Research Article

The comparison of stroke volume variation with central venous pressure in predicting fluid responsiveness in septic patients with acute circulatory failure

Santhalakshmi Angappan, Satyen Parida, Arumugam Vasudevan, Ashok Shankar Badhe

Abstract

Purpose: The present study was designed to investigate the efficacy of stroke volume variation (SVV) in predicting fluid responsiveness and compare it to traditional measures of volume status assessment like central venous pressure (CVP). **Methods:** Forty-five mechanically ventilated patients in sepsis with acute circulatory failure. Patients were not included when they had atrial fibrillation, other severe arrhythmias, permanent pacemaker, or needed mechanical cardiac support. Furthermore, excluded were patients with hypoxemia and a CVP >12. Patients received volume expansion in the form of 500 ml of 6% hydroxyethyl starch. **Results:** The volume expansion-induced increase in cardiac index (CI) was >15% in 29 patients (labeled responders) and <15% in 16 patients (labeled nonresponders). Before volume expansion, SVV was higher in responders than in nonresponders. Receiver operating characteristic curves analysis showed that SVV was a more accurate indicator of fluid responsiveness than CVP. Before volume expansion, an SVV value of 13% allowed discrimination between responders and nonresponders with a sensitivity of 78% and a specificity of 89%. Volume expansion-induced changes in CI weakly and positively correlated with SVV before volume expansion. Volume expansion decreased SVV from 18.86 ± 4.35 to 7.57 ± 1.80 and volume expansion-induced changes in SVV moderately correlated with volume expansion-induced changes in CI. **Conclusions:** When predicting fluid responsiveness in mechanically ventilated patients in septic shock, SVV is more effective than CVP. Nevertheless, the overall correlation of baseline SVV with increases in CI remains poor. Trends in SVV, as reflected by decreases with volume replacement, seem to correlate much better with increases in CI.

Keywords: Acute circulatory failure, central venous pressure, fluid responsiveness, sepsis, stroke volume variation

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Introduction

Volume expansion is the first-line therapy proposed in septic patients in an attempt to improve hemodynamics[1] and is one of the most common maneuvers to increase cardiac output (CO) in patients with circulatory failure. However, if inappropriate, it may have deleterious effects such as volume overload, systemic, and pulmonary edema; and limitation of oxygen diffusion

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to tissues thereby leading to increased tissue hypoxia.[2] It is, therefore, important to obtain reliable information concerning fluid responsiveness in patients with circulatory failure in the intensive care unit. However, clinicians are often faced with imprecise, nonspecific information to guide their therapy. It has been demonstrated that neither the standard preload indices such as central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), heart rate (HR), blood pressure (BP) nor their trends in response to fluid challenge accurately reflected left ventricular (LV) preload or its trends in patients receiving a fluid challenge for hemodynamic instability. Therefore, they are not capable of accurately predicting the hemodynamic response to fluid therapy.[3,4]

Stroke volume variation (SVV) and pulse pressure variation (PPV) have been used as dynamic indices to guide fluid therapy in patients receiving mechanical ventilation.[5] The SVV/PPV are more pronounced during hypovolemia, and the variation decreases if the intravascular volume is restored. These parameters have shown to predict reliably changes in CO.[5] SVV has been repeatedly shown to be a reliable predictor of fluid responsiveness.[6] SVV has been shown to have a high sensitivity and specificity when compared to traditional indicators of volume status such as HR, BP, CVP, or PCWP, and their ability to determine fluid responsiveness.[7] Various devices allow automated clinical assessment of SVV. The Vigileo monitor allows for the continuous monitoring of essential hemodynamic information.[8] The Vigileo monitor, when used with the FloTrac sensor, can display key flow parameters such as CO, SV, SVV, and cardiac index (CI). While SVV, as measured by the Vigileo-FloTrac system of uncalibrated pulse contour analysis, has been found to be a good indicator of cardiac preload, such evidence is not incontrovertible. Several authors have found that SVV, as acquired from either the Vigileo-FloTrac or the PiCCO systems may not reliably predict fluid responsiveness in diverse clinical settings.[9,10] Some of these negative studies have been done in patients with no spontaneous respiratory activity,[9] while others have included spontaneously breathing patients on pressure support ventilation.[10] Hence, in mechanically ventilated patients with acute circulatory failure related to sepsis, the present prospective, interventional study was designed to: (1) Investigate, whether SVV acquired from the Vigileo-FloTrac system, could predict the hemodynamic effects of volume expansion; (2) investigate whether changes in SVV could predict changes in CI; and (3) compare SVV with commonly used methods of determining fluid status such as the CVP as predictors of fluid responsiveness.

