1.1 Article Title

Invasive monitoring of the clinical effects of high intra-abdominal pressure for insertion of the first trocar.

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1.7 Abbreviated Title

Intra-abdominal hypertension for trocar insertion

1.8 Brief Summary Statement

Hemodynamic, blood gas and metabolic parameters in brief hypertensive pneumoperitoneum during first trocar introduction was investigated. It causes variations in MAP, pH, HCO_3 and BE without adverse effects, and it may protect from iatrogenic injury.

Abstract

***Background*:** To analyze the effects of transitory, high intra-abdominal pressure on clinical, hemodynamic, blood gas and metabolic parameters.

***Methods*:** Sixty-seven laparoscopic patients were divided into groups P12 (n = 30, maximum intra-abdominal pressure of 12 mmHg) and P20 (n = 37, maximum intra-abdominal pressure of 20 mmHg). Through radial artery cannulation, mean arterial pressure (MAP) was assessed and blood gas analysis – pH, arterial oxygen tension (PaO_2), arterial carbon dioxide tension ($PaCO_2$), bicarbonate (HCO_3) and base excess (BE) – was performed. These parameters were evaluated in both groups at time point zero, before CO_2 insufflation; at time point one (TP1), when intra-abdominal pressure of 12 mmHg was reached in both groups; at time point two (TP2), 5 minutes after reaching intra-abdominal pressure of 12 mmHg in group P12 and of 20 mmHg in group P20; and at time point three (TP3), 10 minutes after reaching intra-abdominal pressure of 12 mmHg in group P12 and 10 minutes after TP1 in group P20, when intra-abdominal pressure decreased from 20 mmHg to 12 mmHg. Values out of the normal range or the occurrence of atypical phenomena suggestive of organic disease indicated clinical changes.

***Results*:** Significant variations in MAP, pH, HCO_3 and BE were observed in group P20; these changes, however, were within normal limits. Clinical changes were also within normal limits, and no pathological phenomena were observed.

***Conclusions*:** Brief, intra-abdominal hypertension for the insertion first trocar insertion causes variations in MAP, pH, HCO_3 and BE without adverse effects, and it may protect from iatrogenic injury.

Keywords: Pneumoperitoneum, Artificial; Laparoscopy; Surgical Instruments; Monitoring, Intraoperative.

Introduction

Less aggressive methods for gaining access to organs and structures located deeply in the abdomen decrease the impact of the metabolic response to trauma and are more beneficial for patients. This particularly applies to laparoscopy.[1-3] Because laparoscopic techniques are relatively recent, they still spark controversies. One of them concerns the best method for gaining access to the peritoneal cavity. Although there is no consensus regarding the best method for creation of pneumoperitoneum, Veress needle insertion is the most frequently used technique.[4,5] Complications during the blind insertion of the first trocar have also been reported.[5]

Approximately 50% of the complications arising from laparoscopy occur in the beginning of the procedure, i.e., during insertion of the Veress needle and first trocar. Laparoscopic procedures are therefore peculiar, in that surgical access is more dangerous than the surgical intervention itself. A recent literature review of the injuries caused by the Veress needle and the first trocar found a prevalence of 0.04% for gastrointestinal injuries and of 0.02% for vascular injuries among 357,257 patients.[6] Although relatively rare, such iatrogenic injuries have extremely serious consequences, including bleeding, peritonitis, multiple organ failure and death, with medicolegal implications.

Therefore, it is essential to search for alternative techniques that are safer than the most frequently used method, which consists of inserting a Veress needle into the abdomen through a midline incision at the umbilicus, insufflating the abdomen until intra-abdominal pressure reaches levels between 10 and 12 mmHg and blindly inserting the first trocar through the same site where the needle was previously inserted.[4,5]

A systematic literature review revealed that all injuries to the great vessels occurred when the Veress needle was inserted through a midline incision at the umbilicus.[6] The insertion of the Veress needle into the left hypochondrium, however, is safe and effective,[7] and potential injuries are less severe because they do not involve vital structures such as the retroperitoneal vessels.[6]

Nevertheless, the first trocar should be inserted through a midline incision at the umbilicus and not into the left hypochondrium, as recommended for the Veress needle.[7] This recommendation is based on the fact that the trocar is attached to a cannula through which the laparoscope will be inserted.[4,5] Better lighting and better images of intra-abdominal organs and structures are obtained when the laparoscope is positioned near the midline, at the umbilicus. In addition, better visualization of the during insertion of the remaining trocars is obtained when the laparoscope is in such position.

