

# Hemodynamic and pulmonary changes during and after laparoscopic cholecystectomy

## A comparison with traditional surgery

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### Abstract

**Background:** The cardiopulmonary changes experienced by patients who undergo laparoscopic cholecystectomy (LC) and the prognostic value of patient characteristics are not well understood.

**Methods:** Cardiorespiratory changes were investigated in 120 patients undergoing LC or open cholecystectomy (OC). The results and their relation to patient variables were statistically evaluated.

**Results:** The most significant cardiorespiratory changes were (A-a)PO<sub>2</sub> increase during OC; decrease of pH and compliance and increase of peak airway pressure during LC; impairment of arterial blood gas mean values and respiratory muscle strength; atelectasis and pneumonia (five cases) after OC; and lamellar atelectasis (two cases) after LC. Significant adverse prognostic factors related to intra- and postoperative LC cardiorespiratory changes were ASA class greater than I, FEF<sub>75–85%</sub> < 900 ml, and PaO<sub>2</sub> < 10.4 kPa (PPV, 71.4% and 46.6%, respectively).

**Conclusions:** LC carries no significant cardiorespiratory changes provided that intraoperative monitoring of hemodynamics and respiratory parameters is done for the study of blood gas values in all patients at risk.

**Key words:** Laparoscopic cholecystectomy — Hemodynamics — Respiratory function — Prognosis

There is a great deal of disagreement among the authors of the most recent publications on the hemodynamic and ventilatory effects of laparoscopic surgery [2, 6, 23]. Conflicting data has been reported on the incidence of cardiorespiratory complications in patients undergoing cholecystectomy (LC), even though LC is the most common as well as the safest of all laparoscopic procedures [15, 17, 18]. Because

of the growing popularity of this surgery, even for patients who are obese, elderly, or suffering from preexisting cardiopulmonary disease [7, 13, 24], and the increasing need for an extended insufflation period when associated with other endoscopic procedures, the physiologic changes that occur during and after laparoscopic surgery must be monitored carefully [2]. Most of the recent studies are based on the metabolic, inflammatory, and immunological response to acute-phase LC trauma (cytokine and hormonal levels, serum levels of acute-phase reactant protein, nitrogen balance and forearm muscle amino acid fluxes) [8, 10, 12]; these measures are undoubtedly useful in defining the surgical stress response, but they do not help to predict which patients or conditions are at risk for this type of surgery.

The aim of our study was to assess the relative effects of laparoscopic and open cholecystectomy (OC) on intra- and postoperative hemodynamic and respiratory function. We also attempted to evaluate the risk of complications by means of a randomized trial.

### Patients and methods

Between 1993 and 1996, 120 patients with symptomatic cholelithiasis, who were referred to our unit for elective cholecystectomy, enrolled in this study. Of these, those exceeding their ideal body weight by >20% or with a body mass index (BMI) >85th percentile were considered to be obese patients [20]; those aged ≥60 years were defined as elderly patients. After giving informed consent, all patients were randomly assigned to undergo either laparoscopic cholecystectomy (LC) or open laparotomic cholecystectomy (OC). Anesthesia and postoperative control were carried out according to standard protocols. Anesthesia was induced intravenously with thiopental sodium (5 mg pro kg<sup>-1</sup>) and fentanyl (1.5 ng pro kg<sup>-1</sup>); a non-depolarizing muscle relaxant (vecuronium 0.2 mg pro kg<sup>-1</sup>) was used to facilitate endotracheal intubation. For maintenance of anesthesia, patients received isoflurane with 65% N<sub>2</sub>O in O<sub>2</sub>. Small incremental boluses of vecuronium were administered as needed to maintain adequate muscle relaxation. Ventilator settings were adjusted by altering ventilatory frequency and tidal volume if acidosis (pH < 7.20), arrhythmias, or hemodynamic instability were seen. Fentanyl (0.05 mg) was given if arterial blood pressure or heart rate rose by 30% from the previously recorded value (5 min earlier).

