Local Cerebral Blood Flow by Xenon Enhanced CT

To the Editor:

We take exception to the remarks of Gur et al in their recent Progress in Cerebrovascular Disease article (Stroke 13: 750–758, 1982), which may mislead your readership as to the magnitude of the errors associated with xenon CT measurements of CBF. In discussing our error analysis of the single-scan "autoradiographic" approach to xenon CT rCBF measurements (an approach originally championed by Gur and his colleagues), Gur et al fault us for using the fast-flow component, \( f_g \), rather than "total flow" as the reference flow in our definition of percent error. They go on to conclude that "if one corrects for this mistake the errors are significantly lower than reported."

As is apparent from figures 1 and 2, which depict the effect of tissue (gray matter-white matter) heterogeneity in the absence of CT noise, the magnitude of measurement error depends upon (i) whether gray matter (GM) flow, \( f_g \), or "total flow," \( f^* = w f_g + w f_w \) (where \( w \) are proportional weights, and the subscripts g and w refer to gray and white matter (WM), respectively), is used as the reference flow and (ii) whether \( \lambda_g \) or \( \lambda \) \([= w \lambda_g + w \lambda_w] \) is used as the blood-brain partition coefficient for xenon in Kety's blood flow equation. Three definitions of percent error (\( E_1, E_2, E_3 \)) are illustrated: \( E_1 = \frac{f_g - \lambda_g f^*}{f^*} \), \( E_2 = \frac{f_g - \lambda f^*}{f^*} \), and \( E_3 = \frac{f_g - \lambda_g f}{f} \), where \( \lambda_g \) and \( \lambda \) are the solutions to Kety's equation when \( \lambda_g \) and \( \lambda_w \), respectively, are substituted for the partition coefficient. We originally proposed \( E_3 \) to emphasize that, in a one-compartmental model, an anatomic region of interest (ROI) is presumed to be homogeneous; the quantity \( \lambda_g = \frac{f_g}{f} \) was therefore interpreted as estimated GM flow, and the "true" GM flow, \( f_g \), was taken as the reference flow in Equation \( E_1 \). If, however, one measures (or extrapolates) the regional partition coefficient, this "measured \( \lambda \)" is, in a heterogeneous ROI, the weighted average of GM and WM partition coefficients, and the quantity \( \lambda_g = \frac{f_g}{f} \) could be interpreted as estimated "total flow," though the physical meaning of \( f_g \) is problematical. If \( f^* \) is taken as the reference flow, then \( E_2 \) is probably an appropriate definition of percent error. In contrast, the use of \( E_3 \), proposed by Gur et al as the most favorable definition of percent error, cannot be justified; \( \lambda_g \) should not be taken as an approximation of total flow, and the use of \( f^* \) as the reference flow is inappropriate. Using the parameter values in the legend to figure 1, errors predicted by \( E_3 \) are significantly lower than those predicted by \( E_1 \) or \( E_2 \). However, as \( f_g \) and \( \lambda_g \) decrease (as, for example, in stroke), the magnitude of \( E_3 \) may exceed that of \( E_1 \) or \( E_2 \). This is especially apt to occur with the longer inhalation/scanning times required to achieve adequate enhancement under low-flow conditions (fig. 2). Parameter-specific values do not justify the use of \( E_3 \) as an estimate of the accuracy of autoradiographic xenon CT rCBF measurements.

While we applaud the enthusiasm of Gur et al, we must insist that enthusiasm is not an adequate substitute for critical judgement.

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References


The authors reply:

In their letter Drs. Rottenberg and Lu persist in their criticism of the "autoradiographic technique" for measurements of Local Cerebral Blood Flow (LCBF) by Xenon enhanced CT, and they remind the reader that it was "originally championed by Gur and his colleagues." We...
must admit that we are proud to have "originally championed the autoradiographic approach to Xenon/CT LCBF measurements" at a time when most CT scanners were slow (≥ 20 seconds scan time) and interscan delays were much longer (≥ 30 sec) than those currently available on many machines. There is no doubt that this method is now outdated. Since 1979–80 we, along with other groups, have been developing and using more sophisticated multivariable methods of analysis such as the weighted mono- or bi-compartmental least square fits. We believe that the true flow through a tissue volume should be used as a reference flow in error analyses and therefore, we do agree with Rottenberg, et al that what they define as E₂ is the error due to heterogeneity if the mono-compartmental model is used. Rottenberg’s data show that E₂ is significantly smaller than E₁ in tissue with a significant mixture (percent gray < 75%). Therefore, the statement we made in our recent review in STROKE is fully supported by their own data. The rest of the story has already been published and rebattled in the Journal of Cerebral Blood Flow and Metabolism and we see no reason to repeat it here.

