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Diaphragmatic Function before and after Laparoscopic Cholecystectomy

Filippo Erice, M.D.,* Gordon S. Fox, M.D., F.R.C.P.(C.),† Youssef M. Salib, M.B., Ch.B., F.F.A.R.C.S.,‡
Ezio Romano, M.D.,§ Jonathan L. Meakins, M.D., D.Sc., F.R.C.S.(C.),|| Sheldon A. Magder, M.D., F.R.C.P.(C.)#

Background: Diaphragm dysfunction is a primary cause of ventilatory impairment after upper abdominal surgery. Laparoscopic procedures may result in less dysfunction. To test this, diaphragmatic function was studied in ten healthy adult patients undergoing elective laparoscopic cholecystectomy and in five undergoing laparoscopic hernia repair.

Methods: Respiratory gas exchange, ventilation, and breathing pattern were measured before and 3 h after surgery. Respiratory drive was evaluated from the relationship of $P_{0.1}$ to end-tidal carbon dioxide (P_{ETCO_2}) during tidal breathing. Diaphragm contractile function was assessed from maximal transdiaphragmatic pressure ($P_{di_{max}}$), and Pdi during a maximal sniff maneuver ($P_{di_{sniff}}$).

Results: Oxygen consumption and carbon dioxide production did not change after surgery. $P_{di_{max}}$ decreased by more than 50% in the laparoscopic cholecystectomy group, but $P_{di_{sniff}}$ did not change. Tidal volume and the ratio of inspiratory

time over total cycle time decreased by 30% and 13%, respectively, P_{ETCO_2} increased by 9%, and minute ventilation did not change. In contrast, there was no variation in ventilatory function in patients undergoing laparoscopic hernia repair. In both groups, $P_{0.1}$ did not change, which excludes depressed respiratory drive as an explanation for the decreased $P_{di_{max}}$ in laparoscopic cholecystectomy. Contractile failure of the diaphragm was discounted as well, because $P_{di_{sniff}}$ did not change, even in the laparoscopic cholecystectomy group.

Conclusions: Although laparoscopic cholecystectomy does not increase metabolic demands in the early postoperative period, it impairs diaphragm function. The internal site of surgical intervention appears to be the critical variable determining diaphragmatic inhibition after laparoscopic abdominal surgery. (Key words: Measurement technique: transdiaphragmatic pressure; oxygen consumption. Muscle, skeletal: diaphragm. Surgery: laparoscopic cholecystectomy; laparoscopic hernia repair.)

This article is accompanied by a Highlight. Please see this issue of ANESTHESIOLOGY, page 27A.

* Staff Anesthesiologist, Istituto di Anestesia e Rianimazione, Faculty of Medicine, University of Trieste.

† Anesthetist-in-Chief and Associate Professor, Department of Anesthesia, Royal Victoria Hospital, McGill University.

‡ Clinical Fellow, Department of Anesthesia, Royal Victoria Hospital, McGill University.

§ Associate Professor of Anesthesiology, Istituto di Anestesia e Rianimazione; Faculty of Medicine, University of Trieste.

|| Surgeon-in-Chief and Professor and Chairman, Department of Surgery, Royal Victoria Hospital, McGill University.

Director of Medical Division of Critical Care and Associate Professor of Medicine and Physiology, Royal Victoria Hospital, McGill University.

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Address reprint requests to Dr. Magder: Critical Care Division, L3.05, Royal Victoria Hospital, 687 Pine Avenue West, Montreal, Quebec, Canada H3A 1A1.

LAPAROSCOPIC surgery recently has increased in popularity. It is less invasive,¹ seems to produce less stress to the patient,² and can reduce costs related to hospitalization.³ It also has been suggested that laparoscopic surgery may reduce postoperative pulmonary complications²⁻⁶ by avoiding the restrictive pattern of breathing that usually follows upper abdominal surgery. This pattern is characterized by a reduction of inspiratory capacity and vital capacity, and by alveolar hypoventilation,^{7,8} which can occur despite increased metabolic demands⁵ and oxygen consumption.⁹

Craig⁷ proposed that this ventilatory derangement is a consequence of the surgical trauma to the abdominal wall, and Ali and Khan¹⁰ demonstrated that the specific surgical approach and the type of incision affect the incidence of postoperative pulmonary complications. Ford *et al.*¹¹ showed that the reduction in diaphragmatic performance is the main determinant of compromised lung function after upper abdominal surgery. Other investigators,²⁻⁵ in preliminary reports, have proposed that a less pronounced decrease in ventilatory volumes is associated with faster recovery, when laparoscopic cholecystectomy is compared to "open" cholecystectomy. They speculated that laparoscopic

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surgery for upper abdominal procedures can minimize the risk of postoperative ventilatory dysfunction by reducing the tissue trauma.²

However, clinical^{11,12} and experimental¹³⁻¹⁶ evidence suggest that diaphragmatic impairment after upper abdominal surgery is better explained by reflexic inhibition of phrenic nerve efferent activity because of irritation of splanchnic afferents, rather than contractile failure of the diaphragm or surgical trauma to the abdominal wall. If true, laparoscopic cholecystectomy may not spare diaphragmatic function.

