Postoperative spirometry after laparoscopy for lower abdominal or upper abdominal surgical procedures†

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Summary
In this prospective study, we have compared women undergoing laparoscopic cholecystectomy, laparoscopic gynaecological surgery and laparoscopic minor gynaecological procedures (diagnostic, tubal ligation) (n=10 in each group) to determine if lower abdominal laparoscopy results in less postoperative pulmonary dysfunction than upper abdominal laparoscopy. Pulmonary testing was performed before operation, and 3 and 6 h after operation, on the first and second days after surgery. After operation, a significant reduction in forced vital capacity, forced expiratory volume in 1 s and peak expiratory flow rate occurred after laparoscopic cholecystectomy at each time. There were no significant changes after minor gynaecologic laparoscopy, whereas laparoscopic gynaecological surgery resulted in minor pulmonary dysfunction on the day of surgery only. We conclude that postoperative pulmonary function was less impaired after gynaecological laparoscopy than after laparoscopic cholecystectomy. This study suggests that the site of surgery is an important determinant of lung dysfunction after laparoscopy. (Br. J. Anaesth. 1997; 79: 422–426).

Key words

Upper abdominal surgery performed via laparotomy is associated with postoperative respiratory changes, including a restrictive syndrome (reduced vital capacity and functional residual capacity), hypoxaemia, a change from abdominal to rib cage breathing and increased work of breathing. The site and size of incision,1 2 postoperative pain1 3 and diaphragmatic dysfunction1 4–6 are the main factors contributing to the pathogenesis of these postoperative respiratory changes.

Laparoscopy, which avoids large abdominal incisions, allows a significant reduction in surgical trauma, postoperative pain and opioid requirements.7–10 Laparoscopic cholecystectomy also results in less postoperative pulmonary dysfunction,7 9–13 faster recovery of preoperative pulmonary function12 and less atelectasis and hypoxaemia1 11 12 than open cholecystectomy. Nevertheless, postoperative pulmonary dysfunction after laparoscopic cholecystectomy is significant. The role of inflammation of the punctured abdominal wall or gallbladder bed, or both, carbon dioxide pneumoperitoneum or intraoperative patient position in the pathogenesis of this pulmonary dysfunction is unknown. In all of these studies, pulmonary function was evaluated after laparoscopic cholecystectomy. Few data are available after lower abdominal surgical procedures, such as laparoscopic gynaecological surgery, or after minor laparoscopic procedures such as diagnostic laparoscopy or tubal ligation. Such data may be useful to differentiate the influences of the surgical site, parietal trauma, intraoperative position and pneumoperitoneum in the pathogenesis of pulmonary dysfunction.

Therefore, we have compared changes in pulmonary function after laparoscopy for cholecystectomy, gynaecological surgery and minor gynaecological procedures, such as diagnostic laparoscopy or tubal ligation.

Patients and methods
The study was conducted after obtaining approval from our institution’s Ethics Committee and informed patient consent. We studied 30 consecutive women undergoing elective laparoscopic cholecystectomy (group Chol.), laparoscopic gynaecological surgery (group Gyn.) or laparoscopic minor gynaecological procedures (group Min.) (10 in each group). Inclusion criteria were body weight no more than 20% over ideal weight, age between 18 and 60 yr, absence of acute cholecystitis or pelvic inflammatory disease, and no cardiorespiratory disease or medications.

ANAESTHESIA
We used the same anaesthetic technique in all

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groups. All patients were given hydroxyzine 50 mg orally, 2 h before surgery, and an i.m. injection of midazolam 5 mg and atropine 0.25 mg before transfer to the operating theatre. Anaesthesia was induced with sufentanil 15 μg and propofol 2 mg kg⁻¹. After tracheal intubation facilitated by atracurium 0.5 mg kg⁻¹, general anaesthesia was maintained with isoflurane and 50% nitrous oxide in oxygen. Minute ventilation was controlled (Servo 900C, Siemens-Elema) and adjusted to maintain the end-tidal partial pressure of carbon dioxide at 4.0–5.0 kPa. During laparoscopy, intra-abdominal pressure was maintained automatically at 14 mm Hg by a carbon dioxide insufflator. At the end of surgery, residual neuromuscular block was antagonized with neostigmine 40 μg kg⁻¹ and atropine 15 μg kg⁻¹ i.v.

