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# Diaphragmatic Activity after Laparoscopic Cholecystectomy

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***Background:*** **Laparoscopic cholecystectomy is presumed to induce a reduction in diaphragmatic activity. Indirect indices of diaphragmatic function based on tidal changes in pressures and cross-section area measurements can be unreliable in the postoperative phase. The present study evaluates diaphragmatic activity by directly recording diaphragmatic EMG ($EMG_{dia}$) data, along with indirect indices.**

***Methods:*** **Thirteen adult patients (American Society of Anesthesiologists physical status I or II) undergoing laparoscopic cholecystectomy were examined preoperatively for inspiratory tidal changes in gastric ($P_{gas-insp}$) and esophageal ($P_{eso-insp}$) pressures, and tidal changes in ribcage ($V_{thor}$) and abdominal ($V_{abd}$) cross-section areas and then again at 1, 6, and 24 h postoperatively combined with $EMG_{dia}$ recordings. Variations in inspiratory gastric ($\Delta P_{gas-insp}$) and inspiratory transdiaphragmatic ($\Delta P_{di-insp}$) pressures were derived from the above.**

***Results:*** **Laparoscopic cholecystectomy induced a significant reduction in mean $\Delta P_{gas-insp}$, mean $\Delta P_{di-insp}$, and mean $V_{abd}$ indicating a reduction of diaphragmatic activity postoperatively. $\Delta P_{di-insp}$ decreased from 11.8 ± 4.0 cm $H_2O$ preoperatively to 5.7 ± 5.7 cm $H_2O$ at 1 h and 6.6 ± 5.1 cm $H_2O$ at 6 h postoperatively (mean ± SD; $P < 0.05$). $V_{abd}$ decreased from 327.0 ± 113.0 ml preoperatively to 174.0 ± 65.0 ml at 1 h and 175.0 ± 98.0 ml at 6 h postoperatively (mean ± SD; $P < 0.05$). These values had partially recovered at 24 h.**

***Conclusion:*** **The direct and indirect indices of diaphragmatic activity taken together confirm the presence of reduction in diaphragmatic activity after laparoscopic cholecystectomy followed by its partial recovery at 24 h. (Key words: Abdominal dimensions; diaphragmatic EMG; esophageal pressure; gastric pressure; ribcage dimensions.)**

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PATIENTS are prone to a reduction in diaphragmatic activity after upper abdominal surgery,[1] and this is presumed to contribute to postoperative pulmonary complications.[2] Some large epidemiologic studies have reported reduced incidence of postoperative pneumonia after laparoscopic compared with open cholecystectomy.[3] Other studies have shown that the reduction in lung volumes is less pronounced after laparoscopic than open cholecystectomy.[4,5] Erice *et al.*[6] found that diaphragmatic activity is reduced after upper abdominal but not after lower abdominal laparoscopic surgery. Conversely, Couture *et al.*[7] more recently reported intact diaphragmatic activity during quiet breathing in patients after laparoscopic cholecystectomy. Both studies used indirect indices (*i.e.*, pressure and volume-motion changes) to measure diaphragmatic contribution to ventilation. It was reported earlier that an increase in abdominal muscle activity after abdominal surgery can influence these indirect indices of measurements.[8] The present study was therefore designed to analyze the changes in diaphragmatic activity and to define the time course of these changes using not just the indirect indices of diaphragmatic activity but also direct measurements (*i.e.*, diaphragmatic EMG [$EMG_{dia}$) in patients undergoing laparoscopic cholecystectomy.

## Material and Methods

### *Population*

We studied 13 adult patients (10 women, 3 men; age range, 33-67 yr), all American Society of Anesthesiologists physical status I or II, undergoing elective laparo-

**Table 1. Patient Characteristics and Surgical Details**

| Patient Number | Age (yr) | Sex | Operation Time (min) |
|---|---|---|---|
| 1 | 54 | F | 90 |
| 2 | 40 | F | 70 |
| 3 | 50 | F | 125 |
| 4 | 50 | F | 135 |
| 5 | 50 | F | 135 |
| 6 | 38 | F | 145 |
| 7 | 33 | F | 100 |
| 8 | 57 | F | 180 |
| 9 | 67 | F | 195 |
| 10 | 45 | M | 105 |
| 11 | 44 | M | 60 |
| 12 | 46 | F | 195 |
| 13 | 35 | M | 135 |

scopic cholecystectomy (table 1). Patients who had pulmonary disease or morbid obesity (*i.e.*, body mass index > 30) were excluded from the study. The patients gave their informed consent to participate in the investigation, which was approved by the Ethics Committee of the University Hospital, Umeå, Sweden.

