plied when CBF is measured during hypo-
thermia. 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 re-
ported.

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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 mea-
surement errs in using the venous blood con-
centration as an estimate of brain concentra-
tion before equilibrium is attained. An addi-
tional error is introduced when the arterio-
venous concentration integral is estimated be-
fore 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 be-
tween 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 pre-
ferr 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 38Kr where
extrapolation of the arteriovenous integral to
infinite time is possible. We believe this pro-
cedure would result in more rational correc-
tion 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. (ANESTHESIO-
LOGY 36:155-158, 1972) suggest that the liver
protects the fetal brain from high thiopental
levels. Lest 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 con-
centration could occur at a time considerably
later than any study to date has followed it.
We still have no direct evidence on this point,