Therefore, the aim of this study was to evaluate the hypothesis that ventilatory performance is still impaired after laparoscopic cholecystectomy because the site of operation is adjacent to reflexogenic splanchnic areas. Furthermore, we hypothesized that a similar laparoscopic procedure, performed remote from neural afferents, should not be affected by the same respiratory complications and should not be associated with diaphragmatic depression. We tested this by comparing patients undergoing laparoscopic cholecystectomy and patients having laparoscopic hernia repair. These procedures share a similar surgical approach and are performed through small bilateral entry sites in the abdominal wall near the umbilical region.

Materials and Methods

Population

We studied ten adult patients (7 women, 3 men; age range 23–69 yr) undergoing elective laparoscopic cholecystectomy and five patients (all men; age range 32–67 yr) scheduled for laparoscopic hernia repair. All patients were ASA physical status 1 and free of any signs or symptoms of cardiorespiratory disease. This was assessed by a history and physical examination 2 weeks before surgery. Patients with a history of smoking, morbid obesity, and prior thoracic surgery were excluded. The protocol was approved by the Ethics Committee of the Royal Victoria Hospital, McGill University. Written informed consent was obtained from each patient.

Anesthesia

The same anesthetic protocol was used for both groups of patients. Sublingual lorazepam, 2 mg, was administered approximately 30 min before the preoperative test. General anesthesia was induced with 1.5 µg/kg intravenous fentanyl, 4–6 mg/kg thiopental,

and 1.5 mg/kg succinylcholine. Following tracheal intubation, anesthesia was maintained with isoflurane in 70% N₂O and 30% O₂, 0.1 mg/kg vecuronium, and 25–50 µg intermittent intravenous fentanyl. Throughout anesthesia, a Datex Capnomat (Helsinki, Finland) was used to continuously monitor hemoglobin oxygen saturation, end-tidal carbon dioxide (PETCO₂),^{17,18} and end-tidal isoflurane, until the time of extubation. Incremental boluses of vecuronium, 0.5 mg intravenously, were administered on the basis of the results of train-of-four monitoring of abductor pollicis twitch. The same degree of intraperitoneal carbon dioxide insufflation (maximum pressure 15 mmHg) was used for both laparoscopic cholecystectomy and laparoscopic hernia repair. At the end of surgery, after inhalation anesthesia was discontinued, residual neuromuscular blockade was reversed with 50 µg/kg intravenous neostigmine and 0.6 mg atropine. Both types of operations were performed by the same surgeon, and the duration was similar for both procedures: 59 ± 11 min for laparoscopic cholecystectomy and 58 ± 8 min for laparoscopic hernia repair ($P = 0.8$).

Surgery

Both of the laparoscopic procedures were initiated through the umbilicus to create a pneumoperitoneum at a pressure of 15 mmHg. Cholecystectomy required 5-mm ports placed in the right upper and lower quadrants and another 10-mm port in the midline epigastrium, 5 cm below the xiphisternum. The hernia repair required two 10- or 12-mm ports at the lateral edge of the recti muscles, at the level or just inferior to the umbilicus.

Equipment

Measurements were carried out with the Medgraphics CCM (CCM) (Medical Graphics, St. Paul, MN), a critical care monitoring package that incorporates breath-by-breath indirect calorimetry and a respiratory pressure/flow waveform analyzer. Ventilatory volumes were measured with a Hans-Rudolph valve (Kansas City, MO) connected to a Valyline DP250 pressure transducer (Northridge, CA). Volume was calculated by integrating the flow signal with a waveform analyzer and converted from absolute temperature pressure to body temperature pressure. Oxygen concentration was measured by a zirconium oxide electrode and the carbon dioxide concentration by an infrared analyzer (Datex). The inspired and expired gases were sampled continuously at the level of the pneumotachograph and integrated

against time as well as the flow signal. The following parameters were computed on-line, breath-by-breath: oxygen uptake (\dot{V}_{O_2}), carbon dioxide output (\dot{V}_{CO_2}), PET_{CO_2} , tidal volume (V_t), expiratory time, and total breathing-cycle time (T_{tot}). Data were processed on-line by a Mitsubishi computer, and respiratory quotient, inspiratory time (T_i), mean inspiratory flow (V_i/T_i), duty cycle (T_i/T_{tot}), breathing frequency, and minute ventilation were derived. All variables were averaged over 100 breaths.¹⁹

Respiratory pressures, including maximal inspiratory pressure generated at the mouth during a maximal Muller maneuver against a closed shutter, mouth pressure generated at the first 100 ms of a tidal occluded inspiration ($P_{0.1}$) and esophageal pressure, were recorded by three Valyline DP250 differential pressure transducers, the respective ranges of which were ± 300 , ± 150 , and ± 150 cmH₂O. To measure transdiaphragmatic pressure (Pdi),²⁰ we had to modify the commercial system. The two ports of the differential pressure transducer available for the maximal inspiratory pressure were connected to esophageal and gastric balloon-tipped catheters. The reference point for gastric (abdominal) pressure was thus the esophageal (pleural) instead of atmospheric pressure. Unfortunately, with this method, we could not partition, within the same breath, the contribution of ribcage muscles and diaphragm to the development of Pdi.