*Gastric and Esophageal Pressures*

Prior to premedication, a triple lumen nasogastric catheter with its distal orifice located 3 cm from the tip and the other two orifices located 10 and 20 cm proximal to the distal orifice (PVC, esophageal manometric catheter, 3.6 mm OD, 90 cm long, Synectics AB, Stockholm, Sweden) was inserted through one of the nostrils and placed initially with all three orifices in the stomach. Each lumen was connected to a pressure transducer (Ohmeda, DT-XX, Hatfield, Herts, United Kingdom), which was placed at the mid-axillary level and calibrated to the atmospheric pressure and a hydrostatic pressure of 10 cm and flushed thoroughly with normal saline solution to remove air bubbles from the system. The catheter was kept patent by a continuous infusion of normal saline at 3 ml/h. Initially all recorded pressures were the same, *i.e.*, from the stomach. The catheter was then pulled out in a stepwise fashion, noting the recording from the middle hole. The arrival of the middle orifice at the lower esophageal region was marked by a sudden rise in pressure due to the presence of the resting lower esophageal sphincter tone, followed by a trace exhibiting cardiac artifacts and a negative deflection with inspiration. At this point the distal hole was in the stomach and the proximal hole in the esophagus 10 cm proximal to the lower esophageal region. The trace obtained from the distal hole revealed a positive pressure with inspiration and the trace from the proximal hole revealed cardiac artifacts and a negative deflection with inspiration further confirming that the distal and proximal holes were in the stomach and mid-esophagus, respectively. The catheter was then marked at the nasal entry point and secured to the nose by tape. The validation of fluid-filled catheter system for pleural and gastric pressure measurement is described elsewhere.[9,10] The pressure changes with quiet tidal respiration were recorded on a multichannel strip chart recorder (Mingograph 82, Siemens-Elema, Solna, Sweden) which was adjusted to give a 2- or 4-mm deflection for each mmHg pressure change using a test signal. All calibration procedures and the alignment of the fluid jet pens were checked before and after each set of measurements. The changes in gastric pressure ($P_{gas}$) and esophageal pressure ($P_{eso}$) with quiet tidal respiration were recorded from the distal and the proximal hole respectively on a strip chart at three different speeds: 10 mm/s, 25 mm/s, and 50 mm/s. These measurements were obtained preoperatively before premedication and at 1, 6, and 24 h postoperatively, all in the supine position. Tracings recorded at higher speed and with minimal cardiac artifacts were used for measurements as far as possible. Three consecutive pressure curves (and three rib cage and abdominal volume-motion curves recorded parallel to the pressure curves at the same time and on the same paper) were selected and analyzed over the same time period (fig. 1). The beginning and the end of each inspiration was determined after inspection of both the pressure and motion signals. The following measurements were derived from the $P_{gas}$ and $P_{eso}$ values: transdiaphragmatic pressure ($P_{di}$, the difference between simultaneously recorded $P_{gas}$ and $P_{eso}$ values, calculated by manual subtraction technique), gastric pressure variation with inspiration ($\Delta P_{gas\text{-}insp}$, the difference in $P_{gas}$ between the start and the end of inspiration), esophageal pressure variation with inspiration ($\Delta P_{eso\text{-}insp}$, the difference in $P_{eso}$ between the start and the end of inspiration), the ratio of changes in gastric to esophageal pressure variations during inspiration ($\Delta P_{gas\text{-}insp}/\Delta P_{eso\text{-}insp}$), and changes in transdiaphragmatic pressure variation during inspiration ($\Delta P_{di\text{-}insp}$, $\Delta P_{gas\text{-}insp} - \Delta P_{eso\text{-}insp}$). In addition, loops of $P_{gas}$ *versus* $P_{eso}$ were plotted to provide dynamic information of transdiaphragmatic pressure changes over time.

*Rib Cage and Abdominal Dimensions*

Tidal changes in rib cage and abdominal cross-section areas were measured using a respiratory inductive plethysmograph (RIP, Ambulatory Monitoring, Ardsley, NY) and inductor band transducers that were applied circum-

