

The impact of intra-abdominal pressure on the stroke volume variation and plethysmographic variability index in patients undergoing laparoscopic cholecystectomy

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Summary

The purpose of the present study was to evaluate the effect of increasing intra-abdominal pressure (IAP) on stroke volume variation (SVV) and plethysmographic variability index (PVI) in patients undergoing laparoscopic cholecystectomy. PVI examined by Masimo Radical 7 pulse oximeter and SVV determined using FloTrac/Vigileo were monitored simultaneously in forty-five patients undergoing laparoscopic cholecystectomy (LC). Mean arterial blood pressure (MAP), heart rate (HR), cardiac index (CI), perfusion index (PI), airway pressures (P), SVV, and PVI were also recorded at the following predetermined time: 5 min after endotracheal intubation (T₁), 5 min after pneumoperitoneum at 5 mmHg (T₂), 5 min after pneumoperitoneum at 10 mmHg (T₃), 5 min after pneumoperitoneum at 15 mmHg (T₄), and 5 min after the termination of pneumoperitoneum (T₅). Forty-five patients with a total of 225 pairs of measurements were included in the analysis. Compared with the values at T₁, both SVV and PVI showed significant progressive increases as the IAP was adjusted from 5 to 10, 15 mmHg at T₂, T₃, and T₄, respectively. No significant difference was found when the pneumoperitoneum was terminated at T₅. Further regressive analysis indicated strong relationships between SVV and IAP ($r = 0.8118, p < 0.001$), PVI and IAP ($r = 0.8876, p < 0.001$) respectively. Both PVI and SVV showed rapid and IAP correlative changes with increasing intra-abdominal pressure in patients undergoing laparoscopic cholecystectomy.

Keywords: Intra-abdominal pressure, plethysmographic variability index, stroke volume variation

1. Introduction

With the development of mini-invasive technique, laparoscopic operations have been the mainstay of general surgery. Usually artificial pneumoperitoneum is established with carbon dioxide insufflation and the intra-abdominal pressure (IAP) is frequently maintained at 10 to 15 mmHg. Given that the preload is significantly influenced by the increase of IAP, it is important to evaluate the influence of IAP on the volume status of those patients undergoing laparoscopic surgery in clinical practice (1).

Currently there are two ways to monitor the state of volume. The first includes some traditional static

parameters such as central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP). Both indices are invasive and have been shown to reflect the patient's volume status poorly and predict a cardiac response to fluid therapy incompetently (2,3). By contrast, dynamic variable such as stroke volume variation (SVV) has been shown to be a good predictor of volume responsiveness in mechanically ventilated patients undergoing open chest surgery, liver transplantation, and abdominal surgery (4-6). However, it was controversial whether SVV changed when IAP was increased in patients undergoing laparoscopic surgery (7,8). On the other hand, noninvasive plethysmographic variability index (PVI) is a new parameter used for the purpose of fluid responsiveness in patients receiving several kinds of surgeries (9-14). In patients undergoing laparoscopic surgery, a previous study has suggested that PVI increased significantly after pneumoperitoneum was established (7). However, it is still elusive whether PVI and SVV could convincingly reflect the changes

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of intra-abdominal pressure in mechanically ventilated patients.

Therefore, the aim of the present study was to investigate the responsiveness and the relationships between PVI and SVV with the increasing intra-abdominal pressure in patients undergoing laparoscopic cholecystectomy (LC).

2. Materials and Methods

2.1. Subjects

The Institutional Review Board of Jinling Hospital approved the present study and written informed consent was obtained from each patient. Forty five patients undergoing elective LC were included in this prospective observational study from October 2013 to May 2014. Exclusion criteria were arrhythmias, diabetes, pulmonary diseases, intracardiac shunts, valvular heart disease, peripheral vascular disease, long-term taking oral vascular active drugs, body mass index (BMI) smaller than 18 or greater than 30, the blood loss over 100 mL during operation and the duration of surgery over 60 min.

