Nurses commonly experience scenarios where hemodynamic monitoring is focused on hypovolemia (see case study) in clinical practice. In this article, we provide an overview of the use of stroke volume (the amount of blood ejected from the left ventricle with each beat) for hemodynamic management of critically ill patients. We also discuss the limitations of conventional assessment parameters, methods of measuring stroke volume, hemodynamic variables that influence stroke volume, the stroke volume optimization (SVO) replacement algorithm, supporting literature, and nursing considerations.

Much of the supporting literature (mostly studies in perioperative patients) on stroke volume as a primary hemodynamic monitoring parameter focuses on the treatment of hypovolemia, as in the case study. Critical care practices have evolved to rely more on physical assessments for monitoring cardiac output and evaluating fluid volume status because these assessments are less invasive and more convenient to use than is a pulmonary artery catheter. Despite this trend, level of consciousness, central venous pressure, urine output, heart rate, and blood pressure remain assessments that are slow to be changed, potentially misleading, and often manifested as late indications of decreased cardiac output. The hemodynamic optimization strategy called stroke volume optimization might provide a proactive guide for clinicians to optimize a patient's status before late indications of a worsening condition occur. The evidence supporting use of the stroke volume optimization algorithm to treat hypovolemia is increasing. Many of the cardiac output monitor technologies today measure stroke volume, as well as the parameters that comprise stroke volume: preload, afterload, and contractility. (Critical Care Nurse. 2015;35[1]:11-28)
A critical care nurse cares for a patient who had a 3-vessel coronary artery bypass graft approximately 30 minutes earlier. No indications of bleeding are present, and both cardiac output and urine output are within the reference limits. However, the patient’s systolic arterial blood pressure has decreased to 77 mm Hg 3 times in the past 20 minutes. Because the decrease seems to respond to fluid administration, the nurse begins administering a fourth bottle of 5% albumin via the rapid infuser in accordance with the surgeon’s standing orders. Even though the patient’s central venous pressure has remained at 3-4 mm Hg since the patient arrived from surgery, the nurse notes that the stroke volume has remained between 75 and 80 mL since the second bottle of albumin. On the basis of the patient’s hemodynamic profile and this recent sequence of events, the nurse calls the surgeon to inquire about an order to administer a vasoactive agent. In this case, decreased stroke volume response to fluid suggests adequate volume expansion even though low central venous pressure values suggest hypovolemia.

In the following section, we review the clinical importance of hypovolemia that may go undetected (occult hypovolemia) when conventional assessment techniques are used.

### Importance of Occult Hypovolemia

To illustrate the nature of subclinical or occult hypovolemia and to test the sensitivity of gastrointestinal tonometry for detecting such hypovolemia, Hamilton-Davies et al. conducted a study on 6 healthy volunteers in the critical care unit at University College of London Hospitals, London, England. Each of the volunteers had a mean of 25% (21%-31%) of their overall blood volume removed during a 1-hour period, and the volunteers’ response was measured. Variables such as heart rate, blood pressure, serum levels of lactate, and stroke volume were measured every 30 minutes throughout the study. After 90 minutes, decreases in gut intramucosal pH were observed, as well as marked decreases in stroke volume, by a mean of 16.5 mL ($P < .01$). Despite this compromised flow, no clinically significant or consistent postinterventional changes were noted in serum levels of lactate, arterial blood pressure, heart rate, or arterial blood gases according to serial measurements obtained throughout the study period. Retransfusion was started after 90 minutes. The results of this study may provide insight into the reliability of routinely used measurements such as heart rate and systolic blood pressure as volume depletion progressed in these volunteers.

Hypovolemia (defined as inadequate left ventricular filling volumes) affects the cardiovascular system in a characteristic sequence of events as the hypovolemia worsens (Table 1). First, stroke volume decreases when the blood loss exceeds 30% of blood volume, heart rate increases when blood loss exceeds 15%, and pulse pressure decreases. As hypovolemia worsens, mental status changes from slightly anxious to confused and lethargic.

<table>
<thead>
<tr>
<th>Blood loss, %</th>
<th>Class 1</th>
<th>15%-30%</th>
<th>30%-40%</th>
<th>&gt; 40%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, beats per minute</td>
<td>&lt; 100</td>
<td>&gt; 100</td>
<td>&gt; 120</td>
<td>&gt; 140</td>
</tr>
<tr>
<td>Blood pressure, mm Hg</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Normal or increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Respiratory rate, breaths per minute</td>
<td>14-20</td>
<td>20-30</td>
<td>30-40</td>
<td>&gt; 35</td>
</tr>
<tr>
<td>Mental status</td>
<td>Slightly anxious</td>
<td>Mildly anxious</td>
<td>Anxious, confused</td>
<td>Confused, lethargic</td>
</tr>
</tbody>
</table>

* Reprinted from American College of Surgeons, with permission.

