

# Lack of Equivalence Between Central and Mixed Venous Oxygen Saturation\*

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**Study objective:** We compared paired samples of central venous O<sub>2</sub> saturation (ScvO<sub>2</sub>) and mixed venous O<sub>2</sub> saturation (Sv̄O<sub>2</sub>) to test the hypothesis that ScvO<sub>2</sub> is equivalent to Sv̄O<sub>2</sub>. We also compared O<sub>2</sub> consumption (V̇O<sub>2</sub>) computed with ScvO<sub>2</sub> (V̇O<sub>2cv</sub>) to that computed with Sv̄O<sub>2</sub> (V̇O<sub>2v</sub>).

**Design:** Prospective, sequential, observational study.

**Setting:** Combined medical-surgical ICU.

**Patients:** Fifty-three individuals > 18 years of age of either sex who required a pulmonary artery catheter (PAC) to guide fluid therapy. Subjects were identified as postsurgical (32 patients) or medical (21 patients) according to their ICU admission diagnosis.

**Interventions:** A PAC was inserted through the internal jugular or subclavian veins. Care was taken to place the PAC proximal port approximately 3 cm above the tricuspid valve. Blood samples were drawn from the proximal and distal ports in random order. An arterial blood sample also was drawn.

**Measurements:** Cardiac output in triplicate, systemic pressure, and central pressure. We analyzed blood samples for hemoglobin concentration and O<sub>2</sub> saturation (SO<sub>2</sub>). Data were compared by correlation analysis and by the method of Bland and Altman.

**Results:** Sv̄O<sub>2</sub> was consistently lower than ScvO<sub>2</sub> ( $p < 0.0001$ ), with a mean ( $\pm$ SD) bias of  $-5.2 \pm 5.1\%$ . Similar differences in ScvO<sub>2</sub> and Sv̄O<sub>2</sub> were present within each subgroup ( $p < 0.001$ ). A lower Sv̄O<sub>2</sub> resulted in V̇O<sub>2v</sub> values that were higher than the V̇O<sub>2cv</sub> values for all patients in the study (mean V̇O<sub>2v</sub>,  $236.7 \pm 103.4$  mL/min; mean V̇O<sub>2cv</sub>,  $191.1 \pm 84.0$  mL/min;  $p < 0.001$ ) as well as for patients within each subgroup ( $p < 0.001$ ).

**Conclusions:** Measurements of ScvO<sub>2</sub> and Sv̄O<sub>2</sub> were not equivalent in this sample of critically ill patients. Moreover, substituting ScvO<sub>2</sub> for Sv̄O<sub>2</sub> in the calculation of V̇O<sub>2</sub> produced unacceptably large errors. The decrease in SO<sub>2</sub> between ScvO<sub>2</sub> to Sv̄O<sub>2</sub> may result from the mixing of atrial and coronary sinus blood. As such, this difference may be a marker of myocardial O<sub>2</sub> consumption.

(CHEST 2004; 126:1891-1896)

**Key words:** central venous oxygenation; coronary sinus; mixed venous oxygenation; monitoring; myocardial metabolism; oxygen consumption; oxygen delivery; pulmonary artery catheter; resuscitation

**Abbreviations:** APACHE = acute physiology and chronic health evaluation; CI = cardiac index; CO = cardiac output; HC = hemoglobin concentration; PAC = pulmonary artery catheter; PAOP = pulmonary artery occlusion pressure; ScvO<sub>2</sub> = O<sub>2</sub> saturation of pulmonary central venous blood; SO<sub>2</sub> = O<sub>2</sub> saturation; Sv̄O<sub>2</sub> = mixed venous O<sub>2</sub> saturation; V̇O<sub>2</sub> = oxygen consumption; V̇O<sub>2cv</sub> = oxygen consumption calculated with O<sub>2</sub> saturation of pulmonary central venous blood; V̇O<sub>2v</sub> = O<sub>2</sub> consumption calculated with O<sub>2</sub> saturation of pulmonary artery blood

