studies, pointing out the minimal disadvantages and major advantages of this method of relaxant administration, deserve similar emphasis.

The fear that a desensitization block and prolonged apnea may follow SCh when preceded by a nondepolarizing muscle relaxant seems to be groundless, since this was not observed in the 183 patients studied.1–5 That the administration of a nondepolarizing muscle relaxant before SCh necessitates larger doses of SCh for adequate relaxation is substantiated, and full therapeutic advantages can be obtained if the proper doses of both relaxants are chosen. To varying degrees, all three studies demonstrated that paralysis by SCh will be decreased when it is preceded by 3 mg of \(d\)-tubocurarine. However, Freund and Rubin showed that adequate relaxation can be restored by increasing the dose of SCh by 70 per cent.1 So, in a 70-kg patient, the usual dose of SCh of about 60 mg would have to be increased to 100 mg. Since the additional 30–40 mg of SCh does not result in a prolonged neuromuscular block, this seems to be a small price to pay for all the benefits of preceding SCh with \(d\)-tubocurarine. The benefits include decrease or elimination of SCh-induced postoperative muscle pains; elevated intraocular and intragastric pressures; elevated serum creatine phosphokinase; myoglobinuria; and possibly hyperkalemia.

In summary, the similarities of these studies suggest that preceding SCh, 1 to 1.5 mg/kg, with 3 mg of \(d\)-Tc or 20 mg of gallamine is a reasonable clinical approach.

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(Measuring Cerebral Blood Flow by the Nitrous Oxide Method

To the Editor.—The excellent review by Drs. Smith and Wollman (ANESTHESIOLOGY 36:378–400, 1972) requires some comments based on my own experiments.

Several values of CBF measured during hypothermia are cited in their review. Three of them1–5 were measured by the Kety-Schmidt nitrous oxide method or a modification. Bering et al.,4 as well as Adams et al.,5 slightly modified the original nitrous oxide method by lengthening the nitrous oxide inhalation time to 15 minutes so that sufficient equilibration of the nitrous oxide between arterial or cerebral venous blood and cerebral tissue would take place. Zieg and Bender6 pointed out that erroneously low values for CBF would be found if the solubility of nitrous oxide increased at lower temperatures; however, they actually used the original method and a partition coefficient of 1.0 for calculating CBF. Changes in the partition coefficient or the solubility of nitrous oxide were not taken into consideration in other reports.1–3,7

We investigated the uptake of nitrous oxide in cerebral tissue and blood at normothermia and moderate hypothermia. Nitrous oxide did not reach complete equilibrium between the cerebral tissue and blood (arterial as well as cerebral venous at 30 C and 34 C after 30 minutes of inhalation at 50 per cent nitrous oxide in oxygen. The distribution ratio (the ratio of nitrous oxide in cerebral tissue to that in cerebral venous blood, or relative rates of uptake by brain compared with uptake by cerebral venous blood) would be less than 1.0.

The nitrous oxide method, based on the assumption that the brain/blood partition coefficient of nitrous oxide is 1.0, cannot be ap-
applied 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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(Accepted for publication May 25, 1972.)

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 concentra-
tion 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 ^8Kr 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. 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,

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