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### Table 1 Classes of shock by Advanced Trauma Life Support (ATLS) designation

**Class 1**
- < 15% blood loss
- < 100 heart rate
- Normal blood pressure
- Normal pulse pressure
- 14-20 respiratory rate
- Slightly anxious mental status

**Class 2**
- 15%-30% blood loss
- > 100 heart rate
- Normal blood pressure
- Normal pulse pressure
- 20-30 respiratory rate
- Mildly anxious mental status

**Class 3**
- 30%-40% blood loss
- > 120 heart rate
- Decreased blood pressure
- Decreased pulse pressure
- 30-40 respiratory rate
- Anxious, confused mental status

**Class 4**
- > 40% blood loss
- > 140 heart rate
- Decreased blood pressure
- Decreased pulse pressure
- > 35 respiratory rate
- Confused, lethargic mental status

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### Authors

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because of decreased overall circulating volume (class 1). Next, heart rate increases and vasoconstriction occurs to maintain blood pressure and cardiac output (the volume of blood pumped by the heart per minute) (class 2). A surge of endogenous catecholamines helps shunt blood from the periphery and splanchnic circulation to the brain and great vessels to preserve vital organs. Once compensatory mechanisms are exhausted, cellular respiration begins to change from aerobic metabolism to anaerobic metabolism, and tissue oxygenation is threatened. Oxygen extraction rates increase, and mixed venous oxygen saturation (SvO₂) and central venous oxygen saturation (Scvo₂) decrease because of decreased cardiac output, compromised blood flow, and decreased oxygen delivery to tissues (class 3). Finally, urine output, level of consciousness, and blood pressure decrease (class 4). Each event may take minutes to hours. Despite this known sequence, aggressive intervention often is not implemented until hypotension occurs. Traditionally, clinicians are trained to monitor for early indications of decompensation, and the first hemodynamic monitoring parameter to decrease in hypovolemia is stroke volume.

Hypovolemia frequently occurs in patients during surgery and in the critical care unit because of bleeding, hypalbuminemia, capillary leak and interstitial edema, diarrhea, vomiting, and insensible water loss. If the hypovolemia is left untreated (or undertreated), circulatory hypoxia may develop because of the decreased blood flow and hypoperfusion. Compensatory diversion of blood flow centrally, away from the peripheral and splanchnic circulation, often masks hypoperfusion.

If not recognized and treated promptly, decreased circulating volume (particularly at the microvascular level) leads to diminished oxygen delivery, depletion of intracellular energy reserves, acidosis, anaerobic glycolysis, and lactate accumulation. Hypovolemia can also lead to ischemic gastrointestinal complications, including nausea, vomiting, and intolerance of oral intake. Therefore, diligent monitoring, via accurate assessment of cardiac output and stroke volume, for hypovolemia is important for monitoring blood flow.

Limitations of Conventional Assessments

Current conventional assessments such as heart rate, blood pressure, urine output, central venous pressure (CVP), and level of consciousness often lack precision as indicators of changes in a patient’s status. Although the values obtained in these assessments somewhat correlate with hemodynamic variables, the values are slow to change and the changes are often late indications of a patient’s worsening condition. Several studies suggest that using physical assessment to evaluate cardiac output may yield inaccurate findings. More recent data suggest that the predictive power of blood lactate levels for mortality and morbidity are independent of blood pressure and common physiological triage variables (eg, heart rate, blood pressure, mental status, capillary refill).

Despite these limitations, assessments such as blood pressure are still considered a standard of care, and current practice mandates use of the assessments. However, blood pressure itself is a composite of so many factors (Figure 1) that it is of limited value as an early sign of hemodynamic derangements such as hypovolemia. Compensatory mechanisms such as vasoconstriction and tachycardia influence the cardiovascular system to keep blood pressure normal, making the correlation between blood pressure and blood flow slow to change as circulating volume decreases. The terms compensated shock and cryptic shock are now being used to define patient scenarios that meet clinical criteria for shock in
the presence of normal blood pressures. Blood pressure measurements are more useful for conditions that involve treatment of hypertension rather than treatment of hypovolemia or shock. International guidelines such as the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure help guide care providers in the management of hypertension according to a systematic and stepwise approach. However, currently no such guidelines exist for the management of hypotension.

Reconsidering Fluid Replacement End Points

In an article published in 1996, Connors et al suggested that use of a pulmonary artery catheter (PAC) was associated with an increased likelihood of patient death. Since then, use of PACs has generally decreased. Although values obtained via a PAC were once considered the gold standard for bedside hemodynamic monitoring, the precision of a PAC for assessing preload status via filling pressures is limited. As early as 1971, Forrester et al pointed out the inaccuracies of CVP monitoring. In a more recent systematic review of CVP as a predictor of cardiac output and fluid responsiveness, Marik et al concluded that CVP should not be used as a basis for clinical decisions on fluid management. In fact, Marik et al noted that the only published study suggesting CVP could be an accurate indication of preload was done in horses. Even though guidelines such as those of the Surviving Sepsis Campaign recommend using CVP to monitor preload, no study of CVP or pulmonary artery occlusive pressure (PAOP) has shown that these pressures consistently correlate with blood flow or volume status. Early and aggressive use of fluid replacement to preestablished end points such as ScvO₂ is more likely than the measurement of CVP itself to provide patients benefit. The limitations of CVP are further pointed out in the landmark study on septic shock by Rivers et al published in 2001. These investigators randomized 263 patients with septic shock to receive either treatment according to a protocol on fluid replacement known as early goal-directed therapy or conventional care (control group). The patients treated according to the protocol had a 17% reduction in mortality, even though CVP was used as part of the basis for treatment in both the interventional and the control group.

PAOP is also an inaccurate predictor of fluid responsiveness in critically ill patients, further indicating that blood pressures do not correlate with blood flow parameters such as cardiac output and stroke volume. This lack of correlation occurs because many factors can alter the pressure-volume relationship within the heart. For example, conditions that increase PAOP but not preload include, but are not limited to, positive-pressure mechanical ventilation, positive end-expiratory pressure, and decreased ventricular compliance. Conditions that alter cardiac compliance include aging, obesity, diabetes, myocardial ischemia, and sepsis. The challenge encountered with interpreting PAOP is further illustrated in Figure 2; the 3 hearts in the drawing have different cardiomyopathies and various left ventricular end-diastolic volumes (LVEDVs), but each heart has the same PAOP. As a result, the baseline Frank-Starling pressure-volume curves for the 3 hearts differ vastly (Figure 3). When LVEDV increases in normal hearts, pressure increases in a characteristic curvilinear relationship. However, in conditions such as left ventricular hypertrophy, decreased wall compliance increases intracardiac pressure without a concomitant increase in volume. Measurements based on blood flow, such as stroke volume, help clinicians avoid incorrect assumptions based on pressure-volume curves. Ultimately, blood flow is more reliable and precise than are blood pressures, and blood flow can decrease before blood pressures decrease.