Mixed venous O<sub>2</sub> saturation (Sv̄O<sub>2</sub>) is a clinical marker of systemic oxygen utilization,<sup>1,2</sup> and its measurement is part of the routine monitoring of critically ill patients. The calculation of systemic O<sub>2</sub> consumption (V̇O<sub>2</sub>) and of pulmonary shunt fraction requires knowledge of Sv̄O<sub>2</sub>. Decreases in Sv̄O<sub>2</sub> have been associated with a poor prognosis in patients with septic shock<sup>3</sup> or heart failure,<sup>4</sup> and therapeutic interventions that are aimed at raising Sv̄O<sub>2</sub> have been tried during the resuscitation of critically ill patients.<sup>5,6</sup>

The measurement of Sv̄O<sub>2</sub> requires access to blood from the pulmonary artery, the drawing of which is a

highly invasive procedure. Sv̄O<sub>2</sub> may be measured from a sample of blood drawn from the distal port of a pulmonary artery catheter (PAC) or by using a PAC equipped with fiberoptic infrared sensors. Alternatively, the measurement of central venous blood O<sub>2</sub> saturation (ScvO<sub>2</sub>) offers an attractive option to the measurement of Sv̄O<sub>2</sub>, since it avoids some of the possible complications associated PACs.<sup>7</sup> Fiberoptic catheters are commercially available for the continuous measurement of ScvO<sub>2</sub>, and decreases in mortality have been reported when, guided by a goal-oriented algorithm, they have been used to resuscitate patients.<sup>8</sup>

Given the potential advantages of using  $ScvO_2$  as a surrogate for  $S\bar{v}O_2$ , it is important to establish the concordance between these measurements as well as the uncertainty associated with using  $ScvO_2$  as a measure of  $S\bar{v}O_2$ .

In this study, we compared the hemoglobin  $O_2$  saturation ( $SO_2$ ) of paired samples drawn from the proximal and distal ports of PACs in a population sample of critically ill adult patients. We took the proximal port blood sample as being representative of central venous blood. We tested the hypothesis that measurements of  $ScvO_2$  are equivalent to those of  $S\bar{v}O_2$  in critically ill patients, providing that the precision and bias of the  $S\bar{v}O_2$  estimate are within an acceptable clinical range.

## MATERIALS AND METHODS

This was a prospective, sequential, observational study of patients who had been admitted to the George Washington University Hospital ICU over a period of 6 months. The study was approved by the institutional review board, and informed consent for participating in the study was obtained from the patient or from their next of kin.

We enrolled individuals of either sex who were > 18 years of age whose attending physicians determined that a PAC was required to guide fluid therapy. Enrollment in the study occurred when the patient or the nearest relative consented to the introduction of the PAC. Patients who were excluded from the study were those with uncorrected valvular incompetence or intracardiac shunting, or those requiring the insertion of the PAC through the femoral vein. Depending on their diagnosis at the time of ICU admission, patients were identified as being post-surgical (*ie*, in the postoperative group) or medical (*ie*, in the medical group).

A 7.5F, five-lumen PAC that was 110 cm in length and had the right atrial lumen positioned 30 cm from the tip (Edwards Lifesciences; Irvine, CA) was inserted through the internal jugular vein or the subclavian vein using a percutaneous 8.5F sheath introducer (Edwards Lifesciences). On the first appearance of right ventricular pressure waves in the distal port, the catheter was withdrawn until the right ventricular waves disappeared. The catheter distance at the entrance of the sheath introducer was noted. The catheter then was advanced 27 cm

past this point, placing the distal port catheter in the pulmonary artery and the proximal port within the right atrium, approximately 3 to 4 cm above the tricuspid valve. A pressure tracing obtained from the proximal port was used to ascertain correct positioning in the right atrium. A portable chest radiograph and the presence of pulmonary artery pressure tracings confirmed the location of the distal port in the pulmonary artery.

Immediately after the insertion of the PAC, and prior to obtaining measures of pulmonary artery occlusion pressure (PAOP), each patient had one set of paired blood samples drawn in random order and in rapid succession from the proximal and distal port. The first 2 mL blood drawn for each sample was discarded to prevent contamination with flushing fluid. Blood samples were drawn with the catheter balloon deflated to avoid contamination of the distal port sample with pulmonary capillary blood.<sup>9</sup> We drew a blood sample from a previously inserted arterial line immediately after drawing the paired PAC blood samples. The three blood samples were placed on ice and were taken to a central laboratory for the measurement of hemoglobin concentration (HC) and  $SO_2$  (ABL700; Radiometer America Inc; Westlake, OH). We then measured PAOP and cardiac output (CO) by the thermodilution method. Cardiac index (CI) was computed by dividing the average of three sequential measurements of CO by the patient's body surface area.

