ORIGINAL ARTICLE

The effect of laparoscopic pneumoperitoneum on patient's respiratory variation of inferior vena cava and stroke volume index: A randomized controlled study

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Background: The establishment of pneumoperitoneum has impacts on patient's cardiovascular function. In this study, the respiratory variation of inferior vena cava (IVC), stroke volume (SV) index, and other parameters was monitored to determine how the pneumoperitoneum affects the patient's hemodynamic and acknowledge how to resolve it. **Materials and Methods:** Fifty-five patients were randomly divided into Group A (8 mmHg), Group B (10 mmHg), Group C (12 mmHg), Group D (14 mmHg), and Group E (12 mmHg, infusion with 7 mL/kg crystalloid solution). The parameters including IVC variability, SV index, mean artery pressure (MAP), heart rate (HR), cardiac output index (CI), and airway pressure were measured, to compare the changes before and after pneumoperitoneum. **Results:** Among Group A, Group B, Group C, and Group D, there were no significant differences in patients' prepneumoperitoneum information. Compare with prepneumoperitoneum, the difference in IVC variability, SV index, MAP, HR, CI, and airway pressure was statistically significant ($P < 0.05$). After the establishment of pneumoperitoneum, the difference in IVC variability, SV index, and airway pressure among Group A, Group B, Group C, and Group D was statistically significant ($P < 0.05$). Compare with Group C, the change rates of IVC variability, SV index, MAP, HR, and CI were smaller in Group E ($P < 0.05$). **Conclusion:** The establishment of pneumoperitoneum could increase the patient's IVC variability and reduce SV index, and with the increase of pressure, IVC variability had an increasing trend, SV index had a decreasing trend. Fluid transfusion could reduce the relative influence of the pneumoperitoneum.

Key words: Airway pressure, cardiac output, echocardiography, heart rate, hemodynamics, inferior vena cava, laparoscopic, pneumoperitoneum, stroke volumes, transesophageal

How to cite this article: Liu D, Sun J, Chen K, Yao Y, Zhang F. The effect of laparoscopic pneumoperitoneum on patient's respiratory variation of inferior vena cava and stroke volume index: A randomized controlled study. J Res Med Sci 2024;29:46.

INTRODUCTION

Since laparoscopic surgery entered the mainstream of general surgical practice,[1] laparoscopic technique presents less surgical trauma, good visual field, and rapid return to normal activities of patients leading to the rapid adoption by general surgical community.[2,3] Due to advances in technique and the development of new laparoscopic instruments, the indications have been extended to many critically ill patients.[4] Laparoscopic technique brings many advantages, but also presents unwanted effects on patient's physiological status, healthy patients can achieve a new balance through self-regulation after a few minutes, but for some patients with comorbidities, even slight fluctuations in vital signs will induce adverse events.[5] Some studies demonstrated changes in myocardial pump function and performance

Access this article online

Quick Response Code:

Website: https://journals.lww.com/jrms

DOI: 10.4103/jrms.jrms_526_22

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Submitted: 22-Jul-2022; **Revised:** 13-Jun-2023; **Accepted:** 28-Jul-2023; **Published:** 30-Jul-2024

of the entire cardiovascular system after pneumoperitoneum due to increased intra-abdominal pressure,[6,7] knowledge of their principle, and how to prevent it from happening would reduce perioperative mortality and morbidity. The patient's cardiovascular system is determined by the preload, myocardial contractility, afterload, and heart rate (HR), influencing any of these aspects will affect patient's vital signs performed as changes in blood pressure and HR.[8] Intraoperative transesophageal echocardiography proposed as a first-line tool for identifying the mechanisms of cardiovascular function change.[9] Respiratory variation of the inferior vena cava (IVC) is a parameter of the patient's volume responsiveness, which indicates whether we can increase cardiac output (CO) through fluid transfusion and is an index of monitoring the patient's preload.[10,11] The stroke volume (SV) measured by calculating the anterior blood flow of the left ventricular outflow tract (LVOT) is an appropriate parameter for monitoring the contractility of the patient's myocardium.[12] These parameters' variation can directly reflect the mechanism of how the pneumoperitoneum affects cardiovascular function and helps us to determine whether our treatment can effectively improve the impact of pneumoperitoneum on vital signs. Therefore, the objectives of this study were to assess the patients' respiratory variation of IVC and SV index before and after the establishment of the pneumoperitoneum in patients through transesophageal echocardiography and evaluate the effects of pneumoperitoneum on the patients' cardiovascular function.