CVP and PAOP were never intended to be used alone; both are filling pressures meant to guide the optimization of stroke volume. The fundamental reason to administer a fluid bolus to a patient is to increase stroke volume. Although stroke volume monitoring is not considered a standard of care, as is conventional monitoring of vital signs, plotting or documenting stroke volume in response to a fluid challenge may be the closest clinicians can come to using the Frank-Starling curve in routine bedside practice. Stroke volume is more likely to indicate hypovolemia before other monitoring parameters do because the former is not influenced by most compensatory mechanisms. Treatments that include giving fluids and medications such as drugs that improve contractility (inotropes) are often administered with the goal of improving stroke volume. Specifically targeting
The complexity of these changes defies overreliance on parameters such as blood pressure. Ongoing fluid replacement decisions should be based on stroke volume, variations in pulse pressure, cardiac output derived by using a minimally invasive method, and passive leg-raising maneuvers supported by integrated assessment to more...
Methods of Measuring Stroke Volume

Traditionally, echocardiography has been the most commonly used method to measure stroke volume at the bedside. However, this method is expensive and technically difficult and continuous or serial measurements are often not practical in critical care. Several new technologies enable ongoing measurement of stroke volume at the bedside, including noninvasive Doppler imaging (USCOM), esophageal Doppler imaging (Deltex Medical; Figure 4), bioimpedance (SonoSite), endotracheally applied bioimpedance (ConMed Corporation), bioreactance (Cheetah Medical), pulse contour methods (Edwards Lifesciences, LidCo Ltd, Pulsion Medical Systems), an exhaled carbon dioxide method (Philips, Respironics), and the PAC. All use various methods to calculate stroke volume, and the results have various degrees of accuracy. Some devices measure stroke volume directly (eg, esophageal Doppler imaging) and may be considered the preferred method because of the high degree of accuracy of the results. Other technologies simply divide the cardiac output by the heart rate to obtain stroke volume (eg, PAC). Table 2 provides a more detailed comparison.

Clinical application of technology is based on knowledge and experience in obtaining and applying the information received. If a care provider targets the wrong hemodynamic end points or interprets a poor waveform as an accurate tracing, benefits may be limited. These concerns were cited in the technology assessment report published by the Agency for Healthcare Research and Quality in 2008 as some of the most likely reasons studies have collectively suggested no benefit for monitoring with PACs.

Disagreement may exist about which technology is best for monitoring stroke volume because none of the technologies is appropriate for all patients in all situations.

Each technology has a unique profile of advantages and limitations, and a patient’s situation may dictate which technology is best at a given time.
### Table 2 Technology for measuring stroke volume

<table>
<thead>
<tr>
<th>Technology</th>
<th>Manufacturer</th>
<th>Where used</th>
<th>Randomized controlled trials regarding patient outcome</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>External Doppler imaging</td>
<td>USCOM, Sydney, Australia</td>
<td>Anywhere</td>
<td>None</td>
<td>Stroke volume estimate obtained via ultrasound probe placed at the sternal notch or parasternally; ultrasound beam directed at the aortic or pulmonic valve</td>
</tr>
<tr>
<td>Esophageal Doppler imaging</td>
<td>Deltex Medical, West Sussex, England</td>
<td>Operating room, intensive care unit, emergency department</td>
<td>9 trials[^44-52] (2 trials had conflicts of interest to disclose) showing reduced length of stay, complications, use of vasopressors, renal insufficiency, and mortality and lower lactate levels</td>
<td>Stroke volume estimate directly obtained via Doppler signal of descending aorta; typically, patient must be sedated; inserted similarly to a nasogastric tube</td>
</tr>
<tr>
<td>Endotracheal bioimpedance</td>
<td>ConMed Corporation, Utica, New York</td>
<td>Operating room, intensive care unit</td>
<td>None</td>
<td>Bioimpedance technology via endotracheal tube; stroke volume obtained from impedance signal from ascending aorta; patient must be intubated</td>
</tr>
<tr>
<td>Transcutaneous bioimpedance</td>
<td>SonoSite, Bothell, Washington</td>
<td>Anywhere</td>
<td>None</td>
<td>Transcutaneous electrodes placed on neck and chest; electrical impedance between electrodes during cardiac cycle entered into nomogram to compute stroke volume; for best readings, patient must have normal anatomy</td>
</tr>
<tr>
<td>Pulse contour</td>
<td>FloTrac, Edwards Lifesciences, Irvine California</td>
<td>Operating room, intensive care unit</td>
<td>3 trials (each with conflicts of interest to disclose) suggesting decreased complications and length of stay (LidCO),[^53] (FloTrac),[^44] and (PiCCO)[^56] 3 trials suggesting unclear benefit (PiCCO)[^56] (conflict of interest disclosed) or no benefit (FloTrac)[^44] and (PiCCO)[^56]</td>
<td>Stroke volume estimated from arterial pressure waveform via methods such as lithium infusion, pulse pressure variation, or thermodilution; continuous cardiac output and beat-to-beat variability proportional to stroke volume; changes in systemic vascular resistance and arterial pressure necessitate recalibration; dampening, dysrhythmias, and ventilator triggering also limit accuracy</td>
</tr>
<tr>
<td>Exhaled carbon dioxide method</td>
<td>Philips Respironics, Andover, Massachusetts</td>
<td>Operating room, intensive care unit</td>
<td>None</td>
<td>Exhaled carbon dioxide method, with Fick equation; needs controlled mechanical ventilation to work; additional personnel, such as respiratory therapist, may be required; patient must be intubated</td>
</tr>
<tr>
<td>Pulmonary artery catheter</td>
<td></td>
<td>Operating room, intensive care unit</td>
<td>Several trials,[^58-60] with both pro and con findings</td>
<td>Measures cardiac output via thermodilution, temperature sensed by the catheter thermistor; stroke volume calculated by dividing cardiac output by heart rate; central venous access required via catheterization of right side of heart</td>
</tr>
<tr>
<td>Bioreactance</td>
<td>Cheetah Medical, Portland, Oregon</td>
<td>Operating room, intensive care unit</td>
<td>None</td>
<td>Like bioimpedance, uses transcutaneous electrodes; however, signal acquisition eliminates impedance errors present with the first-generation technology</td>
</tr>
</tbody>
</table>