We calculated systemic  $\dot{V}O_2$  by means of the Fick principle, neglecting the effect of dissolved  $O_2$  as follows:  $\dot{V}O_2 = 13.4 \times CO \times HC \times (SaO_2 - \text{venous } O_2 \text{ saturation})$ , where  $SaO_2$  is arterial oxygen saturation. Calculations of  $\dot{V}O_2$  were made by substituting either  $ScvO_2$  ( $\dot{V}O_{2cv}$ ) or  $S\bar{v}O_2$  ( $\dot{V}O_{2v}$ ) for the venous  $O_2$  saturation in the calculation.

### Data Analysis

Paired Student *t* test was used to compare  $ScvO_2$  to  $S\bar{v}O_2$ , and  $\dot{V}O_{2cv}$  to  $\dot{V}O_{2v}$ . Paired samples were compared by correlation analysis,<sup>10</sup> and also by the method of Bland and Altman.<sup>11</sup> Demographic and hemodynamic data were compared for the postoperative and medical groups using the two-tailed Student *t* test with levels of significance adjusted according to the method of Bonferroni for multiple comparisons. Unless otherwise specified, data are shown as the mean  $\pm$  SD, with  $p < 0.05$  deemed to denote a significant difference.

## RESULTS

We enrolled 53 patients in the study, of whom 21 were women. Demographics and diagnoses for individuals are shown in Table 1. Thirty-two patients in the study were in the postoperative group, and 21 were in the medical group. All patients in the medical group were in shock, as defined by the use of vasopressor agents to maintain mean arterial pressure. Sepsis was the predominant diagnosis in the medical group (62%). Coronary artery bypass grafting and aortic or mitral valve replacement represented the majority of the surgical procedures performed in patients in the postoperative group (84%). Only 25% of the postoperative group required vasopressor agents for the maintenance of BP. We found higher APACHE (acute physiology and chronic health evaluation) II scores, heart rates, mean pulmonary pressures, and CIs in the medical

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This study was financed in its entirety by The George Washington University Medical Center Department of Anesthesiology Research Fund.

Manuscript received January 14, 2004; revision accepted June 22, 2004.

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**Table 1—Demographic and Hemodynamic Parameters\***

Parameters	All Patients (n = 53)	Postoperative Group (n = 32)	Medical Group (n = 21)
Age, yr	59.0 ± 13.9	58.2 ± 12.5	60.2 ± 15.9
APACHE II score	14.0 ± 5.7	11.3 ± 3.3	18.2 ± 6.1†
Heart rate, beats/min	93.7 ± 18.0	88.0 ± 15.8	102.4 ± 17.9†
MAP, mm Hg	81.4 ± 12.4	83.3 ± 12.9	78.5 ± 11.2
MPP, mm Hg	28.1 ± 10.1	24.8 ± 9.2	33.1 ± 9.6†
PAOP, mm Hg	17.9 ± 6.7	17.4 ± 6.9	18.7 ± 6.5
CVP, mm Hg	14.9 ± 6.2	13.8 ± 5.4	16.5 ± 7.1
CO, L/min	6.1 ± 2.7	3.7 ± 0.7	7.2 ± 2.5‡
CI, L/min/m <sup>2</sup>	3.2 ± 1.4	2.7 ± 0.7	4.1 ± 1.7‡
SVRI, dynes·s·m <sup>-5</sup>	2014 ± 1254	2410 ± 1389	1410 ± 684†
Hemoglobin, g/dL	10.8 ± 1.9	11.7 ± 1.7	9.4 ± 1.3‡

\*Values given as mean ± SD. CVP = central venous pressure; MAP = mean arterial pressure; MPP = mean pulmonary pressure; SVRI = systemic vascular resistance index.

†p < 0.05 (medical group vs postoperative group).

‡p < 0.01 (medical group vs postoperative group).

group. Systemic vascular resistance and HC were lower in the medical group.

