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ORIGINAL ARTICLE

Pneumoperitoneum affects stroke volume variation in humans

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Abstract
Purpose Stroke volume variation (SVV) is affected by many factors. Although elevated intra-abdominal pressure and a pneumoperitoneum have been shown to increase SVV in animals, a recent human study showed that SVV did not change as a pneumoperitoneum was established. However, we considered the results of this study questionable, and we therefore attempted to study whether SVV changes both before and after pneumoperitoneums in humans.
Methods We performed a prospective observational study in 19 patients undergoing cholecystectomy or colectomy while on mechanical ventilation. Immediately before pneumoperitoneum, baseline registrations of variables were obtained (baseline I), which were measured every min for 5 min after the pneumoperitoneum was initiated. Immediately before the pneumoperitoneum was released, another baseline registration of variables was obtained (baseline II); these variables were then measured every min for 5 min.
Results After the pneumoperitoneum was initiated, there were significant increases in SVV at the 2- to 5-min time points. After release of the pneumoperitoneum, there were significant decreases in SVV at the 1- to 5-min time points.
Conclusion A pneumoperitoneum increased SVV, which is similar to the findings of previous animal studies but is different from a previous clinical study. Upon release of the pneumoperitoneum, SVV decreased significantly, which is new information. SVV values must be estimated cautiously during a pneumoperitoneum.

Keywords Pneumoperitoneum · Stroke volume variation · Hemodynamics

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Introduction

Stroke volume variation (SVV) is one of the most extensively investigated dynamic parameters [1], and Zhang et al. [1] recently demonstrated in their systematic review that (1) the baseline SVV was correlated with fluid responsiveness (changes in cardiac output or stroke volume), and (2) SVV could predict fluid responsiveness. SVV can be affected by various factors, and we recently reported that the rapid infusion of fluid may significantly influence this parameter [2], that SVV can be affected by induced hypertension and hypotension [3] and by induced hypotensive anesthesia [3], and that it is affected by landiolol [4, 5], an

ultra-short-acting adrenergic β_1 receptor blocking agent [4]. SVV can also be affected by factors such as intravascular volume status [6], depth of airway pressure and tidal volume [7, 8], and increased intra-abdominal pressure (IAP) and/or pneumoperitoneum [9–13]. Although elevated IAP and/or pneumoperitoneum have been shown to increase SVV in animals [9–12], Høiseth et al. [13] recently showed in humans that SVV did not change as a pneumoperitoneum was established. However, we considered the results of Høiseth et al. [13] questionable, and our motives for this research were that (1) the results of the animal study were different from those of the human study, and (2) we always experienced SVV changes after pneumoperitoneum is started and after pneumoperitoneum release. We therefore attempted to study whether SVV changes both before and after pneumoperitoneum in humans.

Materials and methods

We conducted a 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 FK-61, 2012-2-24), and we registered this study in the "UMIN Clinical Trial Registry" (ID: UMIN000009316). 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 American Society of Anesthesiologists (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 a preoperative fast for at least 8 h, and no premedication was given to any of the patients.

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 [14, 15]. Anesthesia consisted of 1 % lidocaine epidural anesthesia, and the analgesia level was determined by a pinprick 15 min after administration of the epidural lidocaine.

Induction of general anesthesia was performed with propofol (initial effect-site concentration = 4 μg/mL) administered by a plasma target-controlled infusion, 1-μg/kg remifentanil intravenously (IV), and a 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 patient's lungs were mechanically ventilated by means of a semi-closed circle system at a fresh gas flow of 6 L/min (O_2, 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 ropivacaine, remifentanil at a rate of 0–0.5 μg/kg/min, and rocuronium. We achieved a target bispectral index (BIS) between 40 and 60, and stable circulatory variables during surgery. After surgical skin preparation, the abdomen was insufflated with CO_2 to create and maintain a pneumoperitoneum at 10 mmHg.