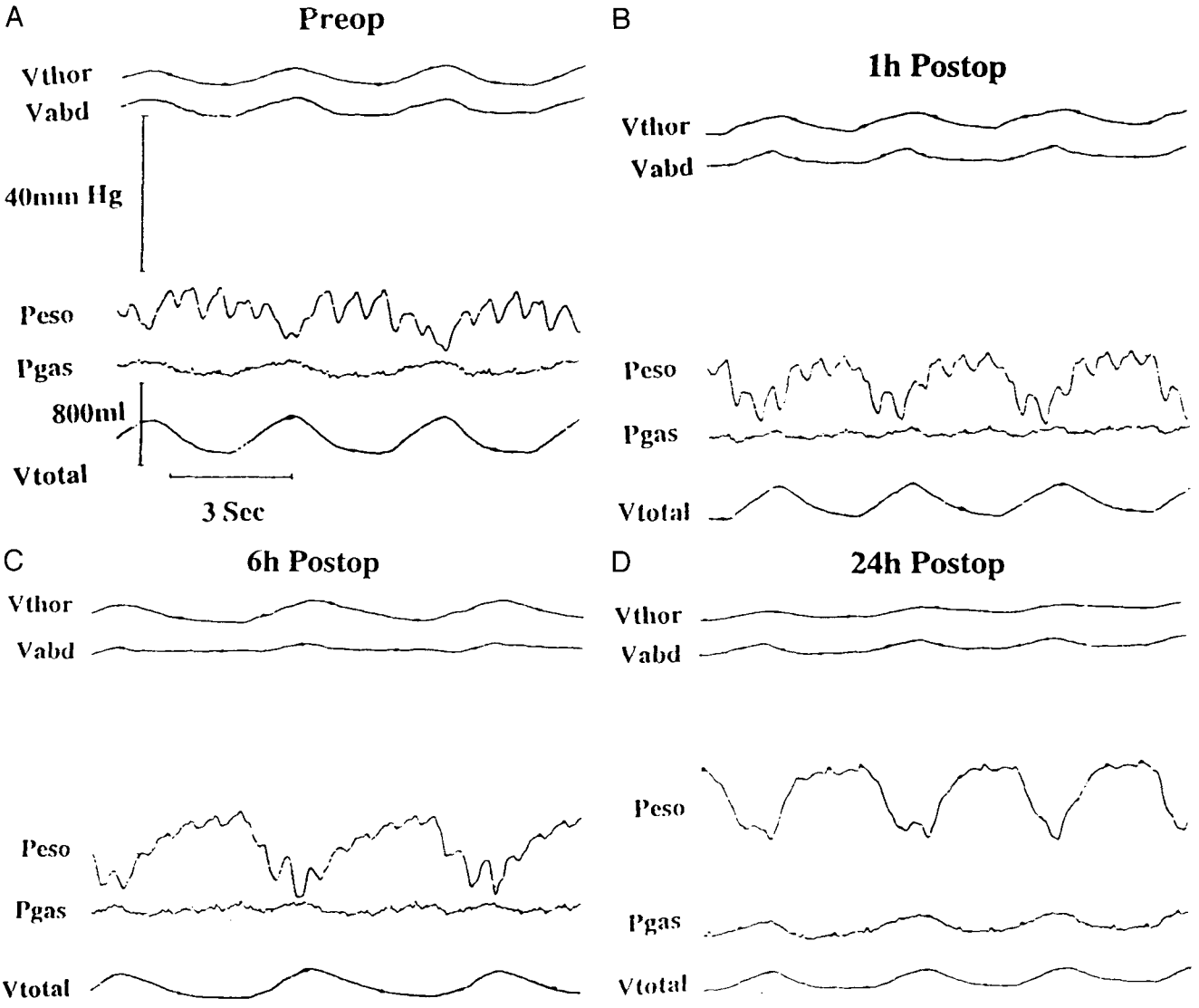


**Fig. 1. Traces of tidal changes in ribcage ($V_{thor}$) and abdominal ($V_{abd}$) cross-section areas and esophageal ($P_{eso}$) and gastric ($P_{gas}$) pressures as recorded on a multichannel strip chart recorder preoperatively (*A*) and 1 h (*B*), 6 h (*C*), and 24 h (*D*) postoperatively in a single patient.**

ferentially over the chest and abdominal wall. The rib cage inductor band was applied with its upper border just below the axillae and the abdominal inductor band was applied with its upper border at the level of umbilicus. Both levels were circumferentially marked on the patient's body with a permanent marker, and the size of inductor band was noted for subsequent measurements. The apparatus was zeroed, adjusted for unity gain using reference voltage, and volume motion coefficients for abdominal and thoracic excursions calculated using the least square method with patients breathing in and out of a fixed volume spirobag in both supine and standing position (before and 6 and 24 h after surgery) and supine and semirecumbent position (1 h after surgery). The calibration procedure, which was repeated before each set of measurements, has been described previously.[11] The multichannel recorder was adjusted to record 10 mm deflection/volt at each channel. The rib cage and abdominal cross-sectional area changes that corresponded to respective tidal volume changes to quiet respiration were recorded along with and at the same time interval as pressure measurements on the Mingograph 82 for approximately 6–8 min, following 5 min of quiet tidal respiration. Tidal volume (Vt) was calculated

