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SCIENTIFIC ARTICLE

Effects of elevated artificial pneumoperitoneum pressure on invasive blood pressure and levels of blood gases

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Abstract

Background and objective: to evaluate the clinical, hemodynamic, gas analysis and metabolic repercussions of high transient pressures of pneumoperitoneum for a short period of time to ensure greater security for introduction of the first trocar.

Methods: sixty-seven patients undergoing laparoscopic procedures were studied and randomly distributed in P12 group: $n = 30$ (intraperitoneal pressure [IPP] 12 mmHg) and P20 group: $n = 37$ (IPP of 20 mmHg). Mean arterial pressure (MAP) was evaluated by catheterization of the radial artery; and through gas analysis, pH, partial pressure of oxygen (PaO₂), partial pressure of CO₂ (PaCO₂), bicarbonate (HCO₃) and alkalinity (BE) were evaluated. These parameters were measured in both groups at time zero before pneumoperitoneum (TP0); at time 1 (TP1) when IPP reaches 12 mmHg in both groups; at time 2 (TP2) after five min with IPP = 12 mmHg in P12 and after 5 min with IPP = 20 mmHg at P20; and at time 3 (TP3) after 10 min with IPP = 12 mmHg in P12 and with return of IPP from 20 to 12 mmHg, starting 10 min after TP1 in P20. Different values from those considered normal for all parameters assessed, or the appearance of atypical organic phenomena, were considered as clinical changes.

Results: there were statistically significant differences in P20 group in MAP, pH, HCO₃ and BE, but within normal limits. No clinical and pathological changes were observed.

Conclusions: high and transient intra-abdominal pressure causes changes in MAP, pH, HCO₃ and BE, but without any clinical impact on the patient.

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Introduction

Minimally invasive methods used to access organs and structures of the abdominal cavity cause a reduction of metabolic

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response to trauma and other benefits for patients. This applies particularly to laparoscopy.¹⁻³ However, being relatively recent, the laparoscopic surgical techniques still show controversy. One of them is the best way of creating the pneumoperitoneum. Although no consensus exists regarding the best method for accessing the peritoneal cavity with respect to the establishment of pneumoperitoneum, the puncture with Veress needle is the technique most often used.^{4,5} The complications occurring during the introduction of the first catheter are still being discussed.

Much of the complications in laparoscopy procedures (in about 50% of them) occur at the beginning of the procedure, during the introduction of the Veress needle and the first trocar. For that reason, laparoscopy is a peculiar surgical procedure, in which the surgical approach is more dangerous than the surgery itself.⁶ In a recent review of the literature concerning injuries caused by the use of a Veress needle and the first trocar in 357,257 patients, a prevalence of 0.04% of gastrointestinal lesions and 0.02% of vascular lesions was found.⁷ These iatrogenic events are relatively rare, but the consequences are exceptionally grave. In such circumstances, bleeding, peritonitis, multiple organ failure, death and medico-legal implications may occur.

Thus, it is essential to seek technical options safer than the most commonly used method, which consists of the Veress needle puncture in the midline of the abdomen, in the vicinity of the umbilicus; abdominal insufflation to obtain intraperitoneal pressure of 10 and 12 mmHg; and the blind introduction of the first trocar in the same location used for needle insertion.^{4,5}

A literature review revealed that the most serious injuries occur when the Veress needle is inserted into the midline of the abdomen at the level of the umbilicus.⁷ The insertion of the Veress needle in the left hypochondriac region, however, is safe and effective⁸ and the likelihood of serious injury is lower, because this place does not involve vital structures, such as the retroperitoneal vessels.⁷

However, the insertion of the first trocar should be done in the midline at the level of the umbilicus, and not in the left hypochondrium, as recommended for the Veress needle.⁸ This recommendation is based on the fact that the trocar is the place where the laparoscopic cannula will be introduced.^{4,5} When the laparoscope is introduced in the midline at the umbilicus, we get better clarity, better images of organs and intra-abdominal structures, and a broader vision for the introduction of the other trocars.

The establishment of a regime of very high pressure by an artificial pneumoperitoneum, during a period just sufficient for the introduction of the first trocar, taken blindly in the closed method, may contribute to the protection of the intra-abdominal structures against injury, but without any organic repercussion in the form of clinical complications.^{9,10} No vascular injury was reported in a study that investigated 3041 patients undergoing blind insertion of the first trocar in the midline with an intra-abdominal pressure below 25–30 mmHg.¹¹

