Letters to the Editor

Plasmapheresis in Heparin-Induced Thrombocytopenia and Thrombosis

To the Editor:

Thrombosis associated with severe thrombocytopenia is an infrequent, but life-threatening side effect of heparin therapy, which can involve any venous or arterial territory including the cerebral circulation. The treatment of this serious complication has not been satisfactorily resolved.1 Recently, Becker and Miller2 reviewed heparin-induced thrombocytopenia with thromboembolism and found one case that had been treated successfully with plasmapheresis.3 In a recent report,4 we described a similar patient who also satisfactorily responded to plasmapheresis therapy. She was admitted to the hospital for right thrombophlebitis with pulmonary embolism. After 13 days on heparin therapy, new thrombotic events in pulmonary and peripheral circulation became evident, and the platelet count fell to 10,000/mm3. We discontinued heparin administration, but the platelet count remained unchanged and the ischemic lesions increased progressively until plasmapheresis therapy was initiated. Shortly thereafter, the number of platelets returned to normal, and the ischemic alterations improved considerably.

The incidence of heparin-induced thrombocytopenia with thrombosis is approximately 1–2%, and it has a mortality of nearly 30%. It is of particular importance to neurologists in the treatment of cerebrovascular disease. An immune-mediated mechanism has been established as the cause of this complication, which is detected in the plasma of most affected patients by the presence of heparin-dependent antiplatelet antibodies that induce platelet aggregation.1,2 Withdrawal of heparin treatment is essential for recovery, but other therapeutic measures must be used as well to prevent progressive development of platelet thrombi. Plasmapheresis therapy, useful in other antibody-mediated diseases,3 can be very effective in severe heparin-induced thrombocytopenia with thrombosis by removing the pathogenic immunoglobulin.

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Lacunar Pontine Infarction With Hemifacial Spasm as the Initial Symptom

To the Editor:

Recently, Ambrosetto et al1 described an unusual case of lacunar pontine infarction producing hemifacial spasm as an isolated clinical finding. We report here our recent experience with a case of lacunar pontine infarction producing hemifacial spasm as the initial symptom.

Our patient was a 76-year-old hypertensive woman who noticed numbness and tightness on the left side of her face when she got up one morning. About 1 hour later, during her breakfast, rapid, involuntary muscular twitches occurred on the left inferior obicularis oculi and soon spread over the left side of her face, occurring intermittently.

Two days later, she was admitted to our hospital. The neurologic examination on her admission revealed a clearly conscious woman who had a slight speech disturbance, a slight right hemiparesis, and intermittent hemifacial involuntary movements similar to the facial spasm that occurs as the common sequel of partial peripheral facial nerve lesions. Computed tomographic scans of her head on admission showed a small lacunar infarct in the left pons.

The hemifacial spasm occurred frequently for the first 4 or 5 days after onset, but thereafter gradually decreased until its disappearance about 2 weeks later.

Ambrosetto et al explain the mechanism of hemifacial spasm as the result of compression of the intrapontine roots of the facial nerve by perifocal ischemic edemas. Since our patient also had slight contralateral hemiparesis, the hemifacial spasm seemed to result from irritation of the nucleus or the facial nerve itself by ischemic edema. Though such a case is quite rare, we believe it important to pay attention to this symptom as a sign of lacunar pontine infarction.

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Changes of Cerebral Blood Flow Velocity During Cognitive Activity

To the Editor:

In the September issue of Stroke, Droste and colleagues1 measured the time course of cerebral blood flow velocity (CBFV) changes in the middle cerebral artery (MCA) to cognitive activity with transcranial Doppler sonography (TCD). Under their conditions of 90 seconds reading aloud followed by a 42-second rest-phase, the authors found an immediate increase of CBFV after onset with a maximum peak (15% increase relative to the steady-state at rest) after about 8 seconds. A steady-state of about 10% increase in CBFV appeared 24–42 seconds after onset. In the rest-phase, the enhanced CBFV continued until a small peak after 6 seconds when it decreased to a minimum and then increased

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again to a steady-state. No differences in response between right and left MCA were observed nor between right-handers and left-handers.

