

Does pneumoperitoneum affect perfusion index and pleth variability index in patients receiving combined epidural and general anesthesia?

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Summary

Plethysmographic variability index (PVI) is a dynamic index used for the purpose of fluid responsiveness in patients, and the effect of pneumoperitoneum on PVI is still unclear. We therefore attempted to determine whether PVI and perfusion index (PI) change before/after pneumoperitoneum in patients receiving combined epidural and general anesthesia, which is a common anesthesia method with intravenous remifentanyl. Twenty patients underwent laparoscopic cholecystectomy or colectomy. Immediately before pneumoperitoneum, variables were measured at baseline I and were then measured every min for 5 min after pneumoperitoneum start. Immediately before pneumoperitoneum release, variables were measured at baseline II and were measured every min for 5 min after pneumoperitoneum release. Compared with baseline I values, after pneumoperitoneum start, significant increases occurred in stroke volume variation (SVV) at 1–5 min, and significant decreases occurred in PI at 1–5 min. PVI did not change. Compared with baseline II values, after pneumoperitoneum release, significant increases occurred in PI at 1–5 min, and significant decreases occurred in PVI at 4–5 min and SVV at 1–5 min. In patients receiving combined epidural and general anesthesia, we newly found that PI decreased but PVI remained unchanged with a sufficient dose of remifentanyl and epidural anesthesia that can block noxious stimuli and also most sympathetic activity. Furthermore, we reconfirmed that PI increased and PVI decreased upon release of pneumoperitoneum. PI and PVI values must be estimated cautiously during and after pneumoperitoneum.

Keywords: Pneumoperitoneum, perfusion Index, pleth variability index, stroke volume variation, combined epidural and general anesthesia, remifentanyl

1. Introduction

Dynamic indices such as stroke volume variation (SVV), pulse pressure variation, and systolic pressure variation have consistently been shown to be more accurate than static indicators such as central venous pressure (CVP) and pulmonary capillary wedge pressure for predicting fluid responsiveness in mechanically

ventilated patients under general anesthesia (1-4). The accurate assessment of intravascular fluid status and measurement of fluid responsiveness have become increasingly important in peri-operative medicine and critical care (4). As a result, these dynamic indices are increasingly used to guide fluid therapy (5).

We recently reported that pneumoperitoneum increased SVV, and furthermore, upon release of the pneumoperitoneum, SVV decreased significantly (6). We have asserted that SVV values must be estimated cautiously during pneumoperitoneum (6). Pleth variability index (PVI) is another dynamic index, and many studies concluded that it is useful for the assessment of fluid responsiveness in patients (2,7-9) although one study concluded that PVI seems inaccurate to predict fluid responsiveness after conventional

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cardiac surgery with cardiopulmonary bypass (10). However, in reference to the studies of Høiseith *et al.* (11) and Liu *et al.* (3), we believe that the effect of pneumoperitoneum on PVI is still unclear. In our recent study, however, we found that pneumoperitoneum increased SVV (6), and this result was very similar to that of several earlier studies (12-15). Høiseith *et al.* (11) showed that SVV did not change as pneumoperitoneum was established, whereas PVI increased in their study, and furthermore, in a recent study describing the effect of pneumoperitoneum on PVI, the baseline was 5 min after endotracheal intubation (3), and we believe that this methodology is questionable for this kind of this study. We therefore attempted to determine whether PVI, which is based on the respiratory variations in the perfusion index (PI) (16), and PI change both before and after pneumoperitoneum in patients receiving combined epidural and general anesthesia with intravenous remifentanyl.

