Letters to the Editor

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Intracerebral Hemorrhage Volume Measurement

We recently demonstrated that the volume of intracerebral hemorrhage, as calculated from the computed tomographic (CT) film using the formula for an ellipsoid, is an accurate and powerful predictor of 30-day mortality following intracerebral hemorrhage.1 Since publication of our article in the July 1993 issue of Stroke, we have received several inquiries regarding clarification of our method of volume measurement. The formula for an ellipsoid is \(4/3 \pi (a \times b \times c)\), where \(a\), \(b\), and \(c\) represent the respective radii of the intracerebral hemorrhage in three dimensions. Although this formula is relatively simple and easy to use, one of our colleagues, Dr Bill Cahill, has pointed out that the formula for an ellipsoid can be further simplified to \(ABC/2\), where \(A\), \(B\), and \(C\) represent the diameters of the hemorrhage in three directions. The latter formula is essentially equal to \(ABC/2\). Grotta and colleagues2 have independently used the formula \(ABC/2\) for estimation of intracerebral hemorrhage volume in another model of outcome following intracerebral hemorrhage. These investigators have also demonstrated the ease and power of this bedside method of volume estimation.

Thus, accurate determination of intracerebral hemorrhage volume can be determined very simply and quickly in the following manner. The CT slice with the largest area of hemorrhage is identified. The longest diameter of the hemorrhage on this slice is measured using the CT measurement scale on the film. The diameter of the hemorrhage that is 90° to the longest diameter represents the second diameter. Finally, the number of 1-cm CT slices on which the hemorrhage is visualized provides the third diameter (eg, a hemorrhage seen on three 1-cm slices would have a diameter of 3 cm). The three diameters are multiplied and then divided by 2 to obtain the volume of intracerebral hemorrhage. This method correlates quite well with a sophisticated but time-consuming planimetric method of volume measurement.3 As noted in our article,1 we recently demonstrated that the volume of intracerebral hemorrhage will be critical for patient selection in future randomized surgical trials.

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TCD Velocities and Arterial Pressures in AVM Feeder Vessels

In a carefully performed study,1 Fleischer et al demonstrated a relationship between cerebral arteriovenous malformation (AVM) feeding mean arterial pressures (FMAP) and parent artery blood flow velocity, measured using transcranial Doppler (TCD). They showed an inverse correlation between FMAP- and TCD-derived velocities, the correlation being closer when peak systolic \(r = -62\) as opposed to mean velocities \(r = -35\) were considered. This correlation is perhaps weaker than the authors had initially hoped; thus, it seems that TCD-derived blood velocities in AVM feeder vessels may not be of such powerful prognostic value. Methodological reasons for the worse-than-expected correlation were discussed, but the authors failed to mention the errors incurred in their TCD measurements through not attempting to correct for the angle of insonation of the feeder vessels. The maximum amplitude TCD signal was analyzed and presumably this was thought to relate to an arterial segment whose vector of blood flow was closest to the direction of the ultrasound beam. Assuming angles of under 30° (cosine 30 = 0.87), the potential error is small (less than 15%). However, at larger angles the error in perceived velocity increases disproportionately. In patients with AVMs, it is our experience (from studies using transcranial color-coded sonography and magnetic resonance angiography) that the anatomy can be distorted with tortuosity of the feeder vessels, which may run at greater angles to the ultrasound beam. In addition, the anterior and posterior cerebral arteries run at greater angles than the middle cerebral artery, and determination of "true" blood flow velocities in these vessels should incorporate a correction for the insonation angle. Because the authors measured the angiographic diameter of the feeder vessels at the point of insonation, they may also be able to measure the angle of the insonated arterial segment to the presumed path of the ultrasound beam. Thus, reanalysis of their data to include a correction for the insonation angle may yield a closer relationship between FMAP and the TCD-derived blood flow velocities.

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References

Response
We thank Martin and Gaunt for their constructive and insightful comments regarding an important possible source of error in
interpretation of TCD velocities in patients harboring an intracranial AVM. We agree that some portion of the variance that accounted for the less-than-perfect correlations may have been attributable to errors related to the angle of insonation.

