Letters to the Editor

Letters to the Editor will be published, if suitable, as space permits. They should not exceed 1,000 words (typed double-spaced) in length and may be subject to editing or abridgment.

Cerebral Vasospasm in Eclampsia

To the Editor:

I read with interest the article by Trommer et al and the letter by Dr. Goodlin. I failed to see a reply from either Dr. Trommer or the editor, but instead saw a letter from Dr. Brick in the next issue of Stroke. I feel both letters need further clarification and offer my comments.

First, I agree in principle with Dr. Goodlin’s points regarding a subspecialty journal and the proper diagnosis of reported patients. However, a strict definition reflects our lack of understanding of eclampsia. Also, defining eclampsia solely in terms of seizures may give a false sense of security and deny early intervention to preeclamptic women with subtle neurologic symptoms (i.e., headache, blurring of vision, etc.). We know through neuroradiologic studies that cerebral lesions may be present even in these patients and that treatment is thereby warranted. In fact, patients with seizures may show no lesions on computed tomograms, with minimal reversible lesions on magnetic resonance images, whereas patients without seizures and only “blurring of vision” may have diffuse permanent lesions. Nobody can predict who will deteriorate or how fast, so even patients with “minor” neurologic symptoms deserve equal attention. Perhaps this is why many physicians still use the older, but all-inclusive, term toxemia and accept the unpopular concept of eclampsia without seizures.

Second, Dr. Goodlin’s comment that “this type of cerebral vasospasm has been recognized for the last 45 years” needs clarification. Though such patients have indeed been known for years, I had no knowledge of direct in vivo evidence to support cerebral vasospasm until a few years ago. Review of the literature shows that angiography was first reported in “toxemia” by Beck and Menezes in 1981. However, this patient had concomitant intracranial hemorrhage. A subsequent report by Will et al also showed hemorrhage in two of three patients. It is possible therefore that the hemorrhage caused the vasospasm. The report of Trommer et al provides some of the first clear angiographic evidence for vasospasm.

Third, although Trommer et al and Brick agree on the possible role of the female reproductive hormones, the authors fail to highlight differences in their cases. One was clearly toxemic with diffuse vascular involvement, whereas the other was not toxemic and showed only focal involvement. These are critical points in the consideration of etiopathogenesis.

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References

The following is in reply:

To the Editor:

The clinical picture of preeclampsia or eclampsia (toxemia) was accurately described in American obstetrical textbooks prior to World War I. In the intervening years, nearly every available laboratory test or diagnostic technique has been found to be abnormal in some of these patients. Yet our understanding and care of these patients has seen little improvement above that available to any seriously ill pregnant woman. In my view, we will make significant progress only when accurate diagnoses are made of the presumed different etiologies of this disease complex.

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Rheological Behavior of White Blood Cells

To the Editor:

We read with interest the short communication by Violi et al regarding the white blood cell (WBC) response in patients suffering from acute stroke and agree that WBC activation occurs in cerebral infarction.

Recently, Nash et al have suggested that WBC activation "in vivo" would have a significant rheological effect detectable by filtration procedures. We are currently studying WBC rheology during the early phase of acute ischemia in humans by measuring the resistance of WBCs to flow through 5-μm pore filters at constant pressure.

Results from our first 10 patients indicate that WBC filterability is impaired in cerebral infarction. When individual WBC subpopulations were analyzed, lymphocytes exhibited the least resistance to flow (not different from 10 matched controls), followed by granulocytes (p<0.01 vs. controls), with monocytes showing most resistance (p<0.01 vs. controls, Table 1).

Our data support previous observations. We conclude that the rheological behavior of WBCs, and in particular the mono-
Cerebral vasospasm in eclampsia.
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Stroke. 1989;20:826
doi: 10.1161/01.STR.20.6.826.a

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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