Shown in Table 2 are the  $SO_2$  values, and  $\dot{V}O_{2cv}$  and  $\dot{V}O_{2v}$  values. For the group as a whole, the values for  $S\bar{v}O_2$  were lower than those for  $ScvO_2$  ( $p < 0.001$ ), with a mean difference (*ie*, bias) of  $-5.2 \pm 5.1\%$ .  $ScvO_2$  was greater than  $S\bar{v}O_2$  ( $p < 0.001$ ) for both the medical and postoperative groups. For all patients, a lower  $S\bar{v}O_2$  resulted in a computed  $\dot{V}O_{2v}$  that was higher than the  $\dot{V}O_{2cv}$  ( $p < 0.001$ ). Both the postoperative and medical groups also had higher  $\dot{V}O_{2v}$  than  $\dot{V}O_{2cv}$  ( $p < 0.001$ ).

Figure 1, *top*, shows the linear correlation of paired  $ScvO_2$  and  $S\bar{v}O_2$  measurements for all patients in the study ( $S\bar{v}O_2 = -2.64 + 0.97 ScvO_2$ ;  $r = 0.88$ ;  $p < 0.0001$ ). Figure 1, *bottom*, shows the Bland-Altman analysis of the paired  $SO_2$  samples. As noted previously,  $S\bar{v}O_2$  was lower than  $ScvO_2$ , with a bias of  $-5.2\%$  (95% confidence interval for the mean,  $-3.8$  to  $-6.6\%$ ) and a 2-SD scatter ranging from  $-15.5$  to  $5.2\%$ . Figure 2, *top*, shows the correlation of  $\dot{V}O_{2cv}$  to  $\dot{V}O_{2v}$  for all patients in the study ( $\dot{V}O_{2v} = 25.5 + 1.1 \dot{V}O_{2cv}$ ;  $r = 0.89$ ;  $p < 0.0001$ ). A Bland-Altman analysis of these data (Fig 2, *bottom*) yields a bias toward a higher  $\dot{V}O_{2v}$  of 44.8 mL/min (95% confidence interval

for the mean, 31.9 to 57.8 mL/min) and a 2-SD scatter ranging from  $-48.5$  to 139.1 mL/min. Similar results were noted when analyzing the data from each subgroup (data not shown).

## DISCUSSION

In the present study, we took blood from the right atrium to be representative of central venous blood. We exercised great care during the insertion of the PAC to position the proximal port approximately 3 cm above the tricuspid valve. Presumably, this position placed the  $ScvO_2$  sampling site anterior to the coronary sinus but sufficiently distal into the right atrium to allow for the mixing of superior and inferior vena cava blood. The location of this sampling site for measuring  $ScvO_2$  differs from that of commercially available, oximetry-capable central venous catheters. In the latter, the infrared transducer lies within the superior vena cava.<sup>12</sup>

Judging from the robust linear relationship that is present between  $ScvO_2$  and  $S\bar{v}O_2$ , it would be reasonable to conclude that these measures are equiv-

**Table 2—Oxyhemoglobin Saturation and Calculated  $\dot{V}O_2$  Computed Using  $ScvO_2$  and  $S\bar{v}O_2$ \***

Variables	All Patients (n = 53)	Postoperative Group (n = 32)	Medical Group (n = 21)
$ScvO_2$ , %	73.9 ± 9.7	71.9 ± 8.4	77.0 ± 10.9
$S\bar{v}O_2$ , %	68.8 ± 10.6†	67.6 ± 9.6†	70.5 ± 12.1†
$\Delta\dot{V}O_2$ , %	5.2 ± 5.1	4.3 ± 4.9	6.5 ± 5.1
$\dot{V}O_{2cv}$ , mL/min	191.1 ± 84.0	202.9 ± 74.1	175.0 ± 96.6
$\dot{V}O_{2v}$ , mL/min	236.7 ± 103.4†	238.1 ± 94.7†	234.5 ± 117.9†
$\Delta\dot{V}O_2$ , mL/min	44.8 ± 47.1	35.2 ± 40.8	59.5 ± 53.1

\*Values given as mean ± SD.

†p < 0.001 (mixed venous vs central venous values by paired *t* test).