Before beginning the study, we validated the Pdi measurements in a healthy volunteer. The subject swallowed two polyethylene catheters, each 94 cm long and with an inner diameter of 1.67 mm. The tubes had multiple holes over the last distal 4 cm. Latex balloons, 5 cm long with a circumference of 3.2 cm, were sealed over the distal portion of both catheters, and these were filled with 0.5 ml. With the subject in the sitting posture and breathing through a mouthpiece connected to the pneumotachograph of the CCM, the position of the esophageal catheter was checked according to the method described by Baydur *et al.*²¹ The proximal ends of both catheters were connected to a short "Y" piece of same inner diameter. One branch of the "Y" connector was attached to the CCM differential pressure transducer, and the other to pressure transducers connected to amplifiers and an eight-channel paper recorder (HP 7758B System, Waltham, MA). The three signals (Pdi from the CCM and esophageal pressure and gastric pressure from the external amplifiers) were sent to an analog-to-digital board and fed into a computer, which allowed simultaneous display

and analysis by an acquisition software package (Codaq Dataq, Akron, OH). The subject then was requested to breathe normally and to perform maximal Muller maneuvers against the closed shutter, while the tracings were displayed. There was good agreement between the two methods of measurement and no phase delay between signals.

Protocol

The pneumotachograph, pressure transducers, and gas analyzers were calibrated before each study. Patients were admitted to the recovery room 1.5 h before surgery and reclined in bed at a 45-degree angle throughout the test. The protocol consisted of three separate trials. In the first, we measured \dot{V}_{O_2} , \dot{V}_{CO_2} , respiratory quotient, PET_{CO_2} and ventilatory pattern (V_t , T_i/T_{tot} , V_i/T_i , breathing frequency, minute ventilation) breath-by-breath, for at least 100 breaths.¹⁹ In the second, we assessed the overall ventilatory drive by recording $P_{0.1}$ during tidal breathing. Particular care was taken to ensure that the patients were unaware of the maneuver.²² This was performed by randomly occluding the airway with a pressurized shutter (a cylindrical balloon) built inside the pneumotachograph. The closure, triggered by the computer, was maintained for less than 200 ms, and the pressure that developed at 100 ms was analyzed.²² All occlusions were performed at the end of expiration.²² Four or five maneuvers were accomplished in each subject at baseline PET_{CO_2} . In the third part, diaphragmatic function was assessed. After the subjects received nasal and pharyngeal topical anesthesia, they were asked to swallow esophageal and gastric catheters. The features of the tubes were the same as described above in the validation of the equipment. The position of the catheters was checked using the software of the CCM package, which refers to the technique of Baydur *et al.*²¹ already mentioned.

We measured maximal Pdi (Pdi_{max}) in all patients by asking them to perform a maximal Muller maneuver against the closed shutter while we observed the pressure tracings on the computer monitor; this provided visual feedback.²⁰ All occlusions were performed at the end of expiration, and each lasted 3 s. The simultaneous tracings of the spirogram allowed inflation of the shutter to be applied always at the same end-expiratory volume, thus ensuring good reproducibility of the maneuver (fig. 1).

In five of the subjects undergoing laparoscopic cholecystectomy and in all of the patients having laparoscopic hernia repair, we also measured Pdi that devel-

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oped during a maximal sniff (Pdi_{sniff})²³ to discriminate between a peripheral (muscular) diaphragmatic dysfunction and a reflexic (neural) inhibition. During this trial, the patients were not connected to the mouth-piece. They were told to keep their mouths closed and perform a sharp maximal sniff from the relaxed expiratory volume, and Pdi was measured. The four best reproducible measurements of a total of ten were used for Pdi_{sniff} and Pdi_{max} . Usually the patients required two or three attempts before becoming confident with the maneuver.

All measurements were repeated 2–3 h after surgery, when the subjects were fully cooperative and able to maintain the same posture as during the preoperative trials. In all but two patients undergoing laparoscopic cholecystectomy, morphine was administered in incremental intravenous boluses of 2.5 mg, up to a total of 15 mg within 2 h postoperatively. The dose depended upon the subject's requirement. All patients were free of pain throughout the postoperative test, and hemoglobin oxygen saturation was normal, as assessed by continuous monitoring with a Biox 3700 pulse oximeter (Ohmeda, Rexdale, Ontario, Canada).

Statistics

Mean values were used for statistical analysis, and \dot{V}_{O_2} , \dot{V}_{CO_2} , V_t , and minute ventilation were normalized per body-surface-area. Paired *t* tests were applied to compare postoperative to preoperative condition, each patient being his/her own control. All differences were considered significant at the $P < 0.05$ level.

