TRANSOESOPHAGEAL ECHOCARDIOGRAPHIC ASSESSMENT OF HAEMODYNAMIC FUNCTION DURING LAPAROSCOPIC CHOLECYSTECTOMY†

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SUMMARY
We have measured cardiovascular changes associated with insufflation of carbon dioxide and the reverse Trendelenburg position during laparoscopic cholecystectomy, using transoesophageal echocardiography in 13 healthy patients. End-tidal carbon dioxide values increased after insufflation of carbon dioxide, with values significantly (P < 0.05) increased after lateral tilt positioning. Creation of a pneumoperitoneum was associated with increases (P < 0.05) in left ventricular end-systolic wall stress, concomitant with increases (P < 0.01) in peak airway pressure and systemic arterial pressure. In addition, left ventricular end-diastolic area decreased (P < 0.05) after reverse Trendelenburg positioning. Left ventricular ejection fraction was maintained throughout the study. (Br. J. Anaesth. 1993; 70: 621-625)

KEY WORDS

Laparoscopic cholecystectomy, described first by Phillipe Mouret in 1988 [1], was reported first in the literature by Perissat, Collet and Belliard [2] and refined and popularized in the United Kingdom by Nathanson, Shimi and Cuschiei [3]. Laparoscopic cholecystectomy has been reported to have the advantages of shorter hospital stay, more rapid return to normal activities, less pain associated with the small, limited incisions and less postoperative ileus compared with open laparotomy [4-6].

Indications for the procedure soon became more liberal after case reports confirmed its efficacy in patients with acute cholecystitis, and in association with obesity, pregnancy and previous abdominal surgery [7-9]. The laparoscopic surgical technique has been advocated for older patients who may have co-existing cardiac or pulmonary disease, or both [10]. Laparoscopic cholecystectomy involves longer periods of intraperitoneal insufflation of carbon dioxide than gynaecological procedures.

The objectives of this study were to assess changes in venous return, myocardial performance and afterload, as determined by transoesophageal echocardiographic assessment of left ventricular end-diastolic area (EDA), left ventricular ejection fraction (LVEF) and left ventricular end-systolic wall stress (LVESWS) in healthy patients undergoing laparoscopic cholecystectomy.

PATIENTS AND METHODS
After obtaining local Human Investigation Committee approval, we studied 13 healthy patients (ASA physical status I-II) undergoing laparoscopic cholecystectomy. All patients gave informed consent. Patients with a history or ECG changes suggestive of previous myocardial infarction or evidence of left ventricular dysfunction were excluded, as were patients with disease states that might preclude transoesophageal echocardiography—coagulation or bleeding diathesis and oesophageal pathology.

A standard anaesthetic technique was used for all patients. All subjects were admitted to hospital on the morning of surgery. Midazolam 1-3 mg was administered i.v. for preoperative sedation before induction of anaesthesia with thiopentone 4.0 mg kg⁻¹ i.v.; vecuronium 0.1 mg kg⁻¹ i.v. was given to facilitate tracheal intubation. Anaesthesia was maintained with 0.5-1.0% end-tidal isoflurane and 50 % oxygen in air, with incremental bolus doses of i.v. fentanyl 1-2 μg kg⁻¹. A urinary bladder catheter and nasogastric tube were inserted.

Intraoperative monitoring included continuous ECG (leads II and V₅), systemic arterial pressure using an automated cuff (PCMS system, Spacelabs Inc., Redmond, WA), pulse oximetry (Spacelabs Inc., Redmond, WA), neuromuscular function, inspired oxygen fraction, peak airway pressure, oesophageal temperature and urinary output. Intermittent positive pressure ventilation (IPPV) was used using an Ohmeda 7810 volume cycled ventilator (BOC Healthcare Co., Madison WI), with an inspiratory flow rate of 25 litre min⁻¹, a ventilatory...
frequency of 8 b.p.m., a tidal volume of 100 ml kg⁻¹ and an inspiratory:expiratory (1:E) ratio of 1:2. A Perkins–Elmer Advantage mass spectrometer was used to measure end-tidal carbon dioxide tension \( (P_{\text{ET} \text{CO}_2}) \) and isoflurane concentrations. End-tidal isoflurane concentrations were maintained in the range 0.5–1.0 %.