[^44-52]: Ref 44-52
[^53]: Ref 53
[^44]: Ref 44
[^56]: Ref 56
[^58-60]: Ref 58-60

References:
- [44-52] None
- [53] 9 trials showing reduced length of stay, complications, use of vasopressors, renal insufficiency, and mortality and lower lactate levels
- [44] 2 trials had conflicts of interest to disclose
- [56] 3 trials suggesting unclear benefit (PiCCO) (conflict of interest disclosed) or no benefit (FloTrac) and (PiCCO)
- [58-60] 3 trials suggesting unclear benefit (PiCCO) (conflict of interest disclosed) or no benefit (FloTrac) and (PiCCO)
that cross the ultrasound transducer beam through the aorta during the systolic phase (Figures 4A and 4B). FTc corresponds to the width of the pulse waveform base and can be used to estimate preload. For example, a longer FTc suggests that the left ventricle is pumping forward an increased amount of blood (ie, increased preload). The width of the pulse wave is measured in milliseconds and represents the amount of time spent in systole compared with total cardiac cycle time, and FTc is also corrected for heart rate. The correction is based on a heart rate of 60/min, although the current heart rate is taken into account. If a patient’s heart rate is 60/min, then each cardiac cycle will last 1 second, or 1000 ms. Normal FTc is 330 to 360 ms. In other words, for a cardiac cycle lasting 1 second, the systolic flow period should last approximately 330 to 360 ms, provided that adequate preload exists. An easy way to remember the reference range is to remember that the heart is in diastole two-thirds of the time and that normal FTc multiplied by 3 equals 1 second, or 1000 ms (Figure 5). But normal reference ranges are really just reference points, not necessarily static physiological targets to be used for all patients. The most important value of FTc is the degree to which it changes in response to intravenous administration of fluids. Increases in FTc in response to volume challenge help confirm hypovolemia, which is manifested as a narrow waveform base and a low FTc (Figure 3).

The accuracy of FTc has been questioned. However, a complete understanding of the variable is critical before FTc and be used effectively in clinical practice. Simply put, FTc is suggestive of the amount of circulating volume that passes the tip of the ultrasound probe during systole. Therefore, conditions such as bleeding (hypovolemia), heart failure (low contractility), and high afterload (eg, vasoconstriction) may contribute to low blood-flow states and thus low FTc. These influences must be considered before FTc is accepted as a surrogate for preload in individual patients. Several investigators have suggested that FTc is as good as or better than PAOP for indicating changes in preload. Most important, however, improvement in stroke volume after fluid administration is what was intended to form the basis on which preload

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**Figure 5** Waveform components for stroke volume optimization (SVO): aortic pulse waveform from an esophageal Doppler examination. Corrected flow time (ie, the time spent in systole) corresponds to the width of the pulse waveform and is an index of preload. Peak velocity corresponds to the height of the wave and is a measure of contractility. Stroke distance represents the area under the curve and is used to compute stroke volume.
responsiveness is ultimately determined (in each of the outcome trials studying SVO). In other words, Ftc (as well as CVP and PAOP) is best used as a decision-making aid for optimizing stroke volume.

Measurement of Contractility: Peak Velocity

Peak velocity, a measure of contractility, is indicated by the amplitude of a Doppler waveform (Figure 5). It indicates the acceleration of blood flow in the systolic phase, or the speed at which a pressure wave goes from baseline to the peak height of contraction. An overall reference range is 50 to 120 cm/s. Peak velocity can be age dependent; the expected range for a 20- to 30-year-old is 90 to 120 cm/s, with gradually decreasing expected peak velocity as a person ages. Patients more than 65 years old are expected to have a peak velocity greater than 50 cm/s. Values less than 50 cm/s are suggestive of poor left ventricular contractility, as in heart failure. However, peak velocity should be evaluated with respect to a patient’s baseline values and how those values respond to treatments. For example, an increase in peak velocity is expected with administration of an inotrope.

A low stroke volume can occur for 1 of 2 main reasons: hypovolemia or decreased ventricular contractility. The immediate measured availability of peak velocity with Doppler techniques provides better information than do the derived contractility parameters of the PAC regarding why stroke volume may be low. For example, if stroke volume is low but peak velocity is normal, the problem most likely is hypovolemia. However, if both stroke volume and peak velocity are low, the problem most likely is left ventricular dysfunction. A patient’s response to medications such as preload reducers, afterload reducers, or inotropes can help differentiate the cause of the left ventricular dysfunction (eg, fluid overload, high afterload, or low contractility, respectively).