2. Materials and Methods

2.1. Subjects

We conducted this prospective study at International University of Health and Welfare Shioya Hospital, Japan. The study protocol was approved by the ethics committee of the International University of Health and Welfare Hospital (protocol number 13-B-31, 2013-12-25), and we registered this study in the "UMIN Clinical Trial Registry" (ID: UMIN000012863). We obtained written informed consent from each patient. Patients were eligible for inclusion in this study if they were to undergo laparoscopic gastrointestinal surgery (cholecystectomy and colectomy). All patients were classified as ASA physical status 1 and 2, and none had known diabetes mellitus; hypertension; cardiovascular (including non-sinus rhythm and 2° or 3° A-V block), pulmonary, endocrinologic, neurologic, or autonomic diseases; or diseases that affect intravascular fluid volume or balance, such as gastrointestinal obstructive or inflammatory diseases. All patients underwent preoperative fast for at least 8 hours, and no premedication was given to any of the patients.

2.2. Anesthesia and monitors

An epidural catheter was placed in one intervertebral space ranging from Th8-9 to Th11-12, at a distance of 4 cm inside the space cephaladly, before induction of general anesthesia. The epidural space was identified by the loss-of-resistance technique using physiological saline (17,18). Anesthesia consisted of 1% lidocaine epidural anesthesia, and the analgesia level was determined by a pinprick 15 min after lidocaine administration.

After establishing an analgesic level from T4 to L1, induction of general anesthesia was performed with

propofol (initial effect-site concentration = 4 µg/mL) administered by a plasma target-controlled infusion method) and 1 µg/kg remifentanyl intravenously (IV) in total, and rocuronium 0.6 mg/kg IV. After induction of anesthesia, a 23-gauge catheter was inserted in the left or right radial artery for direct arterial pressure monitoring, and the patients' lungs were mechanically ventilated by means of a semi-closed circle system at a fresh gas flow of 6 L/min (O₂, 2 L/min and air, 4 L/min). Controlled ventilation was set at 10 breaths/min, with a tidal volume of 8 mL/kg and an inspiratory:expiratory ratio of 1:2. Anesthesia during surgery was maintained with propofol (effect-site concentration ≥ 3 µg/mL), epidural anesthesia with 0.375% ropivacaine, and remifentanyl at a rate of 0-0.5 µg/kg/min, and rocuronium. We achieved a target BIS between 40 and 60 and stable circulatory variables during surgery. After surgical skin preparation, the abdomen was insufflated with CO₂ to create and maintain a pneumoperitoneum at 10 mmHg.

Systolic arterial pressure (SAP), diastolic arterial pressure (DAP), heart rate (HR), cardiac output (CO), and SVV, stroke volume index (SVI), systemic vascular resistance (SVR), and pressure of end-tidal CO₂ (P_{ET}CO₂) were continuously monitored with a standard monitor (S/5 Anesthesia Monitor, GE Healthcare, Helsinki, Finland) and the FloTrac/Vigileo™ system (software version 03.06) (Edwards Lifesciences, Irvine, CA, USA). PVI and PI were also continuously monitored with Radical 7 (software version 7.9.1.0) (Masimo Corporation, Irvine, CA, USA).

We did not insert a central venous catheter into the patients to directly measure central venous pressure (CVP). Rather, we obtained the data for SVR using a fixed CVP equal to 0 mmHg by inputting the pressure into the FloTrac/Vigileo™ system (19).

2.3. Study design

Immediately before pneumoperitoneum, baseline registrations of the variables were obtained (baseline I), and these variables were measured every min for 5 min after pneumoperitoneum started. Immediately before pneumoperitoneum was released, registrations of the variables were obtained again (baseline II), and these variables were also obtained every min for 5 min after release of pneumoperitoneum. The position of the patient during measurements was kept horizontal. CO, SVV, SVI, and SVR were recorded 20 sec after SAP, DAP, HR, and P_{ET}CO₂ were recorded because the Vigileo™ samples the pressure waveform at 100 Hz over 20 sec to capture 2,000 data points for analysis, and parameter calculations are provided at the end of every 20-sec timeframe (20,21).