The operative technique involved i.p. insufflation of carbon dioxide via a Verres needle inserted into a small infra-umbilical incision, with the patient in a 15–20° Trendelenburg position. An electronic variable-flow insufflator terminated flow when an intra-abdominal pressure of 15 mm Hg was reached. A cannula was inserted in place of the needle to provide and maintain insufflation adequate for surgery. A video laparoscope was inserted through the cannula and the operative field was visualized. The patient's position was changed to steep reverse Trendelenburg, with left lateral tilt, to facilitate retraction of the gall bladder fundus. The cystic duct and cystic artery were identified and clamped. The gall bladder was removed by instruments introduced through canulae via three additional small (5–11 mm) skin incisions, including one to the right of the midline to avoid the falciform ligament and two right upper quadrant sites.

Heart rate (HR), systolic/diastolic and mean arterial pressures (SAP/DAP and MAP), and arterial oxygen saturation (SpO₂) were recorded 3 min before induction of anaesthesia. These variables, in addition to peak airway pressures and \( P_{\text{ET} \text{CO}_2} \) were also recorded at the following times: 3 min after induction of anaesthesia; 3 min before carbon dioxide insufflation; 1, 3 and 5 min after insufflation; after reverse Trendelenburg positioning and after left lateral tilt.

Two-dimensional echocardiographic data were obtained with a 5-MHz (64-element) transoesophageal, transverse plane phased array transducer (Imaging System 77020 Hewlett-Packard Corporation, Andover, MA) inserted after induction of anaesthesia and tracheal intubation. Transgastric short axis images were obtained at the previously described left ventricular mid papillary muscle level [11]. Image clarity was optimized by grey scale adjustment.

Measured variables included left ventricular cavity end-diastolic and end-systolic planimetered areas (EDA and ESA) [12] and M-mode determined end-systolic posterior and anterior wall thickness (WT). Derived variables included left ventricular ejection fraction (LVEF) and left ventricular end-systolic meridional wall stress (LVESWS) [13, 14]. An average of three consecutive sinus beats at end expiration was obtained for each value of EDA and ESA. End-diastole was indicated by the peak of the ECG R wave and end-systole was defined as the smallest endocardial area. Left ventricular ejection fraction was determined by the calculation \((\text{EDA} - \text{ESA}) \times 100)/\text{EDA}\). LVESWS was calculated by the formula: \(0.334 \times \frac{P}{(D/WT)(1 + WT/D)}\), where \(P\) = cuff systolic arterial pressure; \(D\) = left ventricular diameter and \(WT\) = wall thickness represented by the average of the anterior and posterior measurements.

RESULTS

We studied 13 patients (five male). Patients' mean age was 52 (17) yr (range 22–78 yr). The mean height and weight were 168 (8) cm and 80 (10) kg, respectively. Preinduction HR was 74 (17) beat min⁻¹, SAP 147 (21) mm Hg, DAP 83 (11) mm Hg, MAP 107 (16) mm Hg and \(\text{SpO}_2\) 97 (2) %.