One study investigated the protective effect of elevated intraperitoneal pressure on intra-abdominal structures facing the aggression shown by the blind introduction of the first trocar into the peritoneal cavity.¹² The authors correlated the distance between the anterior abdominal wall and intra-abdominal viscera with different intraperitoneal

pressures and volumes, and also the observed distances with the required force for insertion of the first trocar into the abdominal cavity. These authors also could observe that high intraperitoneal pressures cause an important increase in these distances and in the volume of gas bubbles and provide a better slippage of the trocar into the cavity. It was also shown that, with the use of high intraperitoneal pressure, the abdominal wall becomes tenser and reduces its elastic deformation caused by a force applied to the trocar.¹²

Despite the absence of clear clinical signs of complications, the artificial pneumoperitoneum with very high pressures over a prolonged period of time can cause hemodynamic and structural changes in the host, directly related to the magnitude of the tensional levels and detectable by monitoring hemodynamic and gas analysis parameters. Thus, under high intraperitoneal pressures, decreases in cardiac output and venous return, increases of mean arterial pressure and systemic vascular resistance and changes in renal perfusion and glomerular filtration were demonstrated, besides ischemic lesion and reperfusion of intra-abdominal organs.¹³⁻¹⁹ Because of these deleterious effects of high intraperitoneal pressures during laparoscopic procedures, most authors recommend maintaining the pressure at a level of 12 mmHg (never more than 15 mmHg, considered as a high pressure).^{5,20-26}

Despite the above considerations, hemodynamic, metabolic and structural changes may occur with elevated intra-abdominal pressures for a prolonged period of time. The literature does not provide important information about gas analysis and metabolic changes in patients undergoing high transient intraperitoneal pressure. This means that laparoscopic surgeons may not have taken into account a safe strategy for the introduction of the first trocar.

The aim of this study is to improve the safety of the introduction of the first trocar and evaluate the clinical, hemodynamic, gas analysis and metabolic effects of high transient pneumoperitoneum pressures for short periods of time.

Materials and methods

For this prospective, randomized clinical trial, authorization was obtained from the Federal University of São Paulo (UNIFESP) Research Ethics Committee under number 1.219/07, and from the University of Taubaté (Unitau) Research Ethics Committee, under number 007/2.007. All patients signed an informed consent. The study was conducted at Hospital Municipal Dr. José de Carvalho Florence (HMJCF) in São José dos Campos (SP).

Between October 2007 and May 2008, 67 patients scheduled for elective laparoscopic surgery, between 20 and 79 years old, classified into ASA I or ASA II according to their physical condition, with no history of abdominal surgery on organs located at the abdominal supramesocolic level, without previously diagnosed peritonitis and with body mass index (BMI) less than 35, were studied.

Upon obtaining odd and even numbers on the upper face of a dice rolling, patients were randomly assigned to P12 group: $n = 30$ (intraperitoneal pressure of 12 mmHg) and P20 group: $n = 37$ (intraperitoneal pressure of 20 mmHg). P12 group consisted of 25 women and five men, between

22 and 72 years (mean \pm SD: 47.2 ± 14.5 years), with BMI between 20.2 and 33.4 kg m^{-2} (mean \pm SD: $26.3 \pm 4 \text{ kg m}^{-2}$). P20 group consisted of 30 women and seven men, aged between 20 and 79 years (mean \pm SD: 46.5 ± 15 years), with BMI between 17.5 and 34.6 kg m^{-2} (mean \pm SD: $26.2 \pm 3.8 \text{ kg m}^{-2}$). No statistically significant difference was observed between groups in the demographic data compared ($p \leq 0.05$).

All patients received pre-anesthetic evaluation in the clinic in a prior date to the surgery. No patient received anesthetic premedication.

Before the start of anesthesia, the modified Allen test was performed.²⁷ The patients were hydrated with Ringer Lactate after venipuncture with a 18G catheter. The patients were monitored by lines installed in order to assess data from cardioscopy, pulse oximetry, non-invasive blood pressure,²⁸ capnometry and intratracheal pressure.

All patients received general anesthesia. The anesthetic procedure was induced with sufentanil 0.5 mcg kg^{-1} , rocuronium 0.6 mg kg^{-1} and propofol 2 mg kg^{-1} . The anesthesia was maintained with sevoflurane in a mixture of oxygen and compressed air. All patients were mechanically ventilated by constant flux in a cycling time fan. Ergo System PC 2700-Shogun Takaoka anesthesia and monitoring machines were used, as well as Fabius GS Dräger anesthesia machine with Dixtal model DX 2010 monitors. Initial ventilation was achieved with a fraction of inspired oxygen of 60%, positive end expiratory pressure (PEEP) = $4 \text{ cm H}_2\text{O}$, tidal volume = 7 mL kg^{-1} , respiratory rate = 15 breaths per minute and inspiration/expiration ratio = 1:2.

With the establishment of an appropriate anesthetic plan and a negative Allen test (modified by Asif),²⁶ the radial artery was catheterized in the non-dominant limb. A maximum of three attempts were done, with exclusion of patients in whom no success was obtained in the procedure.