In patients undergoing LC, the pneumoperitoneum was performed in

**Table 1.** Personal series: clinical parameters in 118 patients undergoing laparoscopic cholecystectomy (LC) and open cholecystectomy

Parameters	LC patients	OC patients	<i>p</i> value
Male/female	16/42	20/40	>0.05
Age (yr)	47.7 ± 17.07	53.52 ± 15.99	>0.05
Obese	20	17	>0.05
Severe COPD	2	3	>0.05
Current smokers	12	23	>0.05
Nonsmokers	40	25	0.005
Former smokers	6	12	>0.05
ASA class			
I	44	45	>0.05
II–III	14	15	>0.05
Operative time (min)	86.6 ± 22	81.0 ± 24.79	>0.05
Hospital stay (days)	4.6 ± 2.9	7.77 ± 3.1	0.0005

the supine position. Carbon dioxide was used for insufflation, and intra-peritoneal pressures were maintained at 1.6–2 kPa. Reverse Trendelenburg (rT) head-up tilt was limited to 10–15°.

Immediately after the end of anesthesia, intravenous analgesia (ketoprofene 100 mg) was started in all cases in the recovery room and continued for 5 h.

During anesthesia ECG, systemic blood pressure, heart and respiratory rate, peak airway pressure (Ppeak), compliance, and end-tidal CO<sub>2</sub> (EtCO<sub>2</sub>) were continuously monitored, while arterial blood gas analysis was repeated every 15 min. In the laparoscopic group, all these parameters were also measured at the induction and at the end of the pneumoperitoneum.

The same parameters [heart and respiratory rates, systemic blood pressure, blood gases together with alveoloarterial oxygen difference (A-a)PO<sub>2</sub>], ventilatory function, body temperature, and inspiratory and expiratory muscle strength were evaluated for each group prior to surgery, and every day for 3 days after surgery. Postoperative pain was assessed using a self-rating visual analog scale (VAS) ranging from 0 = no pain and 10 = worst possible pain. Pain intensity was assessed using a verbal pain score (VPS) ranging from 0 to 5, where 0 = no pain, 1 = mild, 2 = moderate, 3 = discomforting, 4 = stressing, and 5 = horrible. Pain scores were obtained at days 1, 2, and 3 after surgery. On demand, discomforting or continuing pain was treated with an IM injection of 10 mg of ketorolac. At the same predischarge intervals, radiographic examination was performed to evaluate and determine the presence and severity of atelectasis.

Hemodynamic parameters were studied using Lifescope 7 Nihon Khoden (Tokyo); pulmonary parameters were studied prior to and after surgery using a Vitalograph spirometer and monitored during operation using a Capnomar Ultima Datex sidestream spirometer (Buckingham, UK); blood gas analyses were performed using a Radiometer ABL3 blood gas analyzer (Copenhagen, Denmark); postoperative muscle strength was measured with a DHD Medical Products negative inspiratory force monitoring kit. (A-a)PO<sub>2</sub> was calculated using the following formula for alveolar oxygen partial pressure: PaO<sub>2</sub> = (FiO<sub>2</sub> × 94.8) – PaCO<sub>2</sub>/0.8.

All data were expressed as mean ± SD. The significance of the difference between LC and control (OC) groups was tested using Student's *t* test; for nonparametric data the  $\chi^2$  test and Fisher's exact test were employed. A *p* value ≤ 0.05 was considered statistically significant. The efficacy of preoperative parameters in predicting intra- and/or postoperative complications was evaluated by sensitivity, specificity, and positive predictive tests (PPV) using the Galen method.

## Results

In the LC subset, two patients who required conversion of the laparoscopic procedure to standard OC were excluded from the subsequent analyses.

There were no significant differences between the two study groups in age, sex, weight, smoking habits, ASA class, preoperative ventilatory and blood gas parameters, or duration of surgery (Tables 1–4).

The principal intraoperative findings are shown in Table 1. In the LC group, the more significant changes from the