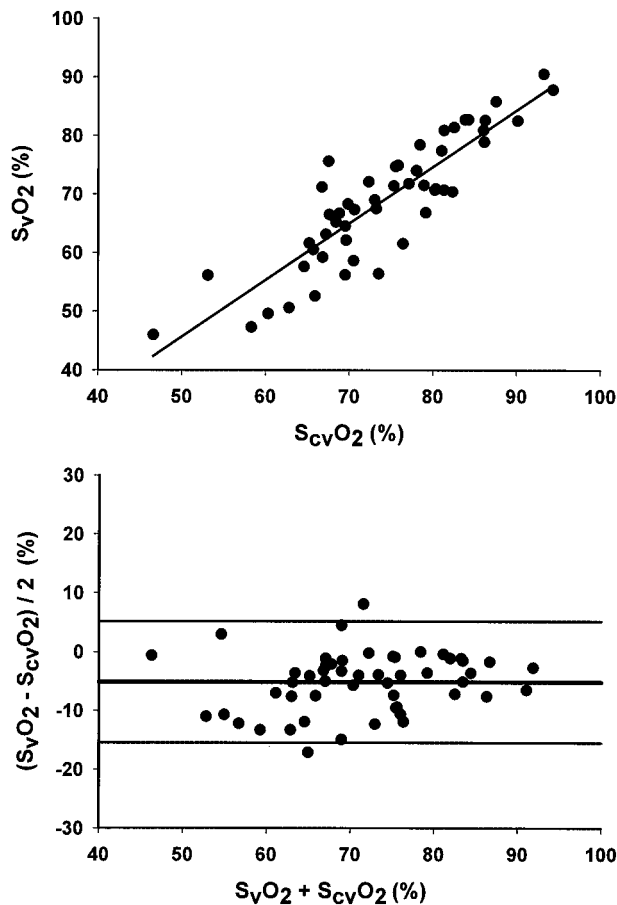


FIGURE 1. *Top*: paired  $\bar{SvO}_2$  and  $ScvO_2$  measurements (percentage of saturation) for all patients in the study. The linear correlation was as follows:  $\bar{SvO}_2 = -2.64 + 0.97 ScvO_2$ ;  $r = 0.88$ ;  $p < 0.0001$ ). *Bottom*: Bland-Altman analysis of the paired  $\bar{SvO}_2$  and  $ScvO_2$  measurements shows a bias toward a lower  $\bar{SvO}_2$  of  $-5.2\%$ , with 95% limits of agreement ranging from  $-15.5$  to  $5.2\%$ .

alent. The strength of this relationship is not surprising, given that  $ScvO_2$  and  $\bar{SvO}_2$  are physiologically tethered. Conversely, a paired-sample comparison of  $ScvO_2$  and  $\bar{SvO}_2$  shows a systematic bias of  $5.2\%$   $SO_2$ , with  $ScvO_2$  higher than  $\bar{SvO}_2$ . This bias is present throughout the span of measurement, implying a greater relative error for  $\bar{SvO}_2$  at lower  $ScvO_2$  values, which is precisely the  $\bar{SvO}_2$  range of greatest interest. Even more troublesome, is the unacceptably wide limit of agreement that exists between  $ScvO_2$  and  $\bar{SvO}_2$ . For example, a measurement of  $74\%$  for  $ScvO_2$  corresponds to an  $\bar{SvO}_2$  of  $68.8\%$ , with an uncertainty of the estimate ranging from  $58$  to  $79\%$ . This estimate is unlikely to be suitable for clinical use, in particular when applied to protocol-guided resuscitation in which decreases in  $\bar{SvO}_2$  of  $5$  to  $7\%$  may trigger therapeutic interventions, such as the use of inotropic agents or the transfusion of blood. We also noted that predictions of  $\dot{V}O_2$  based on  $ScvO_2$  are