Results

Diaphragmatic Function

In patients undergoing laparoscopic cholecystectomy, Pdi_{max} , in the early postoperative period, decreased from 83 ± 10 cmH₂O (mean \pm SEM) to 41 ± 5 cmH₂O ($P < 0.01$; table 1). The magnitude of the decrease ranged from 17% to 79% of the control value. In contrast, in the laparoscopic hernia repair group, Pdi_{max} did not change significantly ($P = 0.58$) and increased in three of the five subjects (table 2). Pdi_{sniff} did not decrease after surgery in laparoscopic hernia repair group ($P > 0.12$) nor in the five patients undergoing laparoscopic cholecystectomy in which it was assessed ($P > 0.77$), thus excluding an alteration of diaphragmatic contractility. Figure 2 is a representative tracing of pre- and postoperative Pdi_{sniff} .

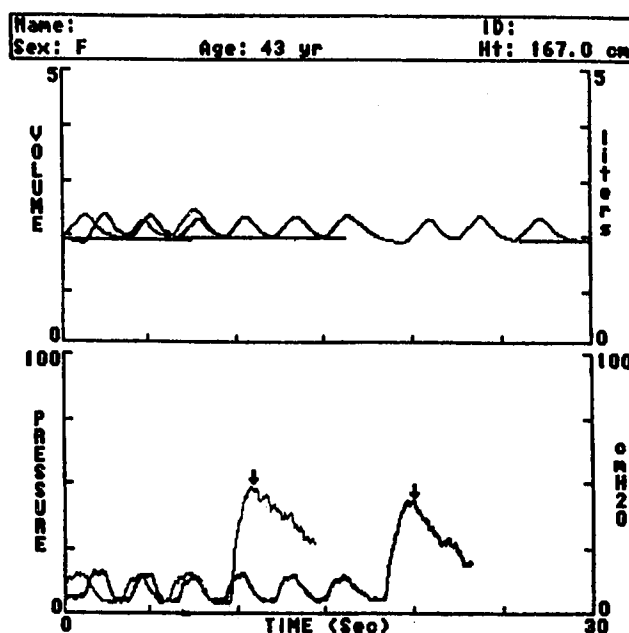


Fig. 1. Pdi_{max} Assessment = tracings obtained from Medgraphics CCM display during preoperative test in patient 6 (laparoscopic cholecystectomy group). (Top) Tidal volume against time (expiration downward); horizontal lines indicate the resting volume level. (Bottom) Tidal Pdi and two Pdi_{max} maneuvers (arrows) performed at the same resting volume.

Ventilatory Pattern

During the postoperative trial, the breathing pattern in the laparoscopic cholecystectomy group changed considerably from the preoperative test (fig. 3, table 1). V_t decreased from 324 ± 24 to 228 ± 16 ml/m² ($P < 0.01$). This decrease was mainly caused by a shortening of the T_i , which decreased T_i/T_{tot} from 0.47 ± 0.01 to 0.41 ± 0.02 ($P < 0.01$). The V_t/T_i values, of 138 ± 12 ml/s/m² before and 149 ± 12 ml/s/m² after surgery, were not significantly different ($P > 0.43$). The breathing frequency increased in laparoscopic cholecystectomy subjects from 12 ± 1 to 16 ± 1 breaths/min ($P < 0.01$). This rapid and shallow pattern of breathing led to a rise of PET_{CO_2} from 35 ± 1 to 38 ± 1 mmHg ($P < 0.01$). Because minute ventilation and \dot{V}_{CO_2} did not change, we can infer that the V_D/V_T ratio increased.²⁴

Respiratory Drive

The drive to breathe, estimated as $P_{0.1}$ at tidal ventilation, was not responsible for the fall of Pdi_{max} and V_t , because it did not vary significantly in both groups of subjects and increased in three of the ten patients undergoing laparoscopic cholecystectomy (fig. 4).

