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 (i.e., 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 insonated vessel can only be approximated.

**References**


**Effect of Nitric Oxide Syntase 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 re-

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their study). In addition, resolution of edema will shrink eventual infarct volume to some extent.

Thus, it is possible that if reperfusion had been allowed to occur in our study, the actual volume of brain infarction would have been less than the amount of early brain injury we observed at 4 hours. However, the exact amount of brain infarction produced in our study would be a basis of the study by de Courten-Myers et al because the anesthetic techniques in the two studies were different. In the study by de Courten-Myers et al, the anesthetic used was pentobarbital, a barbiturate that decreases brain injury in animals exposed to focal ischemia. In contrast, halothane is an inhalational anesthetic that at high concentrations decreases cerebral metabolism but does not appear to decrease brain injury after focal cerebral ischemia. Furthermore, we excluded the cats in which somatosensory evoked potentials were not immediately reduced to ensure a more reproducible insult.

In addition, caution must be taken when comparing results among laboratories. For example, Kolluri et al observed TTC-determined infarct volumes of about 24% in cats subjected to 2 hours of MCA occlusion under ketamine-pentobartoribine anesthe sia followed by 1 week of reperfusion. Comparison of this study with our results would suggest little further change in injury volume after 4 hours of ischemia. In the absence of anesthesia, infarct volumes of 12.8±8.5% have been reported in cats subjected to 4 hours of transient MCA occlusion. This value is greater than that observed by de Courten-Myers et al. Therefore, one cannot conclude that the TTC-determined injury volume of 30% that we observed at 4 hours of occlusion would eventually shrink to the 0.6% infarct volume observed by de Courten-Myers et al.

Our data demonstrate that the amount of early brain injury produced by 4 hours of MCA occlusion is reduced by inhibition of NO synthase. If treatment with L-NAME decreases infarct volume at 4 hours, our data would suggest that inhibition of NO synthase decreases the rate at which brain injury occurs in this experimental model. An alteration in rate of injury may have important clinical implications as it may increase the window of opportunity for use of other therapeutic agents. We believe that it will be important to extend our experiments to evaluate the therapeutic role of NO synthase inhibitors on permanent tissue injury resulting from permanent and transient focal ischemia.

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