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Authors

Ji, Fu-Hai

Li, Wen-Jing

Li, Jiang

et al.

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Original article

Responsiveness of stroke volume variation and central venous pressure during acute normovolemic and hypervolemic hemodilution

JI Fu-hai, LI Wen-jing, LI Jiang, PENG Ke, YANG Jian-ping and LIU Hong

Keywords: *hemodilution;stroke volume variation;central venous pressure*

Background Stroke volume variation (SVV) is a robust indicator of fluid responsiveness during volume change. We compared the sensibility of SVV by Vigileo/Flotrac to central venous pressure (CVP) when volume changes in patients undergoing intraoperative acute normovolemic hemodilution (ANH) and acute hypervolemic hemodilution (AHH).

Methods Forty patients were randomly divided into an ANH group (n=20) and an AHH group (n=20). All patients received general anesthesia and were mechanically ventilated. Data were collected from 7 different time-points in the ANH group: baseline, after withdrawal of 5%, 10%, and 15% of the estimated blood volume (EBV) and after replacement with an equal volume of 6% hydroxyethyl starch 130/0.4 (HES) in 5% EBV increments to baseline. There were four time points in the AHH group: baseline, after 5%, 10%, and 15% expansion of the EBV with 6% HES. At each time-point, CVP, SVV and other hemodynamic parameters measurements were obtained.

Results After removal of 10% and 15% EBV, SVV significantly increased from 10.9±3.0 to 14.1±3.4 and 10.9±3.0 to16.0±3.3 (P <0.01), and returned to a final value of 10.6±3.4 after volume replacement. The CVP value was unchanged after removal and replacement of 15% of the EBV. There were no significant changes in SVV after 5%, 10% whereas there was a significant reduction after 15% (8.2±1.7) expansion of the EBV compared with baseline (9.9±1.8) (P=0.033). However, there was a significant increase in CVP after10% (10.3±2.4), 15% (11.3±2.2) expansion of the EBV compared with baseline (8.2±2.7) (P <0.01).

Conclusion SVV is a more sensitive parameter for volume than CVP during hypovolemia, on the contrary CVP is more sensitive than SVV during hypervolemia.

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Establishing and maintaining optimal intravascular volume is vital in the perioperative period. However, it is difficult to estimate whether there is a volume overload or fluid insufficiency under resuscitation. Conventional hemodynamic variables, such as blood pressure (BP), heart rate (HR), central venous pressure (CVP), and even pulmonary artery occlusion pressure (PAOP), are insensitive and sometimes can be misleading in the assessment of circulatory blood volume.[1-3] CVP has traditionally been used to estimate the preload during surgery, but it could be affected by several other factors, e.g., abnormal heart function, blood pressure, thoracotomy, and mechanical ventilation.[4] Therefore, its usefulness in guiding fluid therapy has been recently challenged.[5,6] Stroke volume variation (SVV), on the other hand, has been reported to have acceptable sensitivity and specificity to predict fluid responsiveness.[7-9] The relationship between SVV and the circulatory volume has been reported in graded (static) hypovolemia and hypervolemia models,[10] but not in clinical practice. The correlation between SVV and left ventricular volume measured by 3D transesophgeal echocardiographic has been reported in ANH,[11] although they did not assess the reponsiveness of SVV and CVP, which is most popular index to monitor volume state during ANH and AHH. Therefore, this study was designed to evaluate the responsiveness of SVV and CVP to intravascular volume change during acute normovolemic hemodilution (ANH) and acute hypervolemic hemodilution (AHH) in the clinical settings.

METHODS

Design and setting

Following approval from the Ethics Committee of First Affiliated Hospital of Soochow University, subjects were enrolled in the study after obtaining their written, informed consent. A prospective randomized comparative design was used. The study was conducted in a tertiary-care center.

