plied when CBF is measured during hypothermia. For the appropriate application of the nitrous oxide method, a distribution ratio which varies with degree of hypothermia must be applied. If the previously published values were corrected in this way, the corrected values would be lower than the values reported.

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REFERENCES

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To the Editor.—Dr. Ikeda’s letter points out a general problem. The Kety-Schmidt nitrous oxide technique of cerebral blood flow measurement errs in using the venous blood concentration as an estimate of brain concentration before equilibrium is attained. An additional error is introduced when the arteriovenous concentration integral is estimated before equilibrium is complete, particularly if no extrapolation of the arteriovenous difference is attempted. One error partially counteracts the other, and both are minimized when flow is high. But errors increase as equilibrium between blood and brain becomes less complete. Thus, small overestimates of cerebral blood flow result when flow is normal, and larger overestimates occur when flow is low.

If cerebral blood flow values measured by the nitrous oxide method during hypothermia were corrected by applying the distribution ratio as suggested by Dr. Ikeda, only part of the problem would be solved. One of the errors would be corrected, and the “corrected” values would still be incorrect. In addition, measurements made with nitrous oxide in other situations where cerebral blood flow is low (e.g., hyperventilation or administration of thiopental) would remain incorrect. We prefer a different approach—composite correction of all errors in the nitrous oxide technique. This can be achieved using correction factors derived from simultaneous measurements of cerebral blood flow with nitrous oxide and with a less soluble gas such as 40Kr where extrapolation of the arteriovenous integral to infinite time is possible. We believe this procedure would result in more rational correction of nitrous oxide values, affecting high flows in minimal degree, reducing normal flows by a small amount, and lowering low flow values substantially.

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Thiopental and the Fetal Liver

To the Editor.—Finster et al. (Anesthesiology 36:155-158, 1972) suggest that the liver protects the fetal brain from high thiopental levels. Let it be concluded that thiopental is therefore theoretically safe in childbirth. I must point out that their experimental results are entirely consistent with a delay-line theory of liver handling of the drug.1 According to this view, a rise to a peak in fetal brain concentration could occur at a time considerably later than any study to date has followed it. We still have no direct evidence on this point,