Systolic arterial pressure (SAP), diastolic arterial pressure (DAP), heart rate (HR), cardiac output (CO), SVV, stroke volume index (SVI), systemic vascular resistance (SVR), and end-tidal CO_2 ($P_{ET}CO_2$) pressure were continuously monitored with a standard monitor (S/5 Anesthesia Monitor, GE Healthcare, Helsinki, Finland) and the FloTrac/Vigileo™ system (Version 03.06; Edwards Lifesciences, Irvine, CA, USA). We did not insert central venous catheters 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 [4].

Immediately before the pneumoperitoneum, baseline registrations of the variables were obtained (baseline I), and these variables were measured every min for 5 min after the pneumoperitoneum was initiated. Immediately before the 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 the pneumoperitoneum. The position of the patient during measurements was horizontal. CO, SVV, SVI, and SVR were recorded 20 s after SAP, DAP, HR, and $P_{ET}CO_2$ were recorded because the Vigileo™ system samples the pressure waveform at 100 Hz over 20 s to capture 2,000 data points for analysis, and parameter calculations are provided at the end of every 20-s timeframe [2, 16].

For laparoscopic cholecystectomy, before general anesthesia/epidural block induction, a crystalloid at a volume of at least 10 mL/kg was infused followed by an additional 10–15 mL/kg during the laparoscopic procedure [17]. For laparoscopic colectomy, before general anesthesia/epidural block induction, a 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/h of a crystalloid plus 3 mL/kg/h of a 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