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Atriopathic Arrhythmias — Sick Sinus Syndrome

To the Editor:
I would like to comment on the paper of Abdon et al in: Stroke 13 (6), 832–837, 1982, by the following letter-to-the-Editor:

The study of Abdon et al demonstrates that the incidence of finding the heart as the source of a focal cerebral infarction is related to the intensity of investigating the heart as such. In their study on long-term electrocardiographic recording they found a significant higher prevalence of "atriopathic arrhythmias" in a population of stroke patients than in a reference group. Sick sinus syndrome was one of these arrhythmias and it was present in 19 of 88 stroke patients and in only 9 of 103 patients of the control group. I wonder what strategy the authors apply when sick sinus syndrome is a fortuitous finding, and when it appears to be present in a stroke patient. Is anticoagulant therapy indicated? The authors use the temporal pattern at stroke onset as the only criterium to decide if the etiological connection is uncertain, have a future risk for cerebral emboli of 7% per individual and year. We therefore find preventive measures advisable where possible and subscribe to the following general policies:

1) If other aggravating drugs are present, i.e. beta blockers or digitals in the case of sinus bradyarrhythmia, they should be withdrawn and arrhythmia re-evaluated.

2) Where possible, anticoagulants should be given.

Due to the high frequency of concomitant disease in this patient group anticoagulants are often inadvisable and individual judgement must be made.

We have followed tradition in classifying stroke which occurs during sleep or with unclear onset as "thrombotic". Some of these strokes may well have been embolic in origin. Only one patient with a "thrombotic" stroke and atrio-pathic arrhythmia demonstrated progressive neurological deficit after admission. We are more inclined to regard the classification of stroke as embolic or thrombotic using only anamnetic data as insufficient than to consider the presence of atrio-pathic arrhythmia in the "thrombotic" group as purely coincidental.

Yours sincerely,

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References


3. Dinsdale,1 in his recent review of hypertensive encephalopathy (HTE) in this journal; mentions sodium nitroprusside (SNP) as often being the initial drug of choice in the treatment of HTE. While this choice is supported by a large segment of the available neurology and internal medicine literature, we feel that a significant amount of theory and objective information exists that make this drug appear less than optimal. The danger in use of SNP is the same mechanisms as for other direct acting vasodilators: cerebral arteriolar dilation allows delivery of more pressure to the microvascular bed, hence increased edema, and increase in cerebral blood volume (CBV), both of which increase intracranial pressure (ICP) when intracranial compliance is low.2,3

There would not be a problem in using SNP in HTE if there was not a problem with either intracranial compliance or ICP. However, this is not the case. In a study reported in 1954, elevated lumbar pressures were found in 39% of patients with HTE.4 More recently, Grisswold measured ICP with a subdural bolt in three children with stage III coma from HTE. ICP was found to be elevated in two of these patients with a range of 32–70 mm Hg.

Studies2,6,7 have already demonstrated a significant increase in ICP and decrease in CPP while using SNP. One study7 looked at ten patients with intracranial masses and demonstrated marked increases in ICP with a diminished CPP while using SNP, leading them to conclude that SNP should not be used in patients with increased ICP unless measures to improve intracranial compliance are instituted prior to its use. In a comparison of 45 patients in which deliberate hypotension was used for neurosurgery, in normocapnic patients SNP caused a significant rise in

Hypertensive Encephalopathy

To the Editor:

The authors reply:

We are pleased to answer the questions raised by Dr. Lodder. In our report the sick sinus syndrome (SSS) was present in 21 of 86 stroke patients. It has been shown that persons with stroke and SSS, even where the etiological connection is uncertain, have a future risk for cerebral emboli of 7% per individual and year. We therefore find preventive measures advisable where possible and subscribe to the following general policies:

1) If other aggravating drugs are present, i.e. beta blockers or digitals in the case of sinus bradyarrhythmia, they should be withdrawn and arrhythmia re-evaluated.

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