induction of anesthesia in terms of mean values were: a 16.6% decrease in arterial pH (*p* = 0.02), an 18.7% increase in Ppeak (*p* = 0.048), and a 33.3% reduction in compliance (*p* = 0.001). In the OC group, the only significant change was a 45.4% increase of (A-a)PO<sub>2</sub> (*p* = 0.001), even if pulmonary shunting remained within normal values (5–10%). There were no changes in mean values for heart rate, systemic blood pressure, SaO<sub>2</sub> and PaO<sub>2</sub> mean values in both LC and OC groups, or in mean ventilatory volume minute (7.35 ± 0.96 l and 6.97 ± 0.42 l, respectively) and mean dose of fentanyl during anesthesia (0.34–0.36 mg). The only significant intergroup variations (comparing LC versus OC) during surgery were observed in pH (0.006), PaCO<sub>2</sub> (*p* = 0.01), and (A-a)PO<sub>2</sub> values (*p* = 0.0002). The latter were markedly altered in the OC group. In terms of individual patient intraoperative cardiorespiratory patterns, during pneumoperitoneum a mild acidosis (pH < 7.35) was observed in eight cases; in two other cases, acidosis was severe (pH < 7.28) and was accompanied by hypercarbia (PaCO<sub>2</sub> = 6.6 kPa), a mild increase of end-tidal CO<sub>2</sub>, and a significant increase of both systolic blood pressure and heart rate. These effects were easily compensated when employing a higher concentration of oxygen and using hyper-ventilation. A significant decrease of systolic blood pressure with bradycardia occurred in two other patients. This situation required the interruption of CO<sub>2</sub> insufflation for a few minutes and, in one case, the administration of plasma expanders. During OC, the transient adverse effects were acidosis (two cases), hypercarbia (two cases), hypertension (five cases), and arrhythmia (three cases). All these abnormalities were easily reversed without further complications.

The postoperative changes are shown in Tables 3 and 4. Regarding PaO<sub>2</sub>, PaCO<sub>2</sub>, (A-a)PO<sub>2</sub>, and pH postoperative mean values, significant differences were observed between the two groups on days 1 and 3. The decrease in PaO<sub>2</sub> and the increase in (A-a)PO<sub>2</sub> were much more consistent following OC (mean variation from baseline values: –17% for PaO<sub>2</sub> (*p* = 0.003); +100 to 90% for (A-a)PO<sub>2</sub> (*p* = 0.00005) than after LC. The significant reduction in FVC, FEV<sub>1</sub>, and in FEF<sub>75–85%</sub> following LC (–46, –45, –45%) on day 1, and their restoration (–29, –28, –38%) on day 3 were similar to those following OC. On day 2, however, FVC and FEF<sub>75–85%</sub> restoration was faster than after OC (*p* = 0.02 and *p* = 0.01, respectively). Considering patient basal characteristics, these differences were more marked when related to obesity. In the latter subset, on days 1 and 2, FEF<sub>75–85%</sub> and PaO<sub>2</sub> reduction following OC was almost twice that following LC. In terms of inspiratory and expiratory muscle strength, no changes with respect to the preoperative values were observed in LC patients, whereas all OC patients showed a mild decrease in inspiratory muscle strength on the 1st postoperative day (–35.2%; *p* = 0.17) and a severe decrease in expiratory muscle strength for the overall postoperative period (–42.5 to –26%; *p* = 0.001 to *p* = 0.037). The difference between the two groups was not significant. In terms of cardiovascular parameters, heart rate and systolic arterial pressure mean values were similar before and after both operations.

Regarding individual changes, cardiac or hemodynamic abnormalities were observed only after OC. Hypertension occurred in three patients and ventricular arrhythmias in another two; all five patients had been classified as ASA I and were >50 years old. As for postoperative pain, there was

**Table 2.** Intraoperative changes of cardiopulmonary parameters (mean  $\pm$  SD) related to laparoscopic cholecystectomy (LC) and open cholecystectomy (OC)