ALGUESIA

Pain intensity was measured using a 100-mm visual analogue scale at 3, 6 and 24 h after surgery. Treatment of postoperative pain was provided on the same nursing unit using a standardized regimen in all groups. During the first 24 h after operation, propacetamol 2 g, a precursor of paracetamol (Prodafalgan, UPSA Medica, Brussels, Belgium: propacetamol 2 g = paracetamol 1 g) were given i.v. every 6 h as necessary. If pain relief was considered inadequate (pain score >40 mm on a 100-mm visual analogue scale at rest) piritramide 0.2 mg kg⁻¹ (Dipidolor, Janssen Pharmaceutica, Beerse, Belgium), a synthetic opioid, was administered i.m. On the second day, paracetamol 1 g with codeine phosphate 60 mg (Dafalgan codeine, UPSA Medica, Brussels, Belgium) were given orally at the patient’s request every 6 h.

SURGERY

For all procedures, the laparoscope was introduced via a 10-mm diameter umbilical port. Laparoscopic cholecystectomies were performed using three additional trocars, one 10-mm and two 5-mm diameter ports, located in the right and left subcostal area, and the epigastric notch. For gynaecological laparoscopies, two trocars, one 10 mm and one 5 mm in diameter, were placed in the hypogastric region in addition to the umbilical port. Patients were tilted to the 10° head-up position for laparoscopic cholecystectomy and to the 20° head-down position for gynaecological laparoscopy. Care was taken to expel a maximum volume of gas from the abdominal cavity at the end of all laparoscopies.

PULMONARY FUNCTION

Pulmonary testing was performed in the sitting position by the same technician, who was unaware of the patient group, the day before surgery, and 3 and 6 h after surgery on days 1 and 2 after operation (except in group Min.; these patients were discharged on day 1). The following variables were recorded: forced vital capacity (FVC), forced expiratory volume in 1 s (FEV₁) and peak expiratory flow rate (PEFR). These variables were measured using a Microlab 3000 series bedside spirometer (Micro Medical Ltd, Rochester, England).

STATISTICAL ANALYSIS

Results are expressed as mean (SEM) except in table 1 where SD values are used. Data were analysed by analysis of variance followed by Scheffé’s test for multiple comparisons. Results were considered to be statistically significant at the 5% level.

Results

Patient data and preoperative pulmonary function were similar in the three groups (table 1). In group Gyn., two patients underwent laparoscopically assisted vaginal hysterectomy, one had bilateral oophorectomy and the other seven had unilateral oophorectomy. In group Min., three women had diagnostic laparoscopy and the others had tubal ligation. Duration of pneumoperitoneum for cholecystectomy was significantly longer than for the other procedures, whereas duration of pneumoperitoneum in group Gyn. was longer than in group Min. (table 1). Pain intensity was similar in the three groups (table 2).

FVC, FEV₁ and PEFR did not change significantly after minor gynaecological laparoscopy. After laparoscopic gynaecological surgery, FVC and PEFR decreased significantly, but only on the day of surgery (19% and 29%, respectively), whereas no significant changes in FEV₁ were observed. After laparoscopic cholecystectomy, FVC, FEV₁ and PEFR decreased significantly at all times, except on day 2, when FEV₁ was no longer different than the preoperative value. In this last group, FVC, FEV₁ and PEFR reached their lowest values 3 h after surgery.

Table 1: Patient data and preoperative pulmonary function (mean (SD or range)) in those undergoing minor gynaecological laparoscopy (group Min.), laparoscopic gynaecological surgery (group Gyn.), or laparoscopic cholecystectomy (group Chol.).

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Group Min.</th>
<th>Group Gyn.</th>
<th>Group Chol.</th>
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<tr>
<td>39 (29–49)</td>
<td>40 (21–60)</td>
<td>39 (18–58)</td>
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<tr>
<td>Weight (kg)</td>
<td>59 (8)</td>
<td>61 (9)</td>
<td>64 (8)</td>
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<tr>
<td>Height (cm)</td>
<td>164 (4)</td>
<td>165 (5)</td>
<td>161 (5)</td>
</tr>
<tr>
<td>Duration of PNO (min)</td>
<td>17 (4)</td>
<td>35 (7)*</td>
<td>57 (20)*†</td>
</tr>
<tr>
<td>FVC (litre)</td>
<td>2.75 (0.49)</td>
<td>2.88 (0.49)</td>
<td>3.04 (0.51)</td>
</tr>
<tr>
<td>FEV₁ (litre s⁻¹)</td>
<td>2.31 (0.39)</td>
<td>2.13 (0.57)</td>
<td>2.36 (0.40)</td>
</tr>
<tr>
<td>PEFR (litre min⁻¹)</td>
<td>295 (88)</td>
<td>308 (95)</td>
<td>352 (92)</td>
</tr>
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Table 2: Pain intensity (mean (SD)) using a 100-mm visual analogue scale after minor gynaecological laparoscopy (group Min.), laparoscopic gynaecological surgery (group Gyn.), or laparoscopic cholecystectomy (group Chol.).

