Comparison of Central Venous to Mixed Venous Oxygen Saturation in Patients With Low Cardiac Index and Filling Pressures After Coronary Artery Surgery

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Objective: **To evaluate the correlation and agreement between mixed venous oxygen saturation (SvO_2) and central venous oxygen saturation ($ScvO_2$) in patients with low cardiac index and filling pressures after coronary artery surgery.**

Design: **Prospective observational study.**

Setting: **Tertiary care academic hospital.**

Participants: **Sixty consecutive patients with a cardiac index <2 L/min/m² and a pulmonary artery occlusion pressure <12 mmHg after coronary artery surgery were included.**

Interventions: **Patients were monitored by a pulmonary artery catheter and a central venous catheter positioned in the superior vena cava.**

Measurements and Results: **SvO_2 and $ScvO_2$ were simultaneously measured before (T0) and after (T1) normalization of the cardiac index (>2.5 L/min/m²) by fluid therapy. Sixty pairs of measures were obtained at T0 and at T1. Bias between SvO_2 and $ScvO_2$ was −0.6% (T0) and −0.8% (T1). Limits of agreement were from −19.2% to 18% (T0) and from −15.6% to 14% (T1), and the correlation coefficient was 0.463 (T0) and 0.72 (T1). SvO_2 and $ScvO_2$ changes from T0 to T1 (ΔSvO_2 and $\Delta ScvO_2$) were calculated. The bias between ΔSvO_2 and $\Delta ScvO_2$ was −0.25. Limits of agreement were from −20% to 19.5%, and the correlation coefficient was 0.6.**

Conclusions: **In patients with low cardiac index and filling pressures after coronary artery surgery, $ScvO_2$ could not be used as a direct alternative for SvO_2. After fluid therapy and normalization of the cardiac index, differences between individual values remained large, and the disagreement between $ScvO_2$ and SvO_2 changes was significant.**

KEY WORDS: cardiovascular monitoring, oxygen consumption and delivery, perioperative care, mixed venous oxygen saturation

MIXED VENOUS OXYGEN SATURATION (SvO_2) is a valuable measurement in hemodynamically unstable patients after cardiac surgery.[1,2] This parameter indicates the balance between oxygen supply and demand and thereby provides an index of tissue oxygenation.[3] Furthermore, it allows calculation of tissue oxygen consumption, oxygen extraction ratio, and the degree of pulmonary venous admixture.[4] However, SvO_2 measurement is obtained only from a correctly positioned pulmonary artery catheter. The insertion of these catheters is associated with significant risks, severe complications as well as increased health care costs,[5,6] and risk/benefit ratio is currently a matter of debate in patients undergoing cardiac surgery.[7]

Central venous oxygen saturation ($ScvO_2$), obtained in a less risky and costly manner from a central venous catheter, may be a useful alternative to SvO_2 measurement. Several studies were performed both in animals[8,9] and in critically ill patients[10-19] to evaluate the correlation between SvO_2 and $ScvO_2$ and the reliability of $ScvO_2$ in clinical decision-making. In cardiac surgery, studies comparing SvO_2 and $ScvO_2$ are limited and have conflicting results.[20-23] Moreover, none of these studies was carried out specifically in patients with low cardiac index and filling pressures after coronary artery surgery. Such a study would be important to determine if, in this specific and very common population of post–cardiac surgical patients, $ScvO_2$ can be used as a surrogate for SvO_2 in therapeutic decisions such as the administration of fluid and vasoactive drugs.

The aim of the present study was to evaluate the correlation and the agreement between the values of SvO_2 and $ScvO_2$ in patients presenting with a low cardiac index and filling pressures after coronary artery surgery. The correlation between changes of SvO_2 and $ScvO_2$ after fluid therapy was also assessed.

METHODS

This prospective observational study was approved by the institutional review board, and all participants gave informed written consent. Three hundred adult patients underwent elective coronary artery surgery in the authors' hospital over a period of 6 months. Anesthesia was induced and maintained by using etomidate, 0.3 mg/kg, pancuronium, 0.2 mg/kg, midazolam, 0.1 mg/kg/h, fentanyl, 5 μg/kg/h, and isoflurane up to 1 minimum alveolar concentration. Invasive homodynamic monitoring was achieved with a 20-G radial artery catheter, a 7.5F pulmonary artery catheter (Edwards Lifesciences, Irvine, CA) inserted through the right internal jugular vein and a 7F central venous catheter (Arrow-Howes, Reading, PA) inserted through the right subclavian vein. Cardiopulmonary bypass was performed under hemodilution and mild hypothermia by using a membrane oxygenator primed with a crystalloid solution. Myocardial preservation was achieved with a cold cardioplegia solution containing 30 mmol of potassium per liter. Coronary anastomoses were done under total aortic cross-clamping.