Patients

A total of 40 subjects were recruited from patients undergoing hip replacement procedures that required ANH or AHH as a routine clinical practice to minimize homologous blood transfusion. Patients with an age of <18 years or >70 years, hematocrit (Hct) <35%, hemoglobin

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Department of Anesthesiology, First Affiliated Hospital of Soochow University, Suzhou, Jiangsu 215006, China (Ji FH, Li WJ, Li J, Peng K and Yang JP)
Department of Anesthesiology and Pain Medicine, University of California, Davis Health System, USA (Ji FH and Liu H)
Correspondence to: JI Fu-hai, Department of Anesthesiology, First Affiliated Hospital of Soochow University, Suzhou, Jiangsu 215006, China (Tel: 86-512-67780055. Email: jifuhai@hotmail.com)
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Conflict of interests: None

(Hb) <12 g/L, arrhythmias, valvular heart diseases, or a history of lung disease, severe coronary artery disease, renal disease, or liver disease were excluded from this study.

Anesthesia

Intravenous (IV) midazolam (0.05–0.01 mg/kg) was administered preoperatively. Upon arrival in the operating room, the patients were oxygenated immediately. Pulse oximetry, echocardiogram (ECG, 5-lead) and noninvasive blood pressure were monitored. General anesthesia was induced by infusing Fentanyl (2–5 μg/kg) and propofol (1–2 mg/kg) after establishing the monitors. Cisatracurium, a neuromuscular blockade, 1.5 mg/kg was used to facilitate endotracheal intubation. Ventilation was controlled to an end tidal CO_2 of 28–32 mmHg by adjusting the respiratory rate, keeping a constant tidal volume of 8 ml/kg and a positive end-expiratory pressure of 5 cmH_2O. No changes to the ventilator settings were made during the study period. General anesthesia was maintained by additional fentanyl and sevoflurane 1.5%-3.0% in a mixture of oxygen and air.

Hemodynamic monitoring

After induction of general anesthesia and before the beginning of surgery, a 14 G central venous catheter was inserted into the right internal jugular. A 20 G arterial catheter was inserted into either the left or right radial artery and connected to a FloTrac/Vigileo system (Edwards Lifesciences, LLC, Irvine, CA), which enables the continuous monitoring of SV, SVV, CO and cardiac index (CI) via the FloTrac™ pressure transducer. Patient data (age, gender, body weight, and height) were entered. After checking the arterial line waveform fidelity, the system was zeroed and the CO measurement was initiated. The FloTrac/Vigileo™ system needs no external calibration and provides continuous cardiac output measurements from the arterial pressure wave. The Vigileo™ (Software version 1.07) records hemodynamic variables at 20-second intervals and performs calculations on the most recent 20 seconds of data. The system calculates SV using arterial pulsatility, resistance, and compliance. The CO is calculated as follows: CO=heart rate×SV. The CO was recorded continuously during the perioperative period. The systemic vascular resistance (SVR) was calculated from CVP and other hemodymanic data. The SVR was calculated as follows: SVR=(MAP-CVP)/CO×80. CVP was measured using a standard central venous catheter inserted via the right internal jugular vein. HR and mean arterial pressure (MAP) were recorded from a standard monitor.

Study protocol

The following hemodynamic variables were recorded 15min post-induction as the baseline data: SBP, DBP, MAP and HR，CVP. The CO, CI, SV, SVI and SVV values were obtained and/or derived from the FloTrac/Vigileo and other standard hemodynamic monitors. The ANH or AHH protocol was then initiated. The method of ANH was the same as the method Kungys and colleagues introduced.[11] The 40 patients were randomly divided into two groups (n=20 in each group): group I, normovolemic hemodilution (ANH group); group II, hypervolemic hemodilution (AHH group). Patients were placed in the supine position. Fifteen percent of the estimated blood volume (EBV) in three 5% EBV aliquots was withdrawn from patients in Group I. Blood was removed via the central venous catheter. The extracted blood was stored in standard citrate phosphate dextrose solution blood packs (Baxter Healthcare Corp, Shanghai, China) and "rocked" to prevent clot formation. The blood was reinfused into patients at the end of their surgeries. After withdrawing 15% of the EBV, an equal volume of 6% HES in sodium chloride solution (Voluven, Fresenius Kabi Corp, Beijing, China) was infused in three 5% EBV aliquots. Each replacement was implemented 5 minutes after hemodynamics were stable. For the patients in group II, 15% of the EBV was expanded with 6% (130/0.5) HES, also in three 5% EBV aliquots. The method of AHH was applied according to Xu's introduce.[12] Ventilator settings were kept constant with a tidal volume of 8 ml/kg during the study. Inotropes or vasopressors were not used.

