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Assessment of left ventricular performance during laparoscopy

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Abstract. – OBJECTIVES: Cardiovascular changes during laparoscopic surgery have been described in several studies. Pneumoperitoneum effects on cardiac performance instead have not been much investigated and are less known. The carbon dioxide insufflation necessary in order to perform laparoscopic procedures represents a higher force against which the myocardial fibers must shorten during ventricular contraction. Hypothesis of this study is that the intra-abdominal pressure at 12 mmHg could acutely affect the left ventricular wall stress and work. Aim of the study was to evaluate the impact of relationship pneumoperitoneum on the echocardiographic measures of left ventricular contractile function.

PATIENTS AND METHODS: We studied 20 healthy, ASA I women, undergoing laparoscopic hysterectomy. Transthoracic echocardiography was performed preoperatively, after induction of anaesthesia, 15' after pneumoperitoneum, 15' after Trendelenburg positioning and 15' after the end of surgery. Left ventricular end-systolic wall stress, stroke work, left ventricular ejection time and mean velocity of fiber shortening were registered.

RESULTS: Carbon dioxide insufflation caused a consistent increase in left ventricular end-systolic wall stress and left ventricular ejection time, while the mean velocity of fiber shortening decreased. The Trendelenburg position did not produce any other signficant effects. These changes were transient with a return to preinsufflation values at the last measurement. Pneumoperitoneum adversely affects left ventricular performance during laparoscopy, leading to an increase of left ventricular end-systolic wall stress and reducing the mean velocity of circumferential fiber shortening. These cardiac consequences appear to be reversible since all the echocardiographic parameters normalized at the end of surgery.

CONCLUSIONS: Our results suggest that there is an adaptation of the cardiac systolic contractile status to the pressure overload during laparoscopy.

Key Words:
Laparoscopy, Pneumoperitoneum, Left ventricular function, Echocardiography.

Introduction

Several authors[1,2] described the cardiovascular changes as consequence of the pneumoperitoneum (Pnp) during laparoscopic procedures. Increases in heart rate, mean arterial pressure (MAP), systemic and pulmonary vascular resistances and a decrease in cardiac index have been described, but its effects on the cardiac performance are less known. A moderate decline in cardiac output, stroke volume, systemic blood pressure and pH may result from retained CO_2, which depend on the volume insufflated and intra-abdominal pressure[3,4]. The compression of the abdominal aorta contributes to the increase in the venous resistance as well the augmented systemic vascular resistances (SVR) is associated with the release of humoral factors like catecholamines, vasopressin, and the enhanced renin-angiotensin activity[2,4]. In a recent study[5] we demonstrated that Pnp during laparoscopic hysterectomy affected the diastolic parameters in a group of healthy women. The pressure overload during the Pnp represents a higher force against which the myocardial fibers must shorten during ventricular contraction. Chronically the heart may become hypertrophic to maintain ventricle wall stress within certain limits. Left ventricular systolic function is the product of the interaction of four variables: myocardial contractile state, end-diastolic myocardial length, preload and afterload. Left ventricular ejection time (LVET) measures the period of blood flow across the aortic valve. It is influenced by heart rate (HR), left ventricular mass, afterload and contractile state. LVET shortens with mitral regurgitation and increases with compensate aortic stenosis and high cardiac output states. It is also used to calculate the mean velocity of fiber shortening (mVcf)[6,7]. Reichek et al[8] demonstrated that end-systolic stress may be determined noninvasively and may be a useful approach to quantify afterload and contractility. Af-

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terload is increased when aortic pressure and SVR augment, and this contributes to an increase in the end-systolic volume (ESV) and a decrease in the stroke volume (SV).

The pathophysiological basis for this is found in the force-velocity relationship for cardiac myocites. Briefly, an increase in afterload could cause a decrease in the mVcf.

Our hypothesis was that the intra-abdominal pressure at 12 mmHg could acutely affect the left ventricular wall stress the contractile response work.

Primary endpoint was the changes on the left ventricular end systolic wall stress (LVESWS). Secondary endpoints were the stroke work (SW) and the relationship between LVET and mVcf.

Patients and Methods

After institutional Ethics Committee approval, we obtained the written, informed consent of 20 female patients scheduled for laparoscopic hysterectomy. All patients were American Society of Anesthesiologists (ASA) physical status I and had no cardiac, renal or respiratory diseases; we also excluded obese subjects (Body Mass Index ≥ 30 kg/m^2).

Anesthesia, Pneumoperitoneum and Surgery

A standardized general anaesthetic protocol was applied for all patients. Thirty minutes before entering the operating room all patients received intravenous midazolam (2 mg). General anaesthesia was induced with fentanyl (2 μg/kg) and propofol (2 mg/kg) followed by administration of vecuronium (0.1 mg/kg) to facilitate tracheal intubation and maintain muscle relaxation. Anaesthesia was then maintained with 50% air in oxygen and sevoflurane (MAC 1). Additional dose (2-3 μg/kg) of intravenous fentanyl was administered after incision.