METHODS

The protocol of this prospective randomized controlled trial is approved by Yongchuan Hospital of Chongqing Medical University (Dated: January 13, 2022) and registered in the Chinese Clinical Trial Registry (registration No: ChiCTR2200055155). Inclusion criteria were patients with age 18–45 years old and those who underwent laparoscopic surgery, including laparoscopic cholecystectomy, laparoscopic appendectomy, the American Society of Anesthesiologists (ASAs) Grade <II, and regardless of gender. Exclusion criteria were patients allergic to coupling agents and who have severe respiratory and circulatory diseases include uncontrolled hypertension, diabetes, heart failure, chronic obstructive pulmonary disease, gastric-esophageal varices, hematological diseases with coagulation disorders, and who cannot cooperate with postoperative follow-up. Fifty-five eligible patients were enrolled in our study between January 2022 and June 2022 with informed consent of the patients and their families. Using envelope method in random grouping, the prepared random grouping scheme was put into the envelope, and the envelopes were opened successively according to the inclusion order. The inclusion patients were determined which group will be included according to the allocation plan in the envelope. Our study was divided into five groups according to different pneumoperitoneum pressure 8 mmHg group (Group A), 10 mmHg group (Group B), 12 mmHg group (Group C), 14 mmHg group (Group D), and a treatment group infusion of 7 mL/kg crystalloid solution before anesthesia (12 mmHg, Group E)[13] [Figure 1].

Anesthesia method

All patients undergo routine preoperative preparation, fasting for 6 h, water deprivation for 2 h.[14] The strategy of anesthesia was as follows: open intravenous access and infusion crystalloid solution, monitoring electrocardiogram, pulse oxygen saturation, noninvasive blood pressure, and radial artery catheterization were performed under ultrasound guidance after local anesthesia to monitor invasive artery blood pressure. Anesthesia induction was performed in Group E after infusion of 7 mL/kg crystalloid solution,[15] and all groups' anesthesia was induced by midazolam 0.05 mg/kg, propofol 2 mg/kg, sufentanil citrate 0.4 mg/kg, rocuronium bromide 6 mg/kg, and endotracheal intubation were performed after 3-min pure oxygen mask ventilation. The ventilation parameters of the anesthesia machine were set about tidal volume 6–8 mg/kg, respiratory rate 12 times, and inspiratory/expiratory ratio 1:2. Continuous pumping of propofol, remifentanil, and inhalation of sevoflurane to maintain anesthesia, intermittent application of rocuronium bromide to maintain muscle relaxation, keep the Bispectral index (BIS) between 40 and 60.

Transesophageal echocardiography

After endotracheal intubation, the transesophageal ultrasound probe (13.0–4.0MHz, SonoScape) was placed through the mouth. After the patient's vital signs stabilized for about 5 min after intubation, the ultrasound sections were obtained by transesophageal ultrasound. The probe was advanced to obtain transgastric (TG) basal short axis (SAX) section at 0° (TG basal SAX), rotate the probe to the right to find the liver and back to find the IVC entering the right atrium (RA). Adjust the probe and angle to identify the hepatic vein entering the IVC to obtain the TG IVC long-axis section. M-mode ultrasound was used to measure the diameter of the IVC at the end of inspiration and expiration at 0.5–1 cm distal to the opening of the hepatic vein into the IVC or 2–3 cm distal to the opening of the RA[16] [Figure 2]. After the variation of IVC was measured, adjust the probe to the TG SAX section, and increase the probe angle to 120°–140° to obtain the TG long axis section (TG LAX), the LVOT and aortic valve were shown, the flow velocity of LVOT was measured by spectral Doppler pulsed wave in this section, and tracing velocity time integral (VTI), than draw back the probe to get the midesophageal LAX section (LAX section) and the inner diameter of LVOT was measured at this section[17] [Figure 3]. After the pneumoperitoneum