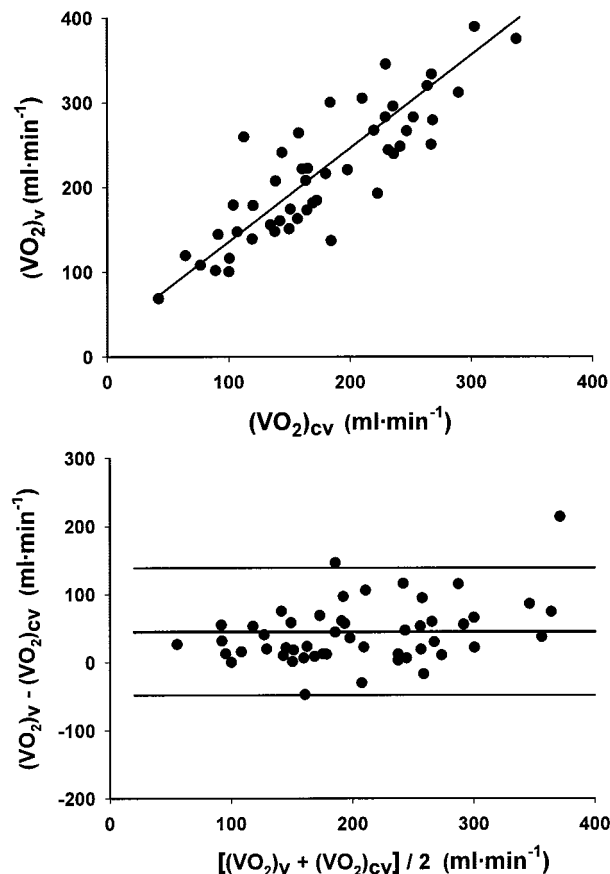


FIGURE 2. *Top*:  $\dot{V}O_{2v}$  shown as a function of  $\dot{V}O_{2cv}$  for all patients in the study. The linear correlation was as follows:  $\dot{V}O_{2v} = 25.5 + 1.1 \dot{V}O_{2cv}$ ;  $r = 0.89$ ;  $p < 0.0001$ ). *Bottom*: Bland-Altman analysis shows a bias toward a greater  $\dot{V}O_{2v}$  of  $44.8$  mL/min, with 95% limits of agreement ranging from  $-48.5$  to  $139.1$  mL/min.

biased toward a lower  $\dot{V}O_2$  and were associated with unacceptably wide limits of agreement.

Others have considered the issue of whether  $ScvO_2$  can be substituted for  $\bar{SvO}_2$ .<sup>13</sup> Experimental studies in animals show an excellent correlation between  $ScvO_2$  and  $\bar{SvO}_2$ . Reinhart et al<sup>14</sup> found a Spearman correlation coefficient of  $0.97$  in anesthetized dogs over a broad range of cardiorespiratory conditions, including hypoxia, hemorrhage, and resuscitation. Schou et al<sup>15</sup> also found a correlation coefficient of  $0.97$  between  $ScvO_2$  and  $\bar{SvO}_2$  in pigs that had been subjected to conditions of graded hypoxemia. Of note, both studies found  $\bar{SvO}_2$  to be consistently lower than  $ScvO_2$ .

The findings presented here agree with those of other studies<sup>16–23</sup> comparing measures of  $ScvO_2$  and  $\bar{SvO}_2$  in critically ill patients. Table 3 shows a compilation of prior clinical studies comparing  $ScvO_2$  to  $\bar{SvO}_2$ , along with their Spearman correlation coefficients and 95% confidence intervals. The weighted average for  $ScvO_2$  is greater than that for  $\bar{SvO}_2$  by

**Table 3—Comparison of Prior Studies Relating ScvO<sub>2</sub> to Sv̄O<sub>2</sub> in Critically Ill Patients\***

Study	No.	r Value	ScvO <sub>2</sub> , %	Sv̄O <sub>2</sub> , %	Method of Measurement	
					ScvO <sub>2</sub>	Sv̄O <sub>2</sub>
Berridge <sup>16</sup>	76	0.93	73.9 ± 1.7	70.8 ± 1.8	SVC catheter	PAC-distal
Edwards and Mayall <sup>17</sup>	30	0.56†	74.0 ± 12.5	71.3 ± 12.7	PAC-sequential	PAC-distal
Ladakis et al <sup>18</sup>	61	0.95	69.4 ± 1.1	68.6 ± 1.2	IVC catheter	PAC-distal
Lee et al <sup>19</sup>	19	0.73	58.2 ± 5.2	56.7 ± 4.9	SVC catheter	PAC-distal
Martin et al <sup>20</sup>	7	0.48	68.0 ± 9.0	67.0 ± 10.0	SVC catheter	PAC-distal
Pieri et al <sup>21</sup>	39	0.90	72.7 ± 8.6	71.8 ± 8.2	PAC-proximal	PAC-distal
Tahvanainen et al <sup>22</sup>	46	0.88	72.0 ± 11.0	70.8 ± 11.0	PAC-proximal	PAC-distal
Turnaoglu et al <sup>23</sup>	41	0.69	76.9 ± 7.2	70.5 ± 9.7	PAC-sequential	PAC-distal
Weighted mean‡			72.3 ± 5.8	68.9 ± 6.1		

\*Values given as mean ± SD, unless otherwise indicated. IVC = inferior vena cava; SVC = superior vena cava; PAC-sequential = samples obtained from the distal port during insertion of the PAC.