With regard to the risk posed by the midline insertion of the first trocar, it has been shown that extremely high intra-abdominal pressure for long enough to insert the first trocar can protect intra-abdominal structures from injury caused by blind insertion of the instrument, without clinical complications.[8,9] No vascular injury was reported in a study investigating 3,041 patients submitted to blind insertion of the first trocar through a midline incision at the umbilicus under intra-abdominal pressure of 25-30 mmHg.[10]

A study investigated the protective effect of elevated intra-abdominal pressure on the intra-abdominal structures at the moment of blind insertion of the first trocar.[11] The authors correlated different pressure levels and intra-abdominal volume with the distance between the anterior abdominal wall and intra-abdominal viscera, as well as with the force required to insert the first trocar into the abdominal cavity. They reported that under high pressure the intra-abdominal volume of gas and the distance between the anterior abdominal wall and intra-abdominal structures increased significantly, allowing the trocar to slide into the peritoneal cavity more easily. In addition, the authors reported that the abdominal wall became tenser, reducing elastic deformation caused by the force applied to the trocar.[11]

It is known, however, that extremely high levels of intra-abdominal pressure for longer periods of time can cause hemodynamic and structural changes, directly related to the tension levels caused by such high pressure.[12-18] High intra-abdominal pressure for longer periods of time

can lead to decreased cardiac output, decreased venous return, increased mean arterial pressure (MAP), increased systemic vascular resistance, altered renal perfusion and altered glomerular filtration rate, as well as ischemia-reperfusion injury of the intra-abdominal organs.[12-18] Therefore, most authors have proposed that intra-abdominal pressure remain at 12 mmHg (and never above 15 mmHg, which is considered a high level) during laparoscopic procedures.[4,19-25]

Although it is known that the aforementioned hemodynamic, metabolic and structural changes can occur when high levels of intra-abdominal pressure are used for longer periods of time, there is a lack of important information regarding the effects of transitory, high intra-abdominal pressure on blood gas and metabolic parameters, which means that an actually useful strategy to improve safety of first trocar insertion perhaps has been overlooked by laparoscopic surgeons.

The objective of the present study was to improve safety of first trocar insertion by evaluating the effects of transitory, high intraperitoneal pressure on clinical, hemodynamic, metabolic and blood gas parameters.

Materials and Methods

The present prospective randomized clinical trial was approved by the Research Ethics Committees of the *Universidade Federal de São Paulo* (UNIFESP, Federal University of São Paulo), São Paulo, Brazil, and of the *Unversidade de Taubaté* (UNITAU, University of Taubaté), Taubaté, Brazil, under research protocol nos. 1219/07 and 007/2007, respectively. All participating patients gave written informed consent. The study was carried out at Dr. José de Carvalho Florence Municipal Hospital, São José dos Campos, Brazil.

Sixty-seven patients scheduled to undergo elective laparoscopic surgery between October of 2007 and May of 2008 were included in the present study. The patients, aged 20-79 years, with

body mass index (BMI) < 35, were classified as P1 or P2 according to the American Society of Anesthesiology Physical Status Classification. They had no history of peritonitis or abdominal surgery in organs located in the supramesocolic compartment.

The patients were randomly divided into two groups. Randomization was achieved by dice toss: odd numbers were assigned to group P12 (n = 30), which comprised patients submitted to a maximum intra-abdominal pressure of 12 mmHg; even numbers were assigned to group P20 (n = 37), which comprised patients submitted to a maximum intra-abdominal pressure of 20 mmHg. Group P12 comprised 25 females and 5 males, aged between 22 and 72 years (mean age of 47.2 ± 14.5 years), with BMI between 20.2 and 33.4 kg/m^2 (mean BMI of 26.3 ± 4.0 kg/m^2). Group P20 comprised 30 females and 7 males, aged between 20 and 79 years (mean age of 46.5 ± 15.0 years), with BMI between 17.5 and 34.6 kg/m^2 (mean BMI of 26.2 ± 3.8 kg/m^2). No statistically significant differences were observed between the groups when the demographic data were compared ($p \leq 0.05$).

