

Evaluation of stroke volume variation and pulse pressure variation as predictors of fluid responsiveness in patients undergoing protective one-lung ventilation

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Summary

In order to investigate whether the hemodynamic indices, including stroke volume variation (SVV) and pulse pressure variation (PPV) could predict fluid responsiveness in patients undergoing protective one-lung ventilation. 60 patients scheduled for a combined thoracoscopic and laparoscopic esophagectomy were enrolled and randomized into two groups. The patients in the protective group (Group P) were ventilated with a tidal volume of 6 mL/kg, an inspired oxygen fraction (FiO₂) of 80%, and a positive end expiratory pressure (PEEP) of 5 cm H₂O. Patients in the conventional group (Group C) were ventilated with a tidal volume of 8 mL/kg and a FiO₂ of 100%. Dynamic variables were collected before and after fluid loading (7 mL/kg hydroxyethyl starch 6%, 0.4 mL/kg/min). Patients whose stroke volume index (SVI) increased by more than 15% were defined as responders. Data collected from 45 patients were finally analyzed. Twelve of 24 patients in Group P and 10 of 21 patients in Group C were responders. SVV and PPV significantly changed after the fluid loading. The receive operating characteristic (ROC) analysis showed that the thresholds for SVV and PPV to discriminate responders were 8.5% for each, with a sensitivity of 66.7% (SVV) and 75% (PPV) and a specificity of 50% (SVV) and 83.3% (PPV) in Group P. However, the thresholds for SVV and PPV were 8.5% and 7.5% with a sensitivity of 80% (SVV) and 90% (PPV) and a specificity of 70% (SVV) and 80% (PPV) in Group C. We found SVV and PPV could predict fluid responsiveness in protective one-lung ventilation, but the accuracy and ability of SVV and PPV were weak compared with the role they played in a conventional ventilation strategy.

Keywords: Stroke volume variation, pulse pressure variation, one lung ventilation, protective ventilation, fluid responsiveness

1. Introduction

One-lung ventilation (OLV) is necessary for thoracic surgery since the surgery requires adequate visualization of the operative field. OLV can cause a series of pathophysiological changes, such as an imbalance in the ventilation-perfusion ratio, increased intrapulmonary shunt, and increased risk of pulmonary edema. In

recent years, along with the advances in the equipment and theory of mechanical ventilation, lung protective ventilation strategies have gained popularity in clinical practice. The core mechanism underlying this strategy is to use low tidal volume ventilation with positive end expiratory pressure (PEEP) in an attempt to reduce shear stresses generated by repeated alveolar inflation through mechanical ventilation, reduce the risk of lung injury, and thereby improve prognosis (1-3).

However, proper fluid infusion in perioperative settings is also critical for maintaining the balance between oxygen supply and demand. Volume monitoring and evaluation of the patient's response to fluid loading are particularly essential in maintaining sufficient blood volume to ensure organ perfusion in

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cases where the risk of pulmonary edema is increased. Conventional hemodynamic parameters cannot fulfill these requirements, and other monitoring strategies such as the use of a floating pulmonary artery catheter and transesophageal echocardiography are not advocated due to concerns about risk, cost, and technology (4,5). Therefore, stroke volume variation (SVV) and pulse pressure variation (PPV) have attracted increasing attention, and a growing number of studies have been conducted on these parameters due to their minimally invasive nature and accuracy (6-9). Previous studies have demonstrated that the hemodynamic parameters represented by SVV can predict fluid responsiveness in patients undergoing thoracic surgery but the accuracy was affected by many factors including the depth of tidal volume, the use of PEEP and so on (10-13), which limits its application in patients undergoing OLV. For this reason, the aim of this study was to investigate the ability of SVV, PPV and other hemodynamic parameters to predict fluid responsiveness in patients undergoing protective OLV.

2. Methods

2.1. Ethics and patients

This study was approved by the Institutional Ethics Committee of People's Liberation Army General Hospital, Beijing, China and written informed consent were obtained from all patients before surgery. Sixty ASA I-II patients (age, 18-70 years; BMI, 18-30 kg/m²) undergoing elective radical esophagectomy of esophageal carcinoma using combined laparoscopic and thoracoscopic approaches were enrolled in this study from December 2013 to July 2014 at People's Liberation Army General Hospital. Patients were otherwise healthy without severe cardiopulmonary diseases, coagulation disorders, hepatic or renal dysfunction prior to surgery.

