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

Sir,—Because there was close correlation between mixed venous (SvO₂) and central venous (ScvO₂) oxygen saturations in his study of largely elective cardiac surgical patients, Dr Berridge [1] asserted that ScvO₂ 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 SvO₂ and ScvO₂ 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 (±2 SEM) of the difference between central and mixed venous oxygen saturation for the entire study group were (ScvO₂ — SvO₂) = 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 the 2 SD of the entire study group and subtract from it. The SD of the entire study group is calculated from the formula: SEM = SD × √n/sample size. The 95 % confidence intervals for an individual patient in the study group (mean ± 2 SD) are therefore (ScvO₂ — SvO₂) = 2.57 to 8.77 %.

Using the above range of measurements for ScvO₂, the variation in derived oxygen transport variables may be demonstrated by calculating oxygen consumption (Vo₂). The range of Vo₂ values may then be compared against Vo₂ calculated using mean ScvO₂ (= 70.8 %). From the study, mean cardiac index (CI) = 3.3 litre min⁻¹ m⁻². If we assume an arterial oxygen saturation (SaO₂) of 97 %, haemoglobin (Hb) = 11.5 g dl⁻¹, Hufner factor = oxygen dissolved in plasma (< 2 %, total oxygen content) then we can calculate, using ScvO₂ that Vo₂ = 133.0 ml min⁻¹ m⁻². Using the range of ScvO₂ values, we can calculate that, for 95 % of patients, Vo₂ may vary between 88.4 and 174.2 ml min⁻¹ m⁻². We consider this to be a significant potential error in estimation of Vo₂.

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 (ScvO₂), which is the most likely sampling point when using jugular or subclavian vein catheters, showed the widest variation from pulmonary artery saturation (SpA₀₂). The mean difference was (3ScvO₂ — ScvO₂) = 7.93 % with a 95 % confidence interval for an individual patient of —18.4 to 24.2 %. Using these measurements we can calculate Vo₂. The patients reported had mean CI = 4.5 litre min⁻¹ m⁻², mean Hb = 11.5 g dl⁻¹ and mean SaO₂ = 97 %. For comparison with the above, we calculated Vo₂ ignoring the small contribution from dissolved oxygen and used oxygen 1.34 ml g⁻¹ Hb as the Hufner factor. If blood is taken from the pulmonary artery, mean Vo₂ = 171.4 ml min⁻¹ m⁻², while, for blood from the superior vena cava, Vo₂ may vary for 95 % of patients between 3.6 and 299.3 ml min⁻¹ m⁻².

We conclude that Dr Berridge has shown, in a heterogeneous group of patients, that there are significant differences between central and mixed venous oxygen saturation and has confirmed that blood from central veins cannot be used as a mixed venous sample for calculation of derived oxygen transport variables.

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Sir,—I suggested, but did not "assert" that, because of a close correlation between central venous and mixed venous oxygen saturations, central venous samples were a useful estimate of mixed venous oxygen saturations. I have already supplied the relevant Bland—Altman data in reply to previous correspondence [1].

Regarding the oxygen transport variables, I have calculated the oxygen consumption index (Vo₂/I) 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 Vo₂/I using ScvO₂ = 126 ml min⁻¹ m⁻² (95 % confidence intervals 116.6—136.1 ml min⁻¹ m⁻²); mean Vo₂/I using ScvO₂ = mean 113 ml min⁻¹ m⁻² (95 % confidence intervals 103.3—125.3 ml min⁻¹ m⁻²). The Bland—Altman plot of the two methods of estimating Vo₂/I 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 pipercuronium by the basis of residual receptor occupancy by the pipercuronium. They attributed it to a presumed plasma concentration of pipercuronium about 50 min after administration of 10 µg kg⁻¹. In our study, we demonstrated prolongation of vecuronium block after 50 % recovery from pipercuronium and a decrease in duration of pipercuronium 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.
by performing the two experiments simultaneously using the two isolated forearms of volunteers [2]. Thus at the time of the second administration of the drugs the background minimal plasma concentration was the same in both arms.