Peak velocity may also help detect acute decompensating systolic heart failure earlier than do other techniques for monitoring cardiac output. In critical illness, poor left ventricular contractility (low ejection fraction) may initially lead to a compensatory increase in end-diastolic volume, a change that implies a normal stroke volume. The ability to monitor peak velocity allows clinicians to recognize this decrease in contractility in real time and intervene before a decrease in stroke volume occurs. Further research is needed to better establish SVO treatment guidelines for patients with heart failure.

Measurement of Afterload: Systemic Vascular Resistance

Systemic vascular resistance (SVR) is the resistance that must be overcome by the ventricles to develop force and contract, propelling blood into the arterial circulation. Most of the newer hemodynamic monitoring technologies (eg, esophageal Doppler imaging, bioimpedance, pulse contour methods) have the capability to calculate SVR. However, SVR was not a major parameter in the algorithms used in any of the SVO trials that showed improved outcomes in surgical patients.

Evidence of lack of inclusion suggests that SVR is a more of a secondary monitoring parameter. Elevated SVR usually occurs in response to systemic hypertension or as a compensatory mechanism due to decreased cardiac output, as in shock states (Figures 1A and 1B). Therefore, nurses must know why the SVR is elevated. If the value is elevated in response to low cardiac output, once cardiac output is improved with treatment (eg, fluid, inotropes), SVR should decrease because of a decreased need for compensatory vasoconstriction. If SVR is elevated because of systemic hypertension, treatment may include administration of an afterload reducer.

When SVR decreases, the left ventricular ejection of blood encounters lower resistance. Low afterload states may be less problematic when blood pressure and cardiac output are normal (Figure 1A). However, attempts to increase low SVR generally include administration of vasopressors. ScvO₂ and stroke volume should also be followed as end points to ensure that blood flow and tissue oxygenation improve in response to the vasopressor (Figure 6). Titrating the dose of a vasopressor used to alter ScvO₂ and stroke volume allows clinicians to focus on optimizing blood flow to both the microcirculation and the macrocirculation. Several studies of fluid replacement protocols that include use of vasopressors suggest that optimizing ScvO₂ and stroke volume improve patients’ outcomes. However, further research is needed to better establish how vasopressors and ScvO₂ are best used in SVO protocols.

Stroke Volume, Stroke Index, and Stroke Distance

Stroke volume is one of the primary end points for detecting fluid responsiveness and guiding goal-directed therapy. Stroke index is a standardized parameter in which a patient’s body surface area is taken into account.
However, monitoring both stroke volume and stroke index is generally not necessary, because they use different units of measure to quantify the same value. Table 3 gives reference ranges for these parameters. However, the ideal stroke volume value is the one that contributes to adequate blood flow for tissue oxygenation without increasing heart rate.

Despite the unique advantages of measuring stroke volume, available technologies to measure this parameter at the bedside have some limitations. Even esophageal Doppler imaging, which provides a highly flow-directed estimation of stroke volume, uses a calculated estimation of aortic diameter based on the patient’s height and weight. Stroke distance may be a more accurate reflection of the Doppler estimation of stroke volume. Stroke distance is the distance a column of blood moves through the descending thoracic aorta during each systolic phase. Because stroke distance is used to calculate stroke volume, the recommendation is that stroke distance be evaluated to determine if the measurement of stroke volume is accurate.

The SVO Algorithm: Putting It All Together

The Frank-Starling principle states that the strength of cardiac contraction is directly related to the length of muscle fibers at end diastole, or preload. Administration of fluid on the basis of stroke volume allows clinicians to directly apply this principle. Figure 7 displays a standard example of an SVO fluid replacement algorithm, cited by Schober et al, that is based on a synthesis of experimental SVO protocols and literature. In this type of algorithm, determination of fluid responsiveness is used: fluid boluses are administered as long as stroke volume continues to improve by 10% or more. When administration of fluid boluses ceases to improve stroke volume by

---

**Table 3** Reference ranges for hemodynamic parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output, L/min</td>
<td>4-8</td>
</tr>
<tr>
<td>Stroke volume, mL</td>
<td>50-100</td>
</tr>
<tr>
<td>Stroke indexb</td>
<td>25-45</td>
</tr>
<tr>
<td>Flow time corrected, ms</td>
<td>330-360</td>
</tr>
<tr>
<td>Peak velocity, cm/s</td>
<td>30-120</td>
</tr>
<tr>
<td>Stroke distance, cm</td>
<td>10-20</td>
</tr>
<tr>
<td>Cardiac indexc</td>
<td>2.8-4.2</td>
</tr>
<tr>
<td>Systemic vascular resistance, dyne sec cm⁻⁵</td>
<td>900-1600</td>
</tr>
<tr>
<td>Saturation of central venous oxyhemoglobin, %</td>
<td>65-80</td>
</tr>
<tr>
<td>Central venous pressure, mm Hg</td>
<td>2-8</td>
</tr>
<tr>
<td>Stroke volume variation, %</td>
<td>&lt; 10-15</td>
</tr>
</tbody>
</table>

a Based on data from Ahrens, Lynn-McHale Wiegand, Lynn-McHale Wiegand and Carlson, and Edwards Lifesciences.
b Calculated as stroke volume in milliliters per heartbeat divided by body surface area in square meters.
c Calculated as cardiac output in liters per minute divided by body surface area in square meters.
10% or more, no more fluid is needed. Using this method of fluid administration can mitigate the risk of pulmonary edema, and bedside clinicians can be better assured that the patient is receiving enough fluid to optimize the macrocirculation but not more fluid than is needed.