Statistical analysis

The data are presented as the mean ± standard error (SE). A mixed model analysis of variance (ANOVA) was used for repeated measurements. Dunnett's multiple comparison tests were used for post hoc analysis to determine the specificity of the changes at each time point compared with baseline (SPSS version 16). The relationships between SVV, CVP and the change of EBV were examined using Pearson's correlation. For all comparisons, a P value of <0.05 was considered statistically significant.

RESULTS

Demographic data

There were no statistically significant differences in age, gender, ASA status, BMI, and the incidence of hypertension and diabetes between two groups (P>0.05).

Hemodynamic parameters

Hemodynamic variables during the study are summarized in Tables 1 and 2. CO decreased from (5.2± 0.4) to (3.5± 0.3) L/min after removal of 15% of the EBV and then increased to a final value of (5.1 ± 0.3) L/min after replacement of 15% of the EBV. CO increased from 5.2±0.2 to 6.4 ±0.3 after expansion of 5%, 10%, 15% of the EBV (P<0.05). There are similar changes in CI (Tables 1 and 2). CI decreased significantly during ANH, but increased gradually following the volume expansion during AHH (P<0.05).

CVP

During ANH, CVP had no significant change when the blood volume decreased (P>0.05), and there was no statistical correlation between CVP and the decrease of EBV (r= -0.142, P>0.05) (Figures 1 and 2). However, during AHH, CVP increased significantly following the 10%, 15% expansion of estimated blood volume (EBVE) (8.2±2.7 to 9.8±2.4 and 11.3±2.2, P=0.023), and there was a significant correlation between CVP changes and the

Table 1. Hemodynamic variables of ANH

Items	Baseline	-5%	-10%	-15%	R-10%	R-5%	Final	*P* values
HR (beats/min)	72.4±15.0	71.6±14.3	71.2±11.9	73.4±14.6	66.3±11.1†	65.1±9.1†	67.4±9.5†	0.044
MAP (mmHg)	82.0±12.5	76.8±13.1	74.6±12.4	72.9±13.1	76.0±10.5	80.3±12.3	81.8±13.4	0.078
CO (L/min)	4.2±1.0	3.8±0.7	3.6±0.8	3.1±1.2†	3.7±1.0	4.7±0.9†	4.6±0.7	0.016
CI ($L \cdot min^{-1} \cdot m^{-2}$) *	2.4±0.5	2.2±0.4	2.1±0.4*	1.9±0.3†	2.5±0.6	2.7±0.5	2.9±0.4*	0.001
Hematocrit (%)*	39±3			38±2			34±1^{+}	<0.001

HR: heart rate; MAP: mean arterial blood pressure; CO: cardiac output; SVV: stroke volume variation; CVP: central venous pressure; CI: cardiac eject index. *: a significant change over time, mixed models analysis of variance, P <0.05. †: a significant difference from the baseline value, Dunnett's test, P < 0.05.

Table 2. Hemodynamic variables of AHH

Items	Baseline	+5%	+10%	+15%	*P* values
HR (beats/min)	76.1±13.1	69.8±13.5^{+}	64.6±13.4^{+}	62.9±12.9^{+}	<0.001
MAP (mmHg)	78.4±10.1	78.8±10.0	78.1±7.5	78.5±8.4	0.064
CO (L/min)	4.2±0.7	4.3±0.7	4.5±0.7^{+}	4.8±0.4^{+}	0.049
CI ($L \cdot min^{-1} \cdot m^{-2}$) *	2.3±0.4	2.4±0.4	2.5±0.4	2.7±0.3	<0.001
Hematocrit (%)*	40±2			36±2	<0.001

HR: heart rate; MAP: mean arterial blood pressure; CO: cardiac output; SVV: stroke volume variation; CVP: central venous pressure; CI: cardiac eject index. *: a significant change over time, mixed models analysis of variance, P <0.05. $^{+}$: a significant difference from the baseline value, Dunnett's test, P < 0.05.

EBVE, (r=0.269, P=0.016) (Figures 3 and 4). CO and CI decreased significantly during ANH (P<0.05), but increased gradually following the expansion during AHH (P<0.05).