For laparoscopic cholecystectomy, before general anesthesia/epidural block induction, crystalloid at a volume of at least 10 mL/kg was infused followed by

an additional 10-15 mL/kg during the laparoscopic procedure (22). For laparoscopic colectomy, before general anesthesia/epidural block induction, colloid (6% hydroxyethyl starch [HES] 70/0.55/4—Saline HES; Fresenius Kabi Japan, Tokyo, Japan) was infused at 5 mL/kg followed intraoperatively by 3 mL/kg/hour of crystalloid plus 3 mL/kg/hour of colloid (6% HES 70/0.55/4), and measured blood loss was compensated with an equal volume of colloid (6% HES 70/0.55/4) until a predetermined critical hemoglobin level for blood transfusion was reached (22). Vasopressors were administered as needed.

We used the almost the same methodology that was used in our previous study (6).

2.4. Statistical analyses

Sample size was estimated from preliminary data obtained from 8 patients, and an assumption was made that a 3-point change in PVI between the baseline II value and that at 5 min after stopping pneumoperitoneum would be clinically relevant. Power analysis suggested that a minimum of 16 patients would be needed for a $\beta = 0.1$ and $\alpha = 0.05$. To compensate for potential dropouts, we enrolled 20 patients in this study. This analysis was performed using GraphPad StatMate 2.00 (GraphPad Software, Inc., La Jolla, CA, USA).

Values are expressed as means \pm standard deviation (SD). Comparisons of SAP, MAP, DAP, HR, SVV, CO, SVI, $P_{ET}CO_2$, SVR, and airway pressure changes were

performed with paired Student t-tests with Bonferroni's correction to determine whether there were significant differences between baseline values and the parameter values during pneumoperitoneum or after release of pneumoperitoneum. A P value of < 0.05 was required to reject the null hypothesis. All analyses were performed with GraphPad Prism 5.04 (GraphPad Software, Inc.).

3. Results

The 20 patients completing the study had an average (mean \pm SD) age of 57 ± 15 years, body weight of 64 ± 18 kg, height of 162 ± 10 cm, and body surface area of 1.67 ± 0.24 m². The male:female ratio was 13:7, and the cholecystectomy:colectomy ratio was also 13:7. No patients received blood transfusion during surgery.

After pneumoperitoneum started, there were significant increases in heart rate (HR) at the 3- to 5-min time points (Figure 1), SVV at the 1- to 5-min time points, and SVR at the 2- to 5-min time points compared with baseline I values (Figure 2). There were significant decreases in $P_{ET}CO_2$ at the 1- to 2-min time points (Figure 1), PI at the 1- to 5-min time points, and SVI at the 1- to 3-min time points compared with baseline I values (Figure 2). Other values including PVI were unchanged (Figures 1, 2).

After release of pneumoperitoneum, there were significant increases in $P_{ET}CO_2$ at the 1-min time point (Figure 3), PI at the 1- to 5-min time points, and SVI at the 1-min time points compared with baseline II