During surgery, minute ventilation was controlled and adjusted to keep the expired end-tidal partial pressure of carbon dioxide (Et-CO_2) between 32 and 35 mmHg. All patients received a continuous intravenous infusion (5 ml/kg/h) of crystalloids solution. The surgical technique was similar for all patients. The CO_2 insufflation was performed with the patients placed in the horizontal position, and intra-abdominal pressure was automatically maintained at 12 mmHg. Intraoperative monitoring included continuous electrocardiography (leads I-II-III), pulse oximetry, non-invasive blood pressure and the mean airway pressures.

Transthoracic Echocardiography

Transthoracic echocardiography (TTE) examinations were performed with a 3.5-MHz transducer (Philips HD15). All echocardiographic measurements were carried out according to the criteria of the American Society of Ecocardiography[9]. We assessed left ventricle end-diastolic and end-systolic volumes in the apical 4- and 2-chamber views, by manual tracing of endocardial border after obtaining the best visualization possible of it to minimize the need for extrapolation. Systolic and diastolic thickness of interventricular septum (IVSs, IVSd) and posterior wall (PWs, PWd) and systolic and diastolic left ventricular diameter (LVDs, LVDd) were measured according to the Penn convention in the parasternal M-Mode view[10,11]. Relative wall thickness (RWT) was computed at end diastole as the ratio of IVS + PW and LVDd. Left ventricular end-systolic meridional wall stress was calculated in M-mode by applying the formulas suggested by Reichek et al[12]. Stroke Work was calculated as the product of SV and mean aortic pressure[12]. Cardiac output (CO) was calculated by multiplying the time velocity integral of the aortic flow (TVI) by the cross sectional area obtained by a 2D measurement of the diameter of the aortic annulus (A) and the HR. For the evaluation of the left ventricular systolic function we measured the ejection fraction (EF) and fractional shortening, calculated by standard formulas (respectively, volumetric and dimensional). LVET was determined from the aortic valve in M-mode; mean velocity of fiber shortening was calculated as the FS/LVET ratio. All measurements were obtained in the following times: before and after induction of anesthesia (respectively T0 and T1), fifteen minutes after Pnp (T2), fifteen minutes after Trendelenburg positioning (T3) and at the end of surgery (T4).

Statistical Analysis

All data were analyzed using the statistical package SPSS 15.0 version (SPSS Inc., Chicago, IL, USA). All data were expressed as mean±SD unless otherwise specified. All data were first analyzed for normality of distribution using the Kolmogorov-Smirnov test of normality. When comparing differences between different timings, the Friedman ANOVA non parametric test was used for non-normally distributed continues variables and analysis of variance for repeated measures was used for normally distributed variables. Categorical variables were compared using the χ^2 test. Correlations were calculated with the Spearman correlation coefficient. A p value of less than 0.05 was considered statistically significant.

Table I. Demographic and clinical data in patients (n = 20).

Age (y)	45.8 ± 11.5
ASA physical status (I/II)	20/0
Weight (kg)	56.6 ± 7.9
Height (cm)	165.3 ± 7.6
Duration of anesthesia (min)	109 ± 31
Duration of surgery (min)	86 ± 9
Duration of CO_2 insufflation (min)	67 ± 15
Duration of Trendelenburg (min)	44 ± 12

ASA: American Society of Anaesthesiologists.

Results

Patient's characteristics and details of operation are listed in Table I. 24 patients were enrolled in the study; four patients were excluded owing to the technical problems in the echocardiographic examination during surgery. No major intraoperative complications occurred in the group. A detailed description of some of the echocardiographic findings is given in Table II.

At T0 measurements represent baseline values. The induction of anaesthesia (T1) did not affect any variables. After the creation of Pnp (T2) we found that MAP increased and SV decreased. At the same time LVEDV significantly diminished ($p < 0.05$), while any variation was found regarding the cardiac output (CO), HR and EF. We observed that capnoperitoneum led to a significant reduction of FS (from 42.5±3.2 to 35.5±1.2 %, $p < 0.05$), while LVESWS significantly increased (from 78±0.8 to 92.5±1.5, $p < 0.05$) (Figure 1).

In Figure 2 are represented the effects of gas insufflation (T2) and Trendelenburg positioning (T3) on LVET and mVcf. When LVET is increased after the creation of Pnp, mVcf significantly decreased, despite the reduction of FS.

Trendelenburg did not affect FS and LVESWS while SW remained raised at this time (T3). mVcf resulted increased at T3 (compared to T2) and LVET resulted slightly higher at T3 than T2. SV and LVEDV are increased after Trendelenburg, while HR resulted slightly reduced, and CO did not change.

