Flow and Velocity During Autoregulation Testing in Humans

To the Editor:

It may be unusual to write a letter to the editor referring to an abstract rather than to an original publication. Nevertheless, the summary of the paper read by Newell et al at the 19th International Joint Conference on Stroke and Cerebral Circulation and published in the January issue of *Stroke* appears to need some comment.

Newell et al measured the percent changes of middle cerebral artery (MCA) velocity by transcranial Doppler sonography and those of internal carotid artery (ICA) flow by electromagnetic flowmetry in seven patients who had undergone carotid endarterectomy. In these patients a drop in arterial blood pressure (ABP) was induced by deflating leg cuffs after 3 minutes of inflation to suprasystolic pressure. From the evidence of identical curves for MCA velocity and ICA flow, Newell et al concluded that changes in time-averaged MCA velocity accurately reflect the changes in ICA flow during autoregulation. This may be true in cases in which autoregulatory dilatation is limited to the small cerebral arteries and does not include the trunks of the basal arteries.

In our own experience, using a type of Doppler signal analysis that includes the measurement of signal power as a function of vessel caliber, there was clear evidence that the middle cerebral artery trunk is included in autoregulatory vasodilation both in orthostasis and lower body negative pressure tests of autoregulation. The same effect can be observed when using the leg cuff technique described by Newell et al for producing a fall in ABP.

Our Figs 1 and 2 show two typical examples in healthy volunteers. In Fig 1, an increase in signal power *p* following the fall of ABP with velocity *v* decreasing can be seen. The product *F* of *p* and *v*, indicating volume flow, remains constant. In the example shown in Fig 2, *v* remains around 100% despite *p* indicating an increase in vessel caliber. Logically, *F* as well increases in this case after the fall in ABP. We view this effect as attributable to an increase in the caliber of the small arteries of the MCA tree in addition to the arterial trunk.

In the abstract of Newell et al, Fig 2 is difficult to interpret. With no clinical comments given regarding carotid stenosis in the case demonstrated, it is entirely possible that there was no autoregulation in the early phase demonstrated on the graph and that the MCA velocity therefore indicates volume flow. Notice, however, that the analysis includes barely one third of a minute during which the MCA velocity and ICA flow returned to normal. Their further evolution, and whether this even included increased flow, cannot be established from the graph.

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### References

The pathogenesis of the neurological manifestations remains incompletely understood, but MRI scans in TTP may provide more insight into the possible mechanisms that are responsible for the neurological manifestations. An additional patient with TTP but with a silent brain infarct detected on serial MRI is described. A 38-year-old executive from Brazil had an inferolateral myocardial infarction in 1992. Results from a subsequent coronary angiogram were normal. A few months later while in his office, he suddenly experienced double vision, left hemiparesis, and difficulty finding words. On admission to another hospital, neurological examination was remarkable for normal visual acuity and normal visual fields, but a rotary nystagmus, an impaired downward gaze, and a left hemiparesis and hypesthesia were found. Laboratory investigation showed a total platelet count of 24,000 and schistocytes on blood smear. Anticardiolipin antibody titer was negative. Renal function tests were normal. MRI imaging showed a right thalamic infarct and scattered small infarcts in the cerebellum (Figure). He was treated with heparin, and he recovered with persistent diplopia and spastic left hemiparesis.

The patient remained asymptomatic for 8 months but continued to have low total platelet counts without evidence of a hemolytic anemia. A repeat MRI scan, however, showed a new occipital infarct that could not be detected on neurological examination. Three months after the second brain MRI, he developed brief episodes of left-hand numbness and word-finding difficulty. Computed tomographic (CT) scan was unremarkable, but MRI was not repeated. He was lost to follow-up after hospital discharge following one series of plasma exchange.

TTP is a source of considerable morbidity in young adults. The clinical entity is often characterized by a pentad of thrombocytopenia, hemolytic anemia, fever, renal failure, and neurologic findings. Neurological manifestations are often a feature of this unusual disease and punctuate the clinical course with a variable degree of central nervous system involvement. Fluctuating confusional states and rapid progression to unresponsive coma dramatically responds to one series of plasma exchange without, in general, visible permanent damage to imaging studies.

The underlying pathophysiological events in TTP have been debated since its original account. The universal histopathologic findings are characterized by hyaline thrombi formed by the aggregation of thrombocytes, mostly in small arterioles and capillaries. Mural or extramural inflammation is typically absent. Abnormalities in the depolymerization of von Willebrand factor

References

Silent Brain Infarct in Thrombotic Thrombocytopenic Purpura

To the Editor: Rinkel and colleagues1 have recently reported magnetic resonance imaging (MRI) abnormalities in relapsing thrombotic thrombocytopenic purpura (TTP). In reviewing the literature, only two case reports of MRI findings of ischemic infarcts have been described since.2,3 The pathogenesis of the neurological manifestations remains incompletely understood, but MRI scans in TTP may provide more insight into the possible mechanisms that are responsible for the neurological manifestations. An additional patient with TTP but with a silent brain infarct detected on serial MRI is described. A 38-year-old executive from Brazil had an inferolateral myocardial infarction in 1992. Results from a subsequent coronary angiogram were normal. A few months later while in his office, he suddenly experienced double vision, left hemiparesis, and difficulty finding words. On admission to another hospital, neurological examination was remarkable for normal visual acuity and normal visual fields, but a rotary nystagmus, an impaired downward gaze, and a left hemiparesis and hypesthesia were found. Laboratory investigation showed a total platelet count of 24,000 and schistocytes on blood smear. Anticardiolipin antibody titer was negative. Renal function tests were normal. MRI imaging showed a right thalamic infarct and scattered small infarcts in the cerebellum (Figure). He was treated with heparin, and he recovered with persistent diplopia and spastic left hemiparesis.

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