Preanesthesia evaluation was carried out at the outpatient clinic of the hospital, one day before the laparoscopic procedure was performed. None of the patients received preanesthetic medication.

Before anesthesia, modified Allen's test[26] was performed. All patients received lactated Ringer's solution for hydration, administered intravenously using an 18-gauge catheter. During anesthesia, besides invasive monitoring, noninvasive monitoring cardioscopy, pulse oximetry, arterial blood pressure monitoring, capnography and intratracheal pressure monitoring were accomplished.

Anesthesia was induced with 0.5 µg/kg of sufentanil, 0.6 mg/kg of rocuronium and 2.0 mg/kg of propofol. Thereafter, general anesthesia was maintained with sevoflurane in a mixture of oxygen and compressed air. All patients were submitted to mechanical ventilation using a time-cycled ventilator. The following equipment was used for anesthesia and monitoring: a PC 2700 Shogun

Ergo anesthesia machine (K. Takaoka Indústria e Comércio Ltda., São Paulo, São Paulo, Brazil); a Fabius GS anesthesia machine (Dräger Medical, Lübeck, Germany) and DX 2010 monitors (Dixtal Biomédica Indústria e Comércio Ltda., São Paulo, São Paulo, Brazil). The ventilator was set to a fraction of inspired oxygen of 60%, a positive end-expiratory pressure of 4 cmH_2O, a tidal volume of 7 ml/kg, a respiratory rate of 15 breaths per minute and an inspiration/expiration ratio of 1:2.

After anesthesia (and provided that the modified Allen's test[26] was negative), radial artery cannulation was performed in the nondominant arm. If cannulation failed after a maximum of three attempts, the patient was excluded from the present study.

A total of six patients were excluded from the present study, for the following reasons: bronchospasm after induction of anesthesia was observed in one patient; orotracheal intubation was difficult in one patient, who required other procedures that were not part of the original study design; radial artery cannulation failed after three attempts in two patients; and the blood samples from two patients had to be discarded due to blood clot formation.

Pneumoperitoneum was created using the closed technique: a Veress needle was inserted into the peritoneal cavity and carbon dioxide (CO_2) was insufflated into the peritoneal cavity at a flow rate of 1 l/min.

During the procedure, MAP was assessed, and arterial blood gas analysis – pH, arterial oxygen tension (PaO_2), arterial CO_2 tension ($PaCO_2$), bicarbonate (HCO_3), and base excess (BE) – was performed (Rapidlab™ 348; Bayer HealthCare LLC, Tarrytown, New York, U.S.A.). These clinical parameters were evaluated at four time points, as follows: time point zero (TP0), before CO_2 insufflation; time point one (TP1), when an intra-abdominal pressure of 12 mmHg was reached in groups P12 and P20; time point two (TP2), 5 min after reaching the intra-abdominal pressure of 12 mmHg in group P12 and of 20 mmHg in group P20; and time point three (TP3), 10 min after

reaching the intra-abdominal pressure of 12 mmHg in group P12 and 10 min after TP1 in group P20, when the intra-abdominal pressure decreased from 20 mmHg to 12 mmHg.

During the anesthetic and surgical procedures, the following parameters were also monitored: heart rate; cardiac rhythm; pulse oximetry; end-tidal carbon dioxide; MAP; and intratracheal pressure. In the postanesthesia recovery room, the following parameters were evaluated: heart rate; cardiac rhythm; MAP; pulse oximetry; level of consciousness and muscle activity. These parameters were monitored until patients were discharged to the infirmary.

Values out of the normal range, or the occurrence of atypical phenomena suggestive of organic disease, indicated clinical changes. The following values were considered normal: MAP between 70 and 120 mmHg; pH between 7.35 and 7.45; $PaCO_2$ between 30 and 45 mmHg; PaO_2 above 80 mmHg; BE between -2 and +2 mmol/l; and HCO_3 between 22 and 26 mmol/l.

Statistical analysis

The descriptive analysis of the data included measures of position for continuous variables and of frequency for categorical variables. The chi-square test was used to compare gender between the two groups, whereas the nonparametric Mann-Whitney test was used to compare age and BMI between the two groups. Repeated measures analysis of variance on ranks was used to compare the measurements of each variable at the different time points. The significance level was set at 5% (*P* values ≤ 0.05).