Discussion

Pneumoperitoneum results in a state of acutely elevated intra-abdominal pressure, which is normally 5 mmHg or less in non obese subjects[13]. The hemodynamic effects of Pnp on cardiac function have not been extensively examined.

In their study Tekelioglu et al[14] exposed that Pnp has adverse effects on cardiac autonomic regulation during postoperative period according to the long-term HR variability and HR turbolence frequency analysis. About left ventricular filling pressures Russo et al[15] demonstrated that Pnp produces a consistent increase of this parameter in a population of hypertensive patients with and without diastolic dysfunction.

The observations in the present study indicate that gas insufflation, during laparoscopic surgery, produced consistent effects on systolic time intervals and left ventricular contractile function. Several factors may influence cardiac function such as preload, afterload, contractility, HR, and myocardial compliance. The main factor specific to laparoscopy which can affect intraoperative cardiac function is the in-

Table II. Hemodynamic and echocardiographic data in patients (n = 20).

	LH (n:20)				
	T0	**T1**	**T2**	**T3**	**T4**
MAP (mmHg)	83.7 ± 4.2	81.0 ± 6.4	104.7 ± 9.3	96.7 ± 3.6	84.1 ± 11.5
HR (bpm)	64 ± 3	66 ± 5	68 ± 4	68 ± 6	71 ± 0
EF (%)	64.7 ± 5.4	61.8 ± 6.7	63.8 ± 5	63.7 ± 5.7	63.5 ± 6.3
CO (L/min)	4.4 ± 1.1	4.5 ± 0.7	4.3 ± 2.3	4.3 ± 4	4.5 ± 0.8
SV (mL)	74.3 ± 2.7	73.6 ± 5.3	66.7 ± 4.8	73.3 ± 2.9	74.6 ± 3.2
LVEDV (mL)	106.3 ± 4.2	105 ± 3.1	99.4 ± 6.1	109.2 ± 2.9	108.5 ± 7.4

Hemodynamic and echocardiographic data of patients at different times of the study are shown in the table.
See "Results" and "Discussion" sections for a detailed comment upon data presented in the table.
T0: baseline, T1: after induction of anaesthesia, T2: 15 min after pneumoperitoneum, T3: 15 min after Trendelenburg, T4: after surgery. CO: cardiac output, EF: ejection fraction; HR: heart rate, LVEDV: left ventricular end-diastolic volume, MAP: mean arterial pressure, SV: stroke volume,
Fonts in bold indicate significantly different values ($p < 0.05$) compared to baseline (T0).

creased intra-abdominal pressure. Fleming et al[16] reported that the afterload, as measured by SVR, increased by 25% of baseline after abdominal insufflation and decreased with desufflation.

Left ventricular performance describes the pumping properties of the ventricle. The performance of the ventricle as a pump has been assessed in our study by measuring the stroke volume ejected by the ventricle, and LVET. The ventricular function includes shortening velocity parameters, mistakenly referred to as indices of ventricular contractility, but these are also sensitive to changes in preload and afterload. In our population study all examinations were made in patients mechanically ventilated. Volume determinations are subject to a variety of criticisms, one of which is that errors in minor-axis dimensional measurements are amplified for volume calculations. For such reason, we have chosen to employ dimensional measurements and fractional shortening determinations derived directly from the dimension. This provides a more accurate representation of myocardial alterations. The relationship between fractional shortening and systolic wall stress can be used as an index of left ventricular contractility. The augmented afterload contributes to the re-

