_d/V_t , physiological dead space estimated according to the Hardman and Aitkenhead equation: $V_d/V_t = 1.14 \times (P_{aCO_2} - P_{E'CO_2}) / P_{aCO_2} - 0.005$.¹⁴ VC_{pno}, VC ventilation after introduction of pneumoperitoneum. VC_{protocol} and PC_{protocol}, VC and PC ventilation, respectively, during established pneumoperitoneum. Data are mean (SD)

	VC _{baseline} (T1)	VC _{pno} (T2)	VC _{protocol} (T3)	PC _{protocol} (T4)	P-value			
					ANOVA	T1 vs T2	T1 vs T3	T3 vs T4
V_t (ml)	639 (96)	633 (94)	642 (100)	630 (89)	0.20			
RR (min^{-1})	9 (1)	9 (1)	9 (1)	9 (1)	0.64			
Flow (litre min^{-1})	18 (4)	17 (3)	17 (3)	48 (8)	<0.0001	0.96	0.98	<0.0001
Airway pressures (cm H_2O)								
P_{peak}	21 (5)	31 (6)	32 (6)	27 (6)	<0.0001	<0.0001	<0.0001	<0.0001
P_{plat}	15 (4)	23 (5)	24 (5)	23 (5)	<0.0001	<0.0001	<0.0001	0.81
P_{mean}	5 (2)	7 (2)	7 (2)	9 (2)	<0.0001	<0.0001	<0.0001	<0.0001
Static compliance of the respiratory system (ml $\text{cm}^{-1} H_2O$)	44 (10)	28 (5)	28 (5)	28 (5)	<0.0001	<0.0001	<0.0001	0.81
Dynamic compliance of the respiratory system (ml $\text{cm}^{-1} H_2O$)	32 (6)	21 (4)	21 (3)	24 (4)	<0.0001	<0.0001	<0.0001	0.0005
$P_{E'CO_2}$ (kPa)	3.7 (0.4)	3.8 (0.4)	4.0 (0.4)	4.0 (0.4)	0.0001	0.73	0.004	0.99
P_{aO_2}/F_{IO_2}	—	—	446 (77)	458 (64)	—			0.16
P_{aCO_2} (mm Hg)	—	—	5.3 (0.6)	5.4 (0.6)	—			0.17
Estimated V_d/V_t (%)	—	—	32 (8)	29 (12)	—			0.09
pH	—	—	7.39 (0.04)	7.40 (0.04)	—			0.06
Temperature ($^{\circ}C$)	35.3 (0.7)	35.1 (0.9)	35.1 (0.7)	35.1 (0.8)	0.04	0.20	0.12	0.99

reveal significant changes. End-tidal CO_2 underestimated P_{aCO_2} levels with a mean gradient of 12 (4) mm Hg in both modes.

The overall results are reported in Table 3. Pneumoperitoneum induced a significant increase in LVESWS and LVESV. A significant decrease in RVSV was also found [-15 (13)%], without any change in RV output impedance estimated by Ac_{mean} obtained at end-expiration. There was no significant variation of filling pressures, estimated by the E/E_a ratio. When the two modes of ventilation were compared, no significant change could be demonstrated in the systolic or diastolic function.

Discussion

Our study demonstrated that, during laparoscopy, PC ventilation decreased P_{peak} and increased P_{mean} slightly when compared with VC ventilation. Dynamic compliance significantly improved after switching from VC to PC. No other significant respiratory change could be noted. Likewise, switching from VC to PC did not lead to modification in systolic or diastolic function. Interpretation of echocardiographic results should be taken into account that 50% of our patients suffered from slight obesity and arterial hypertension.²¹

One expected advantage of PC ventilation could be related to its effect on volotrauma. Thus, in no part of the lung could the pressure be higher than the pre-set pressure.⁶ When compared with VC, the association between PC and a lower P_{peak} was a constant finding in all previous studies.^{5 10–12, 22} This was achieved because of