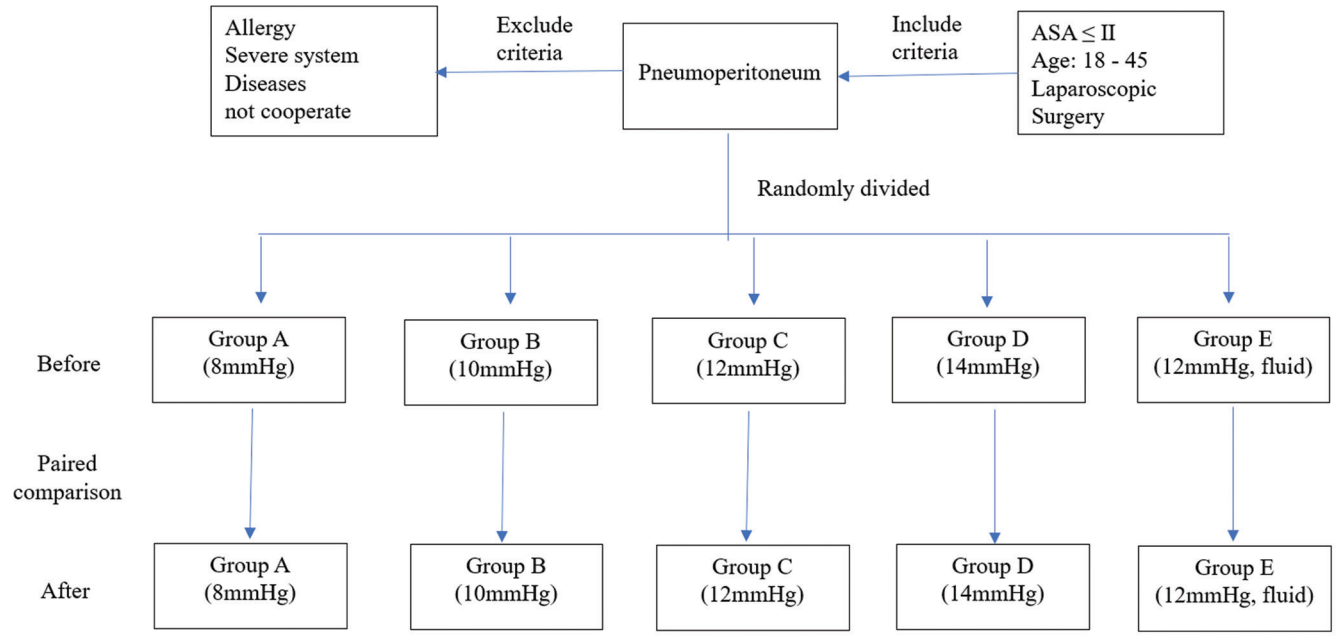

Figure 1: Grouping and handling

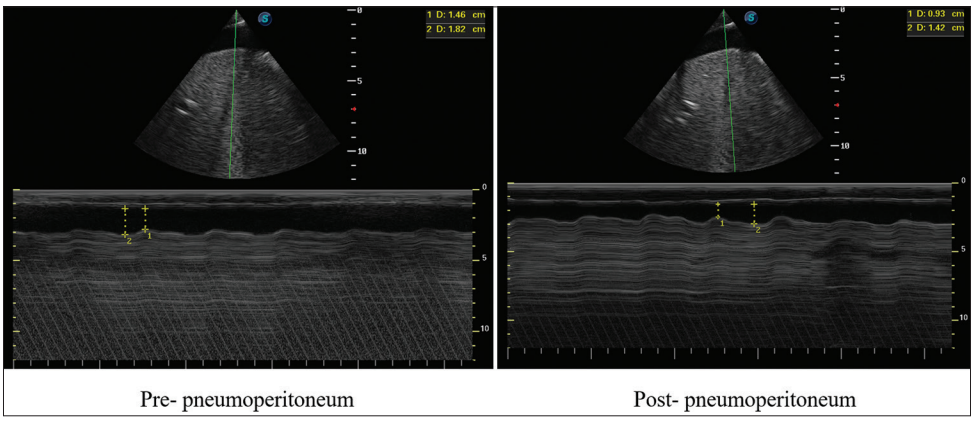

Figure 2: Inferior vena cava variability measurement

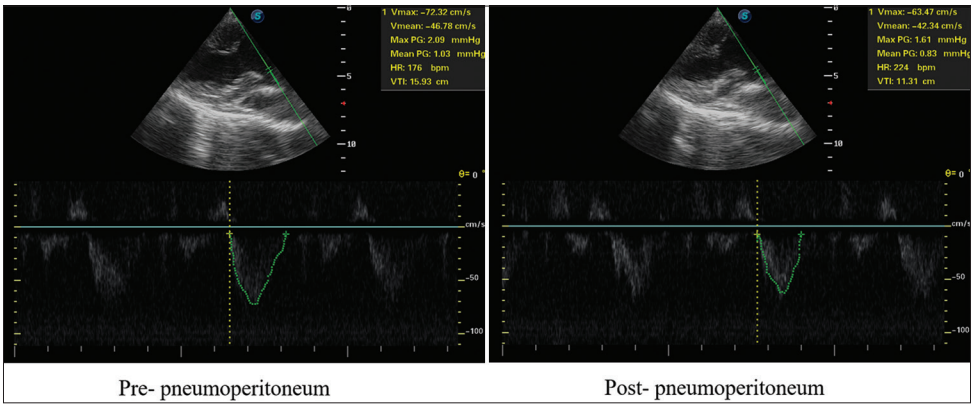

Figure 3: Left ventricular outflow track velocity time integral measurement

was established, the data were collected again by the above methods which include the inner diameter of the IVC at the end of inspiration and expiration, and the VTI of LVOT.

Primary indicators

IVC variability is calculated as the end-inspiratory inner diameter – the end-expiratory inner diameter)/

end-expiratory inner diameter.[15] SV index is calculated as SV/body surface area (BSA). SV = VTI × cross-sectional area (CSA), VTI is the tracing area of blood flow at LVOT and CSA is the CSA at LVOT equals $0.785 \times d^2$,[17] and BSA using the computer calculation.

Secondary indicators

The secondary indicators include prepneumoperitoneum HR, postpneumoperitoneum HR, prepneumoperitoneum cardiac output index (CI), postpneumoperitoneum CI, prepneumoperitoneum mean artery pressure (MAP), postpneumoperitoneum MAP, prepneumoperitoneum airway pressure, and postpneumoperitoneum airway pressure.

Statistical analysis

Statistical analyses were done using SPSS version 26 (SPSS Inc, Chicago, USA), figure was drawn by GraphPad Prism 5, bilateral test was used for testing, and α was set to 0.05. For descriptive data including primary and secondary indicators before and after pneumoperitoneum conforming to the normal distribution were presented as mean ± standard, the paired *t*-test was used for comparison. Independent *t*-test was used to compare descriptive data between Group C and Group E. One-way ANOVA was used for age and BSA comparison between groups. The trend of the studied variables among groups was nonnormal distribution