It is correct that we only showed about 20 seconds of the response. By this time the MCA velocity as well as the ICA flow and velocity during autoregulation testing in humans. Stroke. 1994;25:270. Abstract.


Response

We appreciate the comments to our abstract, which are also appropriate for the larger study published recently in Stroke. However, we disagree with the conclusions and speculations of Dr Müller.

Figs 1 and 2 in Dr Müller's letter indicate that readings during the autoregulatory response were taken at intervals of about 6 seconds. This choice is inadequate, as the autoregulatory response times are typically 5 seconds and may be as short as 3 seconds. Thus, the entire dynamic response may have been missed because of the infrequent sampling. It is correct that we only showed about 20 seconds of the response. By this time the MCA velocity as well as the ICA flow had returned to control values. However, we would like to add that we saw no discrepancy between these parameters in the period after this interval and could find no support for Dr Müller's hypothesis that there may be a delayed dilatation of the MCA in the first minutes of the autoregulatory response to a step decrease in ABP.

We have been using a flow index method since 1986. Our experience with this method is that it is valid only under very specific circumstances. During orthostatic tests, it cannot be used because of the slight movements of probes and shifts in the position of the brain structures. Furthermore, the high-pass filtering of most instruments— including that used by Dr Müller—introduces errors because the flows below the filter levels are neglected in the result. In practice it is also difficult to achieve a sufficient signal-to-noise ratio for the method to be valid. Using our specially designed instrument to compare the Doppler flow index to the spectral outline during autoregulation, we never saw a power or flow index response like the one shown in Figs 1 and 2 in Dr Müller's letter.

Hans R. Müller, MD
Department of Neurology
University Hospital Basel, Switzerland

References


Silent Brain Infarct in Thrombotic Thrombocytopenic Purpura

To the Editor:

Rinkel and colleagues 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.

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 hematologic 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 respond to one series of plasma exchange without, in general, visible permanent damage on 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 agglutination of thrombocytes, mostly in small arterioles and capillaries. Mural or extravascular inflammation is typically absent. Abnormalities in the depolymerization of von Willebrand factor...
Serial T2-weighted magnetic resonance imaging in thrombotic thrombocytopenic purpura. Upper row, Small infarcts in both cerebellar hemispheres and in right thalamus. Lower row, Unchanged small cerebellar infarcts, resolution of thalamic lesion, and a new occipital infarct on the left.

have been hypothesized, and these large multimers of von Willebrand molecules have been shown to clump platelets in vitro.

The neurological manifestations of TTP are diverse, but in most instances an acute confusional state, seizures, transient hemiparesis, and stupor predominate.1,2 The clinicopathologic correlation in patients with decreased levels of consciousness alone is problematic but may be related to widespread vascular involvement in the cortex. CT or MRI findings in TTP have been mostly linked to transient focal neurological deficits, but coma and fluctuating level of consciousness have been correlated with multiple areas of decreased attenuation in white matter.2 Recently, a few reports of patients with TTP who undergo MRI have suggested that although recovery may be complete, small to minute cerebral infarcts can be demonstrated.

Our patient is of particular interest because his clinical course, documented by serial MRI scans, provides presumptive evidence that ischemic infarcts may go unnoticed. The diagnostic value of MRI in TTP has not been systematically evaluated, and in two recently investigated patients with decreased level of consciousness alone and relapsing TTP at our institution, MRI investigation was normal (E.F.M.W., unpublished data, 1994). The observation of unrelenting cerebral infarction may influence the decision of whether to use antiplatelet drugs or high doses of corticosteroids as adjuncts to plasma exchange.6

Eelco F.M. Wijdicks, MD
Department of Neurology
Mayo Clinic and Mayo Foundation
Rochester, Minn

References

Segmental Narrowing of the Supraclinoid Carotid Artery in Young Patients With Ischemic Stroke

To the Editor:

An image of narrowing of the supraclinoid part of the internal carotid artery is occasionally seen on angiography in young patients with ischemic stroke.1,2 Because this angiographic feature is not specific, diagnosis usually remains uncertain except in fatal cases with pathological examination.2 We describe four young patients with cerebral infarction and stenosis of the supraclinoid carotid artery. The regressive nature of the angiographic abnormality made the diagnosis of dissection likely in three of the patients. In comparison with previously reported cases of spontaneous dissection of the intracranial carotid artery, these patients are noteworthy because of their favorable outcome.

Subjects were selected from a series of 141 patients aged 15 to 45 years, consecutively admitted to our department following ischemic stroke. Cerebral angiography, performed in 123 patients (87%), was obtained by transfemoral selective carotid opacification and repeated in all selected patients to assess the evolution of the arterial lesion. Clinical outcome was classified according to the Rankin scale. Clinical and angiographic data are summarized in the Table. The mean age of the patients was 22.5 years. All patients presented with first-ever ischemic stroke. The cerebral infarction was limited to the territory of the anterior choroidal artery in three patients. There was no evidence of subarachnoid hemorrhage, drug addiction, or infection. Cerebrospinal fluid was analyzed in two cases and showed no abnormality. All patients were treated with antiaggregants alone or antiaggregants and then aspirin. Final disability was either absent or slight on the Rankin scale.
Silent brain infarct in thrombotic thrombocytopenic purpura.
E F Wijdicks

*Stroke*. 1994;25:1297-1298
doi: 10.1161/01.STR.25.6.1297

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1994 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/25/6/1297.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Stroke* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Stroke* is online at:
http://stroke.ahajournals.org/subscriptions/