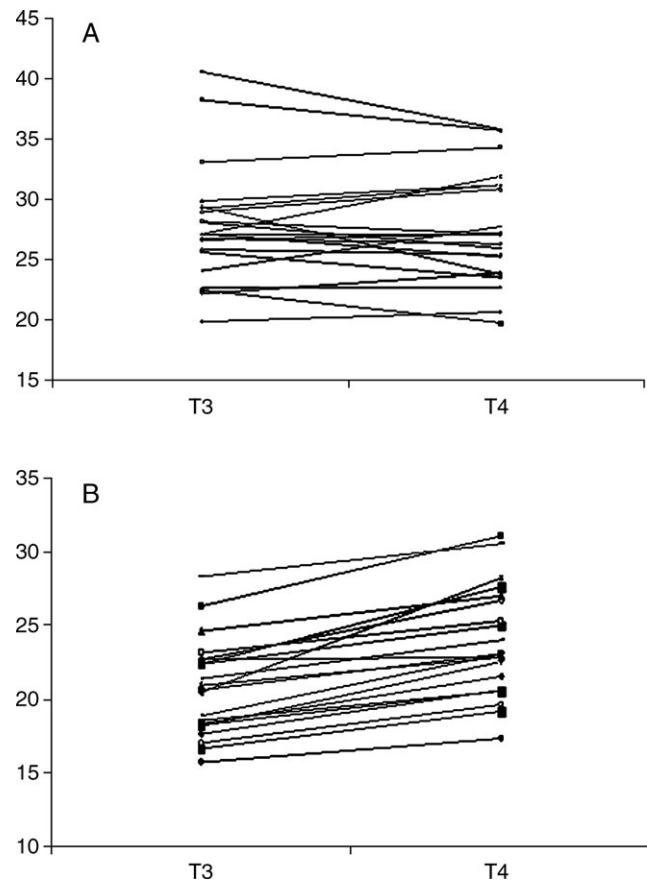


Fig 2 Individual changes in total respiratory static compliance (C_{st}) (A) and in total respiratory dynamic compliance (C_{dyn}) (B) when VC ventilation (T3) was switched to PC ventilation (T4).

Table 3 Comparisons of haemodynamic and echocardiographic measurements during VC and PC ventilation. MAP, mean arterial pressure; HR, heart rate; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LVEF, left ventricular ejection fraction; LVESWS, left ventricular end-systolic wall stress; VTIP, velocity–time integral of pulmonary flow; Ac_{mean} , mean acceleration of the pulmonary artery flow; RVSV, right ventricular stroke volume; E/A mitral, ratio of peak early (E) and peak late (A) velocities; E_a , early diastolic velocity. VC_{pno} , VC ventilation after introduction of pneumoperitoneum. $VC_{protocol}$ and $PC_{protocol}$, VC and PC ventilation, respectively, during established pneumoperitoneum. Data are mean (SD)

Variables	$VC_{baseline}$ (T1)	VC_{pno} (T2)	$VC_{protocol}$ (T3)	$PC_{protocol}$ (T4)	ANOVA	T1 vs T2	T1 vs T3	T3 vs T4
MAP (mm Hg)	85 (11)	104 (15)	98 (14)	96 (13)	<0.0001	<0.0001	0.0003	0.92
HR (beats min^{-1})	62 (13)	61 (13)	64 (13)	63 (13)	0.49			
LVEDV (ml m^{-2})	56 (15)	55 (15)	53 (15)	54 (15)	0.6			
LVESV (ml m^{-2})	17 (5)	20 (6)	18 (6)	18 (7)	0.02	0.04	0.99	0.88
LVEF (%)	70 (4)	68 (7)	70 (5)	70 (7)	0.2			
LVESWS (10^3 $dyn\ cm^{-2}\ m^{-2}$)	58 (15)	69 (17)	66 (16)	63 (14)	0.001	0.002	0.02	0.68
VTIP (cm)	14.0 (2.0)	12.5 (2.0)	12.4 (1.8)	12.2 (1.6)	<0.0001	0.0001	<0.0001	0.95
Ac_{mean} ($m\ s^{-2}$)	7.1 (2.0)	7.6 (2.9)	7.4 (2.8)	7.9 (2.9)	0.70			
RVSV (ml m^{-2})	29 (7)	25 (8)	25 (7)	23 (6)	<0.0001	0.0001	<0.0001	0.29
E/A mitral	1.2 (0.2)	1.1 (0.3)	1.1 (0.3)	1.1 (0.3)	0.21			
E_a ($cm\ s^{-1}$)	10.3 (2.2)	10.6 (2.3)	10.3 (2.5)	10.5 (2.7)	0.7			
E/E_a	6.2 (2.7)	5.7 (2.0)	5.7 (2.2)	5.8 (2.4)	0.1			