SVR and blood pressure are usually not included in SVO algorithms and are considered secondary monitoring parameters in SVO. According to the SVO algorithm, SVR and blood pressure are evaluated only after peak velocity (contractility) and stroke volume are optimized, because SVR and blood pressure are more indirect reflections of cardiac output and are influenced by other factors (see Figures 1A and 1B). Furthermore, when blood flow and tissue oxygenation are measured rather than assumed, doses of vasopressors can be adjusted to optimize the end points of stroke volume (macrocirculation) and ScvO2 (microcirculation) rather than SVR and blood pressure (Figure 6). Stroke volume may improve initially with initiation and escalating doses of vasopressors, but changes in afterload due to further increases in the medication may impede stroke volume and cardiac output. Surveillance of ongoing stroke volume and cardiac output may help clinicians avoid this decrease in stroke volume and cardiac output.

Challenges to SVO implementation may include incorporating new hemodynamic monitoring technology into daily practice (eg, esophageal Doppler imaging, pulse contour method), education of staff members, support from physicians and leaders, and the paucity of literature to support use in nonsurgical patients. However, potential benefits include use of minimally invasive techniques, allowing earlier detection of unstable hemodynamic status, and reductions in morbidity, mortality, and length of stay. More research is needed to determine how values such as peak velocity and ScvO2 can be incorporated into the SVO algorithm. The following case studies illustrate these points and indicate how SVO can be applied in cases involving alterations in preload, afterload, and contractility.

Case Study 1: Decreased Preload

A 59-year-old man was admitted to the surgical intensive care unit after having a partial liver lobectomy...
after a motor vehicle accident (Table 4). On postoperative day 5, he was evaluated for discharge to a general care unit. His urine output had decreased during the preceding 12 hours, suggestive of hypovolemia. The hypovolemia was evidenced by low stroke volume, low FTc, and low ScvO₂ in the presence of a normal peak velocity. After injection of a 1000-mL bolus of physiological saline, stroke volume improved from 34 mL to 48 mL, more than a 10% (3.4 mL) improvement. So, another bolus was given. Satisfactory response to the bolus was manifested by normal FTc and ScvO₂. Stroke volume improved to 49 mL only with the second bolus (<10% improvement), indicating the beginning of the plateau along the Frank-Starling curve where increased stretching of the ventricular myocytes does not improve stroke volume. Thus, no further administration of fluid was indicated.

**Case Study 2: Decreased Preload Leads to Decreased Afterload**

A 55-year-old woman was admitted because of sepsis (Table 5). The patient had a dangerously reduced stroke volume, decreased FTc, decreased ScvO₂, and a normal peak velocity, indicating hypovolemia. She was deemed fluid responsive as indicated by an improvement in stroke volume from 26 mL to 50 mL, a greater than 10% (2.6 mL) improvement, after administration of a bolus of 1000 mL of physiological saline. So, another saline bolus was indicated. However, the patient did not respond to the second bolus, as evidenced by an improvement in stroke volume from 50 mL to only 51 mL (<10%), suggesting that the macrocirculation had been optimized. Norepinephrine was started because of the reduced ScvO₂ and persistent hypotension despite volume correction. The patient responded appropriately as evidenced by the

### Table 4 Interventions used and response of 59-year-old man admitted after a motor vehicle accident

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Stroke volume, mL</th>
<th>Flow time, corrected, ms</th>
<th>Peak velocity, cm/s</th>
<th>Heart rate, beats per minute</th>
<th>Central venous oxygen saturation, %</th>
<th>Central venous pressure, mm Hg</th>
<th>Blood pressure, mean (SD), mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Administer 1000-mL bolus of physiological saline</td>
<td>34</td>
<td>300</td>
<td>96</td>
<td>102</td>
<td>49</td>
<td>3</td>
<td>100/48 (64)</td>
</tr>
<tr>
<td>Administer 1000-mL bolus of physiological saline</td>
<td>48</td>
<td>335</td>
<td>95</td>
<td>100</td>
<td>69</td>
<td>5</td>
<td>94/55 (68)</td>
</tr>
<tr>
<td>Response</td>
<td>49</td>
<td>337</td>
<td>95</td>
<td>99</td>
<td>70</td>
<td>6</td>
<td>100/60 (73)</td>
</tr>
</tbody>
</table>

### Table 5 Interventions used and response of 55-year-old woman admitted for sepsis

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Stroke volume, mL</th>
<th>Flow time, corrected, ms</th>
<th>Peak velocity, cm/s</th>
<th>Heart rate, beats per minute</th>
<th>Central venous oxygen saturation, %</th>
<th>Central venous pressure, mm Hg</th>
<th>Blood pressure, mean (SD), mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Administer 1000-mL bolus of 0.9% normal saline</td>
<td>26</td>
<td>254</td>
<td>78</td>
<td>107</td>
<td>26</td>
<td>4</td>
<td>68/36 (47)</td>
</tr>
<tr>
<td>Administer 1000-mL bolus of physiological saline</td>
<td>50</td>
<td>341</td>
<td>76</td>
<td>105</td>
<td>48</td>
<td>9</td>
<td>76/42 (53)</td>
</tr>
<tr>
<td>Administer norepinephrine 10 μg/min</td>
<td>51</td>
<td>341</td>
<td>76</td>
<td>105</td>
<td>50</td>
<td>9</td>
<td>80/44 (59)</td>
</tr>
<tr>
<td>Response</td>
<td>55</td>
<td>344</td>
<td>72</td>
<td>106</td>
<td>68</td>
<td>8</td>
<td>92/62 (72)</td>
</tr>
</tbody>
</table>
increase in ScvO₂ to 68%, suggesting normalization of the microcirculation.