**Table 2. Basic Ventilatory Measurements and Rib Cage and Abdominal Dimensions (n = 13)**

| Measurements | Control | 1 h Postoperation | 6 h Postoperation | 24 h Postoperation |
|---|---|---|---|---|
| Vt (ml) | 421.0 ± 125.0 | 344.0 ± 84.0 | 347.0 ± 143.0 | 420.0 ± 168.0 |
| $V_{abd}$ (ml) | 327.0 ± 113.0 | 174.0 ± 65.0* | 175.0 ± 98.0* | 260.0 ± 138.0 |
| $V_{abd}$(%) | 76.9 ± 11.0 | 51.0 ± 16.2* | 49.3 ± 16.6* | 59.8 ± 18.8 |
| $V_{thor}/V_{abd}$ | 0.33 ± 0.22 | 1.15 ± 0.68* | 1.31 ± 0.94* | 0.89 ± 0.81 |
| Respiratory rate (breaths/min) | 13.8 ± 4.5 | 14.3 ± 3.9 | 14.7 ± 3.0 | 15.1 ± 4.1 |
| Minute volume (l/min) | 5.60 ± 1.81 | 4.76 ± 1.20 | 5.16 ± 2.69 | 6.15 ± 2.88 |
| Inspiratory time/total respiratory time | 0.38 ± 0.04 | 0.34 ± 0.09 | 0.35 ± 0.05 | 0.33 ± 0.05 |

Values are mean ± SD.

Vt = tidal volume; $V_{abd}$ abdominal volume; $V_{thor}$ = thoracic volume.

* $P < 0.05$ *versus* control (ANOVA for repeated measures followed by *post hoc* Tukey's honestly significant difference test).

from the instantaneous sum of ribcage ($V_{thor}$) and abdominal ($V_{abd}$) volumes. Relative contribution of $V_{abd}$ to Vt was calculated as $V_{abd}$%. The ratio of $V_{thor}$ to $V_{abd}$ ($V_{thor}/V_{abd}$) was also calculated. Konno-Mead plots[12] of $V_{thor}$ *versus* $V_{abd}$ were plotted in all patients.

*Diaphragmatic Electromyograms*

A bipolar myocardial electrode (TME-64Z, 60 cm, Dr. Osypka GmbH, Grenzach-Wyhlen, Germany) was inserted laparoscopically in the muscular part of the cupola of the right hemidiaphragm (n = 13) by the surgeon at the end of surgery. The electrode in its preinsertion state terminates with a needle at each end; its distal end has two bare contact points that are 5 mm and 10-50 mm wide with a gap of 10 mm in between. The 10-50 mm contact point is zigzag-shaped to allow for satisfactory anchoring of the electrode in the muscle mass. The distal end with bare contact points was buried into the muscle, whereas the proximal end was exteriorized through the abdominal wall, sutured in place, and its identification marked. The presence of needles at both ends of the electrode facilitates its insertion with the help of laparoscopy forceps. $EMG_{dia}$ data were filtered and amplified (PS101 and ISO-2104, Braintronics BV, Almere, The Netherlands) and recorded on the Mingograph 82 along with and at the same time interval as the pressure and volume changes postoperatively. The band pass filter (20-2,000 Hz) and amplification were identical at all measurement periods. A qualitative study of the raw $EMG_{dia}$ amplitude was carried out. The electrode was easily withdrawn after the study with minimal discomfort to the patient.

*Anesthesia*

Premedication consisted of 10 mg oral diazepam, and general anesthesia was induced with thiopental (4-6 mg/kg) and maintained with fentanyl (3-4 μg/kg), isoflurane, nitrous oxide (70%), and oxygen (30%). Endotracheal intubation was facilitated with suxamethonium (1 mg/kg), and further muscle relaxation was provided with vecuronium (0.1 mg/kg). The degree of muscle relaxation was monitored with a peripheral nerve stimulator (Microstim plus, P/N7106, Neuro Technology, Houston, TX), and incremental doses of vecuronium (1-2 mg) were given depending on the adductor pollicis response to a train-of-four stimulus. The abdomen was inflated with $CO_2$ to a pressure of 15 mmHg during the surgery, and as much gas as possible was removed toward the end of surgery. The residual muscle relaxation was antagonized with neostigmine (2.5 mg) and atropine (0.5 mg) at the end of the surgery in all patients. Ketorolac and cetobemidone hydrochloride were used for postoperative pain relief. Patients received 10-30 mg Ketorolac intravenously as the first line analgesic. Cetomebidone hydrochloride was reserved for those who had pain despite Ketorolac.

*Statistical Analysis*

Results are reported as mean ± SD. Analysis of variance (one- way ANOVA) for repeated measures followed by Tukey honestly significant difference test were applied where appropriate for comparison between the preoperative and postoperative values. All differences were considered significant at the $P < 0.05$ level.

## Results

All patients were awake 1 h after the operation without residual muscle relaxation.

*Basic Ventilatory Measurements*

The basic ventilatory measurements derived from the respiratory inductive plethysmograph tracing and from the gastric and esophageal pressure tracings are presented in table 2.

