Central venous oxygen saturation monitoring in the critically ill patient

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In the initial treatment of a critically ill patient, blood pressure, heart rate, urine output, and central venous pressure guide resuscitative efforts. Despite normalization of these variables, global tissue hypoxia may still persist and has been implicated in the development of multiorgan failure and increased mortality. Definitive management includes intensive care unit admission, pulmonary artery catheterization using mixed venous oxygen saturation (SvO_2), and hemodynamic optimization. In the absence of or before definitive management, hemodynamic optimization can be performed using central venous oxygen saturation ($ScvO_2$) as a surrogate. The physiology, technology, clinical uses, and rationale for $ScvO_2$ monitoring are reviewed, including issues regarding physiologic equivalence to SvO_2. The clinical use of $ScvO_2$ monitoring, evidence-based outcome implications, and limitations of $ScvO_2$ monitoring will also be examined. Curr Opin Crit Care 2001, 7:204–211

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Abbreviations

DO_2	oxygen delivery
EGDT	early goal-directed therapy
$ScvO_2$	central venous oxygen saturation
SvO_2	mixed venous oxygen saturation

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Shock is defined as the presence of global tissue hypoxia secondary to an imbalance between systemic oxygen delivery (DO_2) and demands. During initial patient management, physiologic variables such as blood pressure, heart rate, and urine output guide resuscitative efforts. Despite normalization of these variables, significant imbalances between DO_2 and demand result in global tissue hypoxia [1,2,3••]. This global tissue hypoxia, if left untreated, leads to anaerobic metabolism, lactate production, and oxygen debt. The magnitude and duration of oxygen debt have been implicated in the development of multisystem organ failure and increased mortality [4–6]. The current review examines the clinical utility of central venous oxygen saturation ($ScvO_2$) as a surrogate for mixed venous oxygen saturation (SvO_2) in the care of critically ill patients.

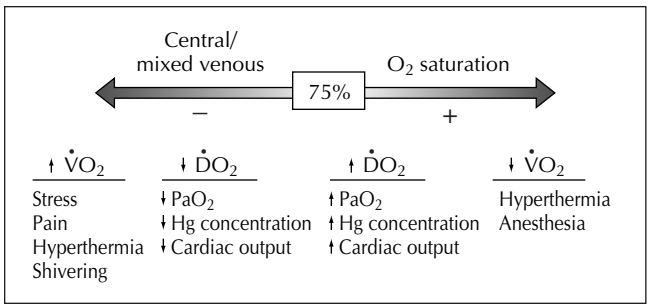
Physiology of mixed venous oxygen saturation

The normal range for SvO_2, which reflects the balance between DO_2 and demands, is 65 to 75% [7,8]. SvO_2 decreases when DO_2 has been compromised or systemic oxygen demands have exceeded supply. When this compensatory mechanism is overwhelmed and SvO_2 remains low, global tissue hypoxia and lactic acidosis ensues. SvO_2 has been used for its prognostic significance as a predictor of hyperlactemia and death (compared with arterial oxygen saturation and cardiac output in patients with severe cardiac and pulmonary disease) [7,9]. The objective of the clinician is to determine which of these variables are deranged and to institute appropriate therapy. Figure 1 illustrates the variables that affect SvO_2.

Technology of venous oximetry

By using infrared oximetry, which is based on reflection spectrophotometry, SvO_2 can be monitored continuously. Light is transmitted into the blood, reflected off red blood cells, and read by a photo detector. The amount of light reflected at different wavelengths varies depending on the concentration of oxyhemoglobin and hemoglobin (Fig. 2). The catheter used to measure venous oxygen saturation can be a pulmonary artery catheter or a 16 to 22 cm central venous catheter capable of fluid or medication administration and pressure measurements.