Methods

Study design and patient population

The study received the approval of our local ethics committee and written informed consent was obtained from the closest relative. The present study was conducted in our critical care unit (CCU) on 45 mechanically ventilated patients of either sex, aged between 20 and 65 years, diagnosed with acute circulatory failure related to sepsis, defined by the criteria of the American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference.[11] In order to be included in the study, patients had to present with the following:

- Acute circulatory failure defined by a systolic BP <90 mmHg or need for vasopressor drugs (dopamine >5 mcg/kg/min or norepinephrine)
- Instrumentation with indwelling radial or femoral artery and central venous catheters
- Relative hemodynamic stability, defined by a variation in HR, BP, and a cardiac index of <10% over the 15 min period before starting the protocol.

Patients were not included if they had atrial fibrillation, severe arrhythmias, a permanent pacemaker or a need for mechanical cardiac support. Patients with hypoxemia (ratio of arterial oxygen pressure to fraction of inspired oxygen PaO_2/FiO_2 <100) and CVP ≥12 mmHg were also excluded.

Hemodynamic and other patient parameters

Electrocardiogram, HR, and pulse oximetry were monitored continuously. Routine clinical monitoring of the patients included a central venous catheter (Arrow International, LLC, Reading, PA) through the right internal jugular vein, with its position confirmed radiologically, a 20G radial artery catheter on the side with a more prominent pulse or a 16G femoral artery catheter. The zero reference level for the supine position was the mid-chest level, and the pressure was measured at the end of expiration. Serial measurements of HR, SpO_2, mean arterial pressure (MAP), mean CVP, SVV, cardiac index (CI), stroke volume index (SVI), and systemic vascular resistance index (SVRI) were undertaken. The Vigileo system (Edwards Lifesciences, LLC, Irvine, CA), software version 3.02, using a FloTrac (Edwards Lifesciences, LLC, Irvine, CA) arterial pressure sensor was used in all the patients to measure SVV, CI, SVI, and SVRI. Each parameter recorded was the average of 10 min preceding the measurement. The measurements were performed in duplicate, first before volume expansion and then 30 min after volume expansion using 500 ml 6% hydroxyethyl starch (HES) (130/0.4). Ventilatory settings and dosages

of inotropic and vasopressor drugs were held constant. All patients were maintained in the 30° head-up position for the duration of the study while hemodynamic measurements were being taken since it has been shown that body position may influence SVV measurements.[12] Apart from these hemodynamic data, we also recorded the cause of sepsis (i.e., bacterial pneumonia, abdominal sepsis, meningitis, etc.), number of patients with or without vasopressor support, underlying disease conditions such as, chronic obstructive pulmonary disease (COPD), diabetes mellitus (DM), ischemic cardiomyopathy, peripheral vascular disease (PVD), and chronic renal failure (CRF), as well as the results of any echocardiography, if performed. The number of patients surviving their stay in the CCU was also recorded.

Therapeutic protocol

The therapeutic protocol is shown in Figure 1. All patients were sedated and mechanically ventilated in a volume-controlled mode with the tidal volume of 8 ml/kg since the tidal volume can significantly affect the SVV value, and an inspiratory/expiratory ratio of 1:2. The level of positive end-expiratory pressure was recorded for each patient. Spontaneous breathing activity was looked for by visual inspection of the airway pressure curve. To ensure that the respiratory changes in SVV measurement reflected only the effects of positive pressure ventilation, patients were sedated or paralyzed if detected to have spontaneous breathing activity. Patients were divided into two groups, according to the percent increase in CI, in response to volume expansion. We assumed that a 15% change in CI was needed for clinical significance according to previous studies.[13,14] Therefore, patients with a CI increase induced by VE >15% and <15% are to be classified as responders and nonresponders, respectively.