Six patients were excluded from the study: one had bronchospasm after induction; one with difficult intubation and with need of additional procedures not included in the study protocol; two, with failure in the third attempt to catheterization of the radial artery; and in the remaining two, the sample was lost by clot formation.

The creation of pneumoperitoneum was obtained by closed technique with abdominal puncture through the Veress needle and CO_2 flow of 1 L/min .

During the procedure, MAP and blood gas analysis – pH, PaO_2 (in mmHg), PaCO_2 (in mmHg), HCO_3^- (in mmol/L), BE (in mmol/L) with a blood gas analyzer Rapidlab 348 Bayer Health Care, Model 348 pH/Analyzer SN 6678. These parameters were evaluated in both groups at time zero, before pneumoperitoneum; at time 1 (TP1), when IPP reaches 12 mmHg in both groups: at time 2 (TP2), after 5 min with IPP = 12 mmHg in P12 and after 5 min with IPP = 20 mmHg in P20; and at time 3 (TP3), after 10 min with IPP = 12 mmHg in P12 and with return of IPP from 20 to 12 mmHg , counted 10 min after TP1 in P20.

All patients were followed during the anesthetic-surgical procedure through the following parameters: heart rate, heart rhythm, pulse oximetry, capnometry (EtCO_2) and mean arterial pressure. In the post-anesthesia recovery room, heart rate, heart rhythm, mean arterial pressure, pulse oximetry, level of consciousness and muscle activity were

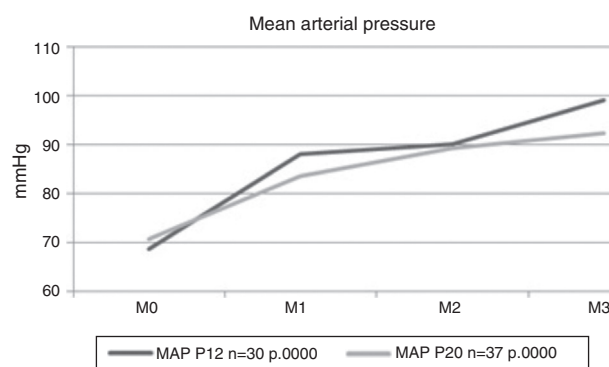


Figure 1 Mean arterial pressure (MAP in mmHg).

the observed parameters observed, until patients' discharge to the ward.

We considered as "occurrence of clinical change" the measured values of the various parameters that extrapolated the limits considered normal for healthy people, or the emergence of atypical phenomena indicative of the presence of organic disease. HR less than 75 beats per minute; MAP between 70 mmHg and 120 mmHg; SaO_2 greater than 93%; EtCO_2 between 30 and 45 mmHg; intrathoracic pressure (ITP) below $35 \text{ cm H}_2\text{O}$; pH between 7.35 and 7.45, PaCO_2 between 30 and 45 mmHg; PaO_2 above 80 mmHg; BE between -2 and $+2$; and HCO_3^- between 22 and 26 mEq L^{-1} were considered normal values.

As for the statistical analysis, in the descriptive analysis, position measurements for continuous variables and frequency for categorical variables were used. To compare gender between groups, we used the chi-squared test, and to compare age and BMI between groups, we used the nonparametric Mann-Whitney test. For comparison among times of variables of interest, we used the analysis of variance (ANOVA) for repeated measures with transformation by posts. A level of 5% ($p = 0.05$) was considered significant.

Results

Mean arterial pressure (MAP in mmHg)

In P12 group, MAP presented the following values (mean and standard deviation) for M0, M1, M2 and M3, respectively: 68.57 ± 10.18 , 88.10 ± 17.68 , 90.10 ± 19.03 and 99.07 ± 18.58 , with statistical difference ($p = 0.0000$). In P20 the mean and standard deviation values of MAP for M0, M1, M2 and M3 were, respectively: 70.57 ± 14.58 ; 83.57 ± 12.86 , 89.30 ± 15.33 and 92.43 ± 14.42 , with statistical difference ($p = 0.0000$) (Fig. 1). In P12 group the statistical difference occurred in M0 with M1, M2 and M3; between M1 and M3 and between M2 and M3. In P20 group a difference was noted in M0 with M1, M2 and M3, and between M1 with M2 and M3.