A further argument against this phenomenon being caused by acetylcholine receptor occupancy is our observation that, at the point of 100% recovery of TI and presumably about 80% residual receptor occupancy, no effect of the conditioning drug upon the second drug was observed. As a result of this and other observations, we concluded that the effect described was the result of biophase binding in the effect compartment and was not directly dependent upon pharmacokinetic events [3].

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2. Feldman SA, Faivel NJ, Hood JR. Recovery from pancuronium and vecuronium administered simultaneously in the isolated forearm and the effect on recovery following administration after cross-over of drugs. Anesthesia and Analgesia 1993; 76: 92-95.

Sir,—We thank Professor Feldman and Dr Hood for drawing our attention to their recent work, which had not been published when our manuscript was submitted to British Journal of Anaesthesia. They have confirmed our clinical finding [1] that neuromuscular blockers administered during partial block have their duration of action modified by the residual drug [2]. We agree that this interaction is not a result of residual plasma concentrations, and did not intend to suggest such a mechanism in our article.

We refered to the ACh receptor in rather loose terms, but concede that the authors are more correct to suggest that this phenomenon takes place in the “biophase”. However, there is no proof of the exact anatomical location at which the biophase interaction occurs.

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2. Feldman SA, Faivel NJ, Hood JR. Recovery from pancuronium and vecuronium administered simultaneously in the isolated forearm and the effect on recovery following administration after cross-over of drugs. Anesthesia and Analgesia 1993; 76: 92-95.

EFFECT OF NITROUS OXIDE ON CEREBRAL BLOOD FLOW

Sir,—We were interested to read the paper by Field and colleagues [1] on the effect of nitrous oxide on cerebral blood flow (CBF) in humans. They interpreted the data to suggest a cerebral concentration of nitrous oxide, they also breathed a smaller concentration of oxygen. This can be avoided only by introducing an inert third gas.

Moreover, we argue that it would be more relevant also to clinical practice to maintain the inhaled oxygen concentration constant: the practical choice is really between (say) 30% oxygen and 70% of either nitrous oxide or nitrogen.

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Sir,—Drs Dirnhuber and Davies have suggested that our data may be interpreted as confirmation of the well-established cerebral vasoconstrictor properties of oxygen.

We thank them for the opportunity to challenge the view that inhalation of 100% oxygen typically reduces cerebral blood flow (CBF) by 10-15%. The studies by Kety and Schmidt [1] and Lambertson and colleagues [2] on which this assumption is based were flawed in that the volunteers taking part hyperventilated whilst inhaling 100% oxygen. A small decrease in mean arterial carbon dioxide tension of 0.13kPa in the former study and a greater change of 0.26 kPa in the latter, whilst not statistically significant, could account for 4% and 8% reductions in CBF, respectively [3]. Lambertson and colleagues pointed out that a 267-kPa increase in arterial oxygen tension together with a 0.67 kPa reduction in arterial carbon dioxide tension do not result in a reduction in CBF greater than that produced by a 0.67-kPa reduction in carbon dioxide tension alone [1]. Furthermore, he concluded that “a physiologically important specific vasoconstrictor action of oxygen probably does not exist in the intact human brain” and that reduced CBF during oxygen inhalation may be explained by a primary hyperventilation and reduction in arterial carbon dioxide tension.

Our volunteers did not hyperventilate as confirmed by a stable end-tidal carbon dioxide concentration throughout the study. We believe that further information is required on the influence of moderate hyperoxia (for instance that produced by inhalation of 40-100% oxygen) on cerebral haemodynamics before the findings of Kety and Lambertson can be validated.

Our purpose was to mimic as closely as possible the clinical situation in which nitrous oxide is administered in varying concentrations with oxygen. The introduction of a third gas in the equation does not conform with current anaesthetic practice. We do not wish to criticize the use of nitrous oxide in neuroanaesthesia, merely to shed some light on the conflicting evidence surrounding its effects on cerebral haemodynamics: we believe, for the reasons explained above, that our data reflect the effect of nitrous oxide on CBF.

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