SVV

During ANH, SVV increased significantly following each EBV reduction (10.9±3.0 to 18.9±2.9, P<0.001), and there was a significant correlation between SVV and the decrease of EBV (r=0.597, P<0.001) (Figure 1 and Figure 5). However, there were no significant changes in SVV after 5%, 10% expansion of the EBV but significant reduction after 15% (8.2±1.7) (P=0.033) compared with baseline (9.9±1.8). In addition, there was no significant correlation between SVV the EBVE (r=–0.163, P>0.05) (Figures 2 and 6).

DISCUSSION

In the present study, we demonstrated that SVV is a sensitive and accurate parameter in detecting hypovolemia during ANH when compared with other cardiac preload parameters. Conversely, CVP is more sensitive than SVV following the change of volume during hypervolemia.

The administration of IV fluid to maintain an effective circulatory volume should be considered a core element of the perioperative practice of anesthesia. [13-15] Hypovolemia may result in tissue hypoperfusion and worsening vital organ functions. However, fluid overload also appears to impede oxygen delivery and compromise surgical outcomes. [16,17] Therefore, it is important to accurately estimate the intravascular volume status in order to guide appropriate volume replacement therapy. The choice of hemodynamic monitoring techniques in different clinical settings is still under debate. Basic hemodynamic variables

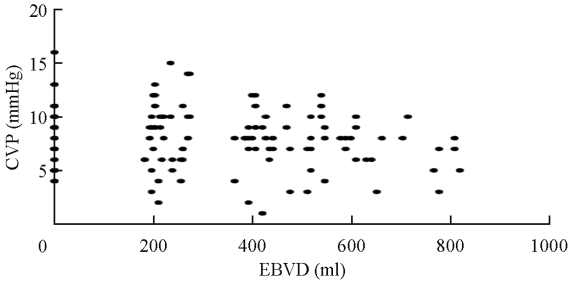

Figure 2. The correlations between central venous pressure (CVP) and the estimated blood volume deficit (EBVD) during acute normovolemic hemodilution (ANH); the solid black dots indicate the real CVP values of each corresponding EBVD. Pearson's correlation r= -0.142 (P>0.05, P=0.056).

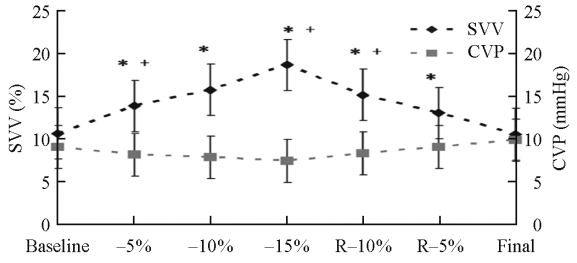

Figure 1. Average changes in stroke volume variation (SVV, left Y axis) and central venous pressure (CVP, right Y axis) at each measurement time during ANH. Values are mean ±SE. *: a significant change over time, mixed models analysis of variance, P <0.05. +: a significant difference from the baseline value, Dunnett's test, P < 0.05.

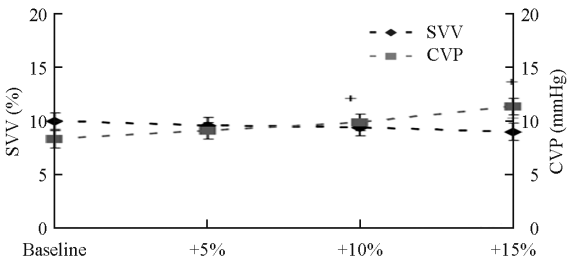

Figure 3. Average changes in stroke volume variation (SVV, left Y axis) and central venous pressure (CVP, right Y axis) during acute hypervolemic haemodilution (AHH). Values are mean ±SE. $^{+}$: a significant difference from the baseline value, Dunnett's test, P < 0.05.