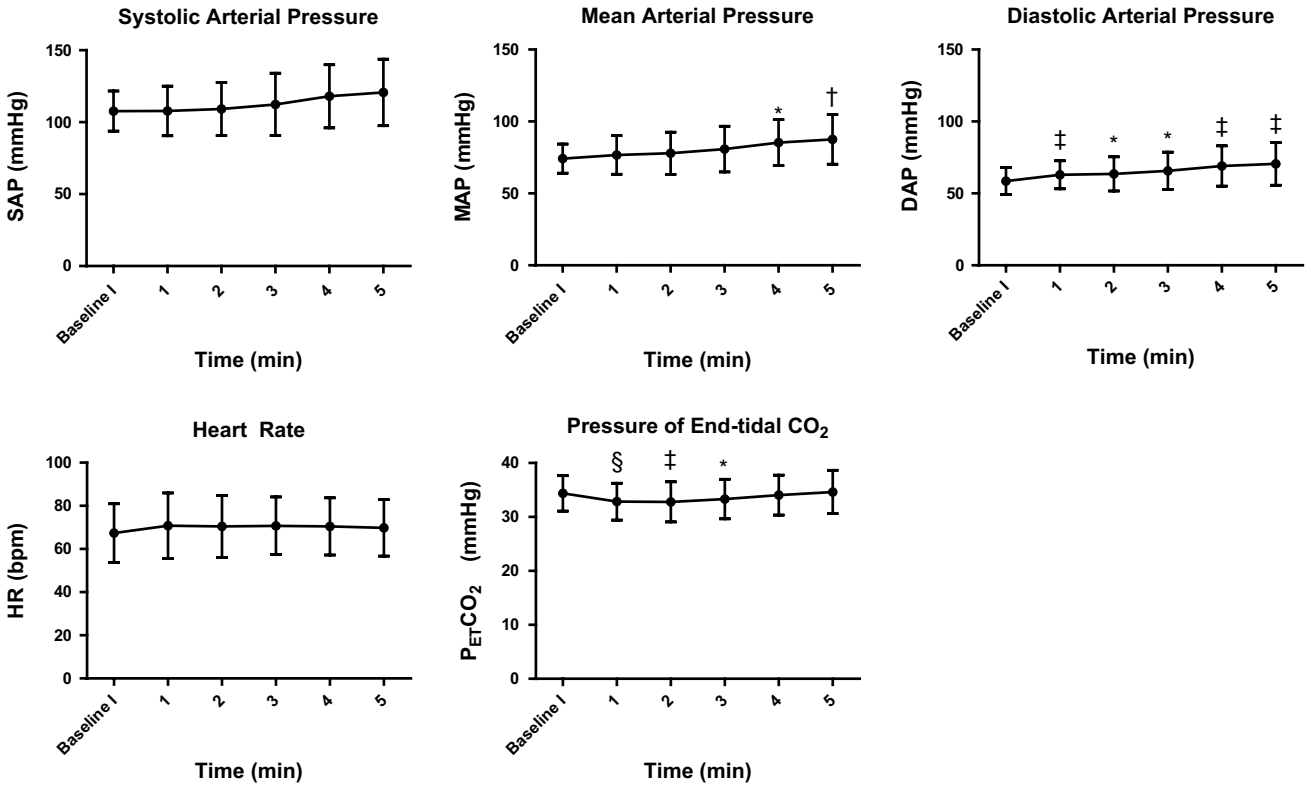

Fig. 1 Sequential changes in systolic arterial pressure (SAP), mean arterial pressure (MAP), diastolic arterial pressure (DAP), heart rate (HR), and end-tidal CO_2 ($P_{ET}CO_2$) pressure at baseline I and after the pneumoperitoneum. Data are expressed as the mean ± standard deviation. *$P < 0.05$ vs baseline I; †$P < 0.01$ vs baseline I; ‡$P < 0.005$ vs baseline I; §$P < 0.001$ vs baseline I

a predetermined critical hemoglobin level for blood transfusion was reached [17]. Vasopressors were administered as needed.

Statistical analyses

Sample size was estimated from preliminary data obtained from eight patients. An assumption was made that a four-point change in SVV between the baseline I values and those 3 min after the pneumoperitoneum started would be clinically relevant. Power analysis suggested that a minimum of 16 patients would be needed for $\beta = 0.1$ and $\alpha = 0.05$. To compensate for potential dropouts, we enrolled 19 patients in this study. This analysis was performed using GraphPad StatMate 2.00 (GraphPad Software, Inc., La Jolla, CA, USA).

Values are expressed as the mean ± 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's *t* test with Bonferroni's correction to determine whether there were significant differences between baseline and the parameters 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.).

Results

The 19 patients completing the study had an average (mean ± SD) age of 62 ± 12 years, a body weight of 66 ± 15 kg, a height of 163 ± 8 cm, and body surface area of 1.71 ± 0.19 m^2. The male:female ratio was 14:5 and the cholecystectomy:colectomy ratio was 11:8. No patients received a blood transfusion during surgery.

After the pneumoperitoneum was initiated, significant increases occurred in MAP at the 4- to 5-min time points, in DAP at the 1- to 5-min time points, in SVV at the 2- to 5-min time points, and in SVR at the 1- to 5-min time points compared with the baseline I values (Figs. 1, 2). Significant decreases occurred in SVI at the 1-min time point and in $P_{ET}CO_2$ at the 1- to 3-min time points compared with the baseline I values (Figs. 1, 2). Other values were unchanged (Figs. 1, 2).

After release of the pneumoperitoneum, significant increases occurred in SVI at the 4- to 5-min time points and in $P_{ET}CO_2$ at the 1-min time point compared with