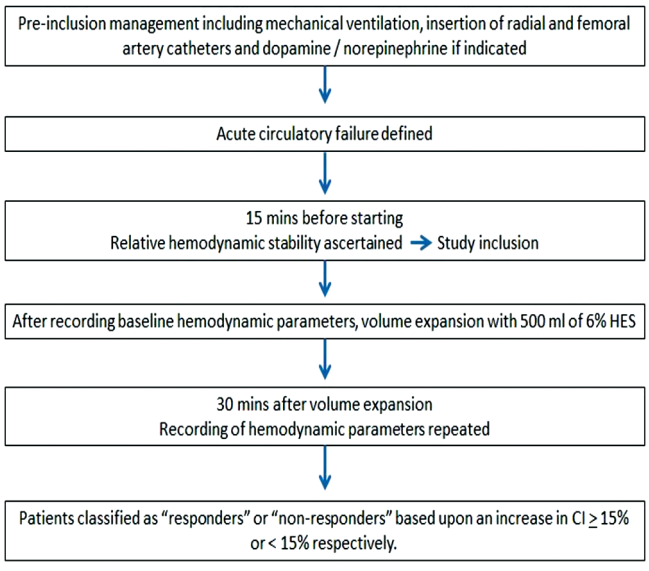

Figure 1: Study synopsis – HES: Hydroxyethyl starch; CI: Cardiac index

Statistical analysis

The hemodynamic data are expressed as mean ± standard deviation. HR, MAP, CVP, SVV, CI, SVI, and SVRI before volume expansion in responders and nonresponders were compared using Student's *t*-test. Correlations between baseline SVV, changes in SVV, and changes in CVP with the changes in CI were tested using the Pearson's correlation. Receiver operating characteristic (ROC) curves were generated for CVP and SVV. The areas under the ROC (AUROC) curves were calculated for each parameter and compared. All statistical analysis were carried out at 5% level of significance, and a $P < 0.05$ was considered significant. SPSS version 16 (SPSS Inc. Released 2007. SPSS for Windows, Version 16.0, Chicago, SPSS Inc.) and MedCalc (MedCalc Software, Ostend, Belgium) were used for statistical analysis.

Results

During the 26-month study period, 45 patients with clear evidence of septic shock, the etiology being abdominal sepsis, admitted in our CCU were given volume expansion, of which 29 patients were responders, and 16 patients were nonresponders. All the 45 patients received vasopressor support (noradrenaline in 8 patients; noradrenaline and dopamine in 37 patients). Underlying diseases included DM (n = 6), DM with hypertension (n = 2), bronchial asthma (n = 1), COPD (n = 1), and CRF (n = 2). Echocardiography was performed in 24 patients, which revealed LV systolic dysfunction in 5 patients. Thirty patients survived the effects of volume expansion on the 45 patients are summarized in Table 1. Table 2 represents the demographic data of the 45 patients are included in the study.

Table 1: Responders and nonresponders

Volume expansion	Number of patients	Percentage
Responders	29	64.4
Nonresponders	16	35.6
Total	45	100.0

Table 2: Demographic data

Variables	Mean±SD
Age	45.2±13.6
Height	170.6±6
Weight	53.7±5.7
BSA	1.5±0.09
Sex (%)	
Female	20
Male	80

Data expressed as mean±SD. SD: Standard deviation; BSA: Body surface area

Hemodynamic measurements

As shown in Table 3, all the hemodynamic variables showed a significant change after volume expansion from their baseline values except the SVRI. Before volume expansion, SVV ranged from 1% to 24% with the 95% confidence intervals between 14% and 23%. Before volume expansion, SVV was not correlated with the CVP. Volume expansion increased CI from 3.11 ± 0.30 to 3.66 ± 0.41 L/min/m^2 ($P < 0.001$). Table 4 represents the hemodynamic variables before volume expansion among responders and nonresponders. Before volume expansion, SVV (20.4 + 2.77 vs. 15.9 ± 4.2, $P = 0.01$) was higher in responder than in nonresponder patients, whereas CVP (9 ± 1.64 vs. 8.7 ± 1.9 mm Hg) was not significantly different between the two groups. The AUROC curves were as follows: 0.716 with 95% CI (0.56–0.84) for SVV and 0.562 with 95% CI (0.41–0.71) for CVP [Figures 2 and 3, respectively]. The area for SVV was significantly greater than the area for CVP ($P < 0.01$). The threshold SVV value of 13% allowed discrimination between responder and nonresponder patients with a sensitivity of 78% and a specificity of 89%.