Table 1. Laparoscopic Cholecystectomy

	BSA (m ²)	V _t (ml · m ⁻²)	PET _{CO₂} (mmHg)	F _R (breaths/min)	V _{O₂} (ml · min ⁻¹ · m ⁻²)	V _{CO₂} (ml · min ⁻¹ · m ⁻²)	RQ	V _I /T _I (ml · s ⁻¹ · m ⁻²)	T _I /T _{tot}	V _E (l · min ⁻¹ · m ⁻²)	Pdi _{max} (cmH ₂ O)	Pdi _{sniff} (cmH ₂ O)
Preoperative												
1	1.95	333	38	8	110	76	0.70	93	0.47	2.7	131	
2	1.77	354	30	16	158	131	0.83	177	0.54	5.7	109	
3	1.88	314	35	15	148	119	0.80	185	0.43	4.7	126	
4	2.21	330	34	12	133	90	0.68	143	0.46	3.9	89	
5	1.68	453	28	10	127	91	0.72	146	0.51	4.5	88	
6	2.06	220	37	15	112	77	0.69	147	0.38	3.3	46	77
7	1.76	281	36	10	106	78	0.74	97	0.48	2.8	60	62
8	1.77	328	37	8	93	68	0.73	89	0.49	2.7	77	92
9	1.53	210	40	17	98	80	0.82	116	0.52	3.6	52	59
10	2.19	419	30	12	127	105	0.83	182	0.46	5	53	55
Mean		324	35	12	121	92	0.75	138	0.47	3.9	83	
SE		24	1	1	7	7	0.02	12	0.01	0.3	10	
Postoperative												
1	1.95	263	39	15	133	98	0.74	165	0.40	3.9	28	-79%
2	1.77	247	34	19	146	97	0.67	190	0.41	4.7	48	-56%
3	1.88	236	39	17	141	101	0.72	168	0.40	4.0	61	-52%
4	2.21	241	38	14	131	79	0.60	115	0.49	3.4	60	-33%
5	1.68	202	37	14	94	61	0.65	112	0.42	2.9	27	-69%
6	2.06	138	41	15	73	49	0.67	99	0.36	2.1	30	-35%
7	1.76	219	36	21	130	93	0.72	169	0.47	4.6	30	-50%
8	1.77	249	43	12	96	69	0.72	113	0.44	3.0	64	-17%
9	1.53	167	44	21	99	69	0.70	139	0.41	3.5	21	-60%
10	2.19	323	33	12	113	85	0.75	215	0.29	3.9	40	-25%
Mean		228	38	16	116	80	0.69	149	0.41	3.6	41	
SE		16	1	1	8	6	0	12	0	0	5.2	
P	<0.01	<0.01	<0.01	<0.01	NS	NS	0.01	NS	<0.01	NS	<0.01	

Values inside the box express the percent decrease in maximum transdiaphragmatic pressure from control (preoperative) values. BSA = body surface area; V_t = tidal volume; PET_{CO₂} = end-tidal carbon dioxide pressure; F_R = breathing frequency; V_{O₂} = oxygen consumption; V_{CO₂} = carbon dioxide production; RQ = respiratory quotient; T_I = inspiratory time; T_{tot} = total respiratory time; V_E = minute ventilation; Pdi_{max} = maximum transdiaphragmatic pressure; Pdi_{sniff} = transdiaphragmatic pressure during sniff maneuver.

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Table 2. Laparoscopic Hernia Repair

		BSA (m ²)	V _I (ml/min)	PET _{CO₂} (mmHg)	F _b (breaths/ min)	\dot{V}_{O_2} (ml·min ⁻¹ · m ⁻²)	\dot{V}_{CO_2} (ml·min ⁻¹ · m ⁻²)	RQ	V _I /T _I (ml·s ⁻¹ · m ⁻²)	T _I /T _{tot}	$\dot{V}E$ (l·min ⁻¹ · m ⁻²)	Pdi _{max} (cmH ₂ O)
Preoperative	1	1.93	230	37	20	144	105	0.74	177	0.42	4.4	61
	2	1.93	350	40	8	137	96	0.70	106	0.44	2.8	104
	3	1.78	406	34	9	102	87	0.84	150	0.41	3.8	86
	4	1.74	351	38	10	109	93	0.84	130	0.45	3.5	109
	5	2.26	496	32	9	123	115	0.94	171	0.44	4.4	123
	Mean		367	36	11	123	99	0.81	147	0.43	3.8	98
	SE		43	1	2	8	5	0.04	13	0.01	0.3	9.1
Postoperative	1	1.93	245	38	18	142	107	0.75	163	0.45	4.4	73
	2	1.93	350	43	10	134	85	0.66	140	0.42	3.0	126
	3	1.78	333	39	10	117	81	0.70	128	0.43	3.3	83
	4	1.74	302	40	12	130	94	0.72	131	0.47	3.6	111
	5	2.26	525	32	8	129	108	0.83	131	0.53	4.2	112
	Mean		351	38	12	130	95	0.73	139	0.46	3.7	101
	SE		47	2	2	4	6	0.03	6	0.02	0.3	9.8
	P	NS	NS	NS	NS	NS	<0.05	NS	NS	NS	NS	

Abbreviations are defined in the footnote to table 1.

Gas Exchange

\dot{V}_{O_2} and \dot{V}_{CO_2} did not change postoperatively in either group. The ratio of $\dot{V}_{CO_2}/\dot{V}_{O_2}$, *i.e.*, the respiratory quotient, increased postoperatively ($P < 0.01$ in laparoscopic cholecystectomies, $P < 0.05$ in laparoscopic hernia repairs) because changes in \dot{V}_{O_2} and \dot{V}_{CO_2} were not proportional.

Postoperative Analgesics

In the cholecystectomy group, eight patients received an average of 11.9 ± 5.5 mg morphine intravenously for postoperative pain and two received an average of 3.5 mg droperidol. In the laparoscopic hernia repair

group, the five patients received an average dose of morphine of 5.5 ± 2.7 mg. There was no relationship between the morphine dose and the decrease in Pdi_{max} postoperatively ($r = 0.198$).