Cardiopulmonary parameters	LC		OC		<i>p</i> value LC vs. OC
	At induction of anesthesia	During pneumoperitoneum	At induction of anesthesia	During operation	
Heart rate (beats/min)	85.7 $\pm$ 10.9	83.2 $\pm$ 12.6	85.1 $\pm$ 12.6	81.0 $\pm$ 6.5	>0.05
Peak airway pressure (kPa)	1.9 $\pm$ 0.6	2.2 $\pm$ 0.4	2.0 $\pm$ 0.5	2.2 $\pm$ 0.6	>0.05
Compliance (ml/kPa)	513.9 $\pm$ 142.7	342.6 $\pm$ 77.7	416 $\pm$ 147.9	364 $\pm$ 95.9	>0.05
SatO <sub>2</sub> (%)	98.3 $\pm$ 1.56	98.4 $\pm$ 0.74	98.6 $\pm$ 0.5	98.4 $\pm$ 0.9	>0.05
End-tidal CO <sub>2</sub> (kPa)	4.0 $\pm$ 0.6	4.3 $\pm$ 0.6	4.3 $\pm$ 0.7	3.8 $\pm$ 0.6	>0.05
pH	7.43 $\pm$ 0.07	7.38 $\pm$ 0.05 <sup>o</sup>	7.43 $\pm$ 0.04	7.44 $\pm$ 0.05 <sup>o</sup>	<sup>o</sup> 0.006
PaCO <sub>2</sub> (kPa)	4.4 $\pm$ 0.7	4.9 $\pm$ 0.8*	4.5 $\pm$ 0.08	4.1 $\pm$ 0.6*	*0.012
PaO <sub>2</sub> (kPa)	20.8 $\pm$ 4.7	19.6 $\pm$ 4.9	18.5 $\pm$ 0.3	18.1 $\pm$ 6.2	0.05
(A-a)PO <sub>2</sub> (kPa)	8.3 $\pm$ 3.1	9.12 $\pm$ 3.2 <sup>^</sup>	9.0 $\pm$ 0.2 <sup>^^</sup>	13.3 $\pm$ 0.8 <sup>^^</sup>	<sup>^</sup> 0.0002 <sup>^^</sup> 0.00003

<sup>o</sup>.\*.^^ Each symbol indicates data with *p* < 0.05 and corresponding *p* value

**Table 3.** Postoperative changes of ventilatory parameters (mean  $\pm$  SD) related to laparoscopic cholecystectomy (LC) and open cholecystectomy (OC)

Ventilatory parameters	LC				OC				<i>p</i> value LC vs. OC
	Preoperative	Postoperative days			Preoperative	Postoperative days			
		1	2	3		1	2	3	
FVC ml	3257 $\pm$ 977	1755 $\pm$ 719	2068 $\pm$ 997*	2334 $\pm$ 878	3104 $\pm$ 959	1435 $\pm$ 668	1503 $\pm$ 388*	2139 $\pm$ 758	*0.02
FEV <sub>1</sub> ml	2642 $\pm$ 947	1455 $\pm$ 652	1580 $\pm$ 773	1868 $\pm$ 758	2602 $\pm$ 763	1251 $\pm$ 542	1202 $\pm$ 409	1726 $\pm$ 635	>0.05
FEF <sub>75-85%</sub> ml	1128 $\pm$ 612	622 $\pm$ 412	769 $\pm$ 535 <sup>o</sup>	694 $\pm$ 376	1056 $\pm$ 531	553 $\pm$ 440	403 $\pm$ 218 <sup>o</sup>	587 $\pm$ 348	<sup>o</sup> 0.01

<sup>o</sup>\* Each symbol indicates data with *p* < 0.05 and corresponding *p* value

**Table 4.** Postoperative changes of arterial blood parameters, heart rate, and muscle strength (mean  $\pm$  SD) related to laparoscopic cholecystectomy (LC) and open cholecystectomy (OC)