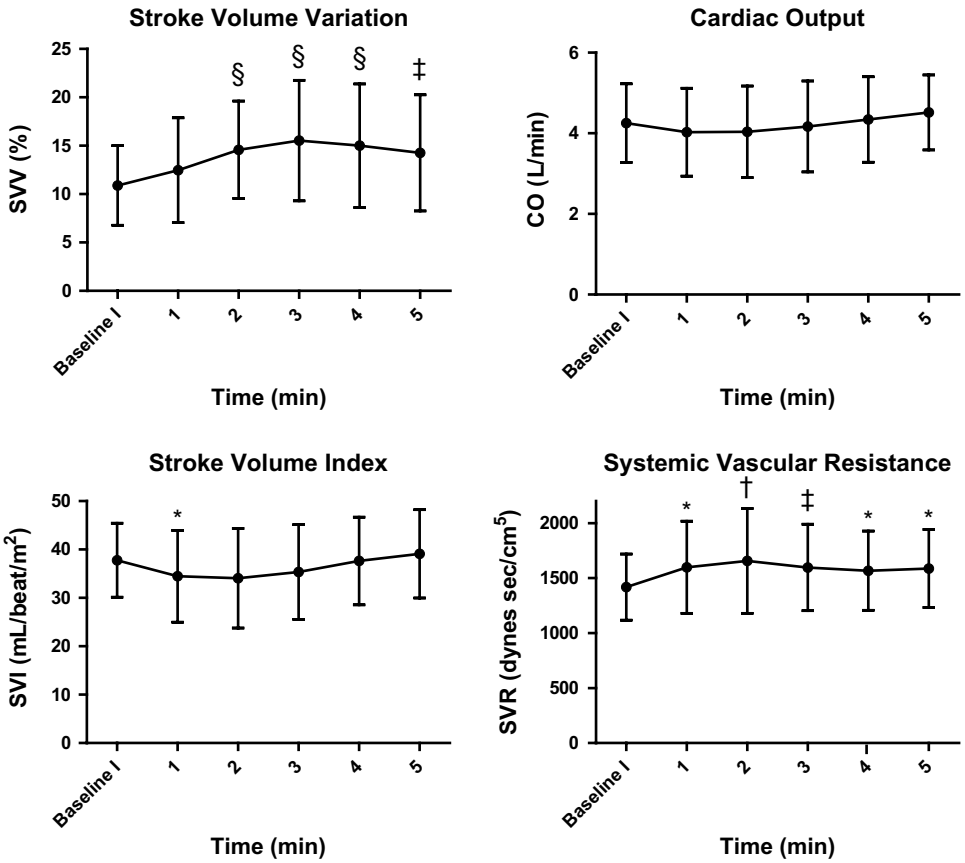

Fig. 2 Sequential changes in stroke volume variation (SVV), cardiac output (CO), stroke volume index (SVI), and systemic vascular resistance (SVR) at baseline I and after the pneumoperitoneum. Data are expressed as the mean ± standard deviation. *$P < 0.05$ vs baseline I; †$P < 0.01$ vs baseline I; ‡$P < 0.005$ vs baseline I; §$P < 0.001$ vs baseline I

the baseline II values (Figs. 3, 4). Significant decreases occurred in DAP at the 1- to 3-min time points, in HR at the 2-min time point, and in both SVV and SVR at the 1- to 5-min time points compared with the baseline II values (Figs. 3, 4). SAP, MAP, and CO values were unchanged (Figs. 3, 4). Airway pressures during measurements are shown in Fig. 5.

Discussion

In this study, a pneumoperitoneum increased SVV, whereas upon release of the pneumoperitoneum, SVV decreased significantly. In all reported animal studies [9–12], SVV increased after elevation of IAP and/or a pneumoperitoneum, similar to our results, but our results were different from those reported by Høiseth et al. [13] in their human study. Many animal studies showed that other dynamic indices such as systolic pressure variation and pulse pressure variation [1, 18] also increased during intra-abdominal hypertension (IAH) [9–12, 19–21]. Furthermore, we found that SVV decreased after the pneumoperitoneum was stopped, which is, to our knowledge, new information for human case studies.

There are several possible mechanisms of SVV increase after a pneumoperitoneum. First, Valenza et al. [12] investigated the effects in pigs of IAH induced by helium inflation of the abdomen on esophageal and central venous pressure considering values obtained at end-expiration (i.e., in static conditions) and during tidal volume delivery (i.e., in dynamic conditions). They commented that the trend of the indices was in favor of a decrease in preload, as shown by a decline in cardiac output and as underlined by the increase in SVV. They also found that the effect of IAH on volumetric indices (independent from changes of pleural pressure) was opposite to that of central venous pressure: intrathoracic blood volume did not change, whereas the mean central venous pressure rose significantly. Second, decreasing inferior vena cava flow (which is "preload dependent") could be one of the mechanisms [9]. Third, IAH induces an increase in right ventricular afterload (which is "preload independent") [10], possibly resulting in an increase in respiratory variations in right ventricular SV [10]. Fourth, IAH induces an increase in pleural pressure swing itself (which is "preload independent") [9]. Furthermore, SVV is defined as SVV (%) = 100 × (SVmax − SVmin)/[(SVmax + SVmin)/2], where SV = stroke volume and maximal and minimal values for SV are determined as