Figure 1. Variables that affect mixed venous oxygen saturation



Central or mixed venous oxygen saturation can be decreased as a result of variables that increase systemic oxygen consumption or a decrease in systemic oxygen delivery. DO_2, systemic oxygen delivery; Hg, mercury; PaO_2, arterial oxygen pressure; VO_2, systemic oxygen consumption.

Clinical uses of mixed venous oxygen saturation monitoring

Used extensively in various clinical scenarios, SvO_2 has been shown to be superior to mean arterial pressure and heart rate in cardiac surgery patients [10]. Declines in SvO_2 preceded the onset of inadequate myocardial function [11], shock, or the development of arrhythmias, although vital signs were normal [12]. SvO_2 has been shown to have diagnostic, prognostic, and therapeutic use in the treatment of critically ill patients who have acute myocardial infarction [13,14] or general medical intensive care unit (ICU) conditions [15]; have undergone postoperative cardiovascular procedures [16••], vascular surgery [20,21], pediatric surgery [11], or lung transplantation [26]; have experienced trauma [17–19], septic shock [22,23], or cardiogenic shock [24,27]; or are neonates [25]. Although SvO_2 has not been shown to have outcome benefit as a goal-directed hemodynamic end-point in a heterogeneous group of patients after ICU admission [28], it has shown to decrease morbidity and health care–resource consumption in postoperative cardiovascular [16••] and trauma [17] patients. There is evidence that the timing of diagnostic and therapeutic intervention using this technology may be a critical outcome determinant that has not been addressed in prior studies [29••].

Why monitor central venous oxygen saturation?

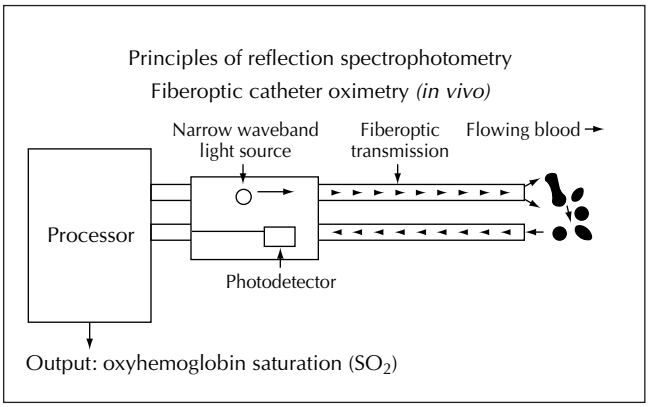
The time between the onset of critical illness and definitive ICU intervention can be significantly long and have outcome implications [30,31••,32]. Measurement of SvO_2 requires placement of a pulmonary artery catheter, which may not be feasible early in resuscitation or in pediatric patients. However, central venous assess can be obtained in both ICU and non–ICU settings, which makes continuous $ScvO_2$ monitoring a convenient surrogate for SvO_2. Up to 50% of patients resuscitated from shock may have continued global tissue hypoxia (*ie*, increased lactate and decreased $ScvO_2$) even with the normalization of vital signs and central venous pressure [3••]. The ability to detect and resolve occult tissue hypoxia early in the course of patient care may have outcome benefit [16••,29••].

Are mixed central venous oxygen saturation clinically equivalent?

The relation between SvO_2 and $ScvO_2$ obtained from the superior vena cava and right atria has been examined in animal and human models (Table 1). Superior vena cava $ScvO_2$ is slightly less than right atria $ScvO_2$ and more accurately reflects SvO_2 when patients are not in shock [33,34••]. Right atria saturations are not significantly different from SvO_2 whether or not in shock [33]. For patients in shock a consistent reversal of this relation occurs and superior vena cava $ScvO_2$ is always greater than SvO_2 [33]; the difference can range from 5 to 18% [33,34••]. Redistribution of blood flow away from the splenic, renal, and mesenteric bed toward the cerebral and coronary circulation, including more desaturated blood (< 30%) from the coronary sinus, contributes to this observation [33]. Thus, superior vena cava $ScvO_2$ consistently overestimates the true SvO_2 under shock conditions.

