

Correspondence

Falsely High Blood Oxygen Content by the Van Slyke-Neill Method

To the Editor:—Fahmy and Laver (ANESTHESIOLOGY 44:6–15, 1976) used the unmodified Van Slyke-Neill method for determining blood O₂ contents of samples containing unspecified amounts of N₂O and halothane. Is this approach acceptable in view of Goldstein's (J Biol Chem 182:815, 1950) and my (ANESTHESIOLOGY 30:325, 1969) demonstration that the use of this method for samples containing ethyl ether and halothane, respectively, results in falsely high values for blood O₂ content? Perhaps the authors assumed that the arterial and mixed venous samples contained identical amounts of N₂O and halothane, which would result in identical false increments in individual O₂ content determinations but an accurate O₂ content difference.

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To the Editor:—The objections raised by Dr. Theye have definite theoretical validity. His study for halothane¹ and that of Goldstein for diethyl ether² have attempted to validate these objections, but in a manner that we cannot consider entirely satisfactory. First, a comment on our data as published.³ We did compare O₂ contents measured by the Van Slyke-Neill method⁴ (without re-extraction) and the fuel-cell technique (Lex-O₂-Con*) in adult human whole blood tonometered with different concentrations of oxygen with or without 1 per cent halothane or a 50 per cent N₂O–50 per cent O₂ mixture. Neither the manometric nor the fuel-cell technique demonstrated a significant O₂ content difference for whole blood tonometered with approximately 7 per cent O₂–5 per cent CO₂ with and without 1 per cent halothane (table 1). Dr. Theye indicated in his orig-

TABLE 1. O₂ Content (ml/100 ml) (Mean ± SD)

	n	Manometric	Fuel Cell
Control	6	11.66 ± 0.4	11.57 ± 0.3
Halothane, 1 per cent	6	11.86 ± 1.2	11.82 ± 1.1

inal paper¹ that the mean difference for "Van Slyke" minus "Goldstein" was 0.4 ml/100 ml, without stating the actual O₂ content. Such presentation is incomplete. For example, if the arterial oxygen content is 15 ml/100 ml, then an error of 0.4 ml/100 ml is of the order of 3 or 4 per cent when mixed venous content is 10 ml/100 ml. We are surprised that Dr. Theye has not commented on his earlier paper³ in which equilibration with 2 per cent halothane increased the mean measured O₂ content from 18.03 to 18.15 ml/100 ml, or by less than 1 per cent. Considering the vagaries of clinical experimentation, these differences may be statistically significant but hardly relevant physiologically. On the other hand, the error will be enhanced, not reduced, for AV_{O₂} content differences if we assume that the halothane concentration is constant but the venous O₂ content is less.

Second, our comparison of contents of whole blood tonometered with 50 per cent N₂O–50 per cent O₂ indicated that N₂O, because of its low solubility in water, remains in the gas phase of the Van Slyke apparatus and will give falsely high O₂ contents compared with the fuel-cell method. The discrepancy is equally high for arterial and mixed venous blood and, as Dr. Theye has suggested, the AV_{O₂} differences are not affected. When calculations are based on a N₂O solubility coefficient (α) of 0.412 in human blood⁶ at one-half atmosphere (P_{N_2O} = 342 mm Hg), the N₂O content will be 342(0.412) · 100/760 = 19 ml/100 ml. Since the discrepancy between our manometric and fuel-cell measurements for O₂ was of the order of 1 to 1.5 ml/100 ml, we must as-

* Lexington Instruments.