Results

Mean arterial pressure

Means and standard deviations for MAP in group P12 were as follows: 68.57 ± 10.18 mmHg; 88.10 ± 17.68 mmHg; 90.10 ± 19.03 mmHg; and 99.07 ± 18.58 mmHg at TP0, TP1, TP2 and TP3, respectively. Significant differences were observed between MAP at TP0 and MAP at all the remaining time points; between MAP at TP1 and MAP at TP3; and between MAP at TP2 and MAP at TP3 (P = 0.0000; fig. 1).

Means and standard deviations for MAP in group P20 were as follows: 70.57 ± 14.58 mmHg; 83.57 ± 12.86 mmHg; 89.30 ± 15.33 mmHg; and 92.43 ± 14.42 mmHg at TP0, TP1, TP2 and TP3, respectively. Significant differences were observed between MAP at TP0 and MAP at all the remaining time points; between MAP at TP1 and MAP at TP2; and between MAP at TP1 and MAP at TP3 (P = 0.0000; fig. 1).

pH

Means and standard deviations for pH in group P12 were as follows: 7.47 ± 0.05; 7.47 ± 0.06; 7.46 ± 0.06; and 7.44 ± 0.06 at TP0, TP1, TP2 and TP3, respectively. Significant differences were observed between pH at TP0 and pH at TP3; between pH at TP1 and pH at TP2; between pH at TP1 and pH at TP3; and between pH at TP2 and pH at TP3 (P = 0.0000; fig. 2).

Means and standard deviations for pH in group P20 were as follows: 7.48 ± 0.06; 7.48 ± 0.06; 7.46 ± 0.06 and 7.45 ± 0.07 at TP0, TP1, TP2 and TP3, respectively. Significant differences were observed between pH at TP0 and pH at TP2; between pH at TP0 and pH at TP3; between pH at TP1 and pH at TP2; and between pH at TP1 and pH at TP3 (P = 0.0000; fig. 2).

Arterial oxygen tension

Means and standard deviations for PaO_2 in group P12 were as follows: 216.80 ± 51.60 mmHg; 192.15 ± 52.73 mmHg; 191.88 ± 51.74 mmHg; and 196.77 ± 46.66 mmHg at TP0, TP1, TP2 and

TP3, respectively. Significant differences were observed between PaO_2 at TP0 and PaO_2 at TP1 (*P* = 0.0057; fig. 3).

Means and standard deviations for PaO_2 in group P20 were as follows: 212.07 ± 72.37 mmHg; 197.73 ± 52.74 mmHg; 202.35 ± 52.46 mmHg; and 203.41 ± 49.20 mmHg at TP0, TP1, TP2 and TP3, respectively. No significant differences in PaO_2 were observed between any of the time points (*P* = 0.4239; fig. 3).

Arterial carbon dioxide tension

Means and standard deviations for $PaCO_2$ in group P12 were as follows: 31.96 ± 5.20 mmHg; 31.48 ± 6.67 mmHg; 32.68 ± 6.82 mmHg; and 32.63 ± 8.30 mmHg at TP0, TP1, TP2 and TP3, respectively. No significant differences in $PaCO_2$ were observed between any of the time points (*P* = 0.3557; fig. 4).

Means and standard deviations for $PaCO_2$ in group P20 were as follows: 32.47 ± 5.36 mmHg; 32.43 ± 4.84 mmHg; 33.19 ± 5.08 mmHg; and 34.09 ± 6.20 mmHg at TP0, TP1, TP2 and TP3, respectively. No significant differences in $PaCO_2$ were observed between any of the time points (*P* = 0.0887; fig. 4).

Bicarbonate

Means and standard deviations for HCO_3 in group P12 were as follows: 22.85 ± 3.11 mmol/l; 22.50 ± 3.85 mmol/l; 22.42 ± 3.34 mmol/l; and 21.96 ± 4.38 mmol/l at TP0, TP1, TP2 and TP3, respectively. No significant differences in HCO_3 were observed between any of the time points (*P* = 0.3629; fig. 5).

Means and standard deviations for HCO_3 in group P20 were as follows: 23.75 ± 3.45 mmol/l; 23.48 ± 2.64 mmol/l; 23.06 ± 3.04 mmol/l; and 23.20 ± 3.17 mmol/l at TP0, TP1, TP2 and TP3, respectively. Significant differences were observed between HCO_3 at TP0 and HCO_3 at TP2 (*P* = 0.0126; fig. 5).