parameters assessed by Kruskal–Wallis *H*-test, and multiple comparisons were used within groups. The continuous variable ASA grading was compared using Kruskal–Wallis *H*-test. The categorical variable gender was compared by Chi-squared test, and the Fisher exact probability method was used for those that did not meet the requirements of the Chi-squared test.

RESULTS

A total of 55 patients were included in this study, 11 patients in each group. There were no statistical differences in the ratio of male to female, age, ASA classification, and BSA among each group [Table 1].

After pneumoperitoneum was established, compared with prepneumoperitoneum, the differences were statistically significant in Group A, Group B, Group D, and Group E with SV index decreased, IVC variability increased, MAP increased, HR increased, CI increased, and airway pressure increased. In Group C, compared with prepneumoperitoneum, the differences were statistically significant with SV index decreased, IVC variability increased, MAP increased, HR increased, and airway pressure increased, and there was no significant difference in CI [Table 2].

Compare the rates of change of the parameters by adjusting post to pre among Group A, Group B, Group C, and Group D, the difference in IVC variability among each group was statistically significant, compared with Group A, IVC variability was higher in Group C and Group D. The difference in SV index among each group was statistically significant, compared with Group A, SV index was lower in Group C and Group D; compared with Group B, SV index was lower in Group C and Group D. The difference in airway pressure among each group was statistically significant, compared with Group A, airway pressure was higher in Group C and Group D; compared with Group B, airway pressure was higher in Group D [Figure 4].