*Rib Cage and Abdominal Dimensions*

The changes in rib cage and abdominal dimensions with quiet tidal respiration are represented in figure 1 and table 2.

During the 1- and 6-h postoperative period, there was a significant reduction in the abdominal component of tidal volume ($V_{abd}$ and $V_{abd\%}$) and an increase in the $V_{thor}/V_{abd}$ ratio, both indicating a shift toward a thoracic pattern of breathing. $V_{abd}$ and $V_{abd\%}$ values returned to at least the control value in 4 of 13 patients at 1 h and 3 of 13 patients at 24 h. Konno-Mead plots of $V_{thor}$ *versus* $V_{abd}$ also displayed a shift toward a thoracic breathing during the postoperative phase in all patients (fig. 2). This pattern of Konno-Mead plot again shifted to predominantly abdominal breathing at 24 h in 5 of 13 patients.

*Gastric and Esophageal Pressures*

The changes in gastric and esophageal pressures are represented in figure 1 and table 3.

Plots of $\Delta P_{eso}$ *versus* $\Delta P_{gas}$ showed a shift to the left of the vertical line at 1 h and 6 h postoperatively in 8 of 13 patients and at only 1 h postoperatively in 2 of 13 patients. At 24 h postoperatively all loops were to the right of the vertical line (fig. 3). $\Delta P_{gas\text{-}insp}$, which was negative (and therefore positive value for $\Delta P_{gas\text{-}insp}/\Delta P_{eso\text{-}insp}$) in 10 of 13 patients at 1 h and 8 of 13 patients at 6 h postoperatively, had recovered to a positive value in all patients at 24 h. The change in mean $\Delta P_{gas\text{-}insp}$ from control is plotted in figure 4. $\Delta P_{gas\text{-}insp}$ values were reduced in 12 of 13 patients at 1 h and 6 h postoperatively. At 24 h postoperatively it had recovered to within 20% of control value in five patients and within 50% of control value in an additional five patients. Mean $\Delta P_{di}$ was significantly reduced at 1 h and 6 h postoperatively. $\Delta P_{di}$ had returned at least to the preoperative value in 9 of 13 patients at 24 h.

Plots of $V_{abd}$ *versus* $\Delta P_{gas}$ in all patients at the measurement periods previously mentioned reflected two broad patterns. In 8 of 13 patients at 1 h and 6 h and in 2 of 13 patients at 1 h after surgery, the loop was shifted to the left of the vertical line (*i.e.*, decrease in $P_{gas}$ with increase in abdominal volume during inspiration; fig. 5) compared with the preoperative loop. In 3 of 13 patients the loops remained to the right of the vertical line at all measurement periods (*i.e.*, increase in $P_{gas}$ with increase in abdominal volume during inspiration; fig. 6). In all patients the loops were seen to the right of the vertical line at 24 h after surgery.

*Diaphragmatic Electromyography*

Diaphragmatic electromyographic data were lacking in 2 of 13 patients due to total loss of contact with one and an absent EMG signal at 24 h in the other.

The EMG recordings were used as a qualitative indicator of diaphragmatic activity. There was no measurable $EMG_{dia}$ activity seen in 4 of 11 patients at 1 h and 1 of 11 patients at 1 h and 6 h. They subsequently reappeared at 6 h (4 patients) and 24 h (1 patient) postoperatively. The five patients with absent $EMG_{dia}$ recordings at 1 h had a significantly greater increase in their thoracic contribution to total tidal volume ($V_{thor\%}$ at 1 h − $V_{thor\%}$ control) compared with the six patients who had recordable $EMG_{dia}$ at 1 h postoperatively. The mean $V_{thor\%}$ increase ± SD was 39.3 ± 14.0 (n = 5) *versus* 15.8 ± 10.8 (n = 6; $P < 0.05$, Mann–Whitney U-test).

## Discussion

In this study both direct and indirect measurements confirm the presence of a reduction in diaphragmatic activity during the immediate postoperative phase in some patients undergoing laparoscopic cholecystectomy. There was a significant reduction in $\Delta P_{gas\text{-}insp}$, $\Delta P_{di\text{-}insp}$, $V_{abd}$, and $V_{abd\%}$ values and a significant increase in $V_{thor}/V_{abd}$ at 1 h and 6 h postoperatively, followed by their partial or complete recovery at 24 h. The $\Delta P_{gas\text{-}insp}/\Delta P_{eso\text{-}insp}$ ratios that were positive in the early postoperative period also returned to a negative value at 24 h. The