A positive but weak linear correlation ($r = 0.32$, $P < 0.05$) was found between SVV before volume expansion and the percentage increase in CI, such that the higher the baseline SVV, the greater was the percentage increase in CI (% change in CI = 0.44 × SVV ± 2.83) [Figure 4], whereas CVP measured before volume expansion was not correlated in any way with the volume expansion-induced changes in CI.

Volume expansion-induced changes in SVV (SVV after volume expansion minus SVV before volume expansion) were moderately correlated with volume expansion-induced changes in CI ($r = 0.5$, $P < 0.05$), such that the greater the decrease in SVV, the greater the increase in CI induced by volume expansion [Figure 5].

Discussion

The effects of volume expansion on SVV, CI, and CVP.

We have demonstrated that the SVV as measured by analysis of peripheral arterial waveform is a better indicator of fluid responsiveness in mechanically ventilated patients with acute circulatory failure related to

Table 3: Effect of volume expansion on hemodynamic variables

Hemodynamic variables	Before volume expansion	After volume expansion	*P*
HR (mean)	131.53±10.62	125.28±9.90	t=18.99; P<0.001**
MAP (mean)	73.33±3.49	87.35±4.37	t=23.14; P<0.001**
CVP (mean)	8.91±1.72	9.82±1.49	t=3.88; P<0.001**
SVV (mean)	18.86±4.35	7.57±1.80	t=22.86; P<0.001**
SVI	23.87±3.36	29.53±4.82	t=13.86; P<0.001**
SVRI	1664.95±144.51	1711.00±195.88	t=1.93: P=0.060
CI (mean)	3.11±0.30	3.66±0.41	t=20.74; P<0.001**

Data expressed as mean±SD. SD: Standard deviation; HR: Heart rate; MAP: Mean arterial pressure; CVP: Central venous pressure; SVV: Stroke volume variation; SVI: Stroke volume index; SVRI: Systemic vascular resistance index; CI: Cardiac index

Table 4: Hemodynamic variables before volume expansion among responders and nonresponders

Hemodynamic variables	Responders	Nonresponders	*P*
HR mean	131.4±11.3	131.6±9.5	0.98
MAP mean	73.2±3.6	73.4±3.3	0.76
CVP mean	9±1.64	8.7±1.9	0.72
SVV mean	20.4±2.77	15.9±4.2	0.010*
SVI	23.5±3.15	24.4±3.7	0.462
SVRI	1684.5±145.8	1629.8±139.2	0.18
CI mean	3.06±0.26	3.2±0.36	0.371

Data expressed as mean±SD. SD: Standard deviation; HR: Heart rate; MAP: Mean arterial pressure; CVP: Central venous pressure; SVV: Stroke volume variation; SVI: Stroke volume index; SVRI: Systemic vascular resistance index; CI: Cardiac index

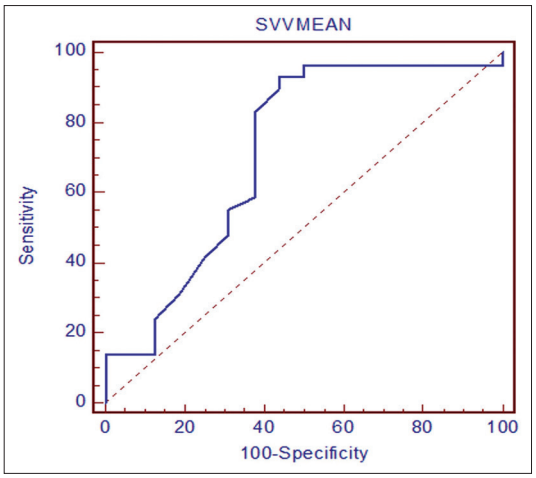

Figure 2: Receiver operating characteristic curve for stroke volume variation with the area under the receiver operating characteristic curve of 0.716 with 95% confidence interval (0.56–0.84). ROC: Receiver operating characteristic; SVV: Stroke volume variation