Pneumoperitoneum pressure in Group C and Group E was the same, the data analysis between them can specify whether our treatment can effectively improve the impact of pneumoperitoneum on vital signs. Comparing the rates of change of the parameters between Group C and Group E, IVC variability, SV index, MAP, HR, and CI were all smaller in Group E than in Group C, and the differences were statistically significant [Table 3].

DISCUSSION

Our results demonstrate that the establishment of pneumoperitoneum can increase the IVC variability and reduce the patient's SV index during mechanical ventilation and increase the HR, CI, MAP, and airway pressure. With the increase of pneumoperitoneum pressure, there is an increasing trend in IVC variability and airway pressure and a downward trend in SV index. Fluid transfusion can reduce the relative influence of the pneumoperitoneum on each parameter and make the circulation more stable.

Blood pressure is one of the most important vital signs of patients. The main factors which affect blood pressure are

Table 1: Patients characteristics and clinical data

Basic information	Group A	Group B	Group C	Group D	Group E	*P*
Total	11	11	11	11	11	
Male/female	4/7	5/6	4/7	5/6	4/7	1.000
Age (year)	39.18±3.401	37.64±3.529	39.27±4.747	39.64±2.111	40.55±0.846	0.551
ASA (I/II)	6/5	6/5	7/4	7/4	8/3	0.901
BSA	1.682±0.143	1.674±0.107	1.687±0.132	1.694±0.138	1.677±0.033	0.996

Data are shown as mean±SD or as an actual number. SD=Standard deviation; ASA=American Society of Anesthesiologists; BSA=Body surface area

Table 2: Comparison of pre and postpneumoperitoneum index

	IVC variability (%)	SV index (mL/m²)	MAP (mmHg)	HR (bpm)	CI (L/min/m²)	Airway pressure (mmHg)
Group A pre	14.27±2.240	42.753±1.548	83.45±3.142	69.36±3.443	2.973±0.189	16.27±1.272
Group A post	23.18±4.020	37.081±1.063	96.18±2.272	86.18±4.262	3.256±0.24	21.73±1.489
T	−13.096	18.584	−15.712	−11.164	−4.986	−26.312
P	0.000*	0.000*	0.000*	0.000*	0.001*	0.000*
Group B pre	13.82±1.834	42.753±1.548	84.45±2.945	71.27±3.875	3.045±0.172	18.82±1.250
Group B post	24.55±2.464	37.081±1.063	95.18±3.459	88.64±5.446	3.286±0.212	22.73±1.737
T	−11.847	18.584	−14.503	−14.942	−4.358	−11.947
P	0.000*	0.000*	0.000*	0.000*	0.001*	0.000*
Group C pre	13.55±1.753	43.241±1.623	84.55±2.945	71.09±5.029	3.136±0.287	16.55±1.036
Group C post	26.09±2.119	35.444±2.319	95.45±3.078	90.55±3.588	3.211±0.269	23.82±1.328
T	−23.000	19.287	−14.201	−13.551	−0.664	−26.667
P	0.000*	0.000*	0.000*	0.000*	0.534	0.000*
Group D pre	14.09±1.700	43.515±1.481	84.64±2.767	70.09±4.571	3.051±0.239	16.45±1.572
Group D post	26.55±2.544	34.468±1.539	94.00±2.280	91.36±5.608	3.15±0.244	25.45±1.214
T	−12.644	26.844	−13.060	−24.317	−2.381	−16.188
P	0.000*	0.000*	0.000*	0.000*	0.039*	0.000*
Group E pre	12.64±1.433	45.881±1.66	83.36±1.912	68.91±3.618	3.157±0.176	16.45±1.036
Group E post	17.91±1.3	43.547±1.837	87.82±1.722	75.27±3.228	3.277±0.18	23.18±1.079
T	−10.808	15.430	−15.815	−14.056	−5.382	−20.215
P	0.000*	0.000*	0.000*	0.000*	0.000*	0.000*

IVC=Inferior vena cava; SV=Stroke volume; MAP=Mean artery pressure; HR=Heart rate; CI=Cardiac output index

