A

cute hemiplegia is often due to ischemia in the territory supplied by the middle cerebral artery (MCA). There is a consensus that if this ischemia persists for longer than a few hours, permanent tissue damage will result. This short time period greatly constrains the opportunity for treatment by emergency revascularization surgery or thrombolysis.

Transcranial Doppler ultrasonography (TCD) offers the possibility in some cases for rapid noninvasive diagnosis of the arterial lesion. Although there is controversy about the quantitative accuracy and significance of the measurement of blood velocity in the accessible intracranial vessels, there can be little doubt that the demonstration of measurable blood velocity in the MCA means that the artery is not occluded. This qualitative fact, relatively easily and quickly demonstrable by TCD, may have practical importance in facilitating some of the decisions that would be necessary for emergency medical or surgical revascularization. The purpose of my report is to present preliminary experience with TCD evaluation in cases of acute hemiplegia.

Subjects and Methods

Fifteen consecutive patients with complete hemiplegia had initial clinical neurologic and TCD examinations within 12 hours of onset of the hemiplegia (early cases) and were then followed daily in the hospital and subsequently up to 6 months as outpatients. An initial noncontrast computed tomogram and lumbar puncture were normal in all 15 patients; arteriography was not done in any. Using an EME TC 2-64 TCD recorder (Uberlingen, F.R.G.) operating with pulsed ultrasound at 2 MHz, mean blood velocity was measured in the horizontal portion of the MCA on each side at 5-mm intervals between the depths of 60 and 40 mm from the temporal scalp. The average of these five measurements in each artery was then calculated and used for comparing the two sides in each patient and the different patients. In three patients it was not possible to obtain a recording from the MCA in the asymptomatic hemisphere. These early cases were compared with 28 in whom the first measurement was made >12 hours after onset of hemiplegia, which had persisted until the time of the examination (late cases).

Results

All 15 early patients were completely hemiplegic at the time of the first TCD examination. Subsequently three made total clinical recovery within 1–4 days. Three made partial useful recovery between 1 and 3 weeks later, becoming able to walk normally, to lift the arm above the head, and to open and close the hand, but they could not oppose the thumb individually to the fingers. The other nine, on examination 3–6 months later, retained total paralysis of the hand and arm, although six had regained limited walking ability.

Among eight patients whose MCA blood velocity was <30 cm/sec, one recovered completely while the other seven retained permanent total paralysis of the hand and arm.

Among eight patients whose MCA blood velocity was >30 cm/sec, five made complete or partial useful recovery of the involved hand and arm. Among eight patients in whom blood velocity was <30 cm/sec, one recovered completely while the other seven retained permanent total paralysis of the hand and arm. (Stroke 1988;19:648–649)
Figure 1. Plot of middle cerebral artery blood velocity and time of transcranial Doppler ultrasonography after hemiplegia.

Discussion

The conclusion from my preliminary data is that the prognosis of acute hemiplegia may be recognized within 12 hours of onset by TCD. Prognosis is relatively good if MCA blood velocity is > 30 cm/sec but poor if it is less than that. If >12 hours have elapsed from onset until the first measurement, TCD has not been very useful for prognosis simply because hemiplegia persisting for >12 hours has itself a poor prognosis. The most that can be said is that if blood velocity at that time is >30 cm/sec there remains a small chance of recovery, whereas if blood velocity is <30 cm/sec there is virtually no chance.

The 30 cm/sec value is the lower limit of normal quoted by others.\(^1,2\) This is also consistent with my own experience with patients who have recovered from stroke or transient ischemic attacks with no residual disability. We suspect that a recorded blood velocity of < 30 cm/sec usually means that though the horizontal portion of the MCA is patent, one or more branches beyond the trifurcation are occluded or that there is a large infarct with intraparenchymal arteriolar occlusions.

A conceptual limitation on interpretation of these data is the inability to distinguish those patients whose MCA had been occluded and then recanalized from those whose proximal MCA was never occluded, their hemiplegia being due to occlusion of a lenticulostriate or anterior choroidal artery, an MCA branch distal to the trifurcation, or even to a brainstem lesion. To argue that these are distinguishable on clinical examination and have a prognosis different from that of proximal MCA occlusion is not really helpful since these criteria are not always reliable when applied by others than the most experienced clinicians. In the evaluation of acute hemiplegia it is at least relevant to want to know if the MCA is or is not occluded. Moreover, from my preliminary data, even allowing for the uncertainty about the site of the lesion, MCA blood velocity appears to have a predictive value. Further application of TCD to the problem of acute stroke appears worthwhile.

References


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