Base excess

Means and standard deviations for BE in group P12 were as follows: 0.15 ± 3.00 mmol/l; -0.08 ± 3.55 mmol/l; -0.53 ± 3.14 mmol/l; and -1.27 ± 3.92 mmol/l at TP0, TP1, TP2 and TP3, respectively. Significant differences were observed between BE at TP0 and BE at TP3; between BE at TP1 and BE at TP2; and between BE at TP1 and BE at TP3 (P = 0.0001; fig. 6).

Means and standard deviations for BE in group P20 were as follows: 1.10 ± 3.27 mmol/l; 0.82 ± 2.74 mmol/l; 0.05 ± 3.22 mmol/l; and -0.03 ± 3.12 mmol/l at TP0, TP1, TP2 and TP3, respectively. Significant differences were observed between BE at TP0 and BE at TP2; between BE at TP0 and BE at TP3; between BE at TP1 and BE at TP2; and between BE at TP1 and BE at TP3 (P = 0.0000; fig. 6).

Neither during the surgical procedure nor during the postanesthesia recovery period did the values obtained for the various parameters evaluated exceed those considered normal for healthy populations.

Discussion

The present study analyzed clinical changes and gas exchange during laparoscopic procedures in which transitory, high intra-abdominal pressure was used for long enough to insert the first trocar.

Patients were randomly divided into groups P12, including patients submitted to a maximum intra-abdominal pressure of 12 mmHg, and P20, including patients submitted to a maximum intra-abdominal pressure of 20 mmHg.

Group P12 was the positive control group, in which clinical changes during the laparoscopic procedure employing standard intra-abdominal pressure (12 mmHg) were investigated. The purpose of assessing the clinical parameters in group P12 was to verify whether the clinical

changes that occurred in group P20 could be attributed to the duration of pneumoperitoneum. Therefore, if changes occurred from one time point to another in group P20 but not in group P12, they were safely attributed to the intra-abdominal pressure of 20 mmHg and not to the duration of pneumoperitoneum, which was equal in the two groups.

Group P20 was the experimental group but also a control group with which it was compared, since the patients in this group were submitted to different levels of intra-abdominal pressure, from 0 to 20 mmHg.

Propofol, rocuronium, sufentanil and sevoflurane were used in the present study because these anesthetics maintain cardiopulmonary parameters stable; they reach the airways rapidly and decrease the incidence of nausea, vomiting and pain in the postoperative period.[27-31]

Initial ventilatory parameters were constant flow, a fraction of inspired oxygen of 60%, a positive end-expiratory pressure of 4 cmH_2O, a tidal volume of 7 ml/kg, a respiratory rate of 15 breaths per minute, an inhalation/exhalation ratio of 1:2 and a volume/minute ratio that compensated for the increased intra-abdominal pressure caused by CO_2.[32]

A study conducted by Abu-Rafea et al.[33] reported no cardiopulmonary complications among 100 healthy women submitted to high intra-abdominal pressure (ranging from 10 to 30 mmHg) for long enough to insert the first trocar. The authors analyzed the volume of CO_2 effectively insufflated into the peritoneal cavity, heart rate, arterial oxygen saturation, MAP and lung compliance, and observed statistically significant changes in MAP and lung compliance; such changes were not, however, clinically significant. Nevertheless, Abu-Rafea et al.[33] did not establish parameters to evaluate changes in respiratory function and gas exchange. In addition, the effect of each preset pressure level (10, 15, 20, 25 and 30 mmHg) was evaluated at the exact moment it was reached, without taking into consideration the cumulative effect of duration of pneumoperitoneum for insertion of the first trocar, which hindered the assessment of the clinical effects resulting from duration of pneumoperitoneum rather than from the level of intra-abdominal pressure reached.

Furthermore, cardiovascular parameters were monitored using noninvasive methods, and arterial blood gas analysis was not performed.

In the present study, a significant increase in MAP was observed in the two groups at all time points analyzed. The increase in MAP in group P12 suggested that this clinical change was caused by the pneumoperitoneum itself, regardless of the intra-abdominal pressure. Even at low pressure levels, e.g., 12 mmHg, vasoconstriction occurs, increasing arterial pressure. However, such increase did not affect the patients clinically (fig.1), and none of the patients presented arterial hypertension.