Hydrogen potential (pH)

In P12 group, the pH values (mean and standard deviation) for M0, M1, M2 and M3 were, respectively: 7.47 ± 0.05 , 7.47 ± 0.06 , 7.46 ± 0.06 and 7.44 ± 0.06 , with statistical difference ($p = 0.0000$). In P20 group the pH values (mean and

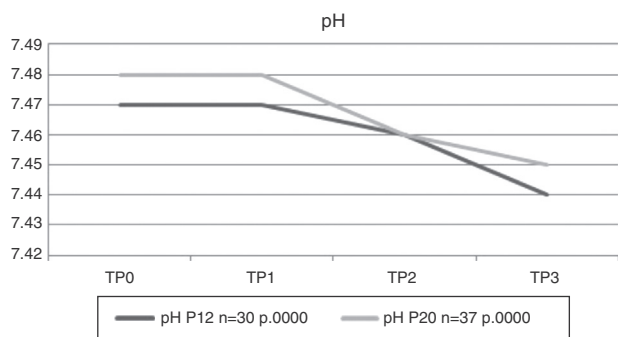


Figure 2 Hydrogen potential (pH).

standard deviation) for M0, M1, M2 and M3 were, respectively: 7.48 ± 0.06 , 7.48 ± 0.06 , 7.46 ± 0.06 and 7.45 ± 0.07 , with statistical difference ($p=0.0000$) (Fig. 2). In P12 group the pH showed significant change between M0 and M3, M1 relative to M2 and M3, and between M2 and M3. In P20 group differences were observed between M0 in relation to M2 and M3, and of M1 compared to M2 and M3.

Partial pressure of oxygen in the arterial blood (PaO₂ in mmHg)

In P12 group, PaO₂ showed the following values (mean and standard deviation) for M0, M1, M2 and M3, respectively: 216.80 ± 51.60 ; 192.15 ± 52.73 ; 191.88 ± 51.74 , and 196.77 ± 46.66 , with statistical difference ($p=0.0057$). In P20 group, PaO₂ showed the following values (mean and standard deviation) for M0, M1, M2 and M3, respectively: 212.07 ± 72.37 ; 197.73 ± 52.74 ; 202.35 ± 52.46 , and 203.41 ± 49.20 , with no statistical difference ($p=0.4239$) (Fig. 3). In P12 group, statistical difference occurred between M0 and M1.

Partial pressure of carbon dioxide (PaCO₂ in mmHg)

In P12 group, mean and standard deviation values of PaCO₂ for M0, M1, M2 and M3 were, respectively: 31.96 ± 5.20 ; 31.48 ± 6.67 , 32.68 ± 6.82 and 32.63 ± 8.30 , with no statistical difference ($p=0.3557$). In P20 group, PaCO₂ had the following values (mean and standard deviation) for M0, M1, M2 and M3, respectively: 32.47 ± 5.36 ; 32.43 ± 4.84 ;

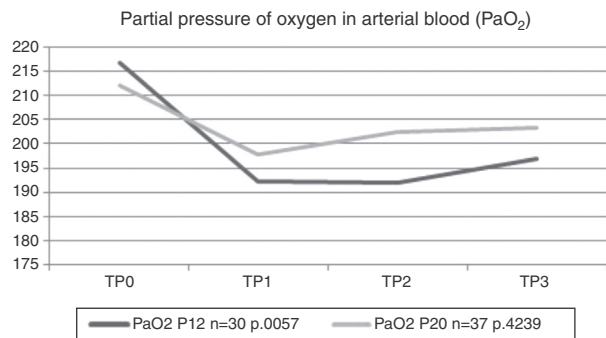


Figure 3 Partial pressure of oxygen in arterial blood (PaO₂ in mmHg).

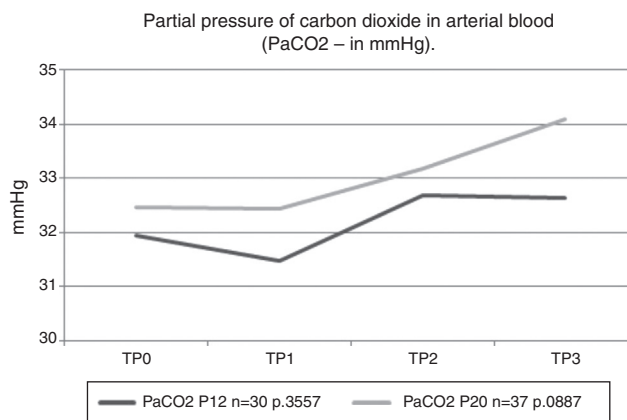


Figure 4 Partial pressure of carbon dioxide in arterial blood (PaCO₂ in mmHg).

33.19 ± 5.08 and 34.09 ± 6.20 , with no statistical difference ($p=0.0887$) (Fig. 4).

Bicarbonate (HCO₃ in mmol L⁻¹)

In P12 group, HCO₃ showed the following values (mean and standard deviation) for M0, M1, M2 and M3, respectively: 22.85 ± 3.11 , 22.50 ± 3.85 , 22.42 ± 3.34 and 21.96 ± 4.38 , with no statistical significance ($p=0.3629$). In P20 group, HCO₃ showed the following values (mean and standard deviation) for M0, M1, M2 and M3, respectively: 23.75 ± 3.45 , 23.48 ± 2.64 , 23.06 ± 3.04 and 23.20 ± 3.17 , with statistical difference ($p=0.0126$) (Fig. 5). In P20 group there was statistical difference between M0 and M2.