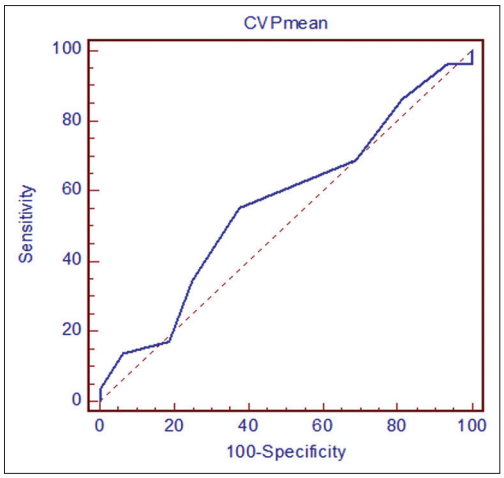

Figure 3: Receiver operating characteristic curve for central venous pressure with the area under the receiver operating characteristic curve of 0.562 with 95% confidence interval (0.41–0.71). ROC: Receiver operating characteristic; CVP: Central venous pressure

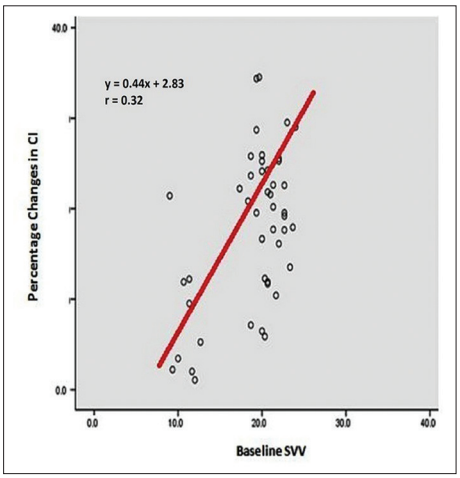

Figure 4: Relationship between baseline stroke volume variation and percentage changes in the cardiac index. SVV: Stroke volume variation; CI: Cardiac index

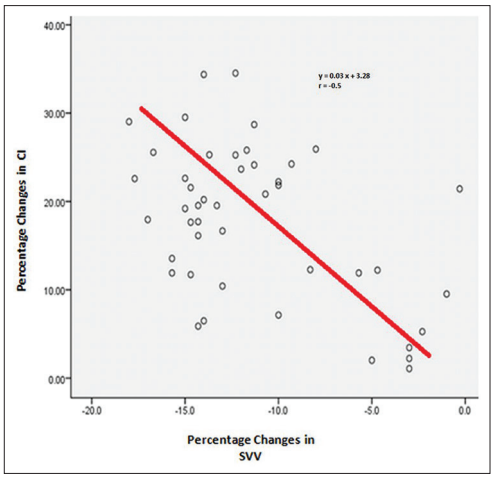

Figure 5: Relationship between the decrease in stroke volume variation produced by volume expansion and the percentage changes in the cardiac index. SVV: Stroke volume variation; CI: Cardiac index

sepsis as compared to CVP, as indicated by the respective AUROC curves for SVV and CVP. Indeed, a patient with a baseline SVV value of more than 13% was very likely to respond to volume expansion by increasing CI by >15%, with a positive predictive value of 89%. However, if SVV was <13%, the decision on whether the patient would respond to fluids was less clear-cut, with a negative predictive value of only 50%. Moreover, SVV before volume expansion, weakly correlated with the volume expansion-induced increase in CI. This was reflected in the scatter in the percent increase in CI induced by the infusion of 500 ml 6% HES when compared to the SVV before volume expansion [Figure 4]. However, overall our findings suggest that analysis of SVV could be helpful in the decision-making process concerning volume expansion in such patients.

The present study further demonstrates that in mechanically ventilated patients with acute circulatory failure related to sepsis, the baseline SVV determined by the Vigileo/FloTrac system has a weak correlation with the percentage change in CI following volume expansion. Our study also demonstrated that changes in SVV measured by the Vigileo system have a moderate agreement and correlation with changes in CI after volume expansion, although there was a lot of scatter. CVP has been proposed for identifying patients who would benefit from volume expansion.[15,16] In the present study, CVP before volume expansion was not significantly different between responders and nonresponders and did not correlate with the volume expansion-induced changes in CI. Moreover, the AUROC curve for CVP indicated that measuring this parameter to assess fluid responsiveness was no better than chance. These findings are in agreement with other reports[17-19] demonstrating that CVP is of little value in predicting the hemodynamic effects of volume expansion in septic patients.