There has been considerable debate regarding whether $ScvO_2$ in a satisfactory substitute for SvO_2, particularly in ranges above 65% [35•,36–39]. Although the absolute values of $ScvO_2$ and SvO_2 differ, studies have shown close tracking of the two sites across a wide range of hemodynamic conditions [40••]. Furthermore, the presence of a pathologically low $ScvO_2$ value (implying an even lower SvO_2) is more clinically important than whether the values are equal. Goldman *et al.* [13] found that $ScvO_2$ less than 60% showed evidence of heart failure, shock, or a combination of the two. Hyperdynamic septic shock

Figure 2. The technology of spectrophotometry



The technology of spectrophotometry involves sending an infrared signal by fiberoptic transmission through the central venous catheter. A receiving fiber in the catheter detects reflected light off of hemoglobin in red blood cells to provide a continuous read-out of central venous oxygen saturation.

Table 1. Studies comparing mixed venous oxygen saturation and central venous oxygen saturation

Study	Study design and subjects	Results	Conclusions
Reinhart *et al.* [40••]	Hypoxia, hemorrhage, and resuscitation in anesthetized dogs.	The correlation coefficient (r) between SvO_2 and $ScvO_2$ was 0.97 with a mean difference of 2.7 ± 2.9% saturation. In each dog the changes in $ScvO_2$ closely paralleled the changes in SvO_2.	Although absolute values of $ScvO_2$ are not identical to SvO_2, close tracking of changes in the two sites across a wide range of hemodynamic conditions.
Scalea *et al.* [57]	Hemorrhage dog model.	Cardiac index and SvO_2 showed linearity as function of measure blood loss. SvO_2 mirrors $ScvO_2$ r values generated by linear-regression anlaysis that ranged from 0.85–0.99 with a mean of 0.95 for SvO_2.	$SvcO_2$ is a reliable substitute for SvO_2 in the hemorrhage shock model.
Baquero-Cano *et al.* [58]	Neonatal piglet sepsis model.	Significant correlation existed between SvO_2 and $ScvO_2$ ($r^2 = 0.88$).	$ScvO_2$ can be a sure, efficient, and easy alternative for SvO_2 in neonatal patients
Schou *et al.* [59]	Hemodilution pig model.	The regression coefficient bwtween SvO_2 and $ScvO_2$ was 0.97 ($r^2 = 0.93$, bias −2.4 ± 5.8%) in the hemodiluted group and 0.99 ($r^2 = 0.97$, bias −3.0 ± 5.0%) in the control group.	During hemodilution, $ScvO_2$ monitoring may be as useful as SvO_2 monitoring.
Davies *et al.* [60]	Pig model of circulatory shock and chemically induced lung damage.	The difference between the overall means of right atria $ScvO_2$ and SvO_2 was 0.91% with a standard error of the estimate of 4.7%, a regression equation of right atria $SvO_2 = SvO_2$ (0.94 ± 2.4)PA SvO_2, and a correlation coefficient of 0.94.	SvO_2 monitoring may be accomplished less invasively and at a lower cost with a right atrial catheter.
Shah *et al.* [61]	Hemorrhagic shock rat model.	Increasing the severity of shock progressively worsened the acidosis, with increased base deficit and lactate and deterioration in $ScvO_2$. $ScvO_2$ predicted subsequent mortality. Lactate levels only predicted irreversibility in late severe shock.	$ScvO_2$ can be used to monitor the progression from the decompensated phase of hemorrhagic shock to irreversibility.
Lee *et al.* [33]	Non shock and shock (hemorrhagic, septic, and neurogenic) patients.	In shock, $ScvO_2$ was significantly greater than SvO_2 ($r^2 =$ 0.73). The correlation between right atria $ScvO_2$ and SvO_2 was 0.95. Right atria $ScvO_2$ was more accurate in reflecting changes in SvO2 (0.81 *vs* 0.27).	$ScvO_2$ levels are consistently greater than those of SvO_2 in shock. Right atria $ScvO_2$ is superior to superior vena cava SvO_2 in reflecting SvO_2.