Alkalinity (base excess [BE] in mmol L⁻¹)

In P12 group, BE showed the following values (mean and standard deviation) for M0, M1, M2 and M3, respectively: 0.15 ± 3.00 , -0.08 ± 3.55 , -0.53 ± 3.14 and -1.27 ± 3.92 , with statistical difference ($p=0.0001$). In P20 group, BE showed the following values (mean and standard deviation) for M0, M1, M2 and M3, respectively: 1.10 ± 3.27 , 0.82 ± 2.74 , 0.05 ± 3.22 and -0.03 ± 3.12 , with statistical difference ($p=0.0000$) (Fig. 6). In P12 group, BE showed statistical difference group when M0 was compared with M3 and M1 was compared with M2 and M3. In P20 group,

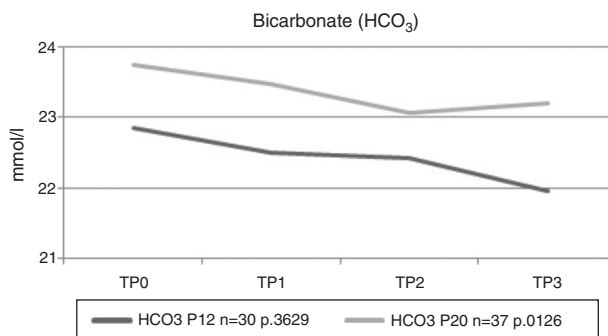


Figure 5 Bicarbonate (HCO₃ in mmol L⁻¹).

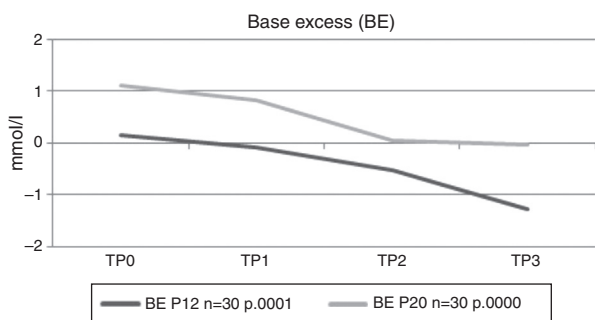


Figure 6 Alkaline reserve (base excess - BE in mmol L⁻¹).

differences appeared in M0 compared with M2 and M3 and in M1 compared with M2 and M3.

The values measured in the different parameters evaluated did not surpass those considered as normal in healthy populations during surgical procedures and in their stay until discharge from post-anesthesia recovery.

Discussion

In this study, the organic alterations and gas exchanges in laparoscopic procedures with high transient pressures of pneumoperitoneum with sufficient time to introduce the first trocar were analyzed.

Patients were divided into two groups, P12 (intra-peritoneal pressure of 12 mmHg) and P20 (intra-peritoneal pressure of 20 mmHg).

The P12 group was the positive control group, in which all events and all possible changes during the surgical procedure with standard (12 mmHg) intra-peritoneal pressure in our population of interest were analyzed. The purpose of the inclusion of P12 group in this study was to clarify the role in isolation of high pressures (20 mmHg) in any change that was to be observed in P20 group, by a comparison among the times of each group studied. The behavior of the parameters was evaluated in P12 group to exclude the factor "exposure time to pneumoperitoneum" as a determinant of organic changes likely to occur in P20 group. Thus, it may be possible to assign exclusively to high intra-peritoneal pressure any such changes observed in P20.

The P20 group was the experimental group with auto-control, because their patients were subjected to different intra-peritoneal pressures, from absence of pneumoperitoneum to an intra-peritoneal pressure of 20 mmHg.

The anesthetic agents propofol, rocuronium, sufentanil and sevoflurane were used with the aim of maintaining the stability of cardiopulmonary parameters, providing quick access to the airways and decreasing the postoperative incidence of nausea, vomiting and pain processes.²⁹⁻³⁴

The initial ventilator settings were: constant flow, end inspired oxygen fraction of 60%, positive end expiratory pressure (PEEP) of 4 cm H₂O, tidal volume of 7 mL kg⁻¹, respiratory rate of 15 breaths per minute, inspiration/expiration ratio of 1:2 and with volume cycling, with the intention of promoting an adequate minute volume to compensate for the patient's exposure to increased intra-peritoneal pressure with CO₂.³⁵