There were no significant changes in either heart rate or arterial oxygen saturation after insufflation of carbon dioxide and changes in patient position. SAP, DAP and MAP were significantly \(P < 0.01\) greater 1, 3 and 5 min after insufflation of carbon dioxide, reverse Trendelenburg and lateral position changes, compared with the pre-insufflation values. \(P_{\text{ET} \text{CO}_2}\) values were greater after insufflation of carbon dioxide, but the values were significantly \(P < 0.05\) increased only after the lateral tilt position change. Peak airway pressures were significantly \(P = 0.0001\) greater than pre-insufflation values at 1, 3 and 5 min after insufflation and after reverse Trendelenburg and lateral tilt positioning (table I). Left ventricular ESA and LVEF were unchanged after insufflation of carbon dioxide and changes in patient position. Left ventricular EDA were unchanged after insufflation. Left ventricular EDA were significantly \(P < 0.05\) reduced after reverse Trendelenburg positioning. LVESWS increased after carbon dioxide insufflation. The increase in LVESWS was statistically significant \(P < 0.05\) 3 min after the insufflation.

A significant linear correlation was observed between MAP and LVESWS before insufflation of carbon dioxide and 3 and 5 min after insufflation and reverse Trendelenburg position \(r = 0.74, 0.64\) and 0.67, respectively. Similarly, significant linear correlations were recorded between the variables MAP and peak airway pressure before carbon dioxide insufflation and 5 min after insufflation and reverse Trendelenburg position \(r = 0.63\) and 0.79, respectively.

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Videotapes recorded in real time simultaneously with 1-cm calibration grids and an electrocardiogram were reviewed on an off-line analyser system providing forward, reverse, slow motion and freeze viewing formats. The areas of interest were traced by hand-held digitizer. Echocardiographic data were obtained before insufflation of carbon dioxide; 1, 3 and 5 min after insufflation; after reverse Trendelenburg position and after left lateral tilt. Measurement reproducibility was assessed by randomly selecting 25 % of profiles for reanalysis. Intra- and interobserver variabilities did not exceed 8 % and 12 %, respectively.

Statistical methods

Paired Student’s t tests and Pearson correlation coefficients were used to compare measured and derived cardiovascular and echocardiographic values before insufflation of carbon dioxide with values 1, 3 and 5 min after insufflation and after reverse Trendelenburg and lateral tilt position changes. Results are expressed as mean (sd). \(P < 0.05\) was considered statistically significant.
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Table 1. Measured and derived haemodynamic and echocardiographic data. Significant differences—compared with before carbon dioxide:
*P < 0.05, **P < 0.01; compared with 5 min after carbon dioxide: †P < 0.05

<table>
<thead>
<tr>
<th></th>
<th>Before CO₂ insufflation</th>
<th>After CO₂ insufflation</th>
<th>Reverse Trendelenburg tilt</th>
<th>Lateral tilt</th>
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<tbody>
<tr>
<td></td>
<td>1 min</td>
<td>3 min</td>
<td>5 min</td>
<td></td>
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<tr>
<td><strong>Heart rate (beat min⁻¹)</strong></td>
<td>69 (16)</td>
<td>70 (17)</td>
<td>74 (12)</td>
<td>71 (10)</td>
</tr>
<tr>
<td><strong>SAP (mm Hg)</strong></td>
<td>110 (13)</td>
<td>131 (19)**</td>
<td>146 (20)**</td>
<td>147 (23)**</td>
</tr>
<tr>
<td><strong>DAP (mm Hg)</strong></td>
<td>67 (16)</td>
<td>85 (10)**</td>
<td>94 (12)**</td>
<td>97 (12)**</td>
</tr>
<tr>
<td><strong>MAP (mm Hg)</strong></td>
<td>81 (11)</td>
<td>100 (13)**</td>
<td>111 (14)**</td>
<td>115 (13)**</td>
</tr>
<tr>
<td><strong>SPw (H₂O, kPa)</strong></td>
<td>98 (1)</td>
<td>98 (1)</td>
<td>98 (1)</td>
<td>98 (2)</td>
</tr>
<tr>
<td><strong>Pw efv, (kPa)</strong></td>
<td>3.7 (0.4)</td>
<td>3.6 (0.8)</td>
<td>3.7 (0.8)</td>
<td>3.7 (0.7)</td>
</tr>
<tr>
<td><strong>Peak airway pressure (cm H₂O)</strong></td>
<td>22 (5)</td>
<td>27 (5)**</td>
<td>31 (7)**</td>
<td>31 (6)**</td>
</tr>
<tr>
<td><strong>Left ventricular end systolic area (cm²)</strong></td>
<td>18.23 (10.14)</td>
<td>16.92 (7.77)</td>
<td>19.04 (9.15)</td>
<td>19.96 (11.11)</td>
</tr>
<tr>
<td><strong>Left ventricular end diaostolic area (cm²)</strong></td>
<td>34.26 (15.09)</td>
<td>34.15 (11.44)</td>
<td>35.17 (11.44)</td>
<td>39.94 (15.53)</td>
</tr>
<tr>
<td><strong>Left ventricular ejection fraction (%)</strong></td>
<td>48.59 (14.54)</td>
<td>47.20 (17.67)</td>
<td>47.54 (15.30)</td>
<td>50.15 (15.43)</td>
</tr>
<tr>
<td><strong>Left ventricular end systolic wall stress (dyne cm⁻²)</strong></td>
<td>234 (161)</td>
<td>258 (139)</td>
<td>306 (157)**</td>
<td>311 (160)</td>
</tr>
</tbody>
</table>