2.2. Anaesthesia management

Atropine (0.5mg) was administered intramuscularly 30 min before surgery. Surgery was performed with the patient in a supine position. An oxygen mask and peripheral venous lines were kept ready, if needed, for further intervention. An electrocardiogram, non-invasive blood pressure measurement and oxygen saturation were recorded. General anesthesia was induced with midazolam (0.03 mg/kg), sufentanil (0.3 µg/kg), propofol (1.5 mg/kg), and rocuronium (0.9 mg/kg). Endotracheal intubation was performed using double-lumen endotracheal tube (Broncho-cath, Tyco Healthcare, Argyle, Mansfield, MA, USA) 3 min after anesthetic induction. After accurate positioning, patients received mechanical ventilation on volume-controlled mode with fractional inspired oxygen concentration (FiO₂)

of 100%, a tidal volume of 8 mg/kg, respiratory rate of 12 breaths/min, respiratory ratio of 1:2, and end-tidal CO₂ partial pressure (P_{ET}CO₂) of 30-35 mmHg. Arterial catheterization was performed *via* a puncture of the left radial artery and was connected to the FloTrac-Vigileo system (Edwards Lifescience, LLC, Irvine, CA, USA). Hemodynamic parameters were recorded after zeroing and entering patient data. Anesthesia was maintained with sevoflurane at an inspired concentration of 1-1.5% and propofol and remifentanyl were delivered with an infusion pump. The depth of anesthesia was controlled to maintain a bispectral index (BIS) (Aspect Medical Systems Inc., Natick, MA, USA) value between 40-60. Rocuronium and sufentanil were administered if needed.

2.3. Study protocol

After the procedure of laparoscopic part, patients were placed in a lateral position and the position of the monitoring system was adjusted accordingly. Five minutes after zeroing the system, SVV, PPV, mean arterial pressure (MAP), heart rate (HR), cardiac output (CO), cardiac index (CI), stroke volume index (SVI), maximum airway pressure (P_{MAX}) and P_{ET}CO₂ were recorded. After starting OLV, patients were randomized into two ventilation strategic groups: Group P (protected group, tidal volume: 6 mL/kg, PEEP: 5 cmH₂O, and FiO₂: 80%) and Group C (conventional group, tidal volume: 8 mL/kg, FiO₂: 100% without PEEP). For the randomization method, the random numbers with odd mantissa were included in the protective group and those with even mantissa were included in the conventional group. For both groups, each patient's respiratory ratio was adjusted to maintain P_{ET}CO₂ ≤ 45 mmHg and plateau pressure ≤ 35 cmH₂O. The first set of data (before fluid loading) was recorded 0.5 h after OLV followed by intravenous infusion of hydroxyethyl starch (130/0.4) 7 mL/mg at a speed of 0.4 mL/kg/min. The second set of data (after fluid loading) was recorded 5 min after stabilization of the data. The test designer was responsible only for data collecting rather than grouping and anesthesia management.

In order to maximize the accuracy and reliability of the data derived from the transducer of the system, the exclusion criterion during the study were as followed: (a) surgical procedures altered; (b) incidents that affects the stability of the respiratory/circulatory system; (c) non-sustainable OLV; (d) repeatedly vasoactive drugs used.

2.4. Statistical analysis

A responder was defined as a subject with a demonstrated increase of > 15% in SVI; otherwise, the patient was defined as a non-responder. Statistical analysis was performed using SPSS 17.0 software (SPSS Inc., Chicago, IL, USA). Quantitative data were presented as mean ± standard deviation ($\bar{x} \pm s$). Normality and

homogeneity of variance were verified. Patient general information and data before and after volume expansion were compared using univariate analysis of variance (ANOVA). For responders, a correlation between pre-fluid loading SVV, PPV and Δ SVI were analyzed, and the predictive capacity of SVV and PPV was tested by receiver operating characteristic analysis (ROC) to determine the specificity, sensitivity and threshold of these parameters.