We would point out the following considerations regarding AVMs. Distortion of the proximal conductance vessels is most likely to occur with mass lesion effects, as Finn et al have described for hydrocephalus; bowing of vessels may cause an underestimation of the true velocity of the blood column. True AVMs present with mass effect only in the rarest of cases. However, because AVMs sometimes result in bizarre changes even in proximal vessels, the authors' point is well taken and should be borne in mind in future studies or the interpretation of values from an individual patient. On an individual basis, this may be especially pertinent because feeding artery pressure, when considered in context with other factors, may influence the incidence of spontaneous intracranial hemorrhage from AVMs and therefore may affect the decision of whether or not to treat a particular lesion.

It is worth noting that Manchola et al performed a study of AVM feeding arteries that was in some respects similar to ours (without pressure measurements). As a part of that study, they described a very careful inspection of the angiograms to optimize the best angle of insonation to minimize this source of error. Of their 40 patients, however, they did not describe any with the remarkable deviations suggested by Martin and Gaunt (ie, angle of insonation of > 30°).

A possible pitfall in trying to determine the actual angle of insonation should also be considered. Unless one performs the TCD during fluoroscopy (which is almost never the case), the exact relationship of probe angulation to the innomated vessel can only be approximated.

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References

Effect of Nitric Oxide Synthase Inhibition on Cerebral Blood Flow and Injury Volume

We commend Nishikawa et al for the care they exercised in their study of middle cerebral artery (MCA) occlusion-induced focal cerebral ischemia in cats, in which they investigated the effects of nitric oxide (NO) synthase inhibition on cerebral blood flow, sensory evoked potentials, and extent of acute cerebral "injury." They tightly controlled relevant variables—a merit of using this large animal model. Their finding of significantly reduced volumes of acutely "injured" caudate nucleus but not of cerebral cortex with no differences in sensory evoked potentials or reductions of cerebral blood flow in the treated versus the control groups contributes new information regarding the mechanisms involved in ischemic brain injury.

The authors assessed tissue "injury" after 4 hours of MCA occlusion using visual evidence of the lack of reduction of 2,3,5-triphenyltetrazolium chloride (TTC) by tissue mitochondrial enzymes and the morphometric quantitation of such nonreactive brain volumes. The authors indicate their awareness that this method's "injury" volume may not necessarily represent actual tissue infarction and that the comparison group's eventual verifiable tissue injury extent might be similar. Because we have carried out studies using the same animal model, our data can provide an estimate of how their acute "injury" volume may compare with permanent tissue infarction volume. We assessed infarct size morphometrically after 2 weeks' survival following 4 hours of temporary, normoglycemic MCA occlusion in 12 cats. Eleven survived and one died acutely from hemispheric edema, cerebral tissue herniation, and brain stem compression. The 11 survivors showed infarction of only 0.6±0.6% of the ipsilateral hemisphere (de Courten-Myers et al, 1989). In comparison, the cats of Nishikawa et al, showed a mean volume of acute "injury" in control animals of 32% of the hemisphere.

Thus, it appears that only a small fraction of the acutely "injured" tissue as measured by the TTC reduction technique evolves into permanent infarcts. Differences in the model can account for only part of this large difference, because both studies were closely controlled and are similar regarding glycemia levels (mean serum glucose concentrations during 4 hours of MCA occlusion in the study of Nishikawa et al versus ours, 163 vs 144 mg/dL, respectively), brain temperature, blood respiratory gases, and other monitored parameters. The two studies did, however, use different anesthetics (halothane versus pentobarbital).

Readers may be tempted to extrapolate the early changes in mitochondrial dysfunction to permanent damage to the brain. However, the data presented suggest that the extent of acute metabolic alterations grossly overestimate the extent of actual brain tissue infarction. Notwithstanding the value of establishing the therapeutic effects of pharmacologic interventions during exposure, follow-up studies determining the extent of permanent tissue injury remain a necessary step in evaluating the effects of drug therapy.

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References

Response
We appreciate the generous comments by Drs de Courten-Myers and Myers. We agree that it would be improper to extrapolate the early changes in mitochondrial dysfunction observed in our study to indicate the volume of actual brain tissue infarction. This question has been directly evaluated by Cole et al, who demonstrated that the histochemical abnormality revealed by TTC staining may not necessarily represent inevitable infarction when used for paradigms of short ischemic periods (3 hours in...
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