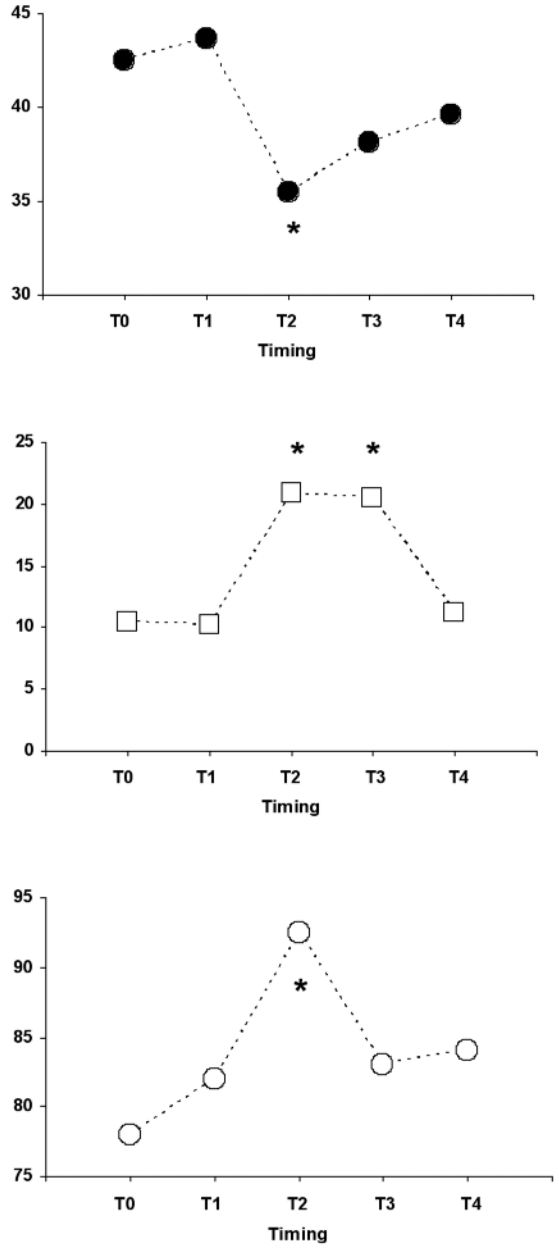

Figure 1. In figure are reported Fractional Shortening (FS) Stroke Work (SW) and Left Ventricular End-Systolic Wall Stress (LVESWS) at different timing. T_0: baseline, T_1: after induction of anaesthesia, T_2: 15 min after pneumoperitoneum, T_3: 15 min after Trendelenburg, T_4: after surgery. *$p < 0.05$ compared with baseline (T_0).

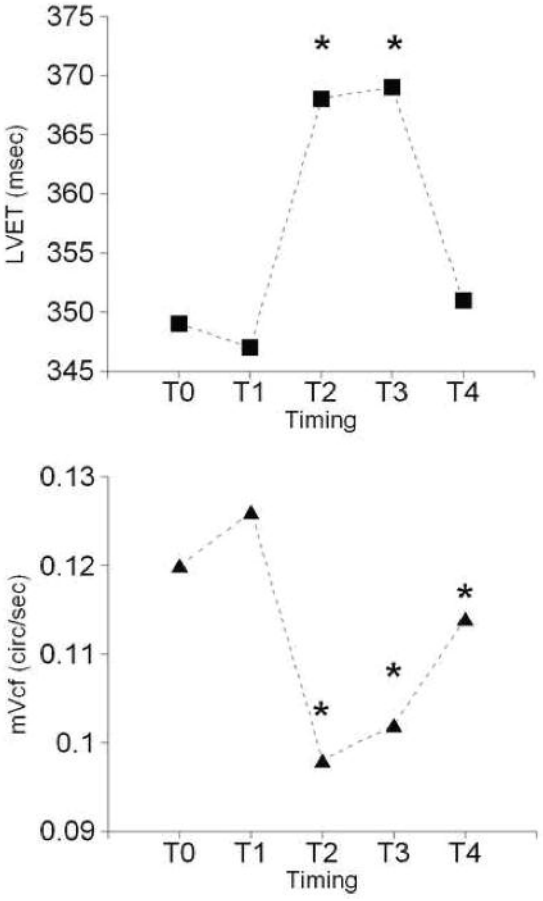

Figure 2. Left Ventricular Ejection Time (LVET) and mean Velocity of fiber shortening (mVcf) at different timing. T0: baseline, T1: after induction of anaesthesia, T2: 15 min after pneumoperitoneum, T3: 15 min after Trendelenburg, T4: after surgery. *$p < 0.05$ compared with baseline (T0).

duced SV (e.g. an increase in the left ventricular end-systolic volume) and the decreased preload caused a reduction of the end-diastolic volume (Table II). Combining the changes in these parameters, a substantial decrease in FS occurred. In accordance with our hypothesis the augmented afterload during Pnp led to increased LVESWS and SW. These findings are in contrast with Hirvonen et al[17] who reported that both right and left ventricular stroke indexes decreased during laparoscopy. Moreover we found that FS decreased after Pnp.

Arterial pressure basically represents the afterload opposing the left ventricle ejection. We found a prolonged LVET with a consequent reduction of the mVcf. The increase in afterload may be attributable to vasoconstriction due to the high intra-abdominal pressure and to the activation of sympathetic nerve system. The increase in SVR is also considered to be mediated by mechanical and neurohumoral factors[18]. The well documented elevation of MAP immediately after the creation of Pnp could be thought as a consequence of this condition. We registered a significant increase of MAP after carbon dioxide Pnp.

Conclusions

Our results suggested that the augmented intra-abdominal pressure during Pnp adversely affects left ventricular performance leading to an increase of the end-systolic wall stress and reducing the mean velocity of circumferential fiber shortening.

Moreover, these cardiac consequences appear to be reversible since all the echocardiographic parameters are normalized at the end of surgery.

Conflict of Interest

The Authors declare that there are no conflicts of interest.

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