Discussion

The major observation of this study is that ventilatory performance is depressed after laparoscopic cholecystectomy. This impairment of ventilatory function was characterized by a decrease of maximal voluntary Pdi and V_I, and by an increase in V_D/V_T ratio, which led to an increase in PET_{CO₂}. The decrease in Pdi was not ap-

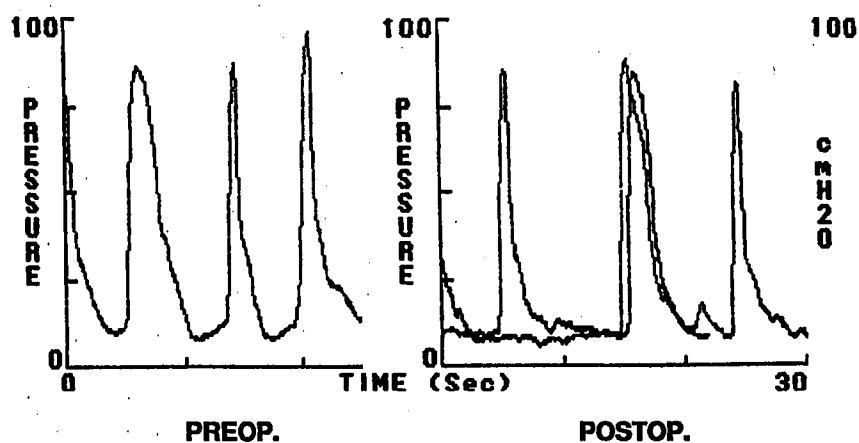


Fig. 2. Pdi_{nltr} = preoperative and postoperative assessment of Pdi_{nltr}. Tracings were obtained from Medgraphics CCM display in patient 8 (laparoscopic cholecystectomy group) as visual feedback was used. The overlapped tracings occur because the screen was not cleared until the trains of maneuvers were completed.

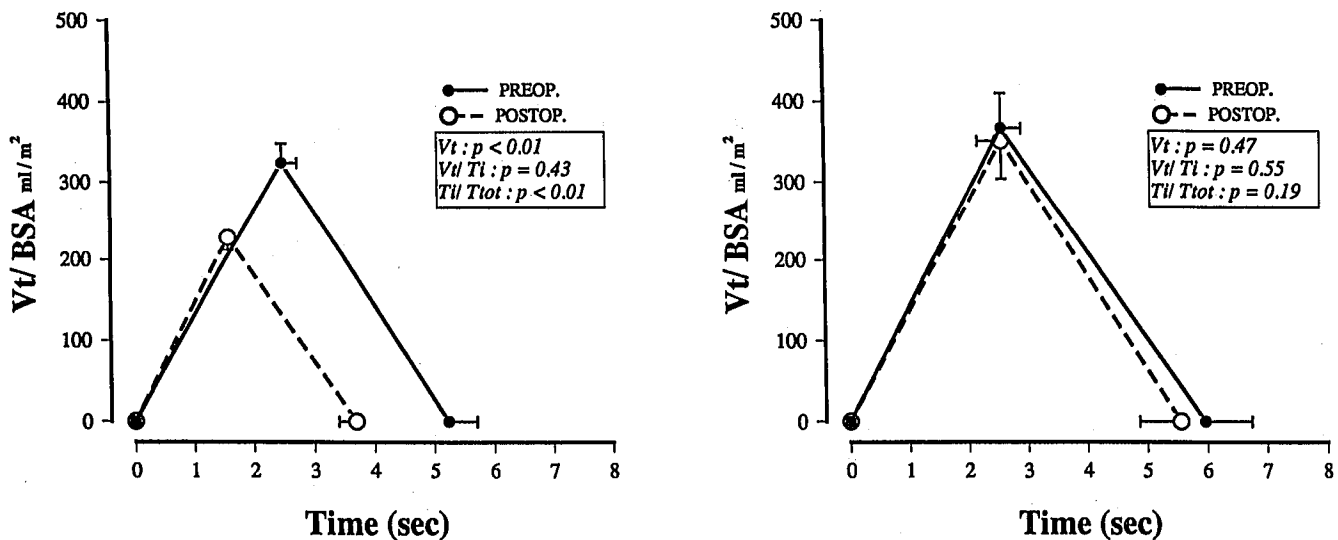


Fig. 3. Breathing Pattern = preoperative and postoperative assessment of V_t against time, from which V_t/T_i and T_i/T_{tot} can be inferred. After laparoscopic cholecystectomy (*left*) V_t decreased because of a decrease in T_i , with no change in V_t/T_i but a decrease in T_i/T_{tot} . Breathing pattern did not change with surgery in laparoscopic hernia repair group (*right*). Bars = SEM. V_t values are normalized per body-surface-area.

parent during a maximal sniff maneuver. To our knowledge, there are no other measurements of Pdi_{max} after laparoscopic cholecystectomy.

It would have been ideal to have included patients undergoing cholecystectomy through a standard laparotomy, but in our institution, "open" cholecystectomies are restricted to patients with complicated cholelithiasis and, hence, the comparison with laparoscopic cholecystectomy group would have been difficult to interpret. On the other hand, a comparison of case-controlled, nonrandomized patients would have included too many biases between groups, including differences in surgeons and patient selection.