the decreasing flow pattern with PC and an earlier dissipation of flow resistance.²² However, P_{plat} was not modified by PC. Similar P_{plat} values suggest that the high flow meeting the endotracheal tube during the use of the constant square waveform was the cause of the elevated P_{peak} . Therefore, during VC, the higher P_{peak} did not reflect a change in lung mechanics or end-inspiratory alveolar peak pressure, and was probably not associated with any ventilatory harm.^{13 23} However, peak inspiratory flow was very different between the two modes of ventilation, and the ventilatory consequences are still a matter of controversy. In a rabbit model, high peak inspiratory flow in PC ventilation induced significantly more severe lung damage than lower peak inspiratory flows in the VC mode.²⁴ In addition, limiting inspiratory flow and altering its pattern of delivery to a constant rate significantly reduced lung injury in normal lung of sheep.²⁵ On the contrary, in patients with acute lung injury, the decelerating flow of PC could reduce lung strain.²³ Another potential advantage of PC was to aid recruitment and to improve distribution of inspired gas.^{12 26} The change in C_{dyn} was of interest here because it was associated with a change in gas distribution.²⁷ However, in isovolumetric conditions, variation of C_{dyn} depended not only on the elastic properties of the respiratory system but also on the resistive (flow-dependent) component of the airways and the endotracheal tube. Thus, neither the Pa_{CO_2} nor the estimated V_d/V_t ratio was different between PC and VC. Interestingly, P_{mean} was slightly higher during PC. Such a result was not surprising. In fact, using a decelerating flow waveform could result in a higher P_{mean} according to the mathematical models.^{7 8} Increases in P_{mean} appeared to be directly related to increases in oxygenation.²⁸ However, the low P_{mean} generated during all steps of our study may have contributed to minimize its positive effect on oxygenation.

The main disadvantage of PC ventilation included variability in delivered V_t .⁶ In contrast to VC, PC ventilation resulted in a smaller delivered V_t when respiratory system

compliance was decreased.²⁹ A smaller V_t might lead to atelectasis and might go undiagnosed because there would be no change in P_{peak} as PC ventilation would be used.²⁹

Monitoring of cardiac function under pneumoperitoneum condition was challenging^{2 3} as illustrated by the poor data recorded in one excluded patient. Investigator's reliability was the other cornerstone condition. The intraobserver and interobserver variabilities reported here (between 3% and 9%) was in accordance with previous published studies.^{2 3 20}

Cardiovascular consequences of laparoscopic surgery have been well documented by TOE. Pneumoperitoneum during VC ventilation caused an increase in mean arterial pressure and in LVESWS.^{2 3} We found the same results as previously reported, but changes were less pronounced.^{2 3} These haemodynamic changes were not sustained throughout the period of pneumoperitoneum.² Our study also showed that filling pressures were stable during the protocol as the ratio of the transmitral E velocity to E_a was significantly related to the filling pressures.¹⁹

Changes in cardiac output are sometimes variable, consistent with the Starling resistor concept of abdominal venous return.³⁰ A significant but small decrease in RVSV was noted 15 min after pneumoperitoneum. As discussed earlier, we failed to detect a significant decrease in preload. We were also unable to show a decrease in Ac_{mean} at the end of expiration, which reflects changes in RV output impedance.¹⁷ Interestingly, elevation of RV afterload was demonstrated during inspiration, whereas no change was observed during expiration.¹⁷ Unfortunately, we have not studied beat-to-beat variations of RV outflow impedance according to the respiratory cycle.¹⁷ However, the pneumoperitoneum led to a decrease in respiratory compliance.^{3 4} This resulted in an increased transpulmonary pressure for delivering the same V_t .³ An increase in RV afterload during inspiration was, therefore, expected^{3 17} as transpulmonary pressure was its main determinant factor. Some authors also reported a decrease in the E/A ratio after

pneumoperitoneum.³ However, we did not find an impairment in LV relaxation. In our study, the induction of pneumoperitoneum was performed when the patients were in a 20° Trendelenburg's position and after an infusion of 500 ml of colloids. These two actions should be taken into account to explain some differences between studies.^{4–18} In order to be unaffected by fluid loading, we used a DTI (Fig. 1).^{19–20} Unlike the E/A ratio which was influenced by loading conditions,¹⁸ the E_a is a preload-independent index of LV relaxation.¹⁹ Thus, E_a did not change during the different steps of our protocol.