DISCUSSION

We found that left ventricular function, as determined by transoesophageal echocardiographic estimation of ejection fraction, was preserved after insufflation of carbon dioxide and changes in patient position, despite variations in loading conditions. However, insufflation of carbon dioxide was associated with increases in left ventricular end-systolic wall stress, concomitant with increases in systemic arterial pressure. In addition, LVEDA decreased after reverse Trendelenburg positioning, while LVEF was maintained. However, it might be speculated that the changes in left ventricular loading conditions might have had deleterious consequences in patients with significant cardiovascular disease.

Laparoscopic cholecystectomy has four potential causes of major physiological changes in the anaesthetized patient—the initial Trendelenburg position, creation of a pneumoperitoneum, the potential for systemic absorption of carbon dioxide, and the reverse Trendelenburg position. Considering the widespread adoption of laparoscopic cholecystectomy, there is a remarkable paucity of data on the haemodynamic and respiratory changes in normal patients and those with cardiorespiratory disorders associated with these manoeuvres. Lack of critical experimental and clinical assessment of the physiological changes associated with laparoscopic cholecystectomy is in marked comparison with data obtained during gynaecological laparoscopy [15, 16].

In laparoscopic procedures, patients are normally placed in a 10–20° Trendelenburg position to keep the small bowel and colon out of the pelvis and to minimize complications associated with blind trocar insertion. Gravity may exert profound effects on the cardiovascular and pulmonary systems. The physiological effects of the Trendelenburg position have been reviewed by Wilcox and Vandam [17]. The associated cardiovascular changes may be influenced by the extent of the head-down tilt, the patient's age, intravascular volume status, associated cardiac disease, anaesthetic drugs and ventilation techniques. However, in our patients, the adoption of the Trendelenburg position was not associated with significant cardiovascular changes, but reverse Trendelenburg positioning was associated with a decrease in indices of left ventricular preload, presumably reflecting a gravitational effect on venous return [18].

The cardiovascular and respiratory changes associated with creation of a pneumoperitoneum have been extensively studied in gynaecological patients. Marshall and colleagues [19] reported no significant changes in cardiac output in anaesthetized, spontaneously breathing patients in whom the intra-abdominal pressure (IAP) was 15–20 cm H₂O (11–15 mm Hg). Smith and colleagues [20] studied the cardiovascular effects of stepwise increases of IAP up to a maximum of 25 cm H₂O in anaesthetized patients undergoing mechanical ventilation. At 25-cm H₂O IAP and increased airway pressure, intrathoracic pressure (ITP), central venous pressure (CVP) and femoral venous pressure were accompanied by hypertension, tachycardia and increased end-tidal carbon dioxide tensions. Based on data obtained from a study of the effects of progressively increasing IAP in horizontal and tilted anaesthetized patients undergoing mechanical ventilation, Kelman and colleagues [21] claimed that moderate increases in IAP (up to 25 cm H₂O) may be accompanied by increased transmural cardiac filling pressure and cardiac output. When IAP was increased further to 40 cm H₂O, tachycardia, hypotension, reduced CVP and cardiac output were observed. These changes were most marked in the horizontal compared with the head-down tilt position. The authors speculated that increased IAP may have two opposite effects on the cardiovascular system—it may force blood out of the abdominal organs and inferior vena cava into the central venous reservoir and may also dam blood back in the legs and thus tend to decrease the central blood volume.