Before discussion of the results, a number of technical aspects need to be reviewed. Although there was a greater proportion of women in the laparoscopic cholecystectomy group compared to the laparoscopic hernia repair group, we believe that this did not negatively influence our findings, for the decrease in Pdi_{max} in the three men of the laparoscopic cholecystectomy group (patients 1, 4, and 10) were large; they were 79%, 33%, and 25%, respectively. Therefore, gender does not appear to be a significant variable.

The impairment of diaphragmatic contractility might have developed from a change in lung volume in the postoperative period. General anesthesia induces a reduction of functional residual capacity,⁶⁻²⁵ and breathing at low lung volumes can increase ventilation/per-

fusion mismatch because of the shift of ventilation from lung bases to apices.^{7,8,11,25} Although we did not measure resting volume in our patients, a reduction in functional residual capacity is unlikely for several reasons. First, it has been demonstrated that, after general anesthesia, functional residual capacity completely returns to preoperative values and does not change for at least 4 h after surgery.^{7,12} Second, Road *et al.*¹⁶ found that the static pressure-volume curves and dynamic compliance of the lungs of dogs undergoing abdominal surgery did not change in the early postoperative hours. Third, an alteration of resting lung volume would have changed the operating length of diaphragmatic fibers as well as their orientation,^{15,20} which would have affected the tension^{12,20} achieved at Pdi_{max} and the tension in the Pdi_{sniff} maneuver but did not. If anything, a decrease in lung volume would have increased diaphragmatic length and increased Pdi_{max} , which would have biased against our results. Fourth, a lung and/or chest wall compliance decrease in the early postoperative period should have been reflected in an increase of effective inspiratory impedance,²⁴ which is defined as the sum of forces that must be overcome during inspiration. These include resistance, lung elastance and inertance, and chest wall deformation.²⁶ Inspiratory impedance can be represented by the ratio $P_{0.1}/(V_t/T_i)^{-1}$,^{24,26} and it did not change after surgery in either group.

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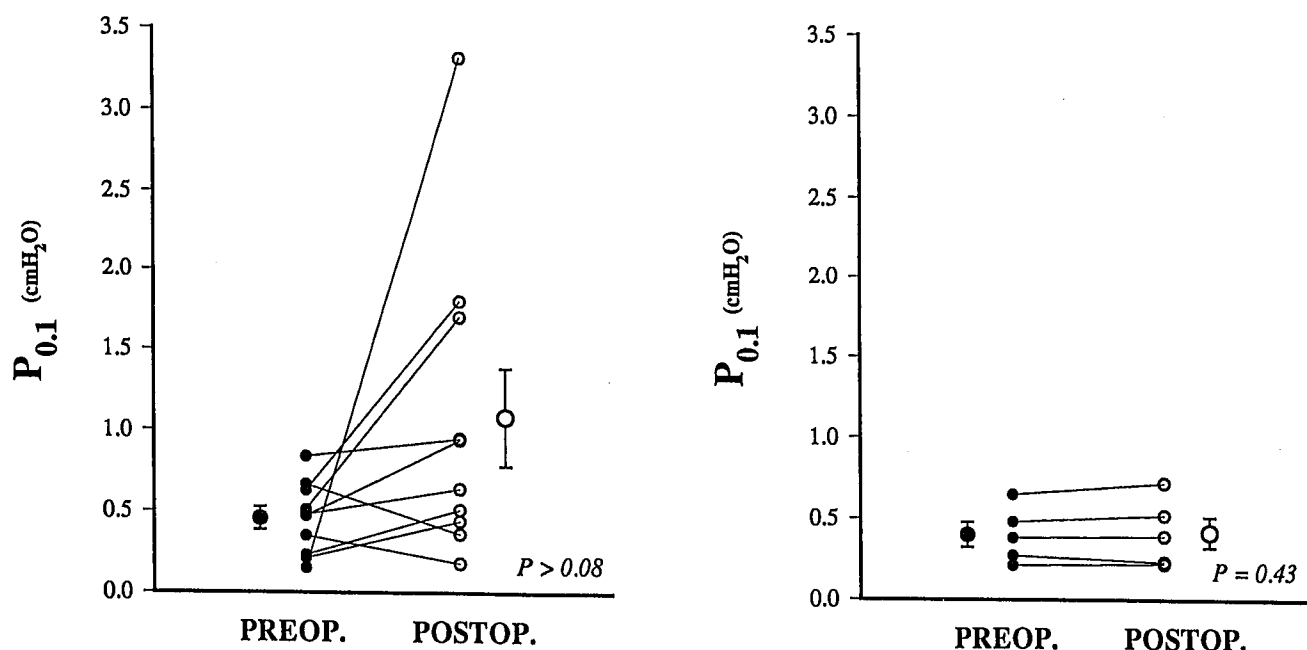


Fig. 4. Respiratory Drive = preoperative and postoperative assessment of $P_{0.1}$ in laparoscopic cholecystectomy (left) and laparoscopic hernia repair (right) group during tidal breathing. Bars = SEM.