Laparoscopic procedures with creation of CO_2 pneumoperitoneum have been linked to a risk of hypercapnia due to increased intra-abdominal pressure and CO_2 absorption by the peritoneum, which can lead to respiratory acidosis.[34-36] In the present study, no significant changes in $PaCO_2$ values were observed in the two groups. As ventilatory parameters did not change during the study, we can infer that CO_2 absorption by the peritoneum does not increase as a result of increased intra-abdominal pressure (i.e., from 12 to 20 mmHg for 5 min) at constant pulmonary ventilation. This might be due to the fact that increased intra-abdominal pressure compresses the peritoneal capillaries, limiting CO_2 absorption[37-39] and decreasing splanchnic blood flow.

In the present study, patients initially developed mild respiratory alkalosis as a result of the ventilatory parameters established for the procedure. As ventilatory parameters did not change and $PaCO_2$ values did not change significantly, the significant decrease in pH values (immediately after initial respiratory alkalosis) might have been due to the slightly elevated $PaCO_2$ values and to the metabolic acidosis resulting from the decreased perfusion of the intra-abdominal organs. It was observed that pH decreased in a more pronounced manner at an intra-abdominal pressure of 20 mmHg than at an intra-abdominal pressure of 12 mmHg. This corroborates the pathophysiological explanation that, in the present study, the decreased perfusion of intra-abdominal structures played a key role in changing pH values, since the other acidosis-related factor, i.e., CO_2 absorption, was

similar in groups P20 and P12, as evidenced by the $PaCO_2$ values obtained through blood gas analysis (fig. 4). Some authors[40] have reported that, at an intra-abdominal pressure of 15 mmHg, pH increases during the first 30 min and subsequently decreases. A similar finding was observed in the present study, both at higher and at lower levels of intra-abdominal pressure (20 and 12 mmHg, respectively). In any case, the changes observed in the present study were not clinically significant (fig. 2).

A statistically significant reduction in HCO_3 was observed in group P20 in TP2 concerning TP0. At lower levels of intra-abdominal pressure, however, no significant reduction in HCO_3 was observed in that group (P12). It means that the 20mmHg pressure itself was responsible for the reduction in HCO_3. It is know that changes in pH is due to alterations of PaCO2 and/or changes in HCO3. In the present research, the fact that pH decreased with statistical significance at an intra-abdominal pressure of 20 mmHg without a significant increase in $PaCO_2$ suggests that there was a greater consumption of HCO_3 in order to attenuate the metabolic acidosis caused by the decrease in blood supply to the splanchnic organs under hypertensive regime. In the study conducted by Sefr *et al*.,[40] HCO_3 production at 10 mmHg was not different from HCO_3 production at 15 mmHg. In the present study, HCO_3 production decreased significantly at an intra-abdominal pressure of 20 mmHg. This change was not, however, clinically relevant – it was under normal limits (fig. 5).

A statistically significant reduction along the time in BE occurred in the two groups. The changes observed were related to the duration of pneumoperitoneum. Such changes manifested earlier at an intra-abdominal pressure of 20mmHg (P20). The association between decreased values in BE values at an intra-abdominal pressure of 20 mmHg with decreased pH, decreased HCO_3 and no significant changes in $PaCO_2$, might have occurred to compensate for the momentary insufficient blood supply to the splanchnic organs. Sefr *et al*.[40] reported decreased BE values at an intra-abdominal pressure of 10 mmHg, as well as increased BE values at an intra-abdominal pressure of 15-mmHg. In the present study, a decrease in BE was observed at intra-

abdominal pressures of 12 and 20 mmHg. Such changes were not, however, clinically significant (fig. 6).

Transitory (5 min), high (20 mmHg) intra-abdominal pressure for the insertion of the first trocar caused changes in MAP, pH, HCO_3 and BE without adverse clinical effects. Therefore, the use of transitory, high intra-abdominal pressure is recommended for the prevention of iatrogenic injury while inserting the first trocar.

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Legends to figures

Fig. 1. Average mean arterial pressure in groups P12 and P20 at TP0, TP1, TP2 and TP3.

Fig. 2. Mean pH in groups P12 and P20 at TP0, TP1, TP2 and TP3.

Fig. 3. Mean arterial oxygen tension in groups P12 and P20 at TP0, TP1, TP2 and TP3.

Fig. 4. Mean arterial carbon dioxide tension in groups P12 and P20 at TP0, TP1, TP2 and TP3.