†Computed from the data shown in Figure 1 of Edwards and Mayall.<sup>17</sup>

‡ρ = 0.88 (95% confidence interval, 0.84 to 0.91).

2.4% SO<sub>2</sub>. The estimate for ρ,<sup>10</sup> the weighted average correlation for the general population, is 0.87. These figures compare favorably with the results of the present study.

A few studies<sup>19,23,24</sup> in healthy volunteers or in patients who were deemed not to be in shock found either no differences between ScvO<sub>2</sub> and Sv̄O<sub>2</sub> or even a small increase in SO<sub>2</sub> in pulmonary artery blood with weighted mean averages of 75.4 ± 5.8% and 76.2 ± 5.7%, respectively, for ScvO<sub>2</sub> and Sv̄O<sub>2</sub>. We did not detect this difference in behavior between the subgroups studied, even though their hemodynamic conditions appeared to be different. The medical group included a sicker group of patients with higher APACHE II scores than those in the postoperative group. The medical group also had higher CO and lower systemic vascular resistance, reflecting a high percentage of patients in septic shock. Most postoperative patients were not in shock and did not receive inotropic agents. Despite these differences, we noted similar findings for SO<sub>2</sub> and V̇O<sub>2</sub> in both populations, that is, both groups showed a 5% step-down from ScvO<sub>2</sub> measurements to Sv̄O<sub>2</sub> measurements.

A possible explanation for the decrease in SO<sub>2</sub> from ScvO<sub>2</sub> to Sv̄O<sub>2</sub> is the myocardial extraction of O<sub>2</sub> as blood flows through the right ventricle into the pulmonary artery. Although, to our knowledge, the rate of O<sub>2</sub> diffusion from ventricular blood into the myocardium has not been quantified, we consider this possibility unlikely.

A more likely hypothesis is that atrial blood, as it moves toward the pulmonary artery, mixes with blood of lower O<sub>2</sub> content. A key element with regard to the significance of this hypothesis is the position of the proximal port of the PAC. The possibility exists that blood drawn from the proximal port of the PAC originated mainly from the superior

vena cava. Should this have been the case, and should inferior vena cava effluent be composed of blood with a lower O<sub>2</sub> content, then the mixing of these effluents could have resulted in a lower Sv̄O<sub>2</sub>.

It is also possible, however, that decreases in ScvO<sub>2</sub> resulted from atrial blood mixing with blood emanating from the coronary sinus and Thebesian veins. Although coronary sinus flow may be but a fraction of total blood flow, the effluent from the coronary sinus has very low SO<sub>2</sub>, since the heart maximally extracts oxygen from the coronary blood flow. We are of the opinion that mixing with coronary sinus blood is the most likely explanation for the decrease in SO<sub>2</sub> from ScvO<sub>2</sub> to Sv̄O<sub>2</sub>.

We conclude that ScvO<sub>2</sub> is not a reliable surrogate for Sv̄O<sub>2</sub> in critically ill medical or surgical patients. Moreover, substituting ScvO<sub>2</sub> for Sv̄O<sub>2</sub> in the calculation of V̇O<sub>2</sub> is prone to unacceptably large errors. We cannot comment on the precision and repeatability of Sv̄O<sub>2</sub> estimates, since we did not measure sequential changes in Sv̄O<sub>2</sub> and ScvO<sub>2</sub>. It is possible that, although biased toward a larger estimate of Sv̄O<sub>2</sub>, the limits of agreement for repeated measures of ScvO<sub>2</sub> and Sv̄O<sub>2</sub> may be narrow enough to allow for continuous monitoring of ScvO<sub>2</sub> as a surrogate for Sv̄O<sub>2</sub> in individual patients.<sup>12</sup>

The step-down from ScvO<sub>2</sub> to Sv̄O<sub>2</sub> propounds the intriguing possibility that differences in blood SO<sub>2</sub> measured at the proximal and distal ports of the PAC may be related to measures of myocardial O<sub>2</sub> utilization. Further studies measuring coronary sinus blood O<sub>2</sub> content and flow are needed to test this hypothesis.

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