3. Results

3.1. Patients demographics

At last, Data collected from forty-five patients were

analyzed. The Group P ($n = 24$) comprised 12 patients who had a response (responders) and 12 patients with no response (non-responders), and Group C ($n = 21$) had 10 responders and 11 non-responders. No significant differences were observed in age, gender, BMI, prior medical history and cardiac ejection fraction either between groups or within groups (Figure1 and Table1).

3.2. Comparasion of hemodynamic variables

The CO, CI, and SVI of responders in both groups significantly increased after fluid loading. Significant changes were observed in SVV and PPV after fluid loading while no significant changes were observed in

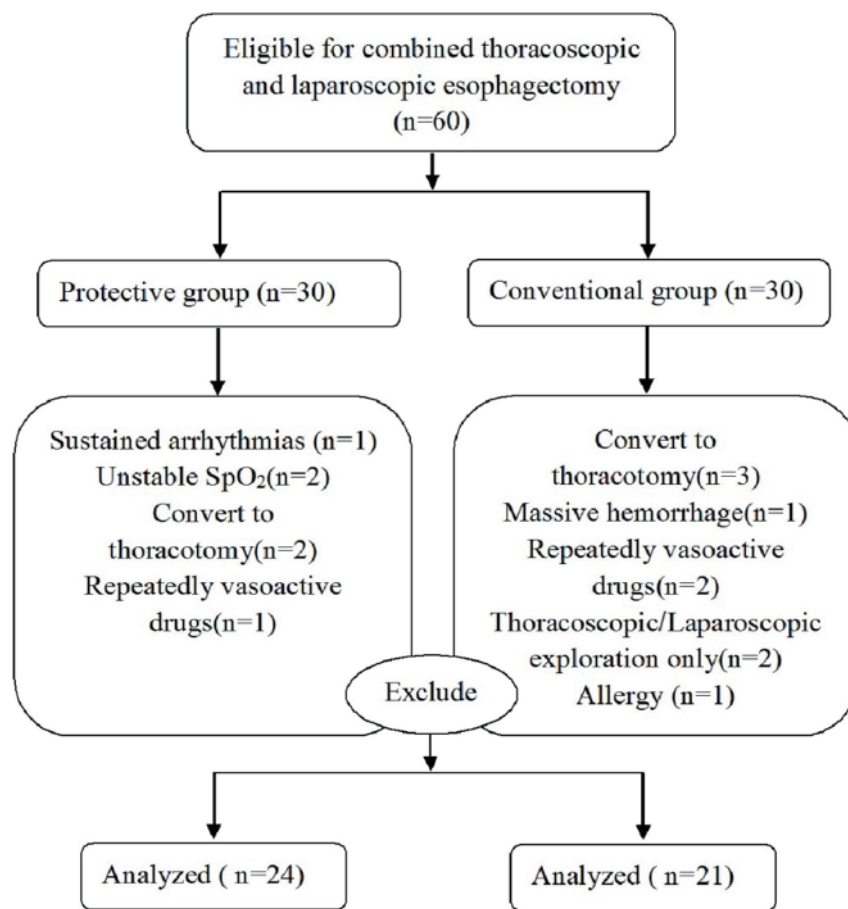


Figure 1. Flow chat of patients selection and distribution.

Table 1. Comparison of patients' general conditions between groups ($\bar{x} \pm s$)

Groups	Group P (n = 24)			Group C (n = 21)			p1	p2	p3
	overall	R (n = 12)	NR (n = 12)	overall	R (n = 10)	NR (n = 11)			
Gender (F/M)	2/22	1/11	1/11	0/21	0/10	0/11	/	/	/
age	60.63 ± 6.16	61.00 ± 6.67	60.25 ± 5.87	61.57 ± 4.91	60.50 ± 4.74	62.55 ± 1.53	0.58	0.79	0.64
BMI	23.03 ± 1.79	23.33 ± 1.73	22.74 ± 1.87	23.31 ± 1.64	23.37 ± 1.71	23.25 ± 1.65	0.59	0.18	0.89
Ejection fraction	63.38 ± 3.50	63.42 ± 3.29	63.33 ± 3.85	63.43 ± 2.73	63.90 ± 2.69	63.00 ± 2.83	0.96	0.92	0.68
History of hypertension	7/24	4/12	3/12	5/21	2/10	3/11	/	/	/
History of diabetes	3/24	2/12	1/12	2/21	1/10	1/11	/	/	/

R: Responders; NR: Non-Responders; p1 value: Group C vs. Group P; p2 value: intra-Group P comparison; p3 value: intra-Group C comparison.