In an attempt to replicate the results of Droste et al,1 using the same transcranial Doppler system (TC2-64, EME, Überlingen, FRG), we failed to record a pulsatile CBFV response during the reading phase because of strong noise resulting from the voice frequency spectrum conducted via the head bones to the temporally placed probe. However, we were able to record CBFV without artifacts if the subjects were instructed to whisper rather than to read aloud. Further, use of a refined Doppler system (trans-Scan, EME, Überlingen, FRG) and improved computer-assistance for the analysis of the Doppler spectra with off-line averaging of these curves over several trials resulted in quite different CBFV response characteristics. In our reading paradigm, we recorded three trials, each consisting of 60 seconds resting and 60 seconds reading, which were averaged after the experiment. In 10 subjects, five right-handers and five left-handers, we measured CBFV curves in two recording sessions of the right and left MCAs, respectively.

Figure 1 shows total averages of the right and left middle cerebral artery CBFV curves of all 10 subjects. Amplitude is given in percent deviation from the mean velocity of the rest and reading phases. Consistent with the results presented by Droste et al,1 we found no left/right differences between CBFV curves of either right- or left-handers. The onset of reading was characterized by a steep increase peaking after 4.5 seconds and the beginning of the rest phase by a smaller peak after 3 seconds. In contrast to their curves, after the initial peak in the reading phase, CBFV fell steeply and reached its steady state 8% below the mean velocity about 40 seconds after reading onset. On the other hand, the resting steady state was 6% larger than the mean velocity.

We believe that voice artifacts have influenced the recordings presented by Droste et al,1 shifting the CVFV curve during the reading phase above its real value. At first glance, their results fit with the response one would expect from investigations of regional cerebral blood flow (rCBF) measurement with the xenon-133 inhalation technique,2 showing that cerebral circulation is increased during a verbal task in selected brain areas. On the other hand, Lassen et al3 showed that rCBF values increase in considerably larger parts of the frontal and prefrontal cortex during a rest state and decrease during mental activity. Since most of these cortices are supplied by the MCA, enhanced frontal circulation during the rest phase may lead to a mean increased CBFV as measured from this vessel. Based on this average, smaller parts of the MCA territory related to verbal tasks may only be detected during the abrupt onset of activity. The dynamics and mechanisms of this obvious rapid and transitory shift of cerebral circulation are to be investigated further.

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References

The following is in response:

To the Editor:

We appreciate the opportunity of responding to the comments of Dr. Diehl et al1 regarding our recent report.2 We agree that vocalization does in fact influence transcranial Doppler recordings; however, we believe its effect occurs more by changes of intrathoracic pressure than by conduction of the voice frequency spectrum to the probe. This influence, together with fluctuation of the measured values due to vegetative, respiratory, and cardiovascular factors, accounts for the substantial standard deviation. Voice artifacts caused by direct conduction have a low frequency and merely alter the envelope curve.

Figure 1 in the report by Diehl et al1 gave us the impression that the initial peak during reading approximately 8 seconds after starting the task was consistent with our findings. Those authors also found an initial peak during rest phases.

In the study conducted by Diehl et al,1 mean rest values were higher than mean task values, which agrees with observations in some of our subjects. Lower values while performing a task have also been recorded in individual persons with the xenon-133 technique, but most of these studies, like ours, showed an increase in values in the study population as a whole.

There are two main differences limiting the comparability of our two studies.1,2 The first concerns the reference baseline, that is, the last 9 seconds of the 42-second rest phase in our study and the mean of all the rest and task values in the study of Diehl et al. We are not sure that the last 9 seconds of the 42-second rest phase really represent a rest steady state; longer monitoring under rest conditions has to be done to confirm this hypothesis. Secondly, the rest phase in the report by Diehl et al preceded the task phase, possibly reflecting increased attention, which might be the reason for the increase of MCA blood flow velocity in their rest phases. In our study, the rest phase followed the task phase, and the decrease in values probably reflects relaxation. Furthermore, the difference in blood flow velocity in the middle cerebral artery before and after vocalization could be due to vegetative, respiratory (e.g., arterial Pco2), and cardiovascular factors.

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Stroke. 1990;21:1236-1237
doi: 10.1161/01.STR.21.8.1236.c

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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