Table 3: Comparison of the rate of change between the two groups

	IVC variability (%)	SV index (%)	MAP (%)	HR (%)	CI (%)	Airway pressure (%)
Group C (post-pre)/pre	94.46±19.41	−18.1±3.34	13.08±3.24	27.93±8.75	9.31±7.18	44.14±6.44
Group E (post-pre)/pre	43.1±16.54	−5.1±1.16	5.36±1.2	9.3±2.42	4.38±2.32	41.22±8.43
T	6.68	−12.192	7.402	6.807	2.168	0.909
P	0.000*	0.000*	0.000*	0.000*	0.042*	0.374

IVC=Inferior vena cava; SV=Stroke volume; MAP=Mean artery pressure; HR=Heart rate; CI=Cardiac output index

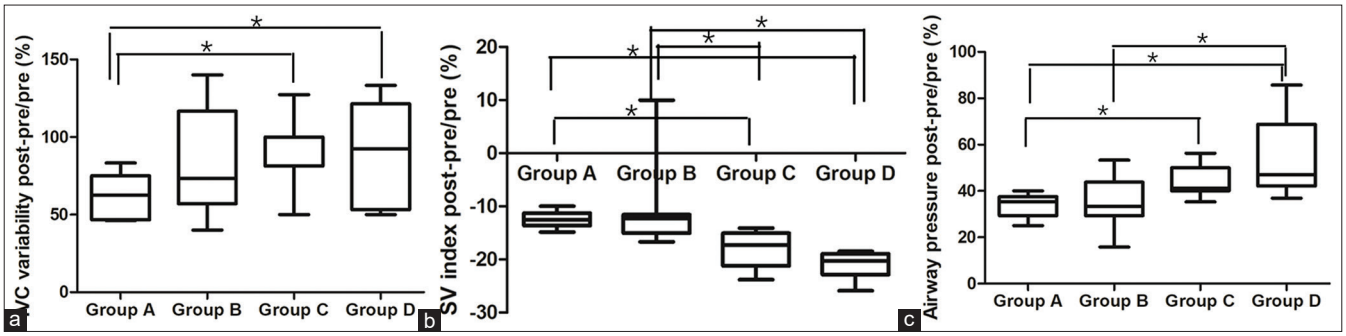

Figure 4: (a) Comparison of inferior vena cava variability (post–pre/pre), (b) Comparison of stroke volume index (post–pre/pre), (c) Comparison of airway pressure (post–pre/pre), **P* < 0.05. IVC = Inferior vena cava; SV = Stroke volume

preload, myocardial contractility, afterload, HR, of which preload is often referred to as the volume load.[8] According to the Frank–Staling curve, the CO can be increased by increasing volume load within a certain range called volume responsiveness.[18,19] During mechanical ventilation, IVC variability <18% defined no volume responsiveness, and conversely >18% defined volume responsiveness.[15] Our study found that IVC variability increased after the establishment of the pneumoperitoneum which makes the patients possess more volume responsiveness. This may be caused by the pneumoperitoneum increased intra-abdominal pressure to shift upward diaphragm increased intrathoracic pressure to reduced venous return,[7] which is manifested as the IVC variability increased. The intrathoracic pressure increased during inhalation and decreased during exhalation, meanwhile, the change of the IVC diameter caused by this simultaneously, the volume of the blood returns to the heart decreased leading to the IVC dilates during inhalation, the volume of the blood returning to the heart increased lead to the IVC contracts during

exhalation.[15,20] When pneumoperitoneum is established, an increase in intra-abdominal pressure may cause decreased peripheral blood return. One study had shown portal venous blood return decreases after pneumoperitoneum,[7] which is similar to our results. Changes in intrathoracic pressure due to elevated intra-abdominal pressure will cause greater fluctuation of intrathoracic pressure during mechanical ventilation, results in a greater variation in the width of IVC diameter. When inhaling, the intrathoracic pressure is much higher, resulting in much less returning blood volume and wider IVC diameter. When exhaling, the intrathoracic pressure decreases instantly, resulting in a sudden increase in returning blood volume and much narrower IVC diameter. After fasting and drinking, patients are usually in a state of insufficient volume, and we found that the IVC variability was relatively reduced by fluid transfusion which indicating that the influence of pneumoperitoneum on patient's IVC variability is affected by the patient's volume status, the effect of the establishment of pneumoperitoneum on blood return is reduced with a situation of sufficient volume.