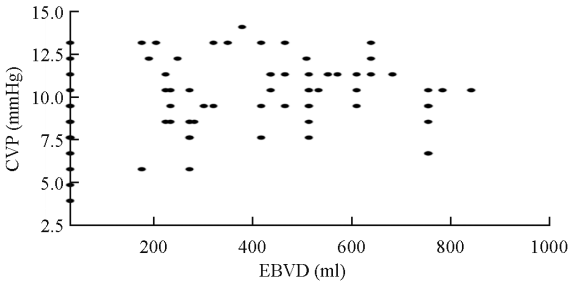

Figure 4. Correlations between central venous pressure (CVP) and the estimated blood volume expansion (EBVE) during acute hypervolemic haemodilution (AHH); the solid black dots stand for the real CVP values of each corresponding EBVE. Pearson's correlation r=0.269 (P<0.05, P=0.016).

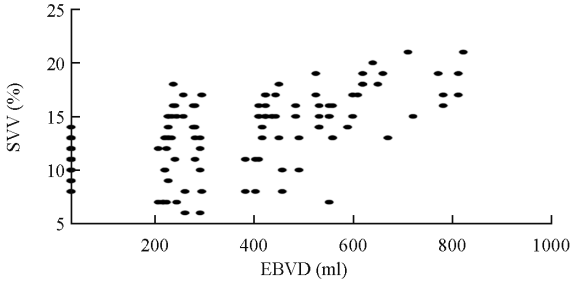

Figure 5. Correlations between stroke volume variation (SVV) and the estimated blood volume deficit (EBVD) during acute normovolemic hemodilution (ANH); the solid black dots represent the real SVV values of each corresponding EBVD. Pearson's correlation r=0.597 (P<0.001).

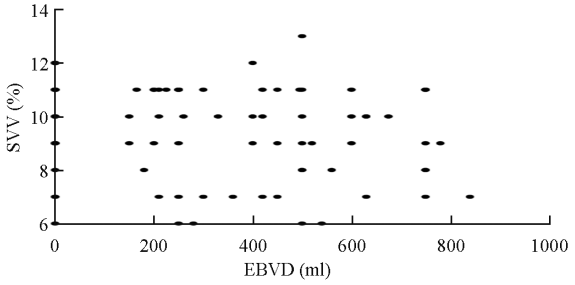

Figure 6. Correlations between stroke volume variation (SVV) and the estimated blood volume expansion (EBVE) during acute hypervolemic haemodilution (AHH); the solid black dots represent the real SVV values of each corresponding EBVE. Pearson's correlation r=-0.163 (P>0.05, P=0.148).

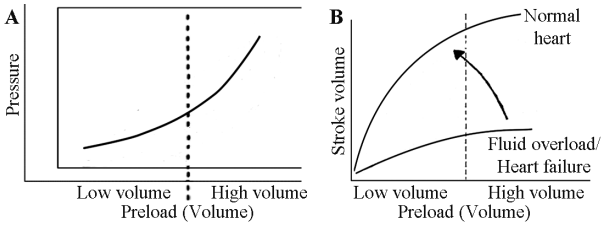

Figure 7. The Frank Starling curve (**A**) shows that an increase in preload (end-diastolic volume) markedly increases stroke volume in hypovolemic state, but slightly elevates pressure (CVP). In conjunction with **A**, Figure **B** also shows that only a modest increase in stroke volume can be achieved with significant pressure increase in hypervolemic state.

such as HR and arterial blood pressure may not have the sensitivity required for optimal care.[18] At the same time, CVP has been used to guide fluid management for many decades. A European survey of intensivists/anesthesiologists reported that more than 90% used the CVP to guide fluid management.[19] However, CVP values do not correlate with the values of measured circulating blood volume or with responsiveness to fluid challenge. Moreover, attempts to assess the differences between the changes in CVP and changes in circulating blood volume also failed to show any significant correlation.[20-22] Due to the changes in venous vessel tone, intrathoracic pressures, ventricular compliance and geometry, there is a poor relationship between the CVP and preload represented by RV end-diastolic volume.[4] However, studies found that measurement of the CVP may be useful in certain selected circumstances. For example, Rex and colleagues found that, after the change from reverse Trendelenburg position to Trendelenburg position, CVP and PAOP increased significantly in post CABG patients.[23] In another study, the authors suggested that regardless of global ejection fraction (GEF), CVP could be useful in predicting fluid responsiveness in patients after coronary and major vascular surgery, provided that positive end-expiratory pressure is low.[24] Changes in CVP and PAOP paralleled changes in CI, particularly when GEF was <20%.[24] Our results indicated that CVP may be useful to predict fluid responsiveness in patients with excessive volume expansion and high CO. The slope of the Frank-Starling curve depends on ventricular contractility and the volume status. On the initial rise of the curve, the slope change steeply (i.e. the slight change in preload brought a bigger change in stroke volume), thus this change can be predicted by volume change rather than by pressure. Conversely, following the excessive increase in preload, the slope change becomes flat (the slight change in stroke volume may bring bigger change in pressure), thus the change can be predicted and monitored by pressure rather than by volume.