Table 2. Comparison of hemodynamic parameters and relevant variables before and after fluid loading during protective OLV ($\bar{x} \pm s$)

Items	Responders (n = 12)			Non-Responders (n = 12)		
	Before	After	p value	Before	After	p value
MAP (mmHg)	77.75 ± 5.82	80.08 ± 6.44	0.36	77.42 ± 6.31	78.83 ± 7.19	0.61
HR (beat/min)	74.92 ± 5.79	72.42 ± 6.54	0.33	68.25 ± 8.06	67.83 ± 5.69	0.89
SVV (%)	9.67 ± 2.01	7.41 ± 2.19	0.01**	6.75 ± 1.96	6.33 ± 1.87	0.60
PPV (%)	8.91 ± 2.06	6.92 ± 1.93	0.02**	5.67 ± 1.23	5.08 ± 1.16	0.25
CO (L/min)	4.85 ± 1.10	6.10 ± 0.95	0.007*	5.79 ± 1.23	6.03 ± 1.32	0.65
CI (L/min/m ²)	2.82 ± 0.53	3.61 ± 0.51	0.001*	3.30 ± 0.57	3.42 ± 0.59	0.63
SVI (mL/m ²)	38.00 ± 7.56	49.08 ± 8.99	0.004*	48.17 ± 8.39	50.17 ± 9.08	0.58
P _{MAX} (cmH ₂ O)	25.75 ± 1.71	25.58 ± 1.72	0.82	26.08 ± 2.50	26.00 ± 2.45	0.94
P _{ET} CO ₂ (mmHg)	35.08 ± 1.93	35.33 ± 1.23	0.71	35.67 ± 1.97	35.58 ± 1.78	0.91

MAP: mean arterial pressure; HR: heart rate; SVV: stroke volume variation; PPV: pulse pressure variation; CO: cardiac output; CI: cardiac index; SVI: stroke volume index; P_{MAX}: maximum airway pressure; * $p < 0.01$, ** $p < 0.05$.

Table 3. Comparison of hemodynamic parameters and relevant variables before and after fluid loading during conventional OLV ($\bar{x} \pm s$)

Items	Responders (n = 10)			Non-Responders (n = 11)		
	Before	After	p value	Before	After	p value
MAP (mmHg)	80.50 ± 3.34	85.00 ± 2.58	0.003*	77.18 ± 5.09	79.73 ± 3.07	0.17
HR (beat/min)	71.70 ± 5.64	69.70 ± 6.06	0.46	71.91 ± 7.50	69.82 ± 6.19	0.48
SVV (%)	9.90 ± 1.52	6.90 ± 2.02	0.001*	7.64 ± 1.57	6.73 ± 2.05	0.26
PPV (%)	9.50 ± 1.43	6.20 ± 2.20	0.001*	6.63 ± 1.69	6.01 ± 2.07	0.51
CO (L/min)	4.99 ± 1.24	6.30 ± 1.34	0.036**	5.52 ± 1.18	5.66 ± 1.26	0.78
CI (L/min/m ²)	2.89 ± 0.54	3.57 ± 0.57	0.013**	3.20 ± 0.56	3.28 ± 0.62	0.75
SVI (mL/m ²)	40.60 ± 6.72	53.10 ± 7.65	0.001*	44.91 ± 6.44	46.00 ± 7.96	0.72
P _{MAX} (cmH ₂ O)	25.50 ± 2.17	25.40 ± 2.79	0.93	25.73 ± 2.28	26.18 ± 2.13	0.64
P _{ET} CO ₂ (mmHg)	34.90 ± 2.23	34.20 ± 2.15	0.48	35.09 ± 1.37	36.18 ± 1.94	0.14

MAP: mean arterial pressure; HR: heart rate; SVV: stroke volume variation; PPV: pulse pressure variation; CO: cardiac output; CI: cardiac index; SVI: stroke volume index; P_{MAX}: maximum airway pressure; * $p < 0.01$, ** $p < 0.05$.

