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Professor Barron has no potential conflicts of interest to disclose. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians ([www.chestjournal.org/misc/reprints.shtml](http://www.chestjournal.org/misc/reprints.shtml)).

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## Mixed vs Central Venous Oxygen Saturation May Be Not Numerically Equal, But Both Are Still Clinically Useful

Mixed venous oxygen saturation ( $S\bar{v}O_2$ ) in sepsis is commonly referred to as an *end point of low impact* on clinical decisions in sepsis patients because of the following common refrain: "S $\bar{v}O_2$  is always increased in septic ICU patients." However, there are fundamental principles that render this modality clinically useful when applying it to the supply-dependent phase of sepsis (*ie*, global tissue hypoxia). The presence of global tissue hypoxia not only has pathologic significance *in vitro*,<sup>1</sup> but there is a pathologic link among the clinical presence of global tissue hypoxia (*ie*, low  $S\bar{v}O_2$  and cardiac index), the generation of inflammatory mediators, and mitochondrial impairment of oxygen utilization that is seen in septic ICU patients.<sup>2</sup> Furthermore, identifying sudden episodes of supply dependency in septic ICU patients (*ie*, sudden decreases in  $S\bar{v}O_2$ ) has diagnostic and prognostic significance.<sup>3</sup> With this background, the rationale for using central venous oxygen (ScvO<sub>2</sub>) saturation as a surrogate for  $S\bar{v}O_2$  to

detect and treat global tissue hypoxia in the most proximal phase of sepsis management (supply dependency) was the basis for its use in the Early Goal Directed Therapy in Severe Sepsis and Septic Shock Study (EGDT).<sup>4</sup>

Early hemodynamic assessment using physical examination, vital signs,<sup>5</sup> central venous pressure,<sup>6</sup> and urinary output<sup>7</sup> fails to detect supply dependency or persistent global tissue hypoxia. Shock patients who are resuscitated to having normal vital signs continue to exhibit evidence of global tissue hypoxia (ScvO<sub>2</sub> < 70% and increased lactate levels) and require additional resuscitation, as shown by Rady et al.<sup>6</sup> Similar findings were confirmed in the EGDT study as 39.8% of the control group vs 5% of the EGDT group continued to have global tissue hypoxia after 6 h of resuscitation despite the fact that all patients attained the same vital sign goals (*ie*, MAP, > 65 mm Hg; CVP, > 8 mm Hg; urine output, 0.5 mL/kg/h). These findings of global tissue hypoxia, or "cryptic shock," in patients<sup>8</sup> have prognostic significance as this state was associated with a 56.5% in-hospital mortality rate. The therapeutic significance was realized as the EGDT patients received early and more aggressive therapy with fluids, RBC transfusion, and inotropic agents.

The question of whether the ScvO<sub>2</sub> is a numeric equivalent to  $S\bar{v}O_2$  has been examined in a number of studies,<sup>9-12</sup> which continues to fuel this debate. These studies, including the trial by Chawla et al,<sup>11</sup> have consistently shown that ScvO<sub>2</sub> values are (on average) approximately 5% higher than  $S\bar{v}O_2$  values, which is likely secondary to the contributions of deoxygenated blood from the coronary sinus. Recognizing this minor, yet consistent, difference allows the clinician to make an accurate assessment of global tissue hypoxia. Furthermore, the clinical utility of an end point of resuscitation is determined by whether it changes clinical practice, morbidity, and mortality in a cohort of patients under the rigors of an appropriately designed clinical trial. In other words, has this end point been calibrated to have clinical utility in the setting in which it is to be used? This was done with ScvO<sub>2</sub> in the EGDT study,<sup>4</sup> in which the range of ScvO<sub>2</sub> values was 48.6 to 49.2%, with lactate levels of 6.9 to 7.7 mmol/L indicating significant supply dependency. Using the finding from Chawla et al,<sup>11</sup> the  $S\bar{v}O_2$  values would be extrapolated to 43 to 45%. Thus, irrespective of whether the ScvO<sub>2</sub> value equals the  $S\bar{v}O_2$  value, the presence of a low ScvO<sub>2</sub> level in patients with early sepsis portends increased morbidity and mortality, and correcting this value according to a consensus-derived algorithm<sup>13</sup> improves morbidity and mortality. It should be further noted that, in this well-designed study by Chawla et al,<sup>11</sup> the majority of the

51 patients were not supply-dependent as the mean  $\text{S}\bar{\text{v}}\text{O}_2$  values ranged from 67.6 to 70.5, and the corresponding  $\text{ScvO}_2$  values ranged from 71.9 to 77.0 with no mention of lactate levels.

Examining studies comparing the numeric equivalency of  $\text{S}\bar{\text{v}}\text{O}_2$  vs  $\text{ScvO}_2$ , while of important academic value, does not address clinical utility. The concept of the approximately 5% numeric difference between  $\text{S}\bar{\text{v}}\text{O}_2$  and  $\text{ScvO}_2$  values is not novel, and the Surviving Sepsis Campaign has acknowledged<sup>14</sup> this by recommending obtaining an  $\text{S}\bar{\text{v}}\text{O}_2$  level of 65% and/or an  $\text{ScvO}_2$  level of 70% in the resuscitation portion of its management of patients with severe sepsis and septic shock bundle.

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Dr. Rivers has done consultant work for Biosite, Inc., Chiron, the Lilly Corporation, and Edwards Lifesciences over the past year. As a consultant, he helped advise the company in developing, but holds no patent rights to, catheters used for early, goal-directed therapy.

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*Chest* 2006;129; 507-508

DOI 10.1378/chest.129.3.507

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