INFLUENCE OF CARDIAC OUTPUT ON THE CORRELATION BETWEEN MIXED VENOUS AND CENTRAL VENOUS OXYGEN SATURATION

Sir,—Because there was close correlation between mixed venous (\(SvO_2\)) and central venous (\(ScvO_2\)) oxygen saturations in his study of largely elective cardiac surgical patients, Dr Berridge [1] asserted that \(ScvO_2\) may be used to estimate derived oxygen transport variables in emergency situations. This is in disagreement with previous studies [2] and, we feel, merits further comment.

Statistically, close correlation between two measurements implies that there is a mathematical relationship between them, not that they are interchangeable. For example, if there had been a constant difference of, say, 20% between \(SvO_2\) and \(ScvO_2\), in all patients, the correlation coefficient would have been 1.0. Clearly, however, the two measurements could not be said to be interchangeable. A more useful test to determine whether two measurements are interchangeable is the method described by Bland and Altman [3]. This examines the differences between measurements made in an individual patient and may be applied to the data in Dr Berridge’s paper. The 95% confidence intervals for the mean (\(\pm \pm 2 \text{ SEM}\)) of the difference between central and mixed venous oxygen saturation for the entire study group were (\(\text{ScvO}_2 - \text{SvO}_2\) = 2.45 ± 3.75%).

To calculate the potential variation in the difference between central and mixed venous oxygen saturation for an individual patient in the study group, we need to calculate \(\text{SD}\) of the 95% confidence intervals for each individual patient in the study group (mean \(\pm 2 \text{ SD}\)). For all patients the 95% confidence intervals for the difference between pulmonary and central venous oxygen saturation are therefore (\(\text{ScvO}_2 - \text{SvO}_2\) = 2.57 to 8.77%).

Using the above range of measurements for \(\text{ScvO}_2\), the variation in derived oxygen transport variables may be demonstrated by calculating oxygen consumption (\(\text{Vo}_2\)). The range of \(\text{Vo}_2\) values may then be compared against \(\text{Vo}_2\) calculated using mean \(\text{ScvO}_2\) (0.87%). From the study, mean cardiac index (CI) = 3.3 litre min\(^{-1}\) m\(^{-2}\). If we assume an arterial haemoglobin concentration of 11.5 g dl\(^{-1}\), Hufner factor = oxygen dissociated in plasma (< 2% total oxygen content) then we can calculate, using \(\text{ScvO}_2\), that \(\text{Vo}_2\) = 133.0 ml min\(^{-1}\) m\(^{-2}\). Using the range of \(\text{ScvO}_2\) values we can calculate that, for 95% of patients, \(\text{Vo}_2\) may vary between 88.4 and 147.2 ml min\(^{-1}\) m\(^{-2}\). We consider this to be a significant potential error in estimation of \(\text{Vo}_2\).

We have previously presented data from our unit comparing the differences between pulmonary artery (mixed venous), right atrial and superior vena cava (central venous) oxygen saturations in critically ill patients (mean APACHE II score = 24.6), and showed large variability [4]. Oxygen saturation was measured with an IL 282 Co-oximeter. Blood from the superior vena cava (\(\text{SV}_{\text{O}_2}\)) is the most likely sampling point when using jugular or subclavian vein catheters, showed the widest variation for the study population using either mixed venous oxygen saturation or central venous oxygen saturation and the actual haemoglobin concentrations of the patients. The results are as follows: mean \(\text{Vo}_2\) using \(\text{SV}_{\text{O}_2}\) = 126 ml min\(^{-1}\) m\(^{-2}\) (95% confidence intervals 116.6-136.1 ml min\(^{-1}\) m\(^{-2}\)) and mean \(\text{Vo}_2\) using \(\text{SV}_{\text{O}_2}\) = 113 ml min\(^{-1}\) m\(^{-2}\) (95% confidence intervals 103.8-121.5 ml min\(^{-1}\) m\(^{-2}\)). The Bland–Altman plot of the two methods of estimating \(\text{Vo}_2\) is shown in figure 1. Mean bias is 13.7 ml min\(^{-1}\) m\(^{-2}\) and limits of agreement are –11.4 to 38.8 ml min\(^{-1}\) m\(^{-2}\). The usefulness or otherwise of such data is for the individual to judge. I cannot explain the difference between my findings and other authors, except that there was a deliberate attempt to position the central venous catheter optimally, clinically, and that a large proportion of the patients I studied were undergoing cardiac surgery and did not have sepsis.

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PIPECURONIUM-INDUCED PROLONGATION OF VECURONIUM BLOCK

Sir,—Drs Smith and White [1] explained the prolongation of vecuronium block after partial recovery from pipecuronium on the basis of residual receptor occupancy by the pipecuronium. They attributed this to a presumed plasma concentration of pipecuronium about 50 min after administration of 10 µg kg\(^{-1}\). In our study [2], we demonstrated prolongation of vecuronium block after 50% recovery from pancuronium and a decrease in duration of pancuronium block after 50% recovery from vecuronium. In these experiments, the influence of the plasma concentration of drug and therefore of any pharmacokinetic influence was obviated.

**References**


**Fig. 1.** Bland–Altman plot of two methods of estimating \(\text{Vo}_2\): using \(\text{SV}_{\text{O}_2}\) or \(\text{SV}_{\text{O}_2}\).

**CORRESPONDENCE**


