The report by Malinow *et al.*[9] that 31 of 66 women developed ultrasonic precordial Doppler changed during cesarean sections under regional anesthesia, and that 14 of the 31 Doppler positive patients developed chest pain and dyspnea within 1–6 min after Doppler sound changes, indicates the need to develop more prospective studies to give us a better idea as to the incidence of VAE not only during and after cesarean section, indicating the influence of methods of anesthesia, but also during and after vaginal deliveries. In the most recent description of maternal deaths in the USA,[10] 25 cases (about 1%) were found to be due to VAE, although there were no details given of the circumstances of these deaths. This is almost the same number of deaths seen from aspiration of gastric contents associated with anesthesia, and, since much effort is expended in attempts to reduce this mortality, we feel that the same level of care is indicated to prevent, diagnose, and treat VAE.

We totally agree with Dr. Rupp that the precordial Doppler should be used routinely in all cesarean sections, since it is non-invasive, sensitive, reliable, and gives an early warning of a potentially dangerous phenomenon.

DAVID A. ROBINSON, F.F.A.R.C.S.
Visiting Assistant Professor of Obstetric Anesthesiology

LEONID BUNEGIN, B.S.
Research Assistant Professor

MAURICE S. ALBIN, M.D., M.Sc. (ANES.)
Professor of Anesthesiology and Neurosurgery

Department of Anesthesiology
The University of Texas
Health Science Center
San Antonio, Texas 78284-7838

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More on Dyes and Pulse Oximeters

To the Editor:—A recent publication by Kessler *et al.* entitled "Spurious Pulse Oximeter Desaturation with Methylene Blue Injection," may contain "spurious" conclusions.[1] We studied the effects of three commonly used intravenous dyes on the Nellcor N-100 pulse oximeter readings.[2] Our results also demonstrated that methylene blue had a striking but transient effect on SaO_2 readings. However, we take issue with the mechanisms proposed by Kessler *et al.* to explain the transient nature of this phenomenon. Kessler *et al.* state that the effect was transient because of the "dilution of the dye and its rapid renal clearance."[1] It is known tht methylene blue is not rapidly cleared (when administered in a dose similar to that used by Kessler *et al.*), and will falsely depress *in vitro* oximetry reading for up to 48 h.[3,4]* *In vitro* oximetry uses a similar technology to calculate hemoglobin saturation, as do pulse oximeters.

We suggest that the transient effect of methylene blue (or any intravenous dye) is due to the manner in which the pulse oximeter calculates arterial (Pulsatile) saturation. The pulse oximeter has an algorithm which calculates arterial hemoglobin saturation by the subtraction of the light absorption of the non-pulsatile (veno-capillary) blood from the light absorption of the pulsatile (arterial) blood.[5] When the concentration of methylene blue (or any dye for that matter) is equal in these two compartments, there will be no effect on the pulse oximetry reading. This is how the pulse oximeter

compensates for the many intra-patient differences, such as skin pigmentation, hemoglobin concentration, and fingernail polish. Only conditions which preferentially affect only one side of the algorith (*e.g.*, the light absorption of the pulsatile or non-pulsatile compartments) will affect pulse oximetry readings.

* IL282 CO-Oximeter Manual, p. 1.5, 1981.

RICHARD UNGER, M.D.
Assistant Clinical Professor of Anesthesiology

MARK S. SCHELLER, M.D.
Assistant Professor of Anesthesiology

Department of Anesthesiology
UCSD Medical Center
225 Dickinson Street
San Diego, California 92103

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In Reply:—Unger and Scheller suggest that the spurious pulse oximetry desaturation associated with intravenous methylene blue administration, initially described by Kessler and Eide,[1] will return to baseline as the dye distributes into venous capillary blood. It is my understanding that the pulse oximeter measures and compares light transmission at the two selected wavelengths (660 nm and 925 nm), and continuously subtracts the non-pulsatile or reference portion of the transmitted light from the total transmission of the pulsating arterialized blood. Therefore, a varying light transmission through non-pulsatile venous blood, whether caused by a changing concentration of dye or deoxyhemoglobin, would be continuously factored out.

Yelderman and New,[2] in their evaluation of pulse oximetry, state that "because the detected pulsatile wave form is produced solely from arterial blood, using the amplitude at each wavelength and Beer's Law allows exact beat-to-beat continuous calculation of arterial hemoglobin oxygen saturation with no interference from surrounding venous blood, skin, connective tissue, or bone."

Intravenous methylene blue pharmacokinetics have been investigated in dogs,[3] and have shown a very rapid and extensive uptake of the dye by tissues (30% of the injected dose taken up by lungs, liver, kidneys, and heart by 3 min in the rat model), where it is bound in the reduced leucomethylene blue form. Additionally, a very rapid and significant decrease in whole blood concentration of the dye has been demonstrated to occur within 10 min after intravenous injection.

Renal excretion of methylene blue in humans[4] has been shown to be present for up to 5 days after oral administration, with 78% excreted as stabilized, colorless, leucomethylene blue. However, the pharmacokinetics and renal excretion of intravenous methylene blue in humans, to my knowledge, have not yet been studied.

Therefore, perhaps my original statement concerning the normalization of pulse oximetry values after intravenous methylene blue administration should be modified to state that the return to normal values of oxygen saturation most likely reflects the dilution of the dye, and its rapid and extensive uptake by body tissues.

THOMAS R. EIDE, M.D.
Assistant Professor

BHARATHI HUMAYUN-SCOTT, M.D.
Assistant Professor

PAUL J. POPPERS, M.D.
Professor and Chairman

Department of Anesthesiology
State University of New York
Stony Brook, New York 11794

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