Scheinman *et al.* [34••]	Critically ill patients	$ScvO_2$ levels in the superior vena cava are significantly greater than those of SvO_2 in shock (58 ± 13 *vs* 47.5 ± 15; (r = 0.55). Although the correlation decreases with the onset of shock, changes in $ScvO_2$ in shock reflect changes in SvO_2 (r = 0.90). ScvO2 from the right atria is similar to SvO_2 (49.2 ± 19 *vs* 49.2 ± 19; r = 0.96).	$ScvO_2$ levels are consistently greater than those of SvO_2 but there is a poor correlation with SvO_2 in heart failure of shock. although right atria SVO_2 levels more accurately replaceSvO_2, superior vena ava $ScvO_2$ is helpful in monitoringtrends or changes in SvO_2.
Goldman *et al.* [13]	Myocardial infarction patients.	Good correlation between $ScvO_2$ in wide range cardiac outputs.	$ScvO_2$ accurately relfecs SvO_2. $ScvO_2 <$ 60% is associated with heart failure or shock.
Tahvanainen *et al.* [62]	Estimated the value of $ScvO_2$ as representative of real changes in pulmonary shunt (Qsp/Qt), SvO_2, and arteriovenous oxygen content difference [$C(a\text{-}v)O_2$] during active phases of adult intensive care.	A significant positive correlation of the measured variables (and especially of the subsequent changes of these variables) in individual patients between PA blood samples and both superior vena cava and right atrial blood samples ($P < 0.001$).	$ScvO_2$ can replace SvO_2 for the aforementioned purposes. However, the exact value of $ScvO_2$ can olny be meausred from blood collected from the PA itself.
Faber [37]	Critically ill patients.	Although the proposed algorithm had a fairly high power of prediction, its merits in comparison to assuming simple proportionality between central venous and mixed venous oxygen content seemed marginal.	Because it is likely that the results so far are mathematically coupled, further prospective studies are necessary.
Herrera *et al.* [63]	Compared SvO_2 and $ScvO_2$ in 23 patients undergoing thoracic surgery	At baseline SvO_2 was > $SvcO_2$ and the mean difference was always < 0.9%. During surgery $SvcO_2$ was always > SvO_2 and the mean difference was < 1.3%. Simple linear correlation was significant ($P < 0.001$) for each of the measurements and for the whole sample. Bias (0.2 and 0.7%) and its standard deviation (2.7 and 2.5%) between the two techniques were small and the differences between all measurements were less than 5%.	For thoracic anesthesia in patients who are not good candidates for catheterization of the pulmonary artery, SvO_2 may be substituted for $SvcO_2$ to monitor the balance of supply and demand.
Edwards *et al.* [35••]	Patients in severe circulatory shock immediately on ICU admission.	Superior vena cava, right atria, and PA values were similar: 74 ± 12.5%, 70 ± 13%, and 71.3 ± 12.7%, respectively. However, the ranges and 95% confidence limits were −19.3 to +23.1% and −19.7 to +16.7%, respectively, and the 95% confidence limits were −18.4 to +24.2% and −18.6 to +17.3%, respectively.	SvO_2 is only reliably measured in samples taken from the PA.
Berridge [64]	Patients in the ICU with low, medium, and high cardiac indexes.	The correlation coefficients of the three groups were: low cardiac index, 0.95; mediaum cardac index; 0.88; and high cardiac index 0.95 ($P < 0.001$).	These results suggest that $ScvO_2$ is a useful estimate of SvO_2 and that the influence of cardiac output on that estimate is minimal.