Thus only minor haemodynamic changes occurred during gynaecological laparoscopic procedures with
insufflation of carbon dioxide when IAP did not exceed 25 cm H₂O (18 mm Hg). However, these short-duration studies were performed in young, relatively healthy, females in the Trendelenburg position. In our study of older, anaesthetized patients undergoing laparoscopic cholecystectomy, IAP of 15 mm Hg in the reverse Trendelenburg position was associated with increased peak airway pressures, arterial pressure and left ventricular afterload. This study and the limited data available on laparoscopic cholecystectomy suggest that the haemodynamic and respiratory function changes may differ from those reported with gynaecological laparoscopic procedures. In our study, end-tidal carbon dioxide partial pressure increased progressively with time—a finding consistent with data reported by Liu and colleagues [22] and Witgen and colleagues [23], who demonstrated similar increases in end-tidal carbon dioxide coincident with parallel changes in arterial partial pressures of carbon dioxide during laparoscopic cholecystectomy. Intraoperative hypercapnia may cause increases in plasma catecholamine concentrations, and increases in systemic arterial pressure and cardiac performance [24]. The latter were manifested by a shortened pre-ejection period (PEP), left ventricular ejection time (LVET), and decreased PEP:LVET ratio. Such neurohumoral mechanisms may have contributed to the increases in afterload observed in our study, and the preservation of cardiac performance in the face of these changes.

Systemic arterial pressure was measured using an automated cuff. Such automatic oscillometric measurements have been shown to correlate closely with invasive arterial cannulation and auscultation determinations [25, 26]. End-tidal carbon dioxide tension and oximetry were used as non-invasive substitutes for arterial PCO₂ and PaO₂ in evaluating adequacy of ventilation and oxygenation. It is well recognized that end-tidal carbon dioxide partial pressure may differ considerably from PaCO₂ because of ventilation–perfusion mismatching [27]. However, in a study of healthy patients undergoing laparoscopic cholecystectomy, Liu and colleagues [22] observed equal and proportional increases in PaCO₂ and PaO₂ after insufflation of carbon dioxide.

The variables LVEDA, LVEF and LVESWS were measured by transoesophageal echocardiography (TOE). Clements and colleagues [28] and Urbanowitz and colleagues [29] have demonstrated a close correlation between TOE-derived estimates of EDA and LVEF and radionuclide measurements. LVESWS is a quantitative index of left ventricular afterload [13]. In our study, these afterload estimates were based on LV echocardiographic end-systolic diameter (D), wall thickness (W) and systolic arterial pressure (P), calculated by the formula: 0.334 × P(D)/WT (1 + WT/D) and expressed as dyn cm⁻². O’Kelly and colleagues [13] have validated the use of TOE measurements in this context. Quinones and colleagues [14] have reported that LVESWS determinations with arterial pressure cuff systolic pressure measurements correlate closely (r = 0.97) with determinations using invasive end-systolic LV micromanometer pressure in patients with chronically sustained hypertension. Reichek and colleagues [30] similarly validated the technique before and after load manipulation with nitroglycerin, nitroprusside, phenylephrine and saline.

It should be noted that our data were obtained in healthy patients. Further studies may be warranted in patients with cardiopulmonary disease.

REFERENCES

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