Fig. 5. Mean bicarbonate in groups P12 and P20 at TP0, TP1, TP2 and TP3.

Fig. 6. Mean base excess in groups P12 and P20 at TP0, TP1, TP2 and TP3.

Figures

Figure 1

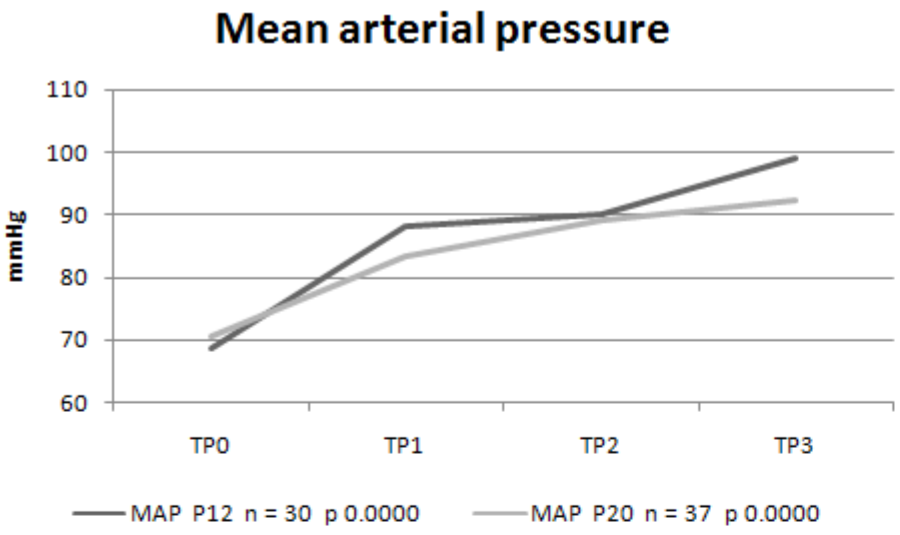