Recently, Bellemare *et al.*²⁷ showed that the contractile properties of the human diaphragm can be studied by bilateral maximal twitch stimulation of the phrenic nerves. Although this technique is feasible in healthy and trained volunteers, its application in postoperative patients is difficult and cumbersome. Another way to test the involuntary response of the diaphragm is the maximal sniff maneuver. Esau *et al.*²³ demonstrated that a short, sharp sniff can approximate the diaphragmatic contraction induced by a brief stimulation of the phrenic nerves. They suggested that this occurs because the same proportion of fast- and slow-twitch diaphragmatic fibers are activated in both circumstances. The fact that Pdi_{sniff} was not altered indicates that the decrease in voluntary Pdi_{max} was not due to an impairment of contractility, and we thus were able to avoid the need for electrical stimulation of the phrenic nerve.

In patients undergoing laparoscopic cholecystectomy, the decrease of Pdi_{max} was associated with decreased V_t , smaller T_i/T_{tot} , and increased breathing frequency; the breathing pattern was more rapid and shallow than in the preoperative period. V_i/T_i did not change after surgery, and $P_{0.1}$ also was not significantly different because of the wide standard deviation, although there was a tendency toward a postoperative increase. Therefore, depressed respiratory drive does

not explain the reduction of Pdi_{max} . The preservation of $P_{0.1}$ occurred even though all patients received narcotics for postoperative analgesia. Thus, the $P_{0.1}$, V_i/T_i , and breathing frequency values may have been even higher without analgesia. Of note is that the total amount of opiates received by each subject after surgery was not related to the magnitude of drop of Pdi_{max} .

All patients were free of pain during the postoperative trial, so that performing the maneuvers did not provoke discomfort. Furthermore, other investigators have documented that epidural analgesia does not prevent postoperative diaphragmatic dysfunction.^{25,26} This excludes nociceptive abdominal and parietal receptors as major contributors to the development of postoperative pulmonary complications.

Postoperative ventilatory dysfunction also could be explained by inhibitory reflexes arising from the abdominal wall as a result of the surgical trauma. To test this hypothesis, we compared the laparoscopic cholecystectomy group to the five patients undergoing laparoscopic hernia repair. The laparoscopic hernia repair patients had no postoperative ventilatory impairment, despite the fact that they underwent the same surgical preparation as the laparoscopic cholecystectomy group. The surgeon performed the same number of incisions, close to the same abdominal regions in all patients. The volume of carbon

dioxide, delivered to inflate the peritoneal cavity, produced the same intraabdominal pressure in both groups of patients, and the time of surgery did not differ between groups. This excludes trauma to the abdominal wall as the possible source of inhibitory reflexes. Our observation is in line with a previous study conducted by Dureuil *et al.*¹² in patients undergoing "open" cholecystectomy. They suggested that the main mechanism for altered diaphragmatic activity, after standard upper abdominal surgery, is reflexic inhibition of phrenic nerve output, rather than the surgical trauma to the abdominal wall. The afferents for the reflex most likely come from the celiac sympathetic plexus or other upper abdominal sympathetic ganglia.

The main difference between laparoscopic cholecystectomy and laparoscopic hernia repair was the different internal site at which the surgeon operated. Of importance, laparoscopic cholecystectomy involves visceral afferents of the mesenteric region. It has been shown that electrical stimulation of mesenteric nerves¹³ and sympathetic afferents,¹⁴ as well as mechanical distension of the small bowel,¹³ can produce inhibition of phrenic nerve efferent discharge and enhancement of external intercostal muscle activity. Therefore, we believe that this is the best explanation for the decrease in $P_{di_{max}}$ with laparoscopic cholecystectomy and not with laparoscopic hernia repair. If true, then the internal anatomic site of intervention is the crucial factor for determining whether postoperative ventilatory dysfunction will occur. Thus, though a laparoscopic intervention is less invasive than the standard approach, ventilatory impairment should be expected after upper abdominal procedures. In support of this, a recent randomized trial showed faster recovery of functional residual capacity in patients undergoing laparoscopic *versus* open cholecystectomy.⁶

We also have excluded that, at least in the early postoperative period, increased metabolic demands complicate ventilatory derangement after laparoscopic surgery. \dot{V}_{O_2} and \dot{V}_{CO_2} did not significantly change in either group, although there was a tendency for \dot{V}_{CO_2} to decrease proportionally more than \dot{V}_{O_2} , resulting in a significantly lower postoperative respiratory quotient in all patients. Continuous monitoring of PET_{CO_2} allowed an early establishment of mechanical hyperventilation during intraoperative carbon dioxide inflation. It appears from previous studies that carbon dioxide clearance is not a problem during laparoscopic surgery, when the amount of mechanical ventilation is set by the level of PET_{CO_2} .

As the overall respiratory drive was unchanged, ventilatory dysfunction may have been a consequence of the reduced diaphragmatic contribution to tidal breathing, with predominant activation of ribcage and accessory muscles. Reflexic inhibition of phrenic nerve output is a very likely mechanism for the depressed diaphragmatic activity after laparoscopic cholecystectomy. Therefore, patients with preexisting limited cardiorespiratory performance still may be prone to postoperative pulmonary complications.^{7,10,11}

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