MAP and HR. As for non-responders, no significant changes were observed in CO, CI, and SVI after fluid loading, either in SVV or PPV (Tables 2 and 3).

3.3. Correlations analysis

No correlations were found between pre-fluid loading SVV/ Δ SVI in responders of Group P (correlation coefficient $r = 0.412$, $p = 0.184$; $r = 0.256$, $p = 0.422$, respectively). As for responders in Group C, correlations were observed between pre-fluid loading SVV/PPV and Δ SVI ($r = 0.697$, $p = 0.025$; $r = 0.637$, $p = 0.047$, respectively) (Figure 2).

3.4. ROC analysis

The threshold values of SVV and PPV used to discriminate between responders and non-responders were determined using ROC analysis of hemodynamic parameters of responders in both groups. For Group P, the threshold of SVV was 8.5%. The ROC-area under the curve (AUC) for SVV was 0.767 (sensitivity, 66.7%; specificity, 50%). The threshold of PPV was 8.5% with AUC of 0.778 (sensitivity, 75%; specificity, 83.3%). For group C, the threshold of SVV was 8.5%

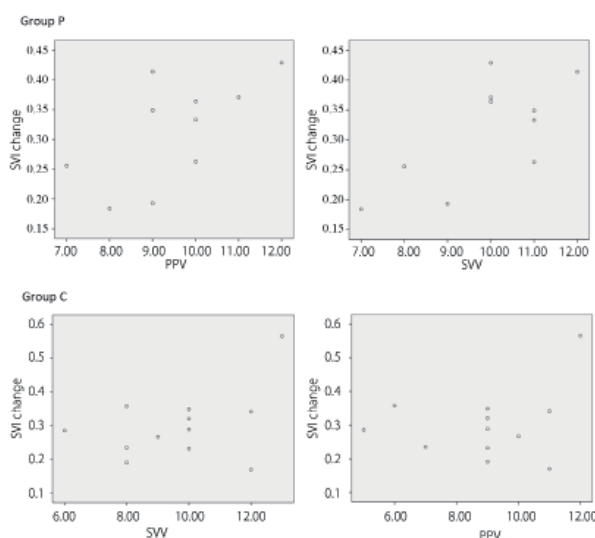


Figure 2. Correlation analysis of the relationship between pre-fluid loading SVV/PPV and Δ SVI in Group P and Group C patients.

with AUC of 0.885 (sensitivity, 80%; specificity, 70%). The threshold of PPV was 7.5% with AUC of 0.890 (sensitivity, 90%; specificity, 80%) (Figure 3).

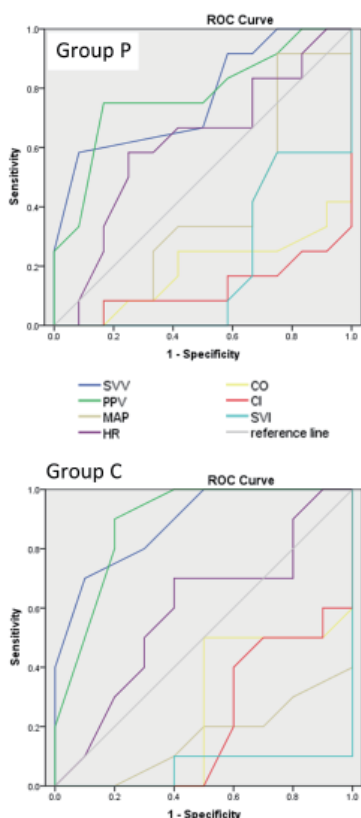


Figure 3. ROC analysis of hemodynamic variables of Responders

4. Discussion

Measurement of SVV by the FloTrac-Vigileo system is derived from variations in venous return blood volume, which is caused by changes in intrathoracic pressure through positive pressure ventilation. Thus, the accuracy of the SVV value depends on the integrity of the pleural cavity, which will be damaged during thoracic surgery and limit the use of SVV monitoring in this kind of procedure. Previous studies have evaluated the predictive value of SVV and other related parameters in open-chest or thoracoscopic surgery and obtained inconsistent results. One study showed that SVV and other dynamic parameters do not necessarily have more predictive potential than static parameters such as central venous pressure (CVP) during OLV (14). However, another study supported the monitoring of these dynamic parameters during thoracoscopic surgery (15). Alternatively, another report demonstrated the limited value of these parameters during open-chest surgery (16) and other volume monitoring strategies were recommended (17,18).