SVV is referred to as a dynamic variable of preload, whereas CVP is a static variable. The use of SVV as a functional hemodynamic variable is based on the heart–lung interactions during mechanical ventilation. Respiratory-induced changes in preload result in cyclic changes in left ventricular stroke volume and arterial pressure. The Vigileo/FloTrac system is able to estimate SVV using only a peripheral arterial pressure waveform without any other invasive monitoring. SVV measured by this method is considered to be a good indicator of fluid responsiveness.[9,25,26] However, our data found that although SVV changed significantly following the blood withdrawal and blood transfusion, SVV did not correlate well to the expansion of blood volume. This was consistent with the results from Fujita and Taguchi's studies in animal models.[3,10] They reported SVV derived by the arterial pulse contour analysis technique are useful indicators of hypovolemia, but not of hypervolemia in mechanically ventilated dogs. Taguchi et al[3] also reported SVV is a sensitive indicator of the dynamic circulatory blood volume

change during both bleeding and transfusion, but not during either the stable circulating blood volume period after blood withdrawal or the fluid-overload period, in mechanically ventilated dogs. These results can be explained by using Frank-Starling theory. During hypovolemia the cardiac filling occurs at the steep portion of the curve. During this period, stroke volume goes up significantly after intravascular fluid administration. During hypervolemia, on the contrary, the cardiac filling occurs at the flat portion of the curve; stroke volume doesn't respond sensitively after volume expansion. Once the left ventricle is functioning near the "flat" portion of the Frank-Starling curve, fluid loading has little effect on cardiac output and only causes an increase in pressure which is reflected by the increases in CVP. This is similar to patients with systolic left ventricular dysfunction and especially true in those patient populations. A right- and downward shift on the Frank-Starling curve and along the curvilinear pressure-volume relationship at end-diastole, preload recruitability may be more dependent on and thus predicted and monitored by pressures than by volumes.[24]

ANH and AHH were useful methods for conserving blood and avoiding an unnecessary blood transfusion perioperatively. In these two situations, the change of intravascular volume is inevitable, although the physiological changes are significantly different between the two. Intravascular volume changes from normovolemia to hypovolemia during ANH, and from normovolemia to hypervolemia during AHH. They serve as good clinical models to study whether the SVV reflects the intravascular volume changes during these dynamic situations. It is important for us to be able to accurately predict the circulation volume status while dynamic circulatory volume changes occur. This study was the first to compare the dynamic changes of SVV and CVP during ANH and AHH periods in the particular clinical settings.

Our study has some limitations. First, CO was obtained only by the Vigileo/FloTrac system. The accuracy of this device has been tested in numerous settings with various results.[9, 27] However, it has been shown that it is able to track changes in SV and CO induced by volume expansion (VE), PEEP and mechanical ventilation. Recent studies evaluating fluid responsiveness used the same system as the reference to define response to VE.[28] Second, we excluded subjects with spontaneous breathing activity or cardiac arrhythmias because respiratory variations in hemodynamic signals are ineffective.[29] Third, the study was performed using subjects sedated and mechanically ventilated with a tidal volume of 8 ml/kg, and SVV is affected by tidal volume under mechanical ventilation.[30] Finally, our low sample size might limit the interpretation of the results.

In conclusion, this study indicated that SVV is a useful indicator of hypovolemia. It can be used to guide preload optimization because it allows estimation of preload and the prediction of cardiac index changes in response to fluid loading. While SVV is more sensitive than CVP during hypovolemia, our data also suggested that CVP is more sensitive than SVV during hypervolemia especially in the range of the plateau portion of the Frank-Starling curve.

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