In summary, volume expansion-induced a significant decrease in SVV in those patients who responded to such expansion by a 15% increase in CI. The decline in SVV produced by volume expansion, at least moderately, correlated with concomitant increases in CI, although, there was a lot of scatter [Figure 5].

It must be stressed that the dynamic indices of preload sensitivity are not appropriate for use in patients who are having severe arrhythmias, as also those having spontaneous breathing activity, due to variable interpretation of the changes in arterial pressure with the phases of respiration in spontaneous and mechanically delivered breaths. Patients with arrhythmias were therefore excluded from our study and those with spontaneous breathing activity were either administered additional sedation or temporarily paralyzed, to eliminate such activity during the period of intervention.

Existing evidence suggests that, CVP should not be expected to represent a reliable marker for fluid responsiveness in disease, although historically, it is the most commonly reported factor used by intensivists to guide decision-making in fluid management. In fact, with an AUROC curve of 0.562 in our study, the predictive power of CVP for fluid responsiveness was no better than a coin-flip. In nonspontaneously breathing patients, dynamic indices such as SVV, PPV, and inferior vena cava (IVC) diameter were far better predictors of fluid responsiveness as compared to CVP, PCWP, and echocardiographic LV end-diastolic area index. Today

a substantial number of studies have confirmed these results in a variety of patient groups. Apart from these dynamic indices, an endogenous fluid challenge, the passive leg raising test as also the conventional mini fluid challenge, have been proposed as predictive tests for fluid responsiveness for intubated patients, as well as patients breathing spontaneously.

The other thing to consider is that for a certain change in LV SV, the SVV might differ depending upon the compliance of the arterial wall. Thus, significant differences in SVV could be observed despite minimal changes in LV SV, if the compliance of the arterial walls is low as in the geriatric age group, with associated PVD. Similarly, it is possible for the SVV to change little despite large alterations in the LV SV if the compliance of the arterial walls is high, as in young patients without any vascular disease, or in vasodilated states, like septic shock, which our patients were in.

Limitations of the study

First, since we studied patients in acute circulatory failure, related to sepsis, the implications of our results can only pertain to the clinical scenario in which the conclusions were drawn. Second, not being an outcome study, it would require a separate outcome study to be able to detect whether any benefits with regard to clinical outcomes can be accrued from this technique. Third, we used the same device to measure both the CI and the SVV, since it was considered unethical to put a pulmonary artery catheter to measure the CO, only for the sake of doing the study, which is not routine practice in our CCU. However, since the Vigileo/Flo Trac has been previously validated against the pulmonary artery based thermodilution technique for measurement of CO,[20] and since both SVV and CO on the Vigileo monitor are measured independent of each other and are not derived quantities from each other, we would like to think that the validity of our results holds good. Further, we admit that our results are not startlingly impressive with regard to the overall performance of SVV to detect preload sensitivity in patients with septic shock, although they do indicate that dynamic indicators of preload responsiveness may be more sensitive than static indicators. Based on these results, we are conducting a prospective randomized study in septic shock patients, comparing SVV as measured by the Vigileo/FloTrac system and ultrasonic assessment of IVC collapsibility index as indicators of fluid responsiveness.

Conclusion

Perhaps the most challenging aspect to the care of a patient with septic shock is to decide, what constitutes adequate fluid management for these patients since such decision has the potential of affecting eventual outcomes. Thus, we attempted to study SVV as a predictor of hypovolemia and fluid responsiveness in sepsis, in comparison with more traditional indicators like CVP. We found that the reduction in SVV moderately correlated with the improvement in CI following a standard volume expansion protocol. Nevertheless, the baseline SVV only had a weak correlation with the degree of CI improvements following volume expansion.

To conclude, we compared static indicators of preload responsiveness such as CVP with dynamic indices like SVV in monitoring and predicting LV preload and circulatory volume and found that the latter is much better in this regard. While the baseline SVV has a weak, albeit positive correlation with the degree of increase in CI, it still manages to identify most patients who would likely benefit from fluid administration in terms of significant increases in CI. Trends in SVV with continued volume expansion could also be used to track concomitant changes in CI.

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Conflicts of interest

There are no conflicts of interest.

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