Figure 2

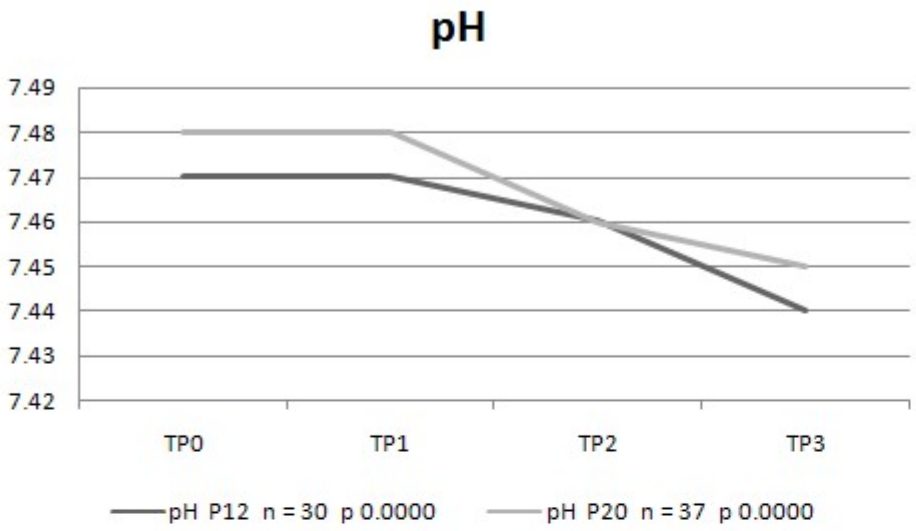

Figure 3

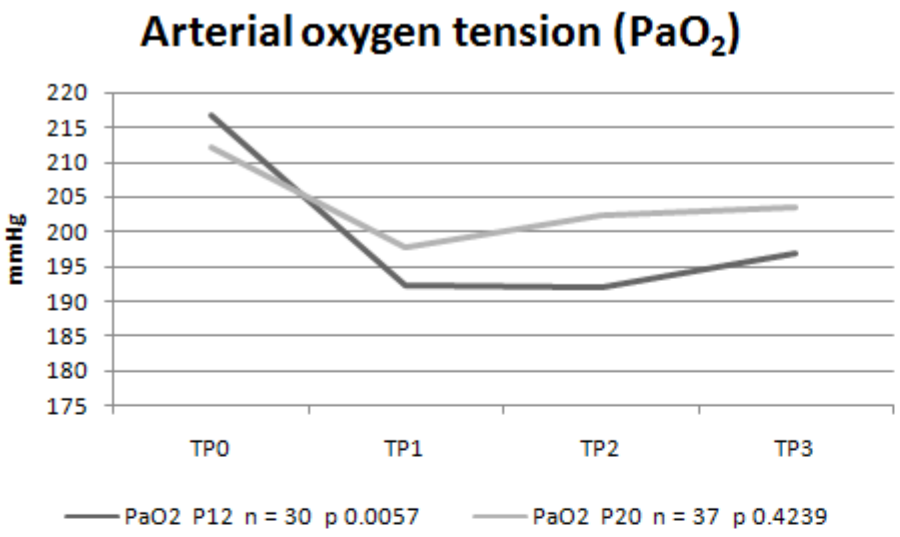

Figure 4

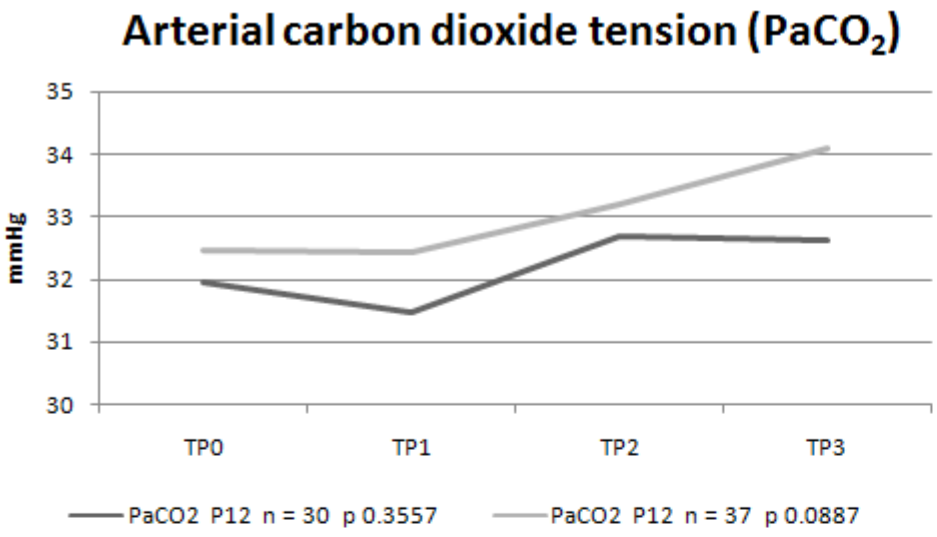

Figure 5

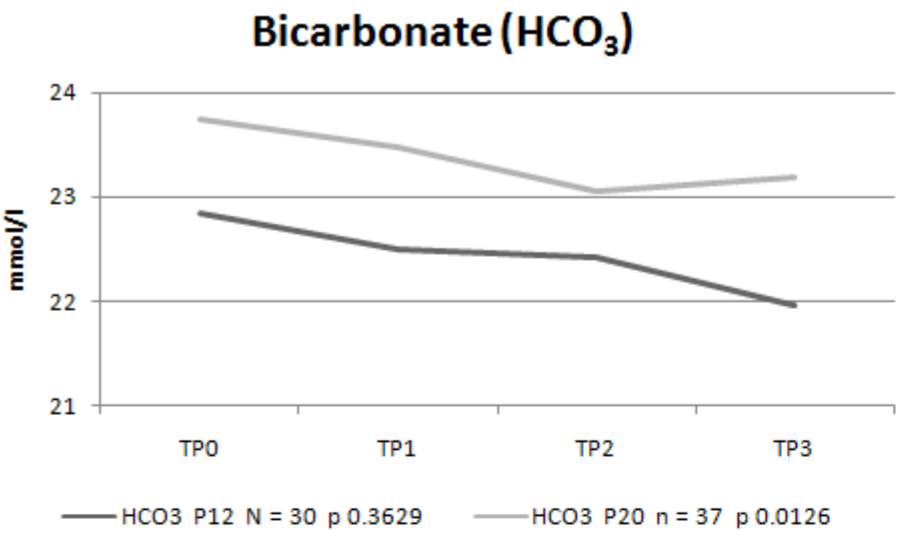

Figure 6