Table 1. Studies comparing mixed venous oxygen saturation and central venous oxygen saturation (continued)

Study	Study design and subjects	Results	Conclusions
Kong *et al.* [65]	Patients with end-stage renal failure.	Cardiac index was lower in patients 2.45 ± 0.42 L/min/m^3) compared with the control subjects (3.74 ± 0.17 L/min/m^3). SvO_2 was 53 ± 8% and $ScvO_2$ (superior vena cava) was 57 ± 6%.	Venous oxygen saturations are similar in end-stage renal failure.
Wendt *et al.* [66]	Patients in the ICU.	The correlation of oxygen partial pressures was 0.687 and the correlation of the saturation reached 0.779. Thecalculation of venous admixture showed a correlation of 0.901.	$ScvO_2$ adequately reflects SvO_2.
Emerman *et al.* [67]	Experimental cardiac-arrest dog model.	PO_2, PCO_2, and pH from the pulmonary artery samples were strongly correlated with those from the central venous (r = .93, .99, and .99, respectively) and from the femoral venous samples (r = .73, .93, and .97, respectively). There were no significant differences in the pulmonary artery, central, or femoral venous gases.	Femoral and central venous samples mirror true mixed venous blood gases from the PA and could be used in their place.
Martin *et al.* [36]	Continuous monitoring in critically ill patients with and without therapeutic interventions	Systematic error was 0.6% and 0.3% and variability was 10%. Differences were greater than or equal to 5% in 49% of values during periods of stability and in 50% of values during periods with therapeutic interventions. Correlation was r = 0.48 without and r = 0.62 with therapeutic interventions. changes n SvO_2 and $SvcO_2$ during periods without and with therapeutic interventions were r = 0.70 and r = 0.77, respectively.	The present study indicates that $ScvO_2$ monitoring was not reliable in the study patients.
Martin *et al.* [68]	Open-chest CPR animal model.	The correlation between $ScvO_2$ and SvO_2 was 0.87 ($P <$ 0.001) before arrest but deteriorated at all times during CPR with values ranging from 0.1589 (P = -0.542) to 0.5781 (P = 0.024).	Although statistically significant at times, the correlation between $ScvO_2$ and SvO_2 during CPR is not consistently high enough to enable the routine substitution in this model.

CPR, cardiopulmonary resuscitation; ICU, intensive care unit; PA, pulmonary artery, $ScvO_2$, central venous oxygen saturation; SvO_2, mixed venous oxygen saturation.

ICU patients seldom exhibit SvO_2 levels less than 60 to 65%, and sustained levels are associated with increased mortality [22,23]. Studies examining the clinical utility of $ScvO_2$ early in the course of disease presentation routinely encounter values less than 50%, which are considered critical [3,29••,41•]. At these values, venous saturations are actually 5 to 18% lower in the pulmonary artery [33,42] and 15% lower in the splanchnic bed [43]. Thus, although not numerically equivalent, these range of values are pathologically equivalent [13] and associated with high mortality [9].

Clinical uses of central venous oxygen saturation monitoring

Rady *et al.* [3••] found that 50% of critically ill patients presenting in shock who were resuscitated to normal vital signs continued to have increased lactate and abnormally low $ScvO_2$, indicating anaerobic metabolism and oxygen debt. These patients required further interventions. Rady *et al.*'s study gave rise to the clinical use of $ScvO_2$ in early management of cardiac arrest, the postresuscitation period, trauma and hemorrhage, severe heart failure, severe sepsis, and septic shock (Table 2).