When switching from VC to PC ventilation, the cardiovascular effects of PC ventilation should be related to airway pressure, through its effects on pleural pressure.⁹ We reported a slight increase in P_{mean} . Expected effects would have been a decrease in RVSV, LV preload, and LVESWS.⁹ However, no echocardiographic change was noted during our study. The most plausible explanation was the absence of a significant increase in intrathoracic pressure after the slight increase in P_{mean} . For the same V_t , RV afterload did not change at the end of expiration. Once again, the main determinant of RV afterload was transpulmonary pressure and not airway pressure in its strict sense at the end of inflation.¹⁷ Interestingly, evaluation of LV relaxation did not show any significant difference between the two modes of ventilation.^{19–20} In summary, our echocardiographic results indicated a lack of association of P_{peak} and inspiratory flow pattern on cardiac function.

There were several limitations of our study. The patients were not randomized to the starting mode of ventilation. However, our intention was to study a ventilatory strategy commonly used in clinical practice. To our knowledge, physicians always started mechanical ventilation with a VC mode. The ventilation in VC or PC mode was maintained for a relatively short time. However, changes in respiratory mechanics, haemodynamics, and gas exchange are usually completed within this time period.^{10–12} Another potential limitation was the lack of pleural pressure measurement.⁹ However, pleural pressure variations would have been minor, taking into account the small changes in respiratory mechanics.

In conclusion, we found no advantage of PC ventilation over VC ventilation regarding respiratory mechanics, gas exchange, or on cardiac function. Specifically, the risk of barotrauma was not decreased by PC ventilation.

References

- Nadu A, Ekstein P, Szold A, *et al.* Ventilatory and hemodynamic changes during retroperitoneal and transperitoneal laparoscopic nephrectomy: a prospective real-time comparison. *J Urol* 2005; **174**: 1013–7
- Decailliot F, Streich B, Heurtematte Y, Duvaldestin P, Cherqui D, Stéphan F. An echocardiography prospective study of hemodynamic effects of portal triad clamping with and without pneumoperitoneum. *Anesth Analg* 2005; **100**: 617–22
- Alfonsi P, Vieillard-Baron A, Coggia M, *et al.* Cardiac function during intraperitoneal CO₂ insufflation for aortic surgery: a transesophageal echocardiographic study. *Anesth Analg* 2006; **102**: 1304–10
- Sharma KC, Brandstetter RD, Brensilver JM, Jung LD. Cardiopulmonary physiology and pathophysiology as a consequence of laparoscopic surgery. *Chest* 1996; **110**: 810–5
- Tugrul M, Camci E, Karadeniz H, Senturk M, Pembeci K, Akpir K. Comparison of volume controlled with pressure controlled ventilation during one-lung anaesthesia. *Br J Anaesth* 1997; **79**: 306–10
- McKibben AW, Ravenscraft SA. Pressure-controlled and volume-cycled mechanical ventilation. *Clin Chest Med* 1996; **17**: 395–410
- Marin JJ, Crooke PS III. A general mathematical model for respiratory dynamics relevant to the clinical setting. *Am Rev Respir Dis* 1993; **147**: 14–24
- Armstrong BW, MacIntyre NR. Pressure-controlled, inverse ratio ventilation that avoids air trapping in the adult respiratory distress syndrome. *Crit Care Med* 1995; **23**: 279–85
- Pinsky MR. Recent advances in the clinical application of heart–lung interactions. *Curr Opin Crit Care* 2002; **8**: 26–31
- Lessard MR, Guérot E, Lorino H, Lemaire F, Brochard L. Effects of pressure-controlled with different I:E ratios versus volume-controlled ventilation on respiratory mechanics, gas exchange, and hemodynamics in patients with adult respiratory distress syndrome. *Anesthesiology* 1994; **80**: 983–91
- Davis K, Jr, Branson R, Campbell RS, Porembka DT. Comparison of volume control and pressure control ventilation: is flow waveform the differences? *J Trauma* 1996; **41**: 808–14
- Prella M, Feihl F, Domenighetti G. Effects of short-term pressure-controlled ventilation on gas exchange, airway pressures, and gas distribution in patients with acute lung injury/ARDS. Comparison with volume-controlled ventilation. *Chest* 2002; **122**: 1382–8
- Stewart TE, Meade MO, Cook DJ, *et al.* Evaluation of a ventilation strategy to prevent barotrauma in patients at high risk for acute respiratory distress syndrome. *N Engl J Med* 1998; **338**: 355–61
- Hardman JG, Aitkenhead AR. Estimating alveolar dead space from the arterial to end-tidal CO₂ gradient: a modeling analysis. *Anesth Analg* 2003; **97**: 1846–51
- Flachskampf FA, Decoodt P, Fraser AG, Daniel WG, Roelandt JR Subgroup on Transesophageal Echocardiography Valvular Heart Disease, Working group on Echocardiography of the European Society of Cardiology. Guidelines from the Working Group. Recommendations for performing transoesophageal echocardiography. *Eur J Echocardiography* 2001; **2**: 8–21
- Grossman W, Jones D, McLaurin L. Wall stress and patterns of hypertrophy in the human left ventricle. *J Clin Invest* 1975; **58**: 56–64
- Vieillard-Baron A, Loubières Y, Schmitt J-M, Page B, Dubourg O, Jardin F. Cyclic changes in right ventricular output impedance during mechanical ventilation. *J Appl Physiol* 1999; **87**: 1644–50
- Cohen GI, Pietrolungo JF, Thomas JD, Klein AL. A practical guide to assessment of ventricular diastolic function using Doppler echocardiography. *J Am Coll Cardiol* 1996; **27**: 1753–60
- Nagueh SF, Middleton KJ, Kopelen HA, Zoghbi WA, Quinones MA. Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressure. *J Am Coll Cardiol* 1997; **30**: 1527–33
- Filipovic M, Wang J, Michaux I, Hunziker P, Skarvan K, Seeberger MD. Effects of halothane, sevoflurane and propofol on left ventricular diastolic function in humans during spontaneous and mechanical ventilation. *Br J Anaesth* 2005; **94**: 186–92
- Pascual M, Pascual DA, Soria F, *et al.* Effects of isolated obesity on systolic and diastolic left ventricular function. *Heart* 2003; **89**: 1152–6
- Edibam C, Rutten AJ, Collins DV, Bersten AD. Effect of inspiratory flow pattern and inspiratory to expiratory ratio on

