

critical illness and comorbid conditions. Without these changes in place, we will remain in the dark ages with respect to universal guideline application. Failure to create these systems will lead to a plethora of unobtainable guidelines that do not benefit our patients. We strongly urge clinicians and information specialists to work toward this essential and important goal to improve patient care and bring evidence-based guidelines to the bedside.

Gerard J. Fulda, MD, FCCM
Surgical Critical Care and Surgical Research
Christiana Care Health System
Wilmington, DE; and
Department of Surgery
Jefferson Medical College
Philadelphia, PA
Antoinette Spevetz, MD, FCCM
Department of Internal
Medicine
Section of Critical Care
Medicine

Cooper University Hospital
Cooper Medical School of
Rowan University
Camden, NJ

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Is it not self-evident that all venous saturations are not created equal?*

The femoral approach to the venous system enjoys widespread use. Having established that central venous pressure measurements obtained from that site generally correlate well with those obtained in the upper body at the end of the last century, we have increasingly relied on this route to not only to monitor fluid status but also to administer vasoactive or other potent medications and importantly to draw blood samples (1).

More recently, central venous catheters (CVC) have come under increasing scrutiny (2). The femoral route is associated with higher rates of infections, and

guidelines advocate avoiding this route and encouraging instead the use of upper body CVC (3, 4). However, the femoral approach remains extremely useful in incompletely resuscitated patients, especially in those in which either the head down position or an accidental pneumothorax would cause immediate potentially lethal patient compromise. The increased risk of subsequent femoral catheter infection, subsequently managed by early replacement, must be weighed against the need for urgent, low-risk CVC access.

Measurement of mixed venous oxygen saturations (SvO₂) from the pulmonary artery reflects the balance between systemic oxygen delivery and demand. Central venous oxygen saturation (ScvO₂) has been used in clinical practice for over 45 yrs as an approximation for SvO₂ (5). In the last decade, the influential Rivers et al (6) article with its description of multifaceted intervention in response to low ScvO₂ during resuscitation of patients

with severe sepsis and septic shock has increased ScvO₂ use in intensive care. Although the overall efficacy and generalized application of such monitoring and interventions remains in dispute, there is no doubt that the presence of low venous saturation is an important prognostic indicator of these patients (7, 8). Sampling from a CVC removed the need for the more complex pulmonary artery flotation catheters, making the “Rivers” protocol and its variants attractive. However, even when sepsis protocols are implemented, pragmatic rationalization and other distractors have resulted in as few as one patient in six having their ScvO₂ measured (9).

Given the difficulties in maintaining compliance with published guidelines, it is likely that many clinicians have unwittingly acted on oxygen saturation results from samples drawn from femoral sites (SfO₂). The study by van Beest et al (10) in this issue *Critical Care Medicine* clearly

*See also p. 3196.

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demonstrates that the inconsistent differences between SfO_2 and ScvO_2 are such that they cannot be substituted, one for the other.

Although the association at a point in time were inconsistent generally, compared to ScvO_2 , SfO_2 tended to be lower in intensive care patients, and higher in postsurgical patients.

SvO_2 reflects the balance between oxygen delivery and demand. Previous studies by the group of van Beest et al (11) have also demonstrated that there is an unacceptably wide range in variance between ScvO_2 in the superior vena cava and SvO_2 . When the physiology is examined, it should come as no surprise that measurements of ScvO_2 , SfO_2 , and SvO_2 were not equivalent in critically ill patients.

The very desaturated coronary blood flow, which at rest comprises 5% of the cardiac output, flowing into the right atrium accounts for a lower SvO_2 than ScvO_2 . Clinical studies measured ScvO_2 in the SVC, but over half the venous return arrives through the inferior vena cava. This larger blood flow means that the oxygen saturations in inferior vena cava blood (Sivc O_2) will have more significant impact on SvO_2 than the similar change in the upper body. Where low global arterial oxygenation, low oxygen carrying capacity or global poor cardiac output exists, and changes in saturation in the upper and lower body are likely to mirror each other. However, when more selective organ oxygen supply-demand balance is affected, upper and lower body venous saturation will differ depending upon the where effected venous blood drainage, and any change seen in the will be attenuated by the addition of the other "normal" blood SvO_2 . Sivc O_2 is normally higher than that of ScvO_2 and SvO_2 , largely because of the major contribution of highly saturated renal venous blood (12)

The positioning of the catheter further complicates Sivc O_2 measurements. Depending upon the length of catheter, entry point, and the patient's size, a longer CVC may include or exclude the renal admixture, further confusing

any potential interpretation of the Sivc O_2 . A previous study using longer, more centrally placed, femoral-sited catheters measuring Sivc O_2 , also found that these did not reflect the ScvO_2 or the SvO_2 (13). The presenting disease, location, and vasopressor dose may all also influence the degree and direction of the difference.

In the van Beest et al (10) study, samples were from the femoral vein, well below where the renal blood admixture occurs. Despite this the results did not reliably reflect either the ScvO_2 or the SvO_2 .

Viewed objectively, it is hardly surprising that the study by van Beest et al demonstrates that we cannot interchange the two sampling sites—but it would easier if we could. The van Beest et al study provides us with high-grade evidence that we must not be tempted to do so. The detection of venous saturation lower than normal for a specific site indicates a supply-demand imbalance that may cause harm; although monitoring multiple sites is attractive, it may be impractical. Unfortunately, van Beest et al study was not sufficiently powered to demonstrate which of the two sample sites best predicts survival, or more importantly what benefit, if any, accrues from manipulation of the various saturations.

The van Beest et al study, while perhaps demonstrating the self-evident, has two major messages. First, using a substitute for ScvO_2 in for any "Riversque like" protocol may lead to inappropriate treatment being given or appropriate treatment withheld. Second, our understanding of the value of the various venous saturations and our knowledge of how to manipulate these somewhat irksome parameters to improve patient outcomes are still far from complete.

Ross Freebairn, MBChB, FCICM
Hawkes Bay Hospital
Hastings, New Zealand

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