Cardiac arrest

Management of the cardiac-arrest patient by advanced cardiac life support guidelines includes physical examination (*ie*, palpation of a pulse) and electrocardiographic monitoring. $ScvO_2$ monitoring during cardiac arrest has been shown to be a diagnostic and therapeutic adjunct [44–46]. Cardiac-arrest patients routinely have $ScvO_2$ values of 5 to 20% during cardiopulmonary resuscitation. Failure to reach $ScvO_2$ of at least 40% during the management of cardiac arrest is associated with a 100% mortality rate even when there is intermittent measurable blood pressure. $ScvO_2$ has been used to confirm the presence or absence of sustainable cardiac activity during electromechanical dissociation (EMD) or a pulseless idioventricular rhythm (where over 35% of patients have been shown to have spontaneous cardiac activity [psuedo-EMD]) [47]. If $ScvO_2$ is greater than 60%, return of spontaneous circulation (ROSC) is likely and the pulse should be frequently rechecked if EMD is present. Between $ScvO_2$ values of 40% and 72%, there is a progressive increase in the rate of ROSC. When $ScvO_2$ greater than 72% is obtained, ROSC has likely occurred. Continuous $ScvO_2$ monitoring provides an objective measure to confirm the adequacy or inadequacy of cardiopulmonary resuscitation in providing DO_2.

Postresuscitation after cardiac arrest

In the immediate postresuscitation period, patients are frequently hemodynamically unstable and have a high frequency of re-arrest. Cuff and intraarterial pressures may be rendered insensitive in the measurement of

Table 2. Clinical studies of central venous oxygen saturation

Study	Study design and subjects	Results	Conclusions
Ander *et al.* [41••]	Examined the use of lactic acid levels and $ScvO_2$ to stratify and treat patients with acutely decompensated end-stage chronic CHF who presented to the emergency department. The patents were divided into a high–lactic-acid group (n = 22), a low–lactic-acid group (n = 5), and a control group (stable patients presenting to a cardiology clinic, n = 17).	There was no statistical difference in vital signs or Killip and New York Heart Association criteria between the three groups. $ScvO_2$ was significantly lower in the high–lactic-acid group (32 ± 12%) than in the normal–lactic-acid (51 ± 13%) and control (60 ± 6%) groups ($P = 0.001$). After treatment there was a significant decrease in lactic acid (–3.65 ± 3.65 mol/L) and an increase in $ScvO_2$ (32 ± 13%) in the high–lactic-acid group compared with the normal–lactic-acid group ($P = 0.001$).	A significant subset of patients with decompensated end-stage CHF present to the emergency department in occult shock and are clinically indistinguishable from patients with mildly decompensated or stable CHF. Once identified, these patients require aggressive alternative management and disposition.
Scalea *et al.* [51]	Patients had injury mechanisms that suggested blood loss but were deemed stable after initial evaluation.	Despite stable vital signs, 10 patients (39%) had $ScvO_2$ saturations < 65%. These patients had more serious injuries and significantly larger estimated blood losses, and required more transfusions than did those patients with $ScvO_2$ saturation > 65%. Linear regression analysis demonstrated the superiority of $ScvO_2$ saturation to predict blood loss ($P < 0.005$) relative to any of the normally allowed parameters.	$ScvO_2$ saturation is a reliable and sensitive method for detecting blood loss. It is useful tool in the evaluation of acutely injured patients.
Kowalenko *et al.* [52]	Critically ill trauma and hemorrhage patients.	Patients initially resuscitated to normal vital signs who had sustained decreases in $ScvO_2$ required acute interventions (procedures or surgery) compared with those who had normal $ScvO_2$.	$ScvO_2$ is clinically useful in the treatment of trauma and hemorrhage.
Nakazawa *et al.* [46]	Compared end-tidal CO_2 monitoring with $ScvO_2$ monitoring for cardiac arrest patients.	During the complete stasis of systemic circulation, when defibrillation was done, $ScvO_2$ did not change, whereas $ETCO_2$ gradually decreased. However, the decrease in $ScvO_2$ temporally occurred when chest compression was resumed. $ScvO_2$ monitoring had great advantage to detecting peripheral tissue oxygenation. $ScvO_2$ seems to be no less accurate and reliable monitoring than capnogram during CPR procedures.	$ScvO_2$ is as accurate and reliable a monitor as the capnogram during CPR procedures. Because the capnogram is noninvasively and easily used in cardiac arrest patients, $ScvO_2$ monitoring combined with the capnogram is the preferred method for assessing the efficacy of ongoing CPR.
Rady *et al.* [3,69]	Critically ill patients in the emergency department admitted to the ICU	Additional therapy is required for 50% of critically ill patients to resolve global tissue hypoxia after initial resuscitation and hemodynamic stabilization in the emergency department to normal blood pressure and heart rate.	The measurement of $ScvO_2$ and lactate can be utilized to guide this phase of additional therapy in the emergency department.
Madsen *et al.* [70]	Compared $ScvO_2$ and CVP as indices of effective blood volume in humans.	CVP decreased from 3 (1–6) to 1 (–3–5) mm Hg ($P = 0.05$) but thereafter remained stable. In contrast, $ScvO_2$ showed a linear decrease with time from 0.75 (0.69–0.78) at rest to 0.60 (0.49–0.67) when presyncopal symptoms appeared ($P = 0.01$).	Reduced central blood volume is reflected more clearly with $ScvO_2$ than in CVP.
Rivers *et al.* [71•]	Out-of-hospital cardiopulmonary arrest patients.	Patients (n = 100) with return of spontaneous circulation (ROSC) had a higher initial mean, and maximal $ScvO_2$ than did those without ROSC. No patient attained ROSC without reaching an $ScvO_2$ of at least 30%. An $ScvO_2$ of greater than 72% was 100% predictive of ROSC.	Continuous central venous oxygen saturation monitoring can reliably indicate ROSC during CPR in humans.
Synder *et al.* [44]	In-hospital cardiopulmonary arrest patients.	Of 14 patient with an initial $PvO_2 > 37$ torr, 12 had ROSC and survived 24 hours; all of the 29 patients with a $PvO_2 < 31$ torr were nonsurvivors.	PvO_2 appears to be a reliable predictor of poor short-term outcome after in-hospital cardiopulmonary arrest and CPR.
Hirschl *et al.* [56]	Animal (rabbit) neonatal model.	Significant changes in $ScvO_2$ were seen with changes in DO_2, peak inspiratory pressure, positive end-expiratory pressure, and progressive hypovolemia.	$ScvO_2$ monitoring may be a powerful tool in the management of critically ill neonates.