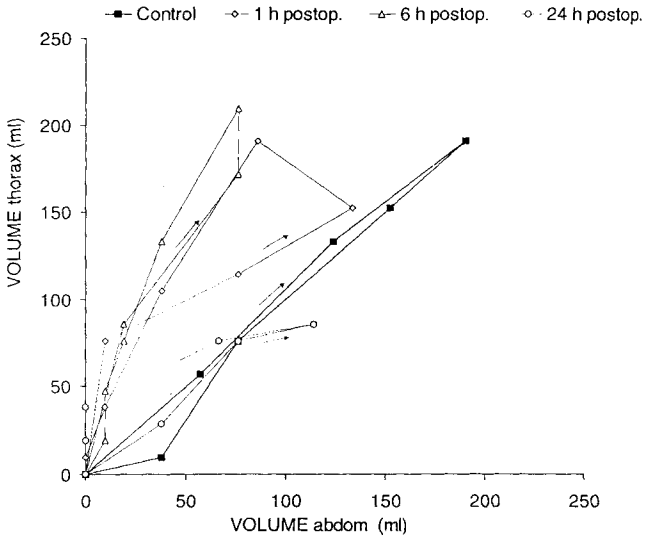


**Fig. 2. Konno-Mead plots of changes in ribcage volume (volume thorax) and abdominal volume (volume abdom) in one patient preoperatively and 1 h, 6 h, and 24 h postoperatively. The arrows indicate the direction of inspiration.**

**Table 3. Gastric and Esophageal Pressure Indices (N = 13)**

| Measurements | Control | 1 h Postoperation | 6 h Postoperation | 24 h Postoperation |
|---|---|---|---|---|
| $\Delta P_{gas\text{-}insp}$ ($cmH_2O$) | 3.22 ± 0.99 | −1.48 ± 3.95* | −0.73 ± 3.63* | 2.29 ± 1.13 |
| $\Delta P_{eso\text{-}insp}$ ($cmH_2O$) | −8.58 ± 3.57 | −7.22 ± 2.93 | −7.33 ± 3.29 | −8.57 ± 3.96 |
| $\Delta P_{gas\text{-}insp}/\Delta P_{eso\text{-}insp}$ | −0.43 ± 0.20 | 0.36 ± 0.77* | 0.37 ± 1.20* | −0.34 ± 0.25 |
| $\Delta P_{di\text{-}insp}$ ($cmH_2O$) | 11.79 ± 3.95 | 5.74 ± 5.73* | 6.60 ± 5.13* | 10.86 ± 3.73 |
| Positive $\Delta P_{gas}/\Delta P_{eso}$ | 0/13 | 10/13 | 8/13 | 0/13 |

Values are mean ± SD.

$P_{gas}$ = gastric pressure; $P_{eso}$ = esophageal pressure; $P_{gas\text{-}insp}$ = difference in $P_{gas}$ between the start and end of inspiration; $P_{eso\text{-}insp}$ = difference in $P_{eso}$ between the start and end of inspiration; $P_{di\text{-}insp}$ = changes in transdiaphragmatic pressure during inspiration.

*$P < 0.05$ *versus* control (one-way ANOVA for repeated measures followed by *post hoc* Tukey's honestly significant difference test).

$V_{thor}/V_{abd}$ values and the Konno-Mead plots showed a thoracic pattern of breathing initially. These values had also partially recovered at 24 h postoperatively. These changes are very similar to those observed during the first postoperative day by Ford *et al.*[1] after open cholecystectomy. The plots of $V_{abd}$ *versus* $\Delta P_{gas}$ in 10 of 13 patients revealed a left shift, indicating powerful abdominal muscle activity rather than changes in diaphragmatic activity in the immediate postoperative period. These findings have been described previously.[7,8,13] The plots of $V_{abd}$ *versus* $\Delta P_{gas}$ had returned to the right of the vertical line at 24 h.

Indirect indices of diaphragmatic activity can be unreliable during the postoperative phase for several reasons. First, $\Delta P_{gas}/\Delta P_{eso}$, which was used to measure the contribution of diaphragm to inspiration, can be reduced by other factors, such as relaxation of abdominal muscles during inspiration and an increase in lung impedance in the postoperative phase.[8,14,15] Second, $\Delta P_{di}$, which was used as an indicator of mechanical activity of the diaphragm, is also affected by the contraction of the abdominal muscles during expiration and by the reduction in lung volume after operation in the abdomen.[8,14] The diaphragm is placed at a higher position in these cases and its fibers are elongated, producing higher tension during contraction and therefore an artificially elevated $\Delta P_{di}$. Finally, the $V_{thor}/V_{abd}$ ratio can be affected by factors such as increased overall respiratory drive, tonic activity of the abdominal muscle, and distortion of abdominal wall or ribcage, all of which have been reported after abdominal surgery.[16]

The two methods that more directly measure diaphragmatic activity are the recording of diaphragmatic EMG and assessment of diaphragmatic contraction using sonomicrometry crystals on the surface of the dia-