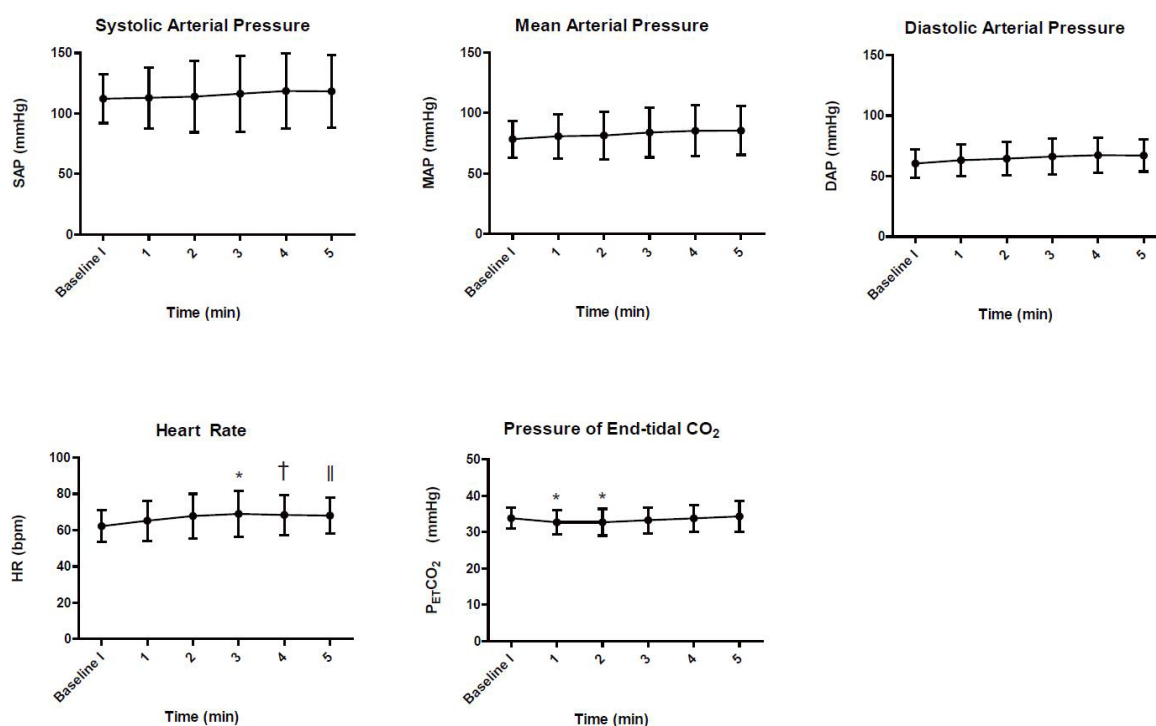


Figure 1. Sequential changes in systolic arterial pressure, mean arterial pressure, diastolic arterial pressure, heart rate, and pressure of end-tidal CO₂ at baseline I and after pneumoperitoneum. Data are expressed as mean \pm standard deviation. * $P < 0.05$ vs baseline I; † $P < 0.01$ vs baseline I; ‡ $P < 0.0005$ vs baseline I.