CHF, congestive heart failure; CPR, cardiopulmonary resuscitation; CVP, central venous pressure; DO_2, oxygen delivery; $ETCO_2$, end tidal carbon dioxide; ICU, intensive care unit; PvO_2, partial venous oxygen pressure; $ScvO_2$, central venous oxygen saturation; SvO_2, mixed venous oxygen saturation; ROSC, restoration of spontaneous circulation.

cardiac output or DO_2 secondary to the high systemic vascular resistance of catecholamine therapy [1,45,48]. An abrupt or gradual decrease in $ScvO_2$ (< 40–50%) indicates likelihood for re-arrest, whereas $ScvO_2$ greater than 60 to 70% indicates hemodynamic stability. A sustained extreme elevation of $ScvO_2$ (> 80%) in the presence of a low DO_2 carries a poor prognosis because it indicates an impairment of systemic oxygen consumption (*ie*, the inability of the tissues to use oxygen). This has been attributed to long periods of

arrest and the use of large doses of vasopressors [49]. If this derangement is not corrected within the early postresuscitation period, the outcome is uniformly fatal [45].

Traumatic and hemorrhagic shock

The standards of Advanced Trauma Life Support focus on normalization of vital signs [50•]. Studies have shown that vital signs are insensitive end-points of resuscitation and outcome predictors in hemorrhage and trauma resuscitation [1,51]. Kowalenko *et al.* [52] and Scalea *et al.* [51] have shown that patients presenting with trauma and hemorrhage required additional resuscitation or surgical procedures when $ScvO_2$ remained less than 65%.