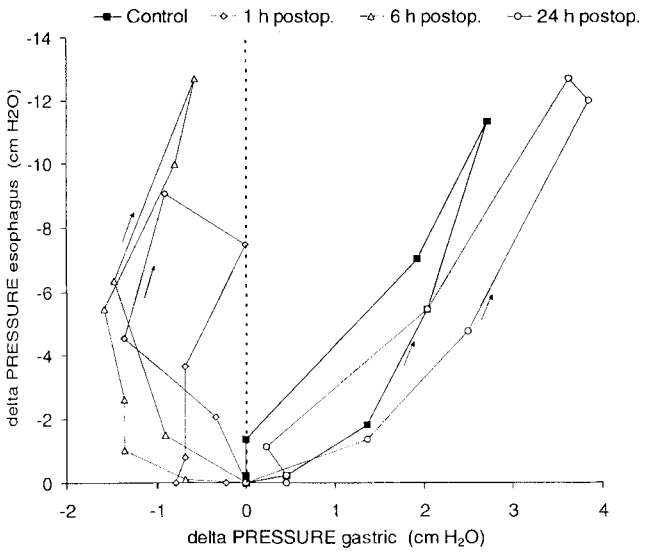


**Fig. 3. Tracing of changes in gastric and esophageal pressures in one patient preoperatively and 1 h, 6 h, and 24 h postoperatively. The arrows indicate the direction of inspiration.**

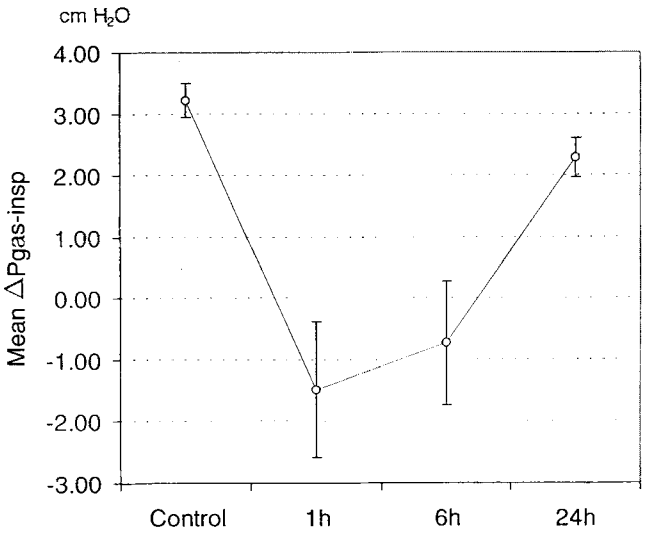


**Fig. 4. Change in mean $\Delta P_{gas\text{-}insp}$ from control at 1 h, 6 h, and 24 h postoperatively.**

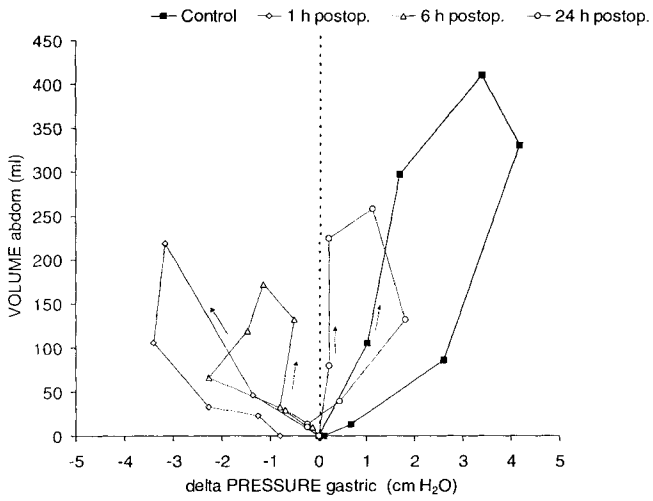


**Fig. 5. Tracing of changes in abdominal volume and gastric pressure in one patient with left shift (at 1 h and 6 h) recorded preoperatively and 1 h, 6 h, and 24 h postoperativly. The arrows indicate the direction of inspiration.**

phragm.[15,17,18] Installation of sensors or electrodes in both these methods is difficult to perform and no preoperative control value is available. The placement of the sensor device itself can induce a local or a general change in the function of the diaphragm. It is difficult to distinguish between an active contraction and a passive movement of the diaphragm with the sonomicrometry technique. We therefore chose direct $EMG_{dia}$ recording and combined it with a number of indirect measurements.