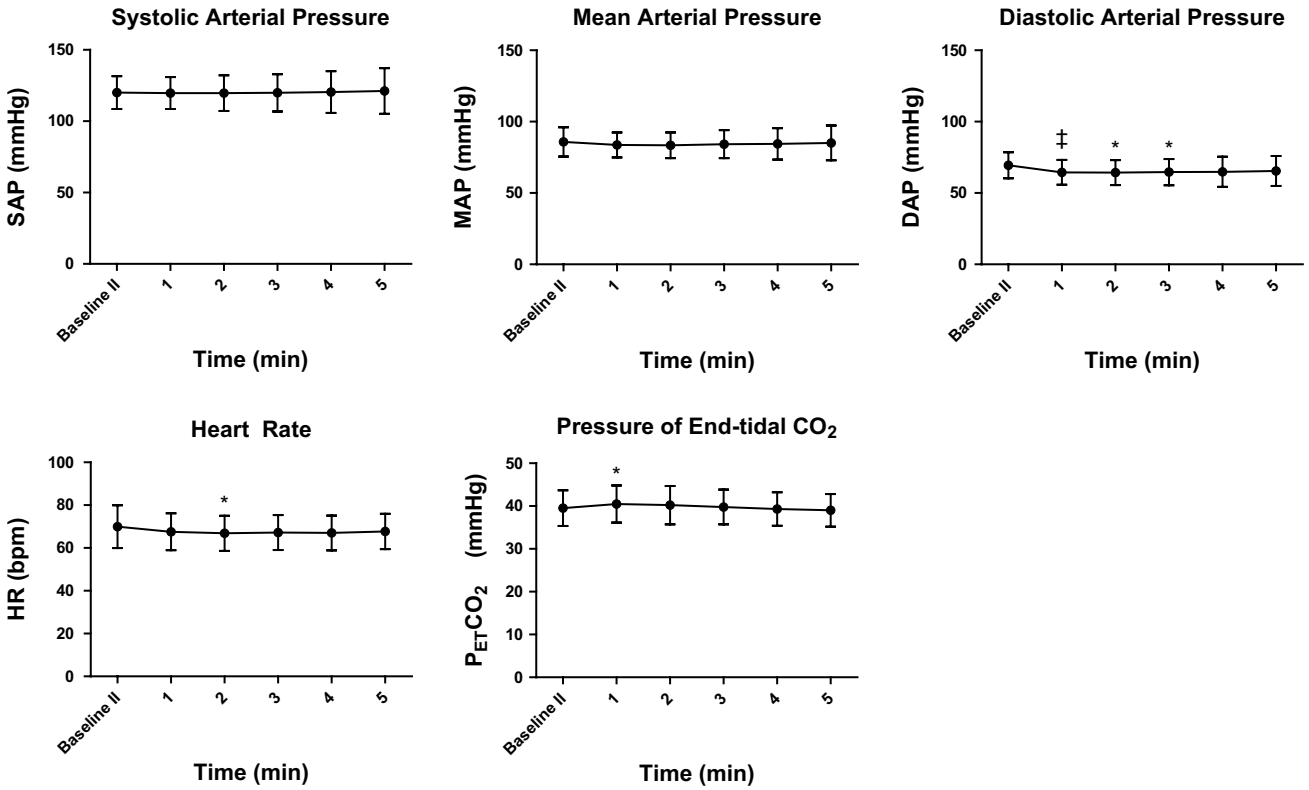

Fig. 3 Sequential changes in systolic arterial pressure (SAP), mean arterial pressure (MAP), diastolic arterial pressure (DAP), heart rate (HR), and end-tidal CO_2 ($P_{ET}CO_2$) pressure at baseline II and after stopping the pneumoperitoneum. Data are expressed as the mean ± standard deviation. *$P < 0.05$ vs baseline II; ‡$P < 0.005$ vs baseline II

SVmax and SVmin, respectively, over a single respiratory cycle of paced breathing [2–4]. In this study, SVI decreased significantly at the 1-min time point after a pneumoperitoneum was initiated (Fig. 2), and SVI increased significantly at the 4- to 5-min time points after release of the pneumoperitoneum (Fig. 4). Some animal studies showed that SV decreased after IAP increased [9, 10].