Occult cardiogenic shock in severe heart failure

Cardiogenic shock is characterized by decreased DO_2 and evidence of tissue hypoxia (*eg*, lactic acidosis, end-organ dysfunction) secondary to myocardial dysfunction [24]. This definition is most commonly seen in acute pump dysfunction (*eg*, acute myocardial infarction). In chronic severe heart failure, this presentation may be insidious. Ander *et al.* [41•] examined patients who presented with decompensated chronic severe heart failure (ejection fraction < 30%) who were stratified into normal and elevated lactate (> 2 mM/L) groups. There was a significant prevalence of "occult cardiogenic shock" with $ScvO_2$ ranging from 26.4 to 36.8% in the presence of normal vital signs. Using a goal-oriented approach of preload, after-load, contractility, coronary perfusion, and heart-rate optimization, these patients required additional therapy whereas their counterparts with normal lactate levels did not [41••].

Severe sepsis and septic shock: an outcome evaluation of early intervention

Previous studies have examined SvO_2–guided goal-directed therapy for severe sepsis and septic shock after ICU admission. In a study evaluating early goal-directed therapy (EGDT) using $ScvO_2$ before ICU admission, patients presenting with severe sepsis and septic shock were randomized to 6 hours of EGDT or standard therapy before ICU admission. Patients in both groups were resuscitated to a central venous pressure greater than 8 mm Hg and mean arterial pressure greater than 65 mm Hg, but those in the treatment group were resuscitated to an $ScvO_2$ greater than 70% using continuous $ScvO_2$ monitoring. Serial end-points of resuscitation, physiologic and organ dysfunction scores, mortality rate, and health care–resource consumption were compared at 0, 6, 12, 24, 36, 48, 60, and 72 hours.

There were no significant differences in 0-hour variables between the EGDT and control group patients. Over the initial 72 hours, there was a higher central venous O_2 saturation (65.4 ± 8.9% *vs* 57.1 ± 13%, $P < 0.001$), lower lactate (3.1 ± 3.0 mmol/L *vs* 4.0 ± 3.8 mmol/L, $P = 0.025$), lower base deficit (2.4 ± 4.6 mEq/L *vs* 5.6 ± 6.0 mEq/L, $P < 0.001$), and higher pH (7.39 ± 0.20 *vs* 7.35 ± 0.14, $P = 0.01$) for those in the EGDT group than for those in the control group. Valus for Acute Physiology and Chronic Health Evaluation Score-II, Simplified Acute Physiology Score-II, and Multiple Organ Dysfunction Syndrome were lower for patients in the EGDT group at all time points than they were for those in the control group (all $P < 0.001$). In-hospital mortality was 26.1% versus 42.4% ($P = 0.009$) and 28-day mortality was 30.5% versus 48.2% ($P = 0.008$) for those in the EGDT group versus the control group, respectively. In survivors, duration of hospital stay and mechanical ventilation were 3.8 ($P = 0.001$) and 1.4 days ($P = 0.001$) less in the EGDT group than in the control group, respectively. Using $ScvO_2$ as a resuscitation end-point in addition to mean arterial pressure and central venous pressure provides significant outcome benefit for patients with severe sepsis and septic shock over standard therapy.

Limitations and future questions

Despite studies questioning the value of SvO_2 in treating ICU patients [45,53–55], considerable evidence suggests that $ScvO_2$ has a beneficial role in the early management of critically ill patients and even neonates [25,56]. The ability to access this information earlier in the phases of critical illness is now a reality and further studies are now in progress to confirm that early recognition and treatment of deranged $ScvO_2$ values have significant outcome benefit.

Conclusions

In the resuscitation of critically ill patients, $ScvO_2$ monitoring has been shown to be a better indicator of tissue oxygenation and derangement of cellular oxygen utilization than vital signs. When applied to the treatment of various shock states, $ScvO_2$ monitoring impacts therapeutic intervention; there is evidence that this may be of outcome benefit over conventional therapy, which uses vital signs and central venous pressure.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • Of special interest
- •• Of outstanding interest

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