Parameters	LC				OC				<i>p</i> value LC vs. OC
	Preoperative	Postoperative days			Preoperative	Postoperative days			
		1	2	3		1	2	3	
PaO <sub>2</sub> (kPa)	11.7 $\pm$ 1.8	10.9 $\pm$ 2.4*	10.2 $\pm$ 1.5	10.5 $\pm$ 1.9	11.6 $\pm$ 1.7	9.7 $\pm$ 1.3*	9.7 $\pm$ 1.7	10.3 $\pm$ 1.5	*0.038
PaCO <sub>2</sub> (kPa)	5.1 $\pm$ 0.4	5.0 $\pm$ 0.4 <sup>o</sup>	4.9 $\pm$ 0.5	5.0 $\pm$ 0.3 <sup>oo</sup>	5.0 $\pm$ 0.5	4.6 $\pm$ 0.5 <sup>o</sup>	4.8 $\pm$ 0.4	4.7 $\pm$ 0.5 <sup>oo</sup>	<sup>o</sup> 0.009 <sup>oo</sup> 0.018
SatO <sub>2</sub> (%)	96.3 $\pm$ 1.37	95 $\pm$ 2.47	94.8 $\pm$ 1.91	95.1 $\pm$ 2.0	95.3 $\pm$ 1.73	94.3 $\pm$ 2.16	94.2 $\pm$ 2.05	94.8 $\pm$ 2.5	>0.05
(A-a)PO <sub>2</sub> (kPa)	2.0 $\pm$ 1.4	3.0 $\pm$ 2.1 <sup>#</sup>	3.4 $\pm$ 1.9	3.2 $\pm$ 2.0	2.2 $\pm$ 1.3	4.4 $\pm$ 1.5 <sup>#</sup>	4.2 $\pm$ 1.9	3.8 $\pm$ 1.6	<sup>#</sup> 0.01
pH	7.40 $\pm$ 0.02	7.41 $\pm$ 0.02 <sup>^</sup>	7.43 $\pm$ 0.02	7.40 $\pm$ 0.03 <sup>^^</sup>	7.40 $\pm$ 0.04	7.43 $\pm$ 0.03 <sup>^</sup>	7.43 $\pm$ 0.04	7.44 $\pm$ 0.03 <sup>^^</sup>	<sup>^</sup> 0.012 <sup>^^</sup> 0.0003
Heart rate (beats/min)	75.8 $\pm$ 11.5	82.1 $\pm$ 13.5	84.9 $\pm$ 9.78	79.3 $\pm$ 8.6	81.0 $\pm$ 10.4	84.0 $\pm$ 12.7	91.6 $\pm$ 12.0	81.9 $\pm$ 13.6	>0.05
Inspiratory muscle strength (kPa)	3.9 $\pm$ 1.8	3.6 $\pm$ 1.9	3.4 $\pm$ 1.6	3.8 $\pm$ 1.8	4.7 $\pm$ 1.1	3.0 $\pm$ 1.8	4 $\pm$ 1.8	3.8 $\pm$ 1.6	>0.05
Expiratory muscle strength (kPa)	4.7 $\pm$ 1.7	4.3 $\pm$ 1.7	3.7 $\pm$ 1.7	4.5 $\pm$ 1.4	5.0 $\pm$ 1.0	2.9 $\pm$ 1.4	3.6 $\pm$ 1.6	3.7 $\pm$ 1.5	>0.05

<sup>oo</sup>.<sup>o</sup>.<sup>#</sup>.<sup>^</sup>.<sup>^^</sup> Each symbol indicates data with *p* < 0.05 and corresponding *p* value

no difference between the groups in the location of pain (i.e., abdominal, shoulder, or thoracic) on days 1 and 3, even though on day 2 the incidence of shoulder pain was higher in the LC group (*p* = 0.006). VAS scores measured at days 1, 2, and 3 were similar in the two groups of patients (1.35  $\pm$  0.9 in, 0.56  $\pm$  0.59 in, 0.12  $\pm$  0.27 in, respectively, after LC; 1.46  $\pm$  0.67, 0.55  $\pm$  0.67, and 0.16  $\pm$  0.35, respectively, after OC). Pain intensity, as indicated by 1–2–3-day VPS (1.5  $\pm$  1.4, 1  $\pm$  1.2, and 0.3  $\pm$  0.6, respectively, after LC; 2.1  $\pm$  1.5, 1.2  $\pm$  1.4, and 0.5  $\pm$  0.9, respectively, after OC) and analgesic requirements after the first 10 h after surgery (17% of both LC and OC cases) were low in the two study groups. No significant difference was observed in the inci-

dence of postoperative fever (>38<sup>o</sup>) (*p* = 0.2): fever occurred in six LC patients and in 12 OC patients. Lamellar atelectasis occurred in two LC patients, whereas three OC patients had segmental atelectasis and another two had pneumonia (pneumonia was considered a major complication). Hospital stay was significantly shorter after LC than after OC (Table 1).

Statistical analysis showed that, of all the preoperative patient characteristics, an ASA class higher than I was the major risk factor, particularly for LC intraoperative cardiopulmonary abnormalities (PPV, 71.4%). An impairment of pulmonary function (FEF<sub>75-85%</sub> < 900 ml and/or PaO<sub>2</sub> < 10.4 kPa) was a mild risk factor for the groups (Table 5).