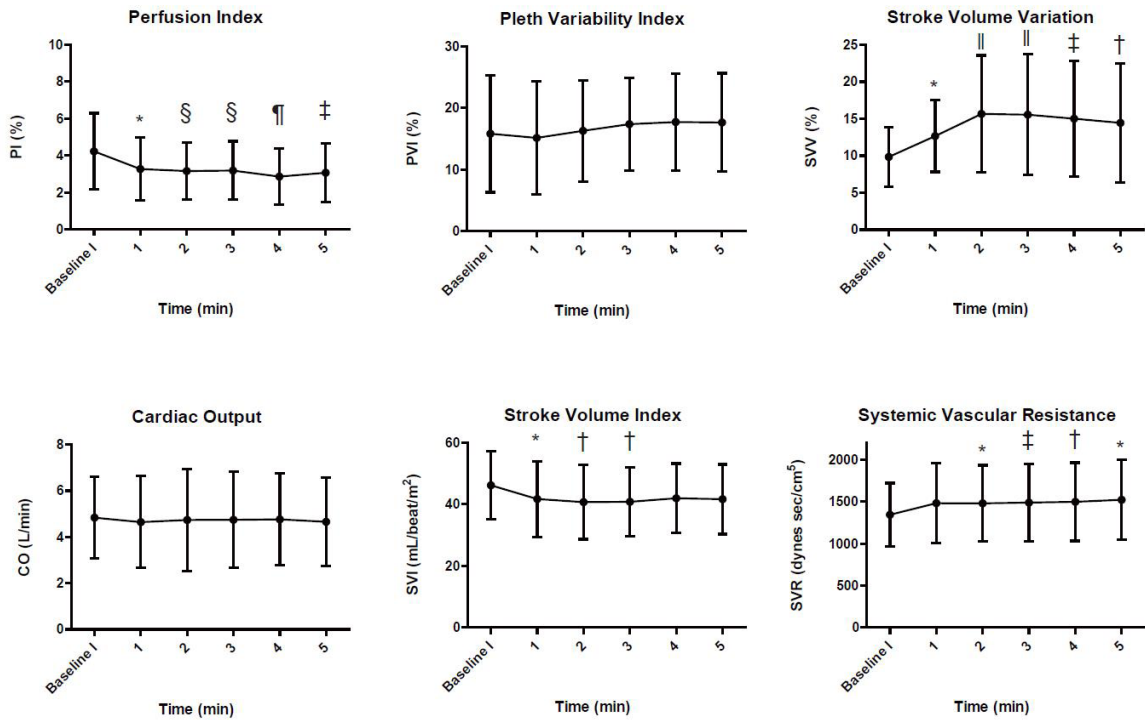


Figure 2. Sequential changes in perfusion index, pleth variability index, stroke volume variation, cardiac output, stroke volume index, and systemic vascular resistance at baseline I and after pneumoperitoneum. Data are expressed as mean ± standard deviation. **P* < 0.05 vs baseline I; †*P* < 0.01 vs baseline I; ‡*P* < 0.005 vs baseline I; §*P* < 0.001 vs baseline I; ¶*P* < 0.0005 vs baseline I; ¶*P* < 0.0001 vs baseline I.