At the time of admission in the cardiac surgery unit (CSU), patients exhibiting a cardiac index ≤2 L/min/m² and a pulmonary artery occlusion pressure ≤12 mmHg were included in the study. Patients presenting a cardiac index ≤2 L/min/m² anytime after admission to the CSU were not enrolled. Pulmonary artery occlusion pressure was displayed on an electronic monitor (Hellige SMU 611; Hellige AG, Freiburg, Germany). Cardiac output was measured by the thermodilution technique. Cardiac index was computed by averaging 3 consecutive measurements of cardiac output divided by the patient body surface area. The position of the pulmonary artery catheter was confirmed by the transduced waveforms. The location of the central venous catheter tip 2 to 3 cm underneath the clavicle was shown by chest radiography. Patients were excluded from the study if they had a left ventricular ejection fraction ≤40%, intracardiac shunting, valvular disease, active bleeding, or inotropic drugs and intra-aortic balloon pumping after cardiopulmonary bypass. Patients enrolled in the study

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had fluid therapy using blood transfusion or isotonic crystalloid solutions according to their baseline hemoglobin concentration. Fluids were administered at a fixed rate of 350 mL/20 minutes. Hemodynamic measurements were performed every 20 minutes to assess the response to fluid therapy. The endpoint for fluid administration was a cardiac index of 2.5 L/min/m^2. Fluid therapy was stopped if pulmonary artery occlusion pressure reached 18 mmHg.

Measurements were taken in enrolled patients at the time of admission to the CSU (T0) and repeated at the end of fluid therapy (T1). Blood samples of 2 mL were simultaneously obtained from the distal lumen of the pulmonary artery catheter and from the central venous catheter after withdrawal of deadspace blood and flushing fluid. All samples were withdrawn over 30 seconds by using a low-negative-pressure technique. The balloon of the pulmonary artery catheter was kept deflated during blood withdrawal. Samples were immediately analyzed for blood gases by using a standard blood gas analyzer, along with central venous and pulmonary hemoglobin oxygen saturation measured by oximetry (Gem Premier 3000; Instrumentation Laboratory, Lexington, MA). Concomitant hemodynamic parameters, arterial blood gases, hemoglobin concentration, and rectal temperature were also recorded. During the study period, all patients were mechanically ventilated, covered with a warming blanket, sedated with midazolam, 0.05 mg/kg/h, and paralyzed with vecuronium, 0.02 mg/kg/h. Arterial oxygenation, acid-base status, hematocrit, and rectal temperature were maintained within normal limits.

Based on a pilot study in the authors' department, a power analysis performed before the study indicated that a sample size of 60 patients was necessary ($\alpha = 0.05$, power $= 0.8$) to detect a difference of $\pm$ 2% between SvO_2 and $ScvO_2$, which was considered as the smallest relevant difference. Data at T0 and T1 were presented as mean $\pm$ standard deviation and were compared by the paired student t test. The correlation between SvO_2 and $ScvO_2$ was evaluated by linear regression analysis and Pearson test. The bias and the precision (standard deviation of the bias) for SvO_2 versus $ScvO_2$ were calculated. Bias was expressed as the mean difference between simultaneously measured SvO_2 and $ScvO_2$ individual values. The Student t test was used to determine whether the mean difference was significantly different from zero. According to Bland and Altman, limits of agreements were calculated as the mean difference $\pm$ 2 standard deviations. Limits of agreements between SvO_2 and $ScvO_2$ were considered to be clinically acceptable if they were within 5%. This difference influences neither patient evaluation nor therapeutic choices. The changes in SvO_2 and $ScvO_2$ values from T0 to T1 were recorded as ΔSvO_2 and $\Delta ScvO_2$. Bias, precision, and limits of agreement for ΔSvO_2 versus $\Delta ScvO_2$ were calculated. The correlation between ΔSvO_2 and $\Delta ScvO_2$ was evaluated by linear regression analysis and Pearson test. All statistics were performed by using the SPSS (version 13.0; SPSS, Chicago, IL) statistical package; $p \leq 0.05$ was considered to be statistically significant.