**Table 5.** Diagnostic efficacy of preoperative parameters in predicting intra- and/or postoperative hemodynamic and pulmonary abnormalities in laparoscopic cholecystectomy (LC) and open cholecystectomy (OC)

Preoperative parameters	Diagnostic efficacy								
	Sensitivity (%) abnormalities			Specificity (%) abnormalities			Positive predictive value (%) abnormalities		
	Intraoperative	Postoperative	Intra- and/or postoperative	Intraoperative	Postoperative	Intra- and/or postoperative	Intraoperative	Postoperative	Intra- and/or postoperative
<b>LC</b>									
current smoking	25.0	25.0	25.0	80.0	80.0	80.9	16.6	16.6	33.3
obesity	20.0	25.0	22.2	62.5	64.0	60.0	10.0	10.0	20.0
ASA class >I	83.3	25.0	55.5	91.3	76.0	90.0	71.4	14.3	71.4
<pulmonary function <sup>a</sup>	83.3	75	83.3	50.0	52.0	55.5	33.3	20	46.6
<b>OC</b>									
current smoking	20.0	16.6	20.0	55.5	52.3	48.6	13.0	13.0	21.7
obesity	46.7	41.1	35.0	77.7	76.7	75.0	41.1	41.1	50.0
ASA class >I	33.3	18.7	22.7	77.7	72.7	73.7	33.3	20.0	33.3
<pulmonary function <sup>a</sup>	53.3	44.4	40.0	66.6	64.3	62.8	34.8	34.7	43.4

<sup>a</sup> FEF<sub>75-85%</sub> < 900 ml and/or PaO<sub>2</sub> < 10.4 kPa

## Discussion

Our experience is in accord with the latest data reported in the literature [6, 22, 24].

The main advantages of LC over OC were found in the postoperative course. As has already been observed by many authors [9, 13, 15, 17, 18, 21, 23], LC patients were characterized by a lesser reduction in overall lung function, lower morbidity, a faster recovery, and earlier discharge from hospital. In particular, our LC series experienced (a) a lower reduction of oxygenation and ventilation-perfusion mismatching, as demonstrated by the lower increase in (A-a)PO<sub>2</sub>; (b) a higher and faster restoration of ventilatory parameters, in particular for FVC and FEF<sub>75-85%</sub> values; (c) no change in inspiratory and expiratory muscle strength (versus a severe decrease of the latter parameter for the overall OC postoperative course); (d) no significant abnormalities in cardiovascular function (versus 8.3% after OC); and (e) no major pulmonary complications and a lesser incidence of subclinical atelectasis (versus 8% after OC). In addition, no patient basal characteristics were seen as statistically valid risk factors for postoperative abnormalities.

In contrast to MacMahon et al. [17], we found no differences in VBS or VAS pain score between the groups. At any rate, requests for analgesic were low in all cases (17% after the first 10 h).

Three aspects of our data are noteworthy. First, the reduction of FEF<sub>75-85%</sub> after LC was half that after OC on day 2. This parameter, unlike VC and FEV<sub>1</sub> is not effort-dependent; therefore it is a better measurement of patient functional condition. As observed by Johnson et al. [13], the fact that in LC cases, this value was only 70% of control on the 3rd postoperative day suggests residual pulmonary dysfunction at 72 h. Second, there were no changes in inspiratory and expiratory muscle strength. This finding was an indirect indication of minimal fatigue, good effort capacity, and no respiratory muscle weakness. It was probably due to the minimal disruption of the abdominal wall muscles and the minimal inhibition of diaphragm reflex characteristic of laparoscopic access [4, 22]. These results, favoring deep

breathing and early mobilization, may explain the low incidence of sublaminal atelectasis found by us even in the obese, elderly and, ASA > I subsets, which are generally considered to be at risk after traditional surgery. This finding is in partial agreement with those of other authors. Johnson et al. [13] observed a low incidence of subclinical atelectasis after LC (10% of cases). However, when they applied multiple regression analysis to patients >60 years of age, patients with smoking habits, or patients with symptoms of respiratory disease, the probability of atelectasis rose to 94%. Using CT, McAllister et al. [16] detected subclinical atelectasis and pleural effusion in one-third of their LC patients and focused on the clinical relevance that this fact may have in patients at risk for postoperative pneumonia.