When venous blood return to the heart is reduced, the volume in the heart and lung is reduced either, which directly leads to a decrease in end-diastolic volume and so the SV.[8] In our study, the change of SV can be better reflected by correcting SV through BSA. We also found that SV index decreased after the establishment of pneumoperitoneum and with the increase of pneumoperitoneum pressure, SV index had a downward trend, Group C and Group D showed a decrease compared with Group A and Group B, and both were statistically significant. The decrease of SV index means a decrease of blood volume entering the arterial system which will cause a decrease in parasympathetic nerve excitability of the carotid body and aortic arch pressure receptors, and increase of the sympathetic nerve signals to the heart and peripheral, leading to an increase of the HR and peripheral vascular contraction.[21] At the same time, when venous return is reduced, the parasympathetic nerve stimulation of cardiopulmonary pressure receptors in the atrium, ventricle, vena cava and pulmonary vessels decreases, resulting in an increase in sympathetic excitability and an increase in HR.[21] In our study, it was also found that after the establishment of the pneumoperitoneum, the HR was significantly higher than before. When patients with a relatively sufficient capacity, the change of cardiac preload is small while establishing the pneumoperitoneum, and so the resulting excitatory changes in pressure receptors as well as the sympathetic and parasympathetic nerves are also not large, finally lead to a relatively stable status in IVC variability, SV index, HR and MAP.

CI is the CO after BSA correction, and CO is calculated by multiplying HR by SV.[17] When SV decreases, the compensatory increase of HR makes up blood volume in the arterial system for the loss caused by the decrease of SV, and even the sudden increase of HR makes the instantaneous CO exceed that before the establishment of pneumoperitoneum. We also found that compared with preestablishment pneumoperitoneum, patients' CI was increased in Group A, Group B, Group D, and Group E. On the basis of little overall change or even increase in CO and peripheral vascular contraction caused by enhanced sympathetic nerve excitability, MAP increased after the establishment of pneumoperitoneum.

Our study also found that the average intrathoracic pressure was significantly higher after the establishment of the pneumoperitoneum, and there was a continuous upward trend with the increase of pneumoperitoneum pressure, which also suggested that the IVC variability and SV index were closely related to the change of intrathoracic pressure.[7] The effects of pneumoperitoneum on patients are complicated, short-term effects include changes in vital signs caused by intra-abdominal and intra-thoracic pressure change in our study, after a period of time, the patients can adjust themselves through self-regulation, such as sympathetic nerve, parasympathetic nerve, self-volume supplementation, so that patients' circulation status tends to be stable.[22] Long-term effects include absorption of CO_2 into the blood, leading to changes in patients' internal environment.[23] For ordinary patients, such physical changes are not enough to cause serious consequences, but for some patients with heart disease, especially for patients with coronary heart disease, the increase in HR may have serious consequences.[24] Therefore, understanding this process also helps us to further prevent the occurrence of this situation, such as appropriate fluid transfusion, slowly establishing the pneumoperitoneum, adjusting ventilator parameters.

Limitation

The combination of transesophageal echocardiography and clinical anesthesia is a feature of this study. The small study sample, single-center design, and all patients included were generally in good condition without serious comorbidities, nor did it study the changes of patient's hemodynamics after the pneumoperitoneum for a period of time were the main limitations of the present study. In the future, we will further study how to avoid the fluctuation of vital signs after the establishment of pneumoperitoneum.

CONCLUSION

The establishment of pneumoperitoneum can increase the patient's respiratory variation of IVC and reduce the SV index, and with the increase of pressure, IVC variability has an increasing trend, and SV index has a decreasing trend. Fluid transfusion can reduce the relative influence of the pneumoperitoneum and make the circulation more stable.

The establishment of pneumoperitoneum has a certain influence on patient's vital signs.

Contributions

DWL contributed in the conception of the work, conducting the study, revising the draft and agreeing for all aspects of the work; JFS contributed in the literature search, conducting the study and data acquisition; KC contributed in conducting the study, data acquisition and statistical analysis; YFY contributed in the literature search, conducting the study and data acquisition; FZ contributed in the conception of the work, approval of the final version of the manuscript and agree for all aspects of the work.

Financial support and sponsorship

This work was supported by the Chongqing Science and Technology Joint Project (No. MSXM20202676).

Conflicts of interest

There are no conflicts of interest.

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