Table 1. Blood Gases, Hemodynamic Parameters, Rectal Temperature, and Hemoglobin

	T0	T1	*p* Value
Arterial oxygen saturation (%)	99.7 ± 0.7	98.7 ± 1.3	NS
$PaCO_2$ (mmHg)	37.1 ± 3.0	35.9 ± 2.1	NS
Bicarbonate concentration (mEq/L)	22.9 ± 2.3	23.9 ± 2.6	NS
Mean arterial pressure (mmHg)	89.8 ± 15.0	83.9 ± 12.0	NS
Cardiac index (L/min/m^2)	1.7 ± 0.2	3.0 ± 0.5	0.001
Pulmonary capillary wedge pressure (mmHg)	8.4 ± 3.3	9.4 ± 2.7	NS
Central venous pressure (mmHg)	5.4 ± 2.8	6.2 ± 2.9	NS
Systemic vascular resistance index (dynes · sec · cm^{-5}/m^2)	3,929 ± 1,307	1,934 ± 475	0.001
Rectal temperature (°C)	36.5 ± 0.5	36.9 ± 1.0	NS
Hemoglobin concentration (g/dL)	11.1 ± 1.6	10.3 ± 1.2	NS

NOTE. Data expressed in mean ± standard deviation.

Abbreviations: TO, before fluid therapy; T1, after fluid therapy; NS, not significant.

Table 2. SvO_2 Versus $ScvO_2$ at T0 and T1

	Bias	Correlation Coefficient (*r*)	Limits of Agreement
T0	−0.6% ± 9.3%*	0.463‡	−19.2% to 18%
T1	−0.8% ± 7.4%†	0.721§	−15.6% to 14%

Abbreviations: T0, before fluid therapy; T1, after fluid therapy.

*$p = 0.64$.

†$p = 0.40$.

‡$p = 0.001$.

§$p = 0.001$.

RESULTS

Sixty patients (53 males/7 females) presented with a low cardiac index and filling pressures on their admission to CSU and were included in this study. Acute physiology and chronic health evaluation II (APACHE) score ranged between 9 and 14. All patients responded to fluid therapy without increasing pulmonary artery occlusion pressure above 18 mmHg. Hemodynamic parameters, arterial blood gases, rectal temperature, and hemoglobin concentration values recorded at T0 and T1 are presented in Table 1. The cardiac index was significantly higher and systemic vascular resistance index was significantly lower at T1 compared with T0. Fluid therapy was achieved over a period of 60 ± 30 minutes, with 2 ± 2 blood units, and 1,000 ± 600 mL of isotonic crystalloid solutions per patient. Vasoactive and inotropic drugs were not used during the study period.

Sixty pairs of simultaneous SvO_2 and $ScvO_2$ measurements were recorded at T0 and at T1. The mean SvO_2 value was 66% ± 10% at T0 and 60% ± 10% at T1. The mean $ScvO_2$ value was 65% ± 8% at T0 and 61% ± 9% at T1. Bias, correlation coefficients, and limits of agreement between SvO_2 and $ScvO_2$ are presented in Table 2. The correlation between SvO_2 and $ScvO_2$ was low at T0, and limits of agreement were clinically unacceptable at T0 and T1. The correlation between SvO_2 and $ScvO_2$ is shown in Figure 1 (T0) and Figure 2 (T1). A plot of the difference between SvO_2 and $ScvO_2$ individual values against their means is presented in Figure 3 (T0) and Figure 4 (T1). Differences between SvO_2 and $ScvO_2$ remained significant over a wide range of mean individual values.

The correlation between ΔSvO_2 and $\Delta ScvO_2$, shown in Figure 5, was positive with an $r = 0.6$ ($p = 0.001$). Bias was −0.25 and limits of agreements for ΔSvO_2 versus $\Delta ScvO_2$ were from −20% to 19.5%. A plot of the difference between ΔSvO_2 and $\Delta ScvO_2$ individual values against their means is presented in Figure 6. Differences between ΔSvO_2 and $\Delta ScvO_2$ remained significant over a wide range of mean individual values.