2.2. Anesthesia and monitors

After arriving the operation room, all patients received routine monitors including electrocardiogram (ECG), pulse oximetry, capnography, and noninvasive blood pressure. General anesthesia was induced by the same anesthesiologist with midazolam (0.05 mg/kg), propofol (2.0-2.5 mg/kg), fentanyl (3 µg/kg) and rocuronium (0.6 mg/kg) to facilitate tracheal intubation. Anesthesia was maintained with continuous infusion of propofol and cis-atracurium supplemented with a bolus of fentanyl 0.3 µg/kg to keep the bispectral index monitor (BIS, Aspect 1000, Aspect Medical System Inc., Natick, MA, USA) between 40 and 60. Mechanical ventilation was adjusted to maintain end-tidal pressure of CO₂ (P_{ET}CO₂) between 30-35 mmHg.

According to the operation manual, SVV was determined by inserting an arterial catheter (20 gauge, Surflo, Terumo, Japan) preoperatively, then connecting *via* a prepackaged arterial pressure tubing set (VAMP Plus, Edwards Lifesciences) to a FloTrac™ sensor (Edwards Lifesciences) and a Vigileo™ monitor (software version 01.12, Edwards Lifesciences). Meanwhile, PVI was measured by a Masimo Radical 7 pulse oximeter with PVI software (version 7.6.2.1) through a pulse oximeter probe placed on the index finger of the other hand and wrapped to prevent outside light from interfering with the signal. Both values were continuously measured.

2.3. Study design

All patients were studied immediately after induction of anesthesia. We avoided any stimulation to the patients

for 5 min before data collection in order to minimize the change of vasomotor tone. PVI and SVV were recorded at a predetermined set of the 5 time points by the other same anesthesiologist during the end-expiratory phase: T₁, 5 min after endotracheal intubation; T₂, 5 min after pneumoperitoneum at 5 mmHg; T₃, 5 min after pneumoperitoneum at 10 mmHg; T₄, 5 min after pneumoperitoneum at 15 mmHg; T₅, 5 min after the termination of pneumoperitoneum. The mean arterial blood pressure (MAP), heart rate (HR), cardiac index (CI = cardiac output/body surface area), SVV, PVI, perfusion index (PI), BIS, airway pressures (P), and P_{ET}CO₂ were also recorded at the aforementioned time points.

To exclude the impact of fluid on the SVV and PVI, 8 mL/Kg balanced solution was infused before anesthesia induction to supplement the insufficient induced by fast, then balanced solution was given at the rate of 4 mL/kg/h.

2.4. Statistical methods

Statistical analysis was performed using the software program Prism version 5.0 (Graphpad, San Diego, CA, USA). Descriptive statistics of the variables are presented as means ± SD. Normality of the distributions was tested using a Kolmogorov-Smirnov test. Data analysis was performed using repeated measurement of analysis of variance (ANOVA) followed by the Bonferroni test for post hoc comparisons. Linear regression was used to measure the relationship between IAP and SVV, IAP and PVI respectively. A *p* value of less than 0.05 was regarded as statistically significant difference.

3. Results and Discussion

In the present study, 45 patients receiving LC were enrolled (24 males and 21 females), with a mean age of 46 years (range, 24-68). No patient met the exclusion criteria, thus 45 patients with a total of 225 pairs of measurements were included in the analysis. The general characteristics of patients were listed in Table 1.

As shown in Table 2, HR, CI and BIS did not alter significantly during the observed period. However, P and MAP increased significantly with the elevation of IAP.

Table 1. Patients characteristics

Parameters	
Gender (male/female)	24/21
Age (year)	46 ± 12
Height (cm)	168 ± 8
Weight (kg)	68 ± 9
ASA (I/II)	19/26
Operation time (min)	37 ± 5
Anesthesia time (min)	57 ± 13

Data are presented as the mean ± standard deviation, or as actual numbers, when appropriate.