A study conducted by Abu-Rafea et al.³⁶ showed no cardiopulmonary complications in 100 healthy women

undergoing high intra-abdominal pressure (between 10 and 30 mmHg) during the introduction of the first trocar. The authors analyzed the volume of CO₂ effectively inflated into the peritoneal cavity, heart rate, blood oxygen saturation, mean arterial pressure and pulmonary compliance, and observed statistically significant changes in MAP and pulmonary compliance, but these changes were not clinically significant. However, Abu-Rafea et al.³⁶ did not set parameters to assess changes in respiratory function and gas exchange. Moreover, the effect of each pressure level (10, 15, 20, 25 and 30 mmHg) was evaluated at the exact moment it was achieved, without taking into account the cumulative effect of the duration of pneumoperitoneum for insertion of the first trocar, and this makes difficult to assess the clinical effects resulting from the duration of pneumoperitoneum, rather than from the level of intra-abdominal pressure reached. Furthermore, the cardiovascular parameters were monitored with noninvasive methods and arterial blood gases were not analyzed. Another study showed that the high intra-abdominal pressure is a safe practice, and no adverse clinical effects were observed by non-invasive monitoring analysis.³⁷

In our results, a statistically significant change was observed in MAP in both groups and throughout artificial pneumoperitoneum. The fact that this change was also observed in P12 group would suggest that its cause was due to the event of exposure of the body to pneumoperitoneum, even with a standard IPP. Even at low pressures (considered) (12 mmHg), a vasoconstriction reflex is triggered, with consequent increase in blood pressure. However, these changes do not represent clinical problems to the patient (Fig. 1). It is noteworthy that there was no case of hypertension in any of the groups.

Laparoscopic procedures with pneumoperitoneum and the use of CO₂ are associated with risk of hypercapnia through IPP increase and of absorption of CO₂ through the peritoneum,³⁸⁻⁴⁰ which can lead to respiratory acidosis. Some studies show that CO₂ absorption is dependent on the intra-peritoneal pressure and on the integrity of the peritoneum to absorb CO₂. In the present study, no statistically significant change in PaCO₂ values in both groups was observed. As the ventilatory parameters were not changed during the study, the findings suggest that there was no increase in CO₂ absorption by peritoneum due to the increase in IPP of 12-20 mmHg during 5 min in the presence of a consistent lung ventilation. This may be due to the fact that the increase in intra-abdominal pressure promotes capillary compression, limiting CO₂ absorption;⁴¹⁻⁴³ on the other hand, it decreases the blood flow to the splanchnic region.

The present study demonstrated that patients initially developed a mild respiratory alkalosis as a consequence of the ventilatory parameters determined for the procedure. Because these parameters were not changed during the study and the measured values of respiratory products (PaCO₂) did not change significantly, the drop in pH values - immediately after the alkalosis - in statistically significant values may have occurred because of the mild elevation of PaCO₂ values and because of the metabolic acidosis generated due to a reduction of intra-abdominal organ perfusion. In the presence of an intra-peritoneal pressure of 20 mmHg, it was noted that the pH reduction occurs more sharply than in patients with intra-peritoneal pressure of 12 mmHg.

This corroborates the pathophysiological explanation that a decreased perfusion of intra-abdominal structures play a major role in the change in pH values observed in this study, since the other factor of acidosis (i.e., CO₂ absorption) was similar in P20 and P12 groups, as may be verified by the PaCO₂ values informed by gas analysis (Fig. 4). Some authors⁴⁴ showed an increase in pH at an intraperitoneal pressure of 15 mmHg in the first 30 min, with subsequent decrease of these values. This result was similar to that found in this study in the presence of higher (20 mmHg) and lower (12 mmHg) intraperitoneal pressures. The changes found in this study had no clinical significance (Fig. 2).

Regarding HCO₃, there was a statistically significant reduction in P20 group after exposure of the patient to an IPP of 20 mmHg, which was not observed at other times of this group with lower IPPs and that also did not happen in P12 group. This shows that the pressure of 20 mmHg is the factor responsible for the changes. Considering also the fact that the pH has shown greater reduction under an IPP of 20 mmHg without significant elevation of PaCO₂, all these may be pointing to a higher consumption of bicarbonate, in order to attenuating the metabolic acidosis by decreasing the irrigation of splanchnic organs. In the study of Sefr et al.,⁴⁴ there was no difference between pressures of 10 and 15 mmHg with respect to the production of HCO₃, while in our study the pressure of 20 mmHg showed a statistically significant decrease in this parameter. However, this change had no clinical significance (Fig. 5).

Regarding the alkaline reserve (BE), there was a statistically significant decrease in both groups. The changes found are related to the exposure time of the body to pneumoperitoneum factor. In the presence of a regime of intraperitoneal pressure of 20 mmHg, these changes appear earlier. The decrease in the values of BE at an IPP of 20 mmHg, associated with decreased pH and decreased HCO₃ factors without significant change in PaCO₂, can point again to alkaline reserve (BE) consumption to compensate for the ischemia of splanchnic organs. Sefr et al.⁴⁴ reported a decrease in BE IPP of 10 mmHg and an increase in the values of BE of 15 mmHg. In this study a decrease in BE was observed at IPPs of 12 and 20 mmHg. These changes had no clinical significance (Fig. 6).