- nonlinear elastic behavior in patients with acute lung injury. *Am J Respir Crit Care Med* 2003; **167**: 702–7
- 23 Dreyfuss D, Saumon G. Ventilator-induced lung injury. Lessons from experimental studies. *Am J Respir Crit Care Med* 1988; **157**: 294–323
 - 24 Maeda Y, Fujino Y, Uchiyama A, Matsuura N, Mashimo T, Nishimura M. Effects of peak inspiratory flow on development of ventilator-induced lung injury in rabbits. *Anesthesiology* 2004; **101**: 722–8
 - 25 Rich PB, Reickert CA, Sawada S, et al. Effect of rate and inspiratory flow on ventilator-induced lung injury. *J Trauma* 2000; **49**: 903–11
 - 26 Ingram RH, Jr, Schilder DP. Association of a decrease in dynamic compliance with a change in gas distribution. *J Appl Physiol* 1967; **23**: 911–6
 - 27 Mang H, Kacmarek RM, Ritz R, Wilson RS, Kimball WP. Cardiorespiratory effects of volume- and pressure-controlled ventilation at various I/E ratios in an acute lung injury model. *Am J Respir Crit Care Med* 1995; **151**: 731–6
 - 28 Marini JJ, Ravenscraft SA. Mean airway pressure: physiologic determinants and clinical importance. Part II. Clinical implications. *Crit Care Med* 1992; **20**: 1604–16
 - 29 Duggan M, Kavanagh BP. Pulmonary atelectasis. A pathogenic perioperative entity. *Anesthesiology* 2005; **102**: 838–54
 - 30 Giebler RM, Behrends M, Steffens T, Walz MK, Peitgen K, Peters J. Intraperitoneal and retroperitoneal carbon dioxide insufflation evoke different effects on caval vein pressure gradients in humans: evidence for the Starling resistor concept of abdominal venous return. *Anesthesiology* 2000; **92**: 1568–80