The $EMG_{dia}$ activity that was absent in 4 of 11 patients at 1 h and 1 of 11 patients at 1 h and 6 h reappeared at 6 h or 24 h, postoperatively further strengthening the view that there is a possible reduction of diaphragmatic activity in the immediate postoperative period. We are unable to tell from the EMG activity data alone if the recovery was complete at 24 h because the experimental method used to record the raw EMG data did not allow us to reliably quantify it, and this interpretation was made worse by the the fact that the patients left the hospital within 24 h of surgery, limiting our duration of recording.

The mechanism for the postoperative inhibition of the diaphragm is unclear. Ford *et al.*[1] suggested three potential mechanisms; anesthesia, local trauma (irritation and inflammation), and local abdominal pain. Animal experiments on nonoperated but anesthetized controls have not induced the type of abnormal breathing pattern that is characteristic of operated animals, perhaps indicating that anesthesia has no role in this inhibition.[19] Similarly, anesthetized patients undergoing laparoscopic hernia repair showed no reduction in diaphragmatic activity.[6] Pain as a cause of this inhibition seems unlikely due to the fact that the abnormal breathing pattern could not be reversed despite adequate analgesia.[20,21] Residual capnoperitoneum probably does not affect diaphragmatic activity because patients undergoing laparoscopic hernia surgery under capnoperitoneum have not been shown to develop a reduction in diaphragmatic activity[6] and, furthermore, patients undergoing cardiac surgery without capnoperitoneum demonstrate a reduction in diaphragmatic activity.[16] Local trauma in the abdomen that is associated with inhibitory phrenic nerve output has been demonstrated in animal studies,[22] making this the most likely cause for the inhibition. This inhibition of the diaphragm was considered due to a decrease in contractility of the diaphragm or a decrease in phrenic nerve activity.[23] Inhibition due to decreased contractility of the diaphragm was ruled out on the basis of restored transdiaphragmatic pressure swings during bilateral phrenic nerve stimulation following upper abdominal surgery[24] and also due to the ability of this group of patients to voluntarily revert to predominantly abdominal breathing when asked to do so in the postoperative period.[1] Erice *et al.*[6] also demonstrated a normal diaphragmatic pressure during the sniff maneuver in their patients, suggesting that the voluntary diaphragmatic activity is intact postoperatively. Reflex inhibition of the phrenic nerve

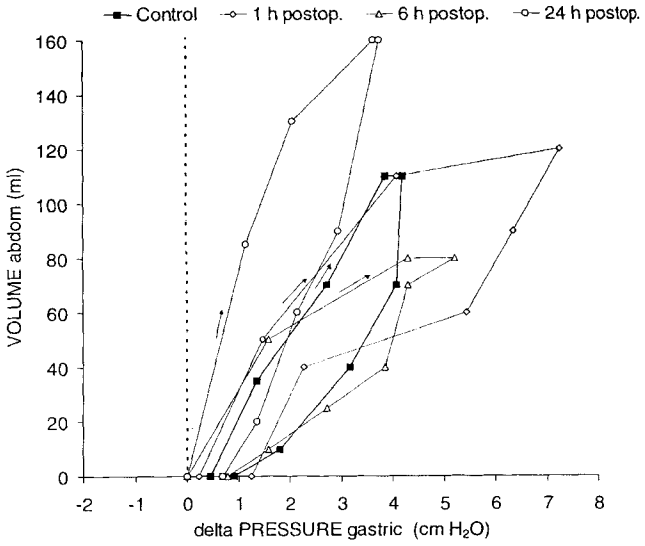


**Fig. 6. Tracing of changes in abdominal volume and gastric pressure in one patient with persistent right shift at all measurement periods (*i.e.*, preoperatively and 1 h, 6 h, and 24 h postoperatively). The arrows indicate the direction of inspiration.**

output therefore seems to be the likely cause of the reduction in diaphragmatic activity. The present study measures the reduction in activity and does not distinguish between voluntary and reflex activity of the diaphragm. The results of our study differ from that of Couture *et al.*,[7] who showed that diaphragmatic activity was preserved after laparoscopic cholecystectomy. This dissimilarity is difficult to explain due to different time frames of data collection and the different data presentation techniques used, but it is worth noting that those authors' patients exhibited a positive gastric pressure in the immediate postoperative period while 10 of 13 of our patients showed a negative gastric pressure in the similar period.

*Critique of the Study*

Due to the limited duration (24 h) of recording in this study, we are unable to say from our measurements if the diaphragmatic recovery was complete. No comparison could be made with patients undergoing open cholecystectomy since this procedure is now rarely carried out in our hospital and if done is usually reserved for complicated cases, which would introduce a bias in the study.

## Conclusion

The present study using both the direct and indirect indices of diaphragmatic activity shows that the diaphragmatic activity is reduced during the immediate postoperative period after laparoscopic cholecystectomy. These measurements also suggest that the recovery of diaphragm is already initiated during the first postoperative day.

The patient clinical data were collected at the Umeå University Hospital, Umeå, Sweden.

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