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<tr>
<td>+3 h</td>
<td>60 (7)</td>
<td>36 (10)</td>
</tr>
<tr>
<td>+6 h</td>
<td>65 (5)</td>
<td>51 (9)</td>
</tr>
<tr>
<td>+24 h</td>
<td>52 (6)</td>
<td>40 (6)</td>
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surgery, and subsequently increased. The increase in FVC became significant relative to 3 h on day 1, and on day 2 for FEV1. FVC, FEV1 and PEFR were significantly lower in group Chol., than in the two other groups. FVC and FEV1 in groups Min. and Gyn. did not differ significantly. PEFR in group Gyn. was significantly reduced compared with group Min. at 6 h after operation (figs 1–3). There were no correlations between duration of pneumoperitoneum and changes in FVC, FEV1 or PEFR at any time.

Discussion

This study suggests that laparoscopy for lower abdominal surgery resulted in minor postoperative pulmonary dysfunction on the day of surgery only. Furthermore, these changes were less severe than those after upper abdominal laparoscopic surgery. Also, carbon dioxide pneumoperitoneum with minimal or no surgery, such as for diagnostic laparoscopy or tubal ligation, and Trendelenburg position did not produce significant changes in pulmonary function.

This study confirms the development of a restrictive pulmonary syndrome after laparoscopic cholecystectomy. Not only expiratory lung volumes,7 9–13 but also functional residual capacity are reduced after laparoscopic cholecystectomy.9 11 13 Postoperative atelectasis on chest x-ray, and decreases in F\text{A\textsubscript{O\textsubscript{2}}} have also been reported.11 12 In addition, changes in respiratory pattern, characterized by shallow breathing at a faster rate, were noted after laparoscopic cholecystectomy.14 15 Therefore, although laparoscopic cholecystectomy is associated with better postoperative lung function than open cholecystectomy,7 9–13 the patterns of alteration after cholecystectomy are qualitatively similar irrespective of the surgical approach. Consequently, the pathogenesis of pulmonary dysfunction observed after open and laparoscopic cholecystectomy should also be similar.

Pain contributes to impairment of pulmonary function after open upper abdominal surgery.13 Several studies, however, failed to demonstrate clinically significant benefits (in term of improved rate of postoperative pulmonary complications) of extradural analgesia compared with conventional systemic opioid analgesia, despite better pain relief.16 17 Similarly, extradural analgesia did not improve lung dysfunction after laparoscopic

Figure 1 Changes in forced vital capacity (FVC) (mean (SEM)) after minor gynaecological laparoscopy (group Min.), laparoscopic gynaecological surgery (group Gyn.) and laparoscopic cholecystectomy (group Chol.). D1 and D2 = Days 1 and 2 after operation. Significant differences (P<0.05) compared with: *group Min., §group Gyn., †before surgery, ‡3 h after surgery.

Figure 2 Changes in forced expiratory volume in 1 s (FEV1) (mean (SEM)) after minor gynaecological laparoscopy (group Min.), laparoscopic gynaecological surgery (group Gyn.) and laparoscopic cholecystectomy (group Chol.). D1 and D2 = Days 1 and 2 after operation. Significant differences (P<0.05) compared with: *group Min., §group Gyn., †before surgery, ‡3 h after surgery.

Figure 3 Changes in peak expiratory flow rate (PEFR) (mean (SEM)) after minor gynaecological laparoscopy (group Min.), laparoscopic gynaecological surgery (group Gyn.) and laparoscopic cholecystectomy (group Chol.). D1 and D2 = Days 1 and 2 after operation. Significant differences (P<0.05) compared with: *group Min., §group Gyn., †before surgery.
cholecystectomy. Minor gynaecological laparoscopies are associated with a similar degree of trauma to the abdominal wall, as ports of similar sizes were used in these procedures. Moreover, laparoscopic tubal ligation, although considered to be a minor procedure, frequently results in severe pain. Accordingly, similar pain intensities were reported by patients in the three groups. Nevertheless, in our study minor gynaecological laparoscopies resulted in no significant changes in pulmonary function. Similarly, laparoscopic hernia repair produces no postoperative ventilatory impairment although the same number of incisions are performed, in close proximity to the abdominal regions involved in laparoscopic cholecystectomy. Therefore, pain, and more particularly incisonal pain, does not seem to play a major role in the lung dysfunction noted after laparoscopy.

