Evaluation of Vasomotor Reactivity by Transcranial Doppler and Acetazolamide Test Before and After Extracranial-Intracranial Bypass

I agree with Karnik et al that the cerebrovascular reactivity as measured with transcranial Doppler (TCD) sonography and acetazolamide test may well become a criterion for selecting those patients suffering from internal carotid occlusion in whom an extracranial-intracranial (EC-IC) bypass could lower the risk of ischemic stroke. Two points, however, must be kept in mind when considering the results of these authors.

First, the response of flow velocity to acetazolamide does not necessarily reflect the response of volume flow. The reason is that constancy of the cross-sectional area of the middle cerebral artery (MCA) cannot be ensured with this test. In fact, measurement of the Doppler signal power (which is proportional to the cross-sectional area of the vessel) along with mean spatial velocity (V) showed that acetazolamide leads to a dilation not only of the small resistance arteries but also of the trunk of the MCA (Figure 1). In the example demonstrated, the percent increase in velocity therefore was barely about half of the percent increase in volume flow, calculated as the product of V and p (TCD flow index [FI]). These findings were very similar to those obtained in CO2 respiration and clearly show that FI rather than V should be used for measuring the cerebrovascular reserve.

Second, and even more important, there is no evidence whatsoever that the cerebrovascular reserve demonstrated by means of the acetazolamide or CO2 respiration tests can be used for judging the integrity of autoregulation. Measurement of the response of the TCD FI to lowering blood pressure by tilting the head up or to lower body negative pressure would therefore appear to be more appropriate approaches to the selection of cases to be considered for carotid endarterectomy or EC-IC bypass surgery.

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References

Applications of Transcranial Doppler Sonography in Acute Ischemic Stroke

The article by Hedera et al suggests that a single transcranial Doppler (TCD) study of the middle cerebral arteries (MCAs) performed up to 48 hours after acute stroke may be clinically helpful, particularly in estimating prognosis over 28 days. We disagree. This is likely to be true only for highly selected patients
with internal carotid artery occlusion, in whom sequential changes are uncommon. Arterial recanalization is more common in patients with intracranial occlusion, and serial transcranial Doppler studies have shown high velocities accompanying such reperfusion, both spontaneously and after thrombolytic therapy. These sequential changes indicate that baseline TCD should be performed early following acute stroke, with repeated studies thereafter. A single study, particularly if delayed, is likely to be of limited value.

We also disagree with the statement that using TCD it is impossible to determine whether an absent signal from MCA is caused by occlusion of the MCA or to other conditions. The identification of ipsilateral posterior or anterior cerebral artery signals, often with increased velocity as a consequence of collateral flow via the leptomeningeal anastomoses, is helpful in confirming that the temporal ultrasound window is adequate and implies true absence of MCA flow.

The conclusions reached in the study are based on results presented in Figure 2 of that article. We are concerned that the data points for mean frequency shift in MCA (first examination) in Figure 2 do not match those in Figure 1, even allowing for the six patients who were excluded from follow-up examination, nor do these data equate with the mean frequency shifts shown in Table 1. We would be grateful for the authors' clarification of these apparent discrepancies.

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References

Response
I appreciate the interest of Drs. Grosset and Lees in our study and welcome this opportunity to clarify several important issues. TCD is a clinically useful methodology with definite limitations. Evaluation of intracranial vessels with TCD in the presence of intracranial occlusion has a limited value for the estimation of blood flow in an area of infarction. Occlusion of the stem of the MCA is associated with an echo-free area from the MCA; occlusion of the peripheral branches of the MCA can cause only mild reduction of blood velocities in the MCA. Even the detection of the presence of collateral flow through the anterior cerebral arteries (ACAs) or posterior cerebral arteries (PCAs) does not allow more exact estimation of adequacy of the perfusion of the infarcted area. However, in our patients with occlusion of extracranial vessels, measurement of flow velocity in the MCA is informative about blood flow in the stroke region. We did not generalize our conclusions to all types of strokes, and we are aware that they are valid for strokes with low flow in the MCA due to occlusion of the extracranial ICA.

Diagnosis of the occlusion of the MCA should not be based solely on the absence of the MCA signal. I agree that undetectable signals from the MCA with higher flow velocities in ACA and PCA ipsilaterally can be considered as diagnostic of MCA stem occlusion. However, ultrasonographic diagnosis of simultaneous occlusion of extracranial segments of ICA and MCA presents a greater challenge. Mattle et al found unchanged or reduced velocities in the ACA and PCA ipsilateral to stenosis or occlusion of the ICA. The amount of collateral flow in the ACA and PCA is highly variable, and globally reduced flow behind the occlusion of the ICA can also reduce flow velocity in the ACA. Moreover, in acute strokes, anatomic position of the MCA can also be changed by edema in the infarction area, which does not allow for detection of the MCA while ACA and PCA signals are present. These factors make the diagnosis of simultaneous occlusion of the ICA and MCA more difficult, and this was the reason for excluding such patients from our study.

I apologize for erroneous plotting of some patients in our Figure 1. I regret this mistake, which leads to discrepancies between Figure 1 and other data. However, this graphical error does not at all change the result presented in our study.

There is no doubt that determination of the prognostic value of TCD in ischemic events with occlusion of intracranial vessels will require further studies. One must keep in mind known patterns of TCD findings under such conditions; certainly they will require different timing for the TCD examinations, as was used in our study.

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Presumed Cardioembolic Lacunar Infarcts
Lacunar infarcts are usually the result of a small-vessel arteriopathy (lipohyalinosis or microatheromatosis) secondary to hypertension that occludes a single perforating artery at the base of the brain. An embolic pathogenesis of lacunar infarct has been discussed in the literature, but the prevalent view is that cardioembolic embolism is unlikely to be a common cause of lacunar infarct. In a recent paper, however, Cacciato and Russo presented two cases of pure motor hemiplegia as an embolic complication of cardiac and arch angiography.

To date, little is known about the frequency of cardiogenic embolism in lacunar infarct. To contribute to the study of the frequency of the cardiogenic lacunar infarct, we carried out a prospective clinical study in 227 consecutive patients with lacunar infarct. In our series, 11 patients (5%) had presumed cardiogenic lacunar infarct. In these cases, a cardioembolic source of stroke was the only risk factor identified. No patients had hypertension, diabetes mellitus, or other stroke risk factors. Computed tomography or magnetic resonance imaging and transthoracic two-dimensional echocardiography had been performed on all patients. Lacunar infarct was defined as in a previous study. The group consisted of six women and five men. The clinical syndromes were pure motor hemiparesis in seven, pure hemisensory stroke in two, the sensorimotor deficit syndrome in one, and modified pure motor hemiplegia with motor aphasia in one.

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