Our results relating to SVV after a pneumoperitoneum were different than those of Høiseth et al. [13], and it is not clear what caused this discrepancy. However, we suppose that the study design was different. For example, Høiseth et al. [13] used the 03.02 version of FloTrac while we used version 03.06. Also, the anesthesia methods were different (tidal volumes [6–8] were different, and Høiseth et al. [13] applied positive end expiratory pressure of 5 cm H_2O, whereas we used zero end expiratory pressure). The patient characteristics were not similar: the height and weight of the Høiseth et al. [13] patients were much higher than those of our patients. Furthermore, the baseline SVV value in the Høiseth et al. [13] study was 9 %, and the value during the pneumoperitoneum increased (10 %), but not significantly so. We wonder whether their sample number might be rather small, and we also believe that it is questionable that the SVV value did not increase significantly during the pneumoperitoneum because the SV in their study decreased by 20 % during the pneumoperitoneum compared to the baseline value. Because we found that SVV decreased after the pneumoperitoneum was stopped, which is new information to our knowledge, we strongly believe that SVV must be changed by a pneumoperitoneum.

We consider the increase in MAP and DAP and the decrease in SVI after the pneumoperitoneum was initiated to be due to the increase in SVR (increase in afterload), and that the decrease in DAP and increase in SVI after release of the pneumoperitoneum are due to the decrease in SVR (decrease in afterload). Further, we surmise that the cause of the decrease in $P_{ET}CO_2$ after the pneumoperitoneum was initiated was due to the decrease of CO (although there were no significant changes in CO after the pneumoperitoneum was initiated), and that of the increase in $P_{ET}CO_2$ after release of the pneumoperitoneum was due to the increase of CO (although CO did not change significantly after release of the pneumoperitoneum). Changes in $P_{ET}CO_2$ qualitatively reflect changes in CO during acute hemodynamic perturbations in anesthetized patients during constant ventilation [22].

There are several limitations associated with our study. We measured SVV values during the 5-min periods