**Literature Supporting Clinical Usefulness of SVO**

Before they adopt a new practice, astute clinicians want to know that the practice is strongly supported in the literature. Randomized controlled trials are the highest-level research design, and the number of well-designed randomized controlled trials is directly correlated with the level of evidence assigned to a given practice.83-85 The findings of 11 randomized controlled trials,44-52,73,74 including 9 prospective trials,44-52 suggest that SVO results in improved patient outcomes. Despite a thorough literature review, we were unable to find a fluid replacement strategy supported by more research. The results of the 9 prospective trials,44-52 which included a total of about 1000 patients, consistently suggested that compared with conventional fluid replacement, SVO fluid replacement protocols contribute to decreases in overall hospital length of stay (by 2 days or more), complication rates, renal insufficiency, infection, use of vasopressors, blood lactate levels, and time-to-tolerance of oral intake. Appropriately implemented SVO programs that replicate these outcomes may also be associated with decreased costs.86

Notably, the sample in all 11 trials44-52,73,74 included perioperative patients. Although 2 of these trials44,47 also focused on postoperative care in the critical care unit, more research is needed to indicate the efficacy of SVO in nonsurgical patients. However, in perioperative patients, the strength of the supporting evidence in favor of SVO has been substantiated by large-scale systematic literature reviews conducted by the Agency for Healthcare Research and Quality,87 the National Health Service,86 and third-party payers such as the Centers for Medicare and Medicaid Services88 and Aetna.89

In 3 of these studies,86-88 the agencies recommended SVO protocols be used for monitoring cardiac output of patients receiving mechanical ventilation in the critical care unit and for surgical patients who require intraoperative fluid optimization. Esophageal Doppler imaging,88 bioimpedance,89 and PACs90 are all reimbursed by the Centers for Medicare and Medicaid Services88 on the basis of systematic literature reviews. However, esophageal Doppler imaging is the only technology also supported by the Agency for Healthcare Research and Quality.87

Similarly, the Cochrane Collaborative59 and the Agency for Healthcare Research and Quality60 have published technology assessments based on meta-analyses of outcomes related to use of PACs. The analyses indicated that the patients studied showed no evidence of benefit or harm from PACs. Among the reasons cited for the perceived lack of benefit was clinician-to-clinician variability in management of hemodynamic data obtained via PACs. In addition, the authors59,60 questioned the accuracy of the interpretation of the hemodynamic information in the studies analyzed and whether or not patient management strategies based on hemodynamic data were appropriate. Furthermore, none of the studies included use of a specific protocol for PAC use. This lack of a protocol is a key difference between PAC studies and SVO studies. Each of the 9 randomized control trials44-52 on SVO included a protocol for use of SVO. Use of a protocol is consistent with other studies of replacement protocols that include fluid therapy, which can be lifesaving when initiated early in the course of treatment. Findings from a meta-analysis of hemodynamic optimization by Poeze et al92 also suggest that replacement strategies such as SVO improve outcomes, including patient mortality, in high-risk surgical patients.

**Nursing Considerations**

Nursing considerations associated with incorporating SVO into bedside practice include acquiring and evaluating hemodynamic data, maintenance of skin integrity, sedation and analgesia, and nursing research.

**Acquisition and Evaluation of Hemodynamic Information**

Clinical proficiency with applying or inserting the hemodynamic monitoring device and adequate signal acquisition are key.83 Each device has its own unique signal acquisition technique and competency requirements. Inappropriate application of the device may produce inaccurate hemodynamic readings, leading to improper treatment decisions.72,73 Once accurate readings are obtained, understanding the appropriate application of “normal”
hemodynamic reference ranges to all patients is crucial. Tracking trends in hemodynamic values over time is generally more useful than is monitoring and treating on the basis of single data points, because transient changes in values may not be clinically importantly.

When readings are considered accurate and hypovolemia is identified, rapid infusion of a fluid bolus may optimize a patient’s response. Fluid infused via a pressure bag often produces a more dramatic increase in stroke volume than does fluid administered via an intravenous infusion pump. A maximum rate of commonly used intravenous pumps is 999 mL/h. Because hypovolemia and hypoperfusion are time-sensitive conditions, the provider’s judgment and the patient’s condition may determine that a more rapid infusion rate is needed.

The latest revision of the Surviving Sepsis Campaign guidelines also suggests an increased emphasis on earlier and more aggressive fluid replacement. For example, the 2008 guidelines recommended a 20 mL/kg crystalloid fluid challenge in a 6-hour replacement bundle. In the 2012 revised guideline, the recommended amount of fluid was increased (to 30 mL/kg) in a shorter time (3-hour bundle). Clinicians must strongly consider strategies to infuse such a volume rapidly enough, in accordance with institutional policy as appropriate.

**Maintenance of Skin Integrity**

Care must be taken to avoid skin breakdown under and around skin electrodes. With bioimpedance and bioreactance, signals are acquired transcutaneously, and skin care should be in accordance with the manufacturer’s recommendations and institutional policy. Mouth ulcerations are also possible with monitoring devices such as those used for esophageal Doppler imaging and endotracheally applied bioimpedance. Diligent oral care should be performed as needed while those devices are in place. Site care is also important when caring for patients monitored with intravenous pulse contour devices or PACs. Catheter infections can be minimized by using sterile conditions during insertion and aseptic technique during dressing changes.

**Sedation and Analgesia**

Sedation is sometimes required with techniques such as the exhaled carbon dioxide method, which requires controlled mechanical ventilation, and esophageal Doppler imaging. These techniques may have limited accuracy when increased respiratory rates or restlessness, respectively, occur. Therefore, sedative agents or analgesics may be administered as needed. Although not a major focus with respect to SVO, pain cannot be overlooked; it is not only an overall priority but can also influence hemodynamic readings.