LC postoperative changes are therefore qualitatively similar but significantly less marked than those seen with OC. Despite these favorable observations, the overall benefit of LC is still in question due to the considerable physiologic cardiopulmonary alterations that accompany surgery. A thorough understanding of these changes is imperative. It is also necessary both to determine the minimum preoperative risk factors and to design a system for monitoring and maintaining a satisfactory intraoperative course for all patients [1, 14, 24, 25]. CO<sub>2</sub> insufflation and rT position are the main problems, followed by abdominal distention, potential hypercarbia, and acidosis [2, 3, 23, 25]. Cardiovascular changes occur frequently, but, since they are the result of opposing local and sympathetic effects, they differ in the various series reported to date, as well as showing considerable variations for individual patients. Reduction of cardiac output, impaired microcirculation of intraperitoneal organs, increase of systemic vascular resistance and heart rate, increase of intracranial pressure, and dysrhythmias have also been reported with different results [5, 6, 11, 14, 24, 25]. Although these changes are easily overcome in healthy patients, they may have a serious clinical impact on patients who are obese, or elderly with limited cardiovascular reserve, or suffering from coexisting lung, renal, or cerebral disease [3, 13, 23, 25]. In our experience, continuous intra-

operative monitoring of cardiovascular and pulmonary parameters was essential (a) to control and modify physiologic responses to the procedure that are not well tolerated, (b) to avoid major complications, and (c) to identify risk factors.

When we compared the intraoperative course of LC with OC, our results confirmed most of the earlier published findings. Operative times were similar for both groups. The use of N<sub>2</sub>O, which has been questioned in laparoscopic surgery due to the possibility that it may cause bowel distension [23], was safe in both groups. The incidence of intraoperative cardiopulmonary changes (soon reversed by increasing minute ventilation and oxygenation) was very low and, at any rate, not statistically different from OC. Most LC respiratory and hemodynamic parameters were maintained near the desired values during the overall intraoperative course.

The main LC changes, as compared with OC, were a major, even if not significantly different, decrease in compliance and increase in pulmonary resistance (Ppeak), as well as a significant decrease in pH and increase in PaCO<sub>2</sub>. As for monitoring tools, it was significant that, in contrast to OC, end-tidal PCO<sub>2</sub> was not a reliable index of PaCO<sub>2</sub>. In our series, monitoring arterial PCO<sub>2</sub> was essential, since in most cases there was a great individual difference between the two values (from 10% to 40%). This result was in agreement with many earlier reports; the normal gradient of 0.4–0.67 kPa between PaCO<sub>2</sub> and EtCO<sub>2</sub> was markedly increased in ASA III–IV patients, in those with a high ventilation/perfusion rate, as well as during hyperventilation, decreased cardiac output, or hypovolemia [1, 3, 25].

Among patient basal characteristics the only significant factor of risk was an ASA class higher than one with a sensitivity of 83% and a PPV of 71.4%. A preoperative pulmonary impairment (FEF<sub>75–85%</sub> < 900 ml and PaO<sub>2</sub> < 10.4 kPa) had the same sensitivity (83.3%) but a low PPV (33%).

## Conclusions

The potential benefit of LC on postoperative cardiopulmonary function must be weighed carefully against the detrimental intraoperative effects of CO<sub>2</sub> pneumoperitoneum, at least in patients with a high ASA class and in those with pulmonary dysfunction. But even in these cases, our experience showed that LC is a safe procedure. We think that intra- and postoperative cardiopulmonary abnormalities may be avoided by maintaining a low insufflation pressure (1.3–1.6 kPa) and a 10–15° head-up tilt; by intraoperative monitoring of hemodynamic and respiratory parameters (mainly compliance, Ppeak, and overall PCO<sub>2</sub> by arterial blood sampling); and by maintaining strict control of the intravascular volume in patients with cardiovascular problems. Some authors [14, 19] have suggested that preoperative hydration may attenuate the reduction in preload caused by pneumoperitoneum, whereas bioimpedance cardiography may be useful in monitoring patients susceptible to myocardial ischemia. In addition, a multimodal approach to postoperative pain may help to promote pulmonary recovery decrease the probability of atelectasis in patients at risk.

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