Table 2. Values of mean arterial blood pressure (MAP), heart rate (HR), cardiac index (CI), perfusion index (PI), airway pressures (P), and bispectral index (BIS) at five time points

Parameters	T ₁	T ₂	T ₃	T ₄	T ₅
MAP (mm Hg)	79 ± 7	80 ± 7	90 ± 8*	102 ± 10*	80 ± 8
HR (beats/min)	78 ± 10	77 ± 7	77 ± 7	76 ± 11	77 ± 6
CI (L·min ⁻¹ ·m ⁻²)	2.7 ± 0.7	2.6 ± 0.6	2.5 ± 0.9	2.4 ± 0.6	2.9 ± 0.7
PI (%)	5.0 ± 0.4	3.7 ± 0.3*	2.1 ± 0.2*	1.2 ± 0.1*	4.8 ± 0.4
P (cmH ₂ O)	15.0 ± 2.6	16.6 ± 3.1	19.3 ± 3.4*	22.0 ± 3.9*	15.5 ± 2.7
BIS	51 ± 5	51 ± 4	50 ± 5	51 ± 5	51 ± 4

Data are presented as the mean ± standard deviation. T₁: 5 min after endotracheal intubation; T₂: 5 min after pneumoperitoneum at 5 mmHg; T₃: 5 min after pneumoperitoneum at 10 mmHg; T₄: 5 min after pneumoperitoneum at 15 mmHg; T₅: 5 min after the termination of pneumoperitoneum. * $p < 0.05$ vs. T₁.

In contrast, PI decreased significantly with the increase of IAP. After termination of pneumoperitoneum, these parameters returned to the level before insufflation.

As shown in Figure 1A, SVV measured at T₂ ($13.73 \pm 2.84\%$), T₃ ($19.20 \pm 2.84\%$) and T₄ ($24.67 \pm 3.93\%$) were significantly increased when compared with the value at T₁ ($12.47 \pm 3.13\%$). No significant difference was found between SVV at T₅ ($11.39 \pm 3.96\%$) with SVV at T₁. Similarly, the levels at T₂ ($17.96 \pm 3.02\%$), T₃ ($26.07 \pm 4.22\%$) and T₄ ($34.67 \pm 5.56\%$) was significantly enhanced compared with the PVI at T₁ ($13.09 \pm 3.36\%$). There was no significant difference between PVI at T₅ ($14.64 \pm 4.24\%$) with PVI at T₁.

As shown in Figure 2, both SVV and PVI correlated significantly with the level of IAP (between SVV and IAP, $r = 0.8118$, $p < 0.001$; between PVI and IAP, $r = 0.8876$, $p < 0.001$).

The major finding of the present study was that increasing IAP induced rapid and correlative changes for both SVV and PVI. After termination of insufflation, both SVV and PVI returned to the level without pneumoperitoneum.

Estimating the volume status of patients is a frequent problem for the anesthesiologist and surgeons. The estimation is usually obtained by means of static as well as dynamic variables. Frequently used static preload variables, including CVP and PCWP, often fail to provide reliable information on volume status and are not capable of predicting a cardiac response to fluid therapy (2,3). By contrast, dynamic variables such as PVI and SVV could reliably predict the response to fluid administration during mechanically ventilation (6,9,11,15). Although both SVV and PVI could reflect the fluid loading, it is known that both are disturbed by several confounder factors such as: arrhythmia, vasomotor tone, vasoactive drugs, pleural effusion, intra-abdominal pressure and so on (16-18).

In patients undergoing laparoscopic surgery, intra-abdominal pressure is deliberately increased to 10-15mmHg by carbon dioxide insufflation. It is well known that pneumoperitoneum could induce significant disturbance of hemodynamics, which are manifested as decreases in cardiac output, increases of systemic and pulmonary vascular resistances, elevated arterial

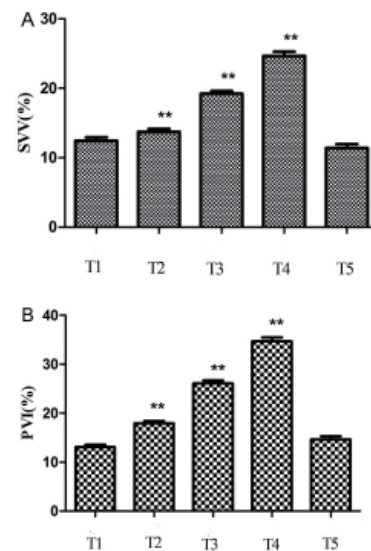


Figure 1. The values of stroke volume variation (SVV) and plethysmographic variability index (PVI) at different levels of intra-abdominal pressure (IAP). T₁: IAP = 0 mmHg before pneumoperitoneum; T₂: IAP = 5 mmHg after pneumoperitoneum; T₃: IAP = 10 mmHg after pneumoperitoneum; T₄: IAP = 15 mmHg after pneumoperitoneum; T₅: IAP = 0 mmHg termination of pneumoperitoneum. ** $p < 0.01$, compared with the level of SVV (A) or PVI (B) at the T₁, $n = 45$.

