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 space) in length, and may be subject to editing or abridgement.

Middle Cerebral Artery Stenosis

To the Editor:

In the recent paper of Corston, Kendall and Marshall on middle cerebral artery stenosis published in the March-April 1984 STROKE, the authors proposed that the prognosis of this condition was not as benign as that previously reported by Hinton et al. 1 In fact, a critical review of the series of Corston et al. does reveal a surprisingly benign clinical course. Of the eleven patients in their series treated with aspirin, aspirin and dipyrimadole, or nothing at all, six suffered no new deficits during follow-up. There were three deaths, two due to cancer, and one from unknown causes, and there were only two strokes. I would have anticipated many more neurologic complications in this group.

Because the paper of Hinton et al. suggests that Warfarin might be an effective treatment for middle cerebral artery stenosis, I was interested in the outcome of the patients in Corston, Kendall and Marshall’s group who were treated with this medication. Only two patients in their series appear to have been treated with Warfarin throughout the entire duration of their follow-up. If my interpretation of their paper is correct, only a single patient, badly impaired with an initial stroke, went on to have two additional strokes on Warfarin therapy. Could the authors tell us if their patient’s Warfarin therapy was managed appropriately and if the strokes were suffered in the hemisphere ipsilateral to the stenosis? It was also interesting to note that of the four patients dying because of an infarct on the side of the middle cerebral stenosis, only one of these patients appears to have been on Warfarin at the time of the fatal stroke.

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References

Editor’s Note: The following letter is the author’s response to Dr. Scott’s comments.

To the Editor:

We would not ourselves subscribe to the view put forward by Dr. Scott that the outcome in our series of middle cerebral artery stenoses was surprisingly benign. Five of the 21 patients had a further stroke. The numbers are clearly too small to permit any meaningful comparison with existing data in the literature on the risk of stroke in general but an incidence of 25% can hardly be called benign.

The patient treated with Warfarin who had two further strokes attended an anticoagulant clinic elsewhere regularly until his death. No problems about control was noted but his records of this prothrombin time test were not available. His second stroke was in the territory of his middle cerebral stenosis; the site of his final fatal stroke is less certain.

I am aware of the pitfalls to be aware of in these patients and should not dissuade cautious immediate anticoagulation in selected patients. Earlier reports cited by the authors, recommend avoidance of anticoagulation in patients with massive deficits and alterations in consciousness such as Case 1 with spontaneous decerebrate posturing. Cases 3 and 4 illustrated the dangers of excessive anticoagulation. Case 5 does not really apply to immediate anticoagulation. The remaining patient, Case 2, although not benefiting from immediate anticoagulation did not appear to be an example of “catastrophic hemorrhage” either, since the focal neurologic deficit described did not worsen after the transient lethargy noted. This case also points out the potential benefits of prophylactic anticoagulation prior to elective cardioversion. Ideally, this patient’s embolic stroke could