Some studies have suggested that the tidal volume was at least 8 mL/kg during ventilation while using these dynamic parameters as predictors of fluid responsiveness (19,20). However, in an effort to reduce the risk of lung injury, OLV with low tidal volume and proper PEEP protective mode is gradually becoming the mainstream strategy (1-3,21). Some studies have explored the

predictive value of hemodynamic parameters in protective ventilation mode. For instance, Lee *et al.* compared protective ventilation (tidal volume: 6 mL/kg, FiO₂: 50%, PEEP: 5 cmH₂O) with conventional ventilation (tidal volume: 10 mL/kg, FiO₂:100%, no PEEP) in patients undergoing thoracotomy. PPV obtained by transesophageal echocardiography was employed as the predictor of fluid responsiveness. The results showed that PPV could predict fluid responsiveness but only in protective ventilation mode (22). Despite the conflicting results of this study with those of previous studies, the issue of whether or not the PPV calculated directly from echographic imaging findings is more accurate and reliable requires further study.

The FloTrac-Vigileo system obtains the pressure wave signal from any standard peripheral arterial line and automatically adjusts actual vascular compliance based on patient demographic characteristics (age, gender, height and body weight) to obtain the relevant stroke volume. During OLV intervention, the pleura on the operated side is damaged whereas the pleura on the non-operated side remains intact. Mechanical ventilation-induced cyclic changes in intrathoracic pressure can still, to some extent, transmit to pulmonary vessels and the right atrium, thereby affecting cardiac output (23), which is a possible reason for the positive results observed in some studies. Ventilation with a lower tidal volume leads to insufficient cyclic pressure, which definitely influences the accuracy of dynamic parameters. One study has demonstrated that sternotomy, OLV and lateral patient positioning alone can affect the values of dynamic parameters (24), which is the reason for the requirement of a minimum tidal volume of > 8 mL/kg. However, when implementing protective ventilation strategy, increased intrathoracic pressure through persistent PEEP support might, to a certain extent, compensate for the insufficiency of low tidal volume, which explains the result of this study demonstrating that SVV and PPV still predicted fluid responsiveness in patients of Group P, but with poorer sensitivity and specificity than those of Group C.

OLV-induced hypoxic pulmonary vasoconstriction and intrapulmonary shunt can partially affect changes in airway pressure (25), which is the reason for employing P_{MAX} in this study. However, no significant differences were observed in P_{MAX} between patients in Group P and Group C, which is likely a consequence of the specific surgical procedure of combined laparoscopy and thoracoscopy, the specific model of the double-lumen endotracheal tube and male-dominated subjects. More studies are needed to resolve this issue.

There are several limitations of this study. First, the sample size may be insufficient. This is especially important in correlation studies, where sample size might influence the correlation coefficient. Second, esophageal surgery requires more enhanced visualization of the surgical field than pulmonary surgery. Intraoperative

traction used in other operations might cause a variation in the values of detected parameters, thereby affecting the accuracy of the results. Third, the settings of protective ventilation may lack of individualization, which can be resolved by using a dynamic pressure-volume curve to determine the appropriate tidal volume and PEEP value, thus achieving individualized protective OLV (26,27).

In summary, hemodynamic parameters including SVV and PPV can predict fluid responsiveness in patients on protective OLV during radical esophagectomy using a combined laparoscopic and thoracoscopic approach. However, the predictive values of these parameters were not superior to those values detected in conventional OLV mode in terms of sensitivity, specificity and correlation with cardiac output. Their application in clinical practice is still controversial and further studies are required.

Acknowledgements

Dr. Qiang Fu was supported by a Japan-China Sasagawa medical research fellowship. Thanks to Dr. Junko Kouhei and Prof. Makoto Ozaki (Tokyo Women's Medical University) for their kind guidance and help.

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- (Received July 15, 2015; Revised August 16, 2015; Accepted August 25, 2015)*