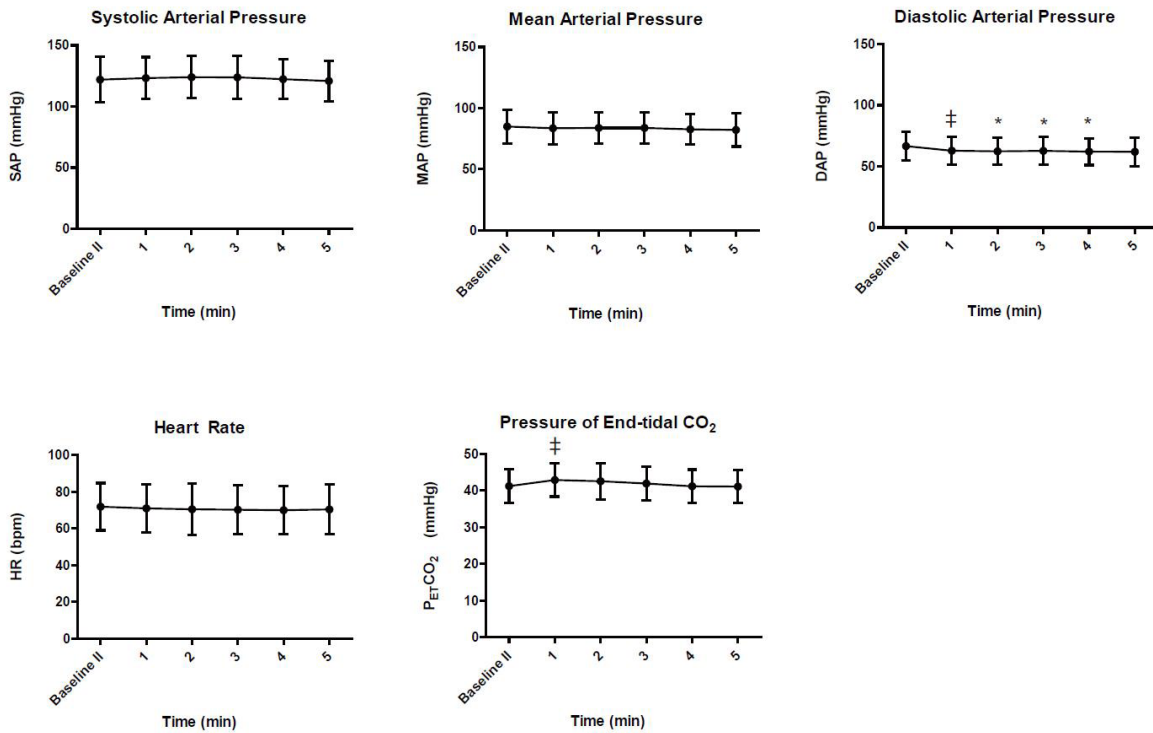


Figure 3. Sequential changes in systolic arterial pressure, mean arterial pressure, diastolic arterial pressure, heart rate, and pressure of end-tidal CO₂ at baseline II and after stopping pneumoperitoneum. Data are expressed as mean ± standard deviation. **P* < 0.05 vs baseline II; ‡*P* < 0.005 vs baseline II.

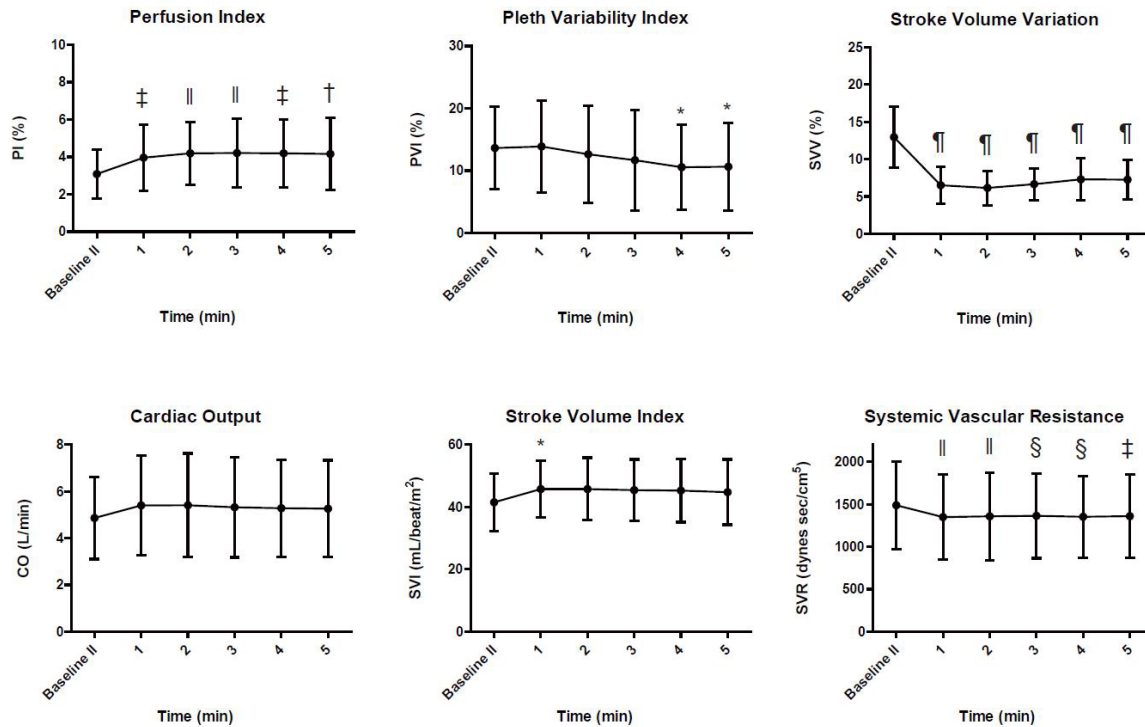


Figure 4. Sequential changes in perfusion index, pleth variability index, stroke volume variation, cardiac output, stroke volume index, and systemic vascular resistance at baseline II and after stopping pneumoperitoneum. Data are expressed as mean \pm standard deviation. * $P < 0.05$ vs baseline II; † $P < 0.01$ vs baseline II; ‡ $P < 0.005$ vs baseline II; § $P < 0.001$ vs baseline II; ¶ $P < 0.0005$ vs baseline II; * $P < 0.0001$ vs baseline II.

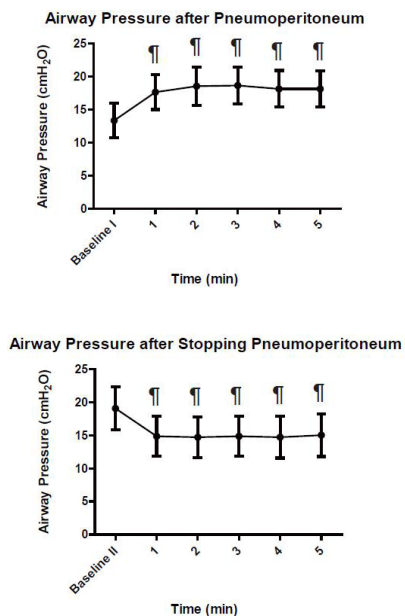


Figure 5. Sequential changes in airway pressure after pneumoperitoneum start and after stopping pneumoperitoneum. Data are expressed as mean \pm standard deviation. ¶ $P < 0.0001$ vs baseline I and baseline II.

(Figure 4). There were significant decreases in DAP at the 1- to 4-min time points (Figure 3), PVI at the 4- to 5-min time points, SVV at the 1- to 5-min time points, and SVR at the 1- to 5-min time points compared with baseline II (Figure 4). SAP, MAP, HR, and CO were

unchanged (Figures 3, 4). Airway pressures during measurements are shown in Figure 5.

4. Discussion

In this study, pneumoperitoneum decreased PI, did not change PVI, and increased SVV, whereas upon release of pneumoperitoneum, PI increased and both PVI and SVV decreased significantly in patients receiving combined epidural and general anesthesia, which is a common anesthesia method, with intravenous remifentanyl, a very potent opioid. Liu *et al.* (3) recently showed that both PVI and SVV increased and PI decreased significantly after pneumoperitoneum, and these values returned to the baseline level after release of pneumoperitoneum. However, they defined baseline as the values measured 5 min after endotracheal intubation, and we believe that this methodology is questionable for this kind of study because the values at 5 min after endotracheal intubation are considered to be unstable in terms of hemodynamics. Høiseith *et al.* (11) found that as pneumoperitoneum was established, PVI increased, PI decreased significantly, and SVV was unchanged. This reported lack of increase in SVV is questionable because in all reported animal studies (12-15), SVV increased after elevation of intra-abdominal pressure and/or pneumoperitoneum. We recently found that pneumoperitoneum increased SVV in humans (6), and furthermore, many animal studies showed that other

dynamic indices such as systolic pressure variation and pulse pressure variation (23,24) also increased during intra-abdominal hypertension (12-15,25-27). Therefore, the results of Liu *et al.* (3) and Høiseith *et al.* (11) might be questionable, including the change in PVI and PI values.

Our results relating to PVI and SVV after pneumoperitoneum were different than those of Høiseith *et al.* (11) and Liu *et al.* (3), and the reason for the discrepancy is unclear. However, we suppose this relates to differences in study design. For example, the anesthesia methods were quite different: we gave our patients combined epidural and general anesthesia using propofol, remifentanyl, rocuronium, and epidural 0.375% ropivacaine as local anesthetics. In contrast, Høiseith *et al.* (11) inserted an epidural catheter in 9 of 20 patients but maintained just general anesthesia using desflurane and fentanyl, and Liu *et al.* (3) induced general anesthesia with midazolam, propofol, fentanyl, and rocuronium, and maintained general anesthesia with propofol, cisatracurium and a bolus of fentanyl as supplement. Furthermore, tidal volume, which is one of the deciding factors that can change dynamic indices values (28-30), was different with Høiseith *et al.* (11) (Liu *et al.* (3) never referred to tidal volume), and Høiseith *et al.* (11) applied positive end expiratory pressure of 5 cm H₂O, whereas we used zero end expiratory pressure. The patient characteristics were also not similar: the height and weight of the Høiseith *et al.* (11) patients were much higher than those of our patients. Moreover, the baseline SVV value in their study was 9%, and it increased non-significantly to 10% during the pneumoperitoneum. We also believe that it is questionable that the SVV value did not increase significantly during pneumoperitoneum because the SV in their study decreased by 20 % during pneumoperitoneum compared to the baseline value; SVV is defined as $SVV (\%) = 100 \times (SV_{max} - SV_{min}) / [(SV_{max} + SV_{min}) / 2]$, where SV = stroke volume and maximal and minimal values for SV are determined as SV_{max} and SV_{min}, respectively, over a single respiratory cycle of paced breathing (19,20,31).

Although the PVI did not change after pneumoperitoneum in our study, Høiseith *et al.* (11) found that PVI increased when pneumoperitoneum was established and explained the mechanisms of this result as follows: "This result may be caused by sympathetic activity (32) induced by surgery or possibly release of norepinephrine induced by pneumoperitoneum per se (33). The finger photoplethysmographic waveform is affected by vasoconstriction induced by cold pressor test, stimulating sympathetic stimulation (34). Both inflation of CO₂ and surgical stimulation may contribute to the changes observed in the photoplethysmographic variables. The same mechanisms probably explain the reduction in PI. These findings are supported by a study on PVI and PI during skin incision, in which

PVI increases and PI decreases with incision (16)." Høiseith *et al.* (11) did not use remifentanyl (although we administered it properly and also gave epidural anesthesia, which can block sympathetic activity in fingertip (35)), and MAP and cardiac index increased significantly after pneumoperitoneum, whereas in our study, SAP, MAP, DAP, and CO were unchanged probably because we administered remifentanyl properly, and epidural anesthesia as a noxious stimuli and also most sympathetic activity was blocked. Therefore, we believe that this is also one of the most probable reasons the PVI did not change significantly after the start of pneumoperitoneum in the present study. Furthermore, the mechanism of PI increase and PVI decrease after release of pneumoperitoneum in the present study owes to the decrease of sympathetic activity induced by the pneumoperitoneum (11,32-34).

There are several limitations associated with our study. We measured PI, PVI, and SVV values during the 5-min period immediately after the start and end of pneumoperitoneum, and we did not record these values during the time of pneumoperitoneum. However, we can surmise the values of PI, PVI, and SVV during surgery from the values of baseline II. Although Høiseith *et al.* (11) showed that SVV predicted fluid responsiveness relatively poor during ongoing laparoscopic surgery, we believe that reevaluation is needed. Furthermore, we did not insert central venous catheters into the patients to directly measure CVP, but we obtained the data for SVR using a fixed CVP (= 0 mmHg) by inputting the pressure into the FloTrac/Vigileo™ system as described above (19). Donati *et al.* (36) reported that after induction of pneumoperitoneum (endoabdominal pressure = 11-15 mmHg; patient in head-down position), CVP increased by 3.7 mmHg, and we thought this value would be negligible when the SVR was calculated by the FloTrac/Vigileo™ system because our endoabdominal pressure was 10 mmHg, and also the position of the patients during measurements was kept horizontal.

In conclusion, although some studies showed that pneumoperitoneum decreases PI and increases PVI under general anesthesia, in this study, in patients receiving combined epidural and general anesthesia, PI decreased but PVI remained unchanged using a sufficient dose of remifentanyl and epidural anesthesia that can block noxious stimuli and also most sympathetic activity. This is newly found information. Because we believe that blockade of noxious stimuli can change these values as Takeyama *et al.* (16) insisted, further studies are needed, e.g., those in which the dose of remifentanyl is changed. Furthermore, we could reconfirm that PI increased and PVI decreased upon release of the pneumoperitoneum. Because PVI based on a plethysmographic waveform can be affected by several factors, PI and PVI may be more fragile than SVV, and therefore, PI and PVI values must be estimated cautiously during and after pneumoperitoneum.

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