The high (20 mmHg) and transient (5 min) intra-abdominal pressure for insertion of the first trocar causes changes in MAP, pH, HCO₃ and BE without clinical consequences for the patient and should be used to prevent the occurrence of iatrogenic injuries in the introduction of the first trocar.

Conflicts of interest

The authors declare no conflicts of interest.

References

- Schippers E, Ottinger AP, Anurov M, et al. Laparoscopic cholecystectomy: a minor abdominal trauma? *World J Surg.* 1993;17:539–42.
- Roll S, Azevedo JLMC, Campos F, et al. Two-ports technique of laparoscopic cholecystectomy. *Endoscopy.* 1997;29:543.
- Novitsky YW, Kercher KW, Czerniach DR, et al. Advantages of mini-laparoscopic vs. conventional laparoscopic cholecystectomy: results of a prospective randomized trial. *Arch Surg.* 2005;140:1178–83.
- Neudecker J, Sauerland S, Neugebauer EB, et al. The European Association for Endoscopic Surgery clinical practice guideline on the pneumoperitoneum for laparoscopic surgery. *Surg Endosc.* 2002;16:1121–43.
- Molloy D, Kaloo PD, Cooper M, et al. Laparoscopic entry: a literature review and analysis of techniques and complications of primary port entry. *Aust N Z J Obstet Gynaecol.* 2002;42:246–53.
- Neves JFNP, Monteiro GA, Almeida JR, et al. Lesão vascular grave em colecistectomia videolaparoscópica. Relato de dois casos. *Rev Bras Anesthesiol.* 2000;50:294–6.
- Azevedo JL, Azevedo OC, Miyahira SA, et al. Injuries caused by Veress needle insertion for creation of pneumoperitoneum: a systematic literature review. *Surg Endosc.* 2009;23:1428–32. <http://dx.doi.org/10.1007/s00464-009-0383-9>.
- Azevedo OC, Azevedo JLMC, Sorbello AA, et al. Veress needle insertion in the left hypochondrium in creation of the pneumoperitoneum. *Acta Cir Bras.* 2006;21:296–303.
- Reich H, Rasmussen C, Vidali A. Peritoneal hypertension for trocar insertion. *Gynaecol Endosc.* 1999;8:375–7.
- Tsaltas J, Pearce S, Lawrence A, et al. Safer laparoscopic trocar entry: it's all about pressure. *Aust N Z J Obstet Gynaecol.* 2004;44:349–50.
- Reich H, Ribeiro SC, Rasmussen C, et al. High-pressure trocar insertion technique. *J Soc Laparoendosc Surg.* 1999;3:45–8.
- Phillips G, Garry R, Kumar C, et al. How much gas is required for initial insufflation at laparoscopy. *Gynaecol Endosc.* 1999;8:369–74.
- Koivusalo AM, Lindgren L. Effects of carbon dioxide pneumoperitoneum for laparoscopic cholecystectomy. *Acta Anaesthesiol Scand.* 2000;44:834–41.
- Safran DB, Orlando R. Physiologic effects of pneumoperitoneum. *Am J Surg.* 1994;167:281–6.
- Indberg F, Bergqvist D, Bjorck M, et al. Renal hemodynamics during carbon dioxide pneumoperitoneum. *Surg Endosc.* 2003;17:480–4.
- MacDougall EM, Monk TG, Wolf JS, et al. The effect of prolonged pneumoperitoneum on renal function in an animal model. *J Am Coll Surg.* 1996;182:317–28.
- Akbulut G, Polat C, Aktepe F. The oxidative effect of prolonged CO₂ pneumoperitoneum on renal tissue of rats. *Surg Endosc.* 2004;18:1384–8.
- Ozmen MM, Kessaf Alsar A, Besler HT. Does splanchnic ischemia occur during laparoscopic cholecystectomy? *Surg Endosc.* 2002;16:468–71.
- Zulfikaroglu B, Koc M, Soran A. Evaluation of oxidative stress in laparoscopic cholecystectomy. *Surg Today.* 2002;32:869–74.
- Dexter SP, Vucevic M, Gibson J, et al. Hemodynamic consequences of high and low pressure capnoperitoneum during laparoscopic cholecystectomy. *Surg Endosc.* 1999;13:376–81.
- Rosen DMB, Lam AM, Chapman M, et al. Methods of creating pneumoperitoneum: a review of techniques and complications. *Obstet Gynecol Surv.* 1998;53:167–74.
- Motew M, Ivankovich AD, Bieniarz J, et al. Cardiovascular effects and acid–base and blood gas changes during laparoscopy. *Am J Obstet Gynecol.* 1973;115:1002–12.
- Greim CA, Broscheit J, Kortlander J, et al. Effects of intra-abdominal CO₂-insufflation on normal impaired myocardial function: an experimental study. *Acta Anaesthesiol Scand.* 2003;47:751–60.
- Ivankovich AD, Albrecht RF, Zahed B, et al. Cardiovascular collapse during gynecological laparoscopy. *Ill Med J.* 1974;145:58–61.
- Gutt CN, Oniu T, Mehrabi A. Circulatory and respiratory complications of carbon dioxide insufflation. *Dig Surg.* 2004;21:95–105.