**Nurse Research**

The implications of patient advocacy extend beyond routine patient care and include nurses’ participation in designing and implementing future research on the clinical usefulness of SVO in critical care. Critical care nurses monitor and treat hypovolemia daily and have a unique opportunity to contribute to the existing scientific body of knowledge through participation in SVO studies in medical critical care patients.

**Summary**

The growing body of evidence supporting SVO suggests that implementation of SVO into daily practice should be considered. A new era is emerging in which blood-flow monitoring is taking precedence over the monitoring of blood pressures. Cardiac pressures help provide estimates of blood volume; however, normal cardiac pressures can be observed in a patient in shock and provide little information about blood flow. Interpretation and treatment of blood pressures incorporate assumptions, whereas stroke volume may be considered a more precise measure of fluid responsiveness and an earlier warning sign of volume depletion than are urine output, altered mental status, CVP, heart rate, and blood pressure. Earlier signals allow clinicians to anticipate rather than react to changes, improving the likelihood for maintaining a stable metabolic state at the organ and cellular level. In addition to the evidence supporting SVO, minimally invasive applications and improvements in accuracy also add to safety advantages when inherent limitations of the various methods are considered.

For years, strategies for use of SVO were not feasible because no practical measurement method for SVO...
existing for bedside clinicians. Fortunately, technology has improved the hemodynamic monitoring landscape. Compared with old devices, newer technology is less invasive, safe, evidence based, flow directed, cost-effective, easier to use, and accurate. Although further research on SVO and dynamic indices are needed to establish the clinical efficacy of SVO in critical care units, the current body of literature indicates that SVO is associated with fewer complications and reduced hospital lengths of stay, particularly in patients receiving mechanical ventilation and in surgical patients. Until more randomized trials on the impact of SVO protocols on the outcomes of critical care patients are published, SVO is supported by more evidence than is use of filling pressures for fluid replacement in critical care units. On the basis of our review of the current available literature, we suggest that the SVO algorithm for fluid replacement be considered in place of use of cardiac filling pressures for patients in critical care, as appropriate, with attention to outcomes. In the meantime, more research is needed to evaluate the impact of SVO on patients other than perioperative patients and on nonintubated patients.

Financial Disclosures

Tom Ahrens has lectured for hemodynamic monitoring companies (including Deltec Medical Inc) and is a hemodynamic monitoring consultant.

Acknowledgments

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63. Singer M. The FTc is not an accurate marker of left ventricular preload: reply to the comment by Chemla and Nitenberg. Intensive Care Med. 2006;32(9):1456-1457.


1. Which of the following hemodynamic values help determine responsiveness to fluid replacement?
   a. Stroke volume within normal values
   b. Normal stroke volume with normal filling pressures
   c. Adequate blood flow for tissue oxygenation without increasing heart rate
   d. Afterload and preload stabilized

2. Which of the following is the first hemodynamic parameter to decrease in hypovolemia?
   a. Pulmonary artery occlusive pressure (PAOP)
   b. Central venous pressure (CVP)
   c. Cardiac output
   d. Stroke volume

3. After mitral valve replacement, your patient’s urine output decreases over 3 hours. The monitor displays sinus tachycardia with heart rate 108 beats/min, CVP 8 mm Hg, cardiac output 4 L/min, and mean arterial pressure (MAP) 80 mm Hg. The MAP remains within normal range because of which of the following?
   a. Normal cardiac output
   b. No change in circulating volume
   c. Compensatory mechanisms
   d. MAP is an independent parameter

4. Monitoring stroke volume gives the clinician insight into which of the following?
   a. Blood flow and circulating volume
   b. Cardiac filling pressures
   c. Oxygenation
   d. Contractility

5. Which of the following explains why clinicians prefer blood flow monitoring versus blood pressure monitoring?
   a. Compensatory mechanisms mask hypoperfusion
   b. Afterload, preload, and contractility are influenced by medications
   c. Blood pressure, MAP, and heart rate are late indicators of hypoperfusion
   d. All of the above

6. Which of the following changes is expected in a patient who received a 1000-mL fluid challenge that confirms hypovolemia?
   a. Increase in PAOP
   b. Increase in urine output
   c. Increase in stroke volume, corrected flow time, and cardiac output
   d. Increase in CVP and MAP

7. Volume and vasopressors are often the treatment of choice in a patient with shock. Which of the following parameters are best used as end points to guide therapy?
   a. Stroke volume, peak velocity, and corrected flow time
   b. Stroke volume, systemic vascular resistance, and central venous oxygen saturation (ScvO₂)
   c. ScvO₂, corrected flow time, and peak velocity
   d. ScvO₂ and stroke volume

8. Nursing implications in obtaining hemodynamic data include which of the following?
   a. Proficiency in application of monitoring device
   b. Application of data obtained to patient population
   c. Achieving optimal signal acquisition
   d. All of the above

9. Mechanical ventilation with positive end-expiratory pressure can impede blood flow and increase PAOP. How can clinicians determine proper interventions for this patient population?
   a. Trend and optimize stroke volume
   b. Trend cardiac filling pressures
   c. Closely follow pulmonary vascularity on the chest radiograph
   d. Trend blood pressure

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

1. [ ] a  [ ] b  [ ] c  [ ] d
2. [ ] a  [ ] b  [ ] c  [ ] d
3. [ ] a  [ ] b  [ ] c  [ ] d
4. [ ] a  [ ] b  [ ] c  [ ] d
5. [ ] a  [ ] b  [ ] c  [ ] d
6. [ ] a  [ ] b  [ ] c  [ ] d
7. [ ] a  [ ] b  [ ] c  [ ] d
8. [ ] a  [ ] b  [ ] c  [ ] d
9. [ ] a  [ ] b  [ ] c  [ ] d