Whereas surgical trauma to the abdominal wall and the type of incision have been proposed as important causes of pulmonary dysfunction after open upper abdominal surgery, Ford and colleagues showed that a reduction in diaphragmatic performance was the main determinant of impaired lung function. Several clinical and experimental studies have suggested that diaphragmatic dysfunction possibly results more from reflex inhibition of afferent phrenic nerve activity secondary to irritation of splanchnic afferents, than from contractile failure of the diaphragm or surgical trauma to the abdominal wall. After laparoscopic cholecystectomy, functional diaphragmatic impairment has also been documented. Diaphragmatic electromyography using electrodes implanted in the diaphragm during surgery confirmed diaphragmatic dysfunction in the early postoperative period. The magnitude of this dysfunction appears, nevertheless, to be less than that after open cholecystectomy. Impairment of intrinsic diaphragmatic contractility after laparoscopic cholecystectomy has also been excluded. Inflammation of the punctured parietal peritoneum, peritoneal inflammation secondary to carbon dioxide pneumoperitoneum, residual carbon dioxide accumulated under the diaphragm after operation and inhibitory reflexes arising from the gallbladder bed are all potential causes of diaphragmatic dysfunction after laparoscopic cholecystectomy. The lack of an effect of extradural analgesia on the restrictive syndrome after laparoscopic cholecystectomy excludes noxious abdominal stimuli as major determinants of diaphragmatic dysfunction.

Moreover, in this study, gynaecological laparoscopic surgery produced no or only minor postoperative pulmonary impairment despite similar trauma to the abdominal wall and a similar postoperative pain intensity compared with laparoscopic cholecystectomy. Although the extent of abdominal wall trauma was similar, the sites of trocar insertion were different for gynaecological laparoscopy and laparoscopic cholecystectomy, and might therefore contribute to differences in pulmonary function observed after these laparoscopic procedures. Nevertheless, Erice and colleagues did not observe diaphragmatic dysfunction after laparoscopic inguinal hernia repair, although sites for trocar insertion for laparoscopic hernia repair and cholecystectomy involved the same abdominal region. These observations exclude parietal trauma as a possible source of inhibitory reflexes. The longer duration of pneumoperitoneum might be responsible for the more intense pulmonary dysfunction observed after laparoscopic cholecystectomy. However, we did not observe a significant correlation between duration of pneumoperitoneum and any of the spirometric variables measured. The position of the patient may induce changes in lung volumes. Whereas the Trendelenburg position reduces functional residual capacity (FRC) and vital capacity in anaesthetized spontaneously breathing volunteers, the head-up position increases FRC. Thus postoperative lung volumes might be expected to be affected more after laparoscopic procedures in the head-down position. However, there were no significant changes in lung function in group Min. In addition, carbon dioxide pneumoperitoneum alone, such as for diagnostic laparoscopy, does not impair diaphragmatic function. Accordingly, we did not observe pulmonary dysfunction in patients undergoing minor gynaecological laparoscopy. Therefore, the intra-abdominal site of surgery itself appears to be the main determinant of diaphragmatic inhibition after laparoscopic abdominal surgery, as seen in open surgery. The exact reflex neural pathways that cause this dysfunction need to be defined. Experimental data suggest that afferents for reflex inhibition of phrenic output possibly originate in the coeliac sympathetic plexus or other upper abdominal sympathetic ganglia.

Taken together, these observations raise the question of the mechanism(s) accounting for the less severe lung dysfunction seen after laparoscopic cholecystectomy compared with open procedures. During laparoscopy, the most apparent reduction in surgical trauma results from the absence of large abdominal incisions. Consequently, postoperative pain, more particularly during mobilization and coughing, is less after laparoscopy. Moreover, whereas incision of the abdominal wall results in persistent pain after laparotomy, pain after laparoscopy is more short-lived. Finally, visceral pain accounts for most of the pain experienced after laparoscopic cholecystectomy, compared with parietal pain which is predominant after open cholecystectomy. These changes in pain characteristics may explain why the contribution of incisonal pain to the respiratory restrictive syndrome after laparoscopy is insignificant. Visceral trauma, the main determinant of postoperative diaphragmatic dysfunction, would be expected to be unchanged whatever the surgical approach. Nevertheless, handling and dissection of intra-abdominal viscera are more gentle during laparoscopy, probably leading to less visceral trauma. The consequent visceral inflammatory reaction might be reduced compared with open surgery. In support of this concept, it is notable that postoperative adhesions are less after laparoscopy compared with laparotomy.
References