26. Barczynski M, Herman RM. A prospective randomized trial on comparison of low-pressure (LP) and standard-pressure (SP) pneumoperitoneum for laparoscopic cholecystectomy. *Surg Endosc.* 2003;17:533–8.
27. Asif M, Sarkar PK. Three-digit Allen's test. *Ann Thorac Surg.* 2007;84:686–7.
28. Amaral JLG, Ferreira ACP, Ferez D, et al. Monitorização da respiração: oximetria e capnografia. *Rev Bras Anesthesiol.* 1992;42:51–8.
29. Turazzi JC, Bedin A. Sevoflurano em cirurgia videolaparoscópica. *Rev Bras Anesthesiol.* 1999;49:299–303.
30. Filipovic M, Michaux I, Wang J, et al. Effects of sevoflurane and propofol on left ventricular diastolic function in patients with pre-existing diastolic dysfunction. *Br J Anaesth.* 2007;98:12–8.
31. Filipovic M, Wang J, Michaux I, et al. Effects of halothane, sevoflurane, and propofol on left ventricular diastolic function in humans during spontaneous and mechanical ventilation. *Br J Anaesth.* 2005;94:186–92.
32. Dobson AP, McCluskey A, Meakin G, et al. Effective time to satisfactory intubation conditions after administration of rocuronium in adults. Comparison of propofol and thiopentone for rapid sequence induction of anaesthesia. *Anaesthesia.* 1999;54:172–97.
33. Dershwitz M, Michalowski P, Chang Y, et al. Postoperative nausea and vomiting after total intravenous anesthesia with propofol and remifentanyl or alfentanil: how important is the opioid? *J Clin Anesth.* 2002;14:275–8.
34. Thomson IR, Harding G, Hudson RJ. A comparison of fentanyl and sufentanil in patients undergoing coronary artery bypass graft surgery. *J Cardiothorac Vasc Anesth.* 2000;14:652–6.
35. Kaba A, Joris J. Anaesthesia for laparoscopic surgery. *Curr Anaesth Crit Care.* 2001;12:159–65.
36. Abu-Rafea B, Vilos GA, Ahmad R, et al. High-pressure laparoscopic entry does not adversely affect cardiopulmonary function in healthy women. *J Minim Invasive Gynecol.* 2005;12:475–9.
37. Hypólito O, Azevedo J, Caldeira FLA, et al. Creation of pneumoperitoneum: noninvasive monitoring of clinical effects of elevated intraperitoneal pressure for the insertion of the first trocar. *Surg Endosc.* 2010;24:1663–9.
38. Gándara V, Vega de DS, Escriú A, et al. Acid-base balance alterations in laparoscopic cholecystectomy. *Surg Endosc.* 1997;11:707–10.
39. Iwasaka H, Miyakawa H, Yamamoto H. Respiratory mechanics and arterial blood gases during and after laparoscopic cholecystectomy. *Can J Anaesth.* 1996;43:129–33.
40. Pearce DJ. Respiratory acidosis and subcutaneous emphysema during laparoscopic cholecystectomy. *Can J Anaesth.* 1994;41:314–6.
41. Ishizaki Y, Bandai Y, Shimomura K, et al. Changes in splanchnic blood flow and cardiovascular effects following peritoneal insufflation of carbon dioxide. *Surg Endosc.* 1993;7:420–3.
42. Lister DV, Rudston-Brown B, Wriner B. Carbon dioxide absorption is not linearly related to intraperitoneal carbon dioxide insufflation pressure in pigs. *Anesthesiology.* 1994;80:129–36.
43. Mullet CE, Viale JP, Sagnard PE. Pulmonary CO₂ elimination during surgical procedures using intra or extraperitoneal CO₂ insufflation. *Anesth Analg.* 1993;76:622–6.
44. Sefr R, Puszkailer K, Jagos F. Randomized trial of different intraabdominal pressure and acid-base balance alterations during laparoscopic cholecystectomy. *Surg Endosc.* 2003;17:947–50.