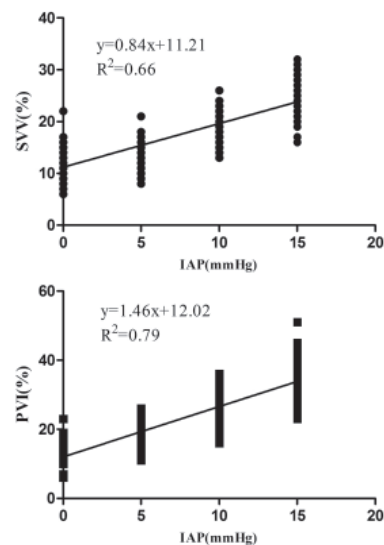


Figure 2. Correlation between stroke volume variation (SVV) and plethysmographic variability index (PVI) with the different levels of intra-abdominal pressure (IAP) from the data of 45 patients.

pressure and decreases in venous return (1). In the present study, we also found the increase of MAP and the decrease of PI. Using SVV and PVI, we further observed the effect of IAP on the volume status. In 2012, Høiseth *et al.* reported that pneumoperitoneum (10-12 mmHg) only changed the PVI significantly, but did not change the SVV (7). However, a recent study by Wajima *et al.* indicated that SVV was significantly increased at 2 to 5 min after pneumoperitoneum, and significantly decreased at 1 to 5 min after release of pneumoperitoneum (8). Compared with those studies, our work had three features that the level of IAP was not sole, but adjusted to three stages as 5, 10 and 15 mmHg; the values of SVV and PVI were recorded simultaneously at 5 min after the establishment of the different levels of pneumoperitoneum and the infused fluid was restricted at low speed to avoid the impact on the SVV and PVI. We found that both SVV and PVI increased progressively as the level of IAP was adjusted from 0 to 5, 10 and 15 mmHg. After termination of pneumoperitoneum, both SVV and PVI showed no difference with the level before pneumoperitoneum. Furthermore, the regressive analysis demonstrated that there existed positive regression between SVV and IAP, PVI and IAP, respectively.

Although both SVV and PVI could reflect the influence of IAP effectively, PVI might be the better choice for clinical setting. Firstly, PVI could be continuously and noninvasively monitored. As for SVV, it was invasive and not routinely available in daily clinical practice. Secondly, PVI showed a more consistent response to the IAP. In the present study, we found that the individual values of SVV and PVI at T_2 were actually smaller than those at T_1 in 17.8% (8/45) patients for SVV and 4.4% (2/45) patients for PVI. When IAP was 10 or 15 mmHg, there were no exceptions for both indices. Lastly, PVI showed a more steep response to the increase of IAP compared with the SVV.

There also existed some limitations in our study. Firstly, the IAP before pneumoperitoneum was arbitrarily determined as 0 mmHg, but it was well known that the IAP correlated with BMI in the healthy people (19). Thus the inclusion criteria for BMI was between 18 and 30. Furthermore our repeated measure study design should greatly overcome this shortcoming. Secondly, BIS could not effectively measure the degree of stress response, which would affect both SVV and PVI significantly. Unfortunately, the devices that could monitor the stress response are not available in the current clinical setting. Lastly, in the present study increasing IAP was induced by carbon dioxide insufflation, which might be different from conditions occurred secondary to abdominal compression in critically ill patients. Therefore, future studies are warranted to determine the change of SVV and PVI with increased intra-abdominal pressure induced by different circumstances.

In conclusion, our data suggested that both SVV and noninvasive PVI demonstrated rapid and correlative changes with increased intra-abdominal pressure resulting from carbon dioxide insufflations. Furthermore, it seemed that PVI was more sensitive to reflect the change of IAH.

Acknowledgements

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