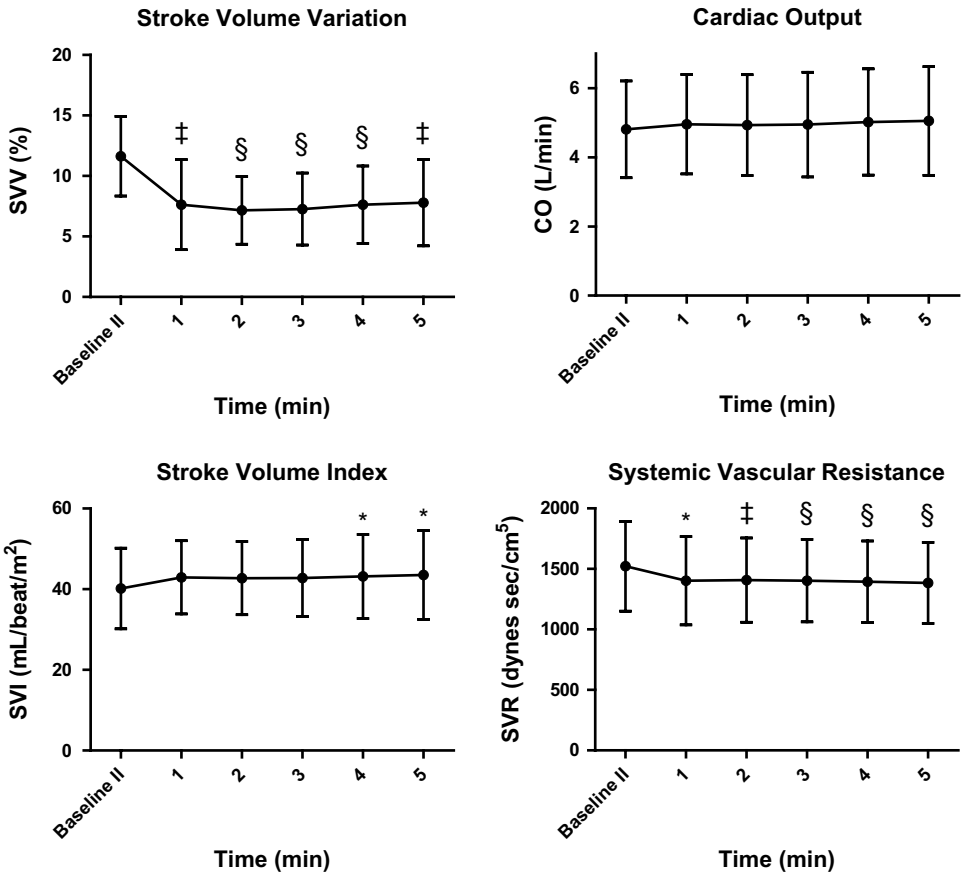

Fig. 4 Sequential changes in stroke volume variation (SVV), cardiac output (CO), stroke volume index (SVI), and systemic vascular resistance (SVR) at baseline II and after stopping pneumoperitoneum. Data are expressed as the mean ± standard deviation. $^{*}P < 0.05$ vs baseline II; $^{\ddagger}P < 0.005$ vs baseline II; $^{\S}P < 0.001$ vs baseline II

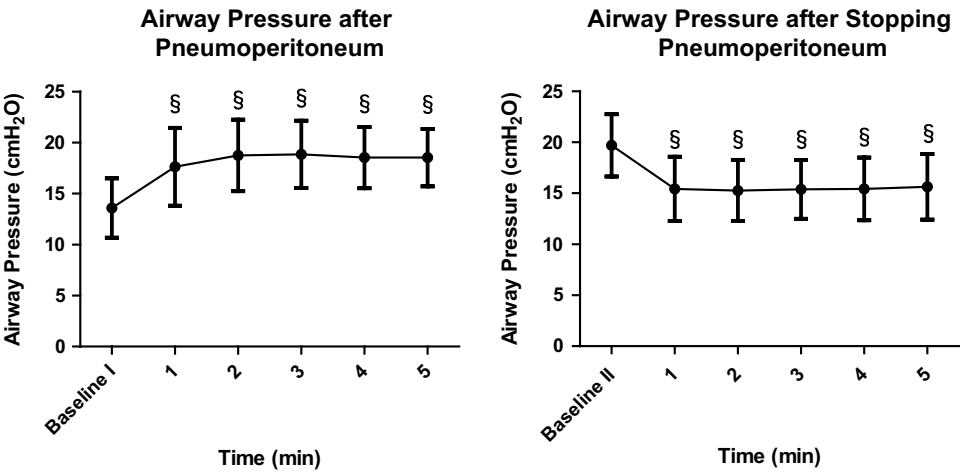

Fig. 5 Sequential changes in airway pressure after the pneumoperitoneum and after stopping the pneumoperitoneum. Data are expressed as the mean ± standard deviation. $^{\S}P < 0.001$ vs baseline I and II

immediately after a pneumoperitoneum was initiated and stopped, and we did not record SVV values during the interval when the pneumoperitoneum was present. However, the SVV values generally remained higher than baseline I values during surgery. We can surmise the SVV values during surgery from baseline II values. Zhang et al. [1] reported that SVV can predict fluid responsiveness across a wide spectrum of clinical settings and showed that the SVV cutoff value ranged from 8.5 to 15.5 %. However, we did not try to determine whether SVV could predict fluid responsiveness in our mechanically ventilated patients undergoing laparoscopic surgery. Although Høiseth et al. [13] showed that SVV predicted fluid responsiveness relatively poorly 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, rather we obtained the data for SVR using a fixed CVP (= 0 mmHg) by inputting the pressure into the

FloTrac/Vigileo™ system as described above [4]. Donati et al. [23] reported that after induction of a 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 summary, pneumoperitoneum increased SVV, which is similar to the findings of previous animal studies but opposite that of a previous clinical study. Upon release of the pneumoperitoneum, SVV decreased significantly, which is new information. We believe that there are several mechanisms for the increase in SVV after a pneumoperitoneum in humans and that SVV values must be estimated cautiously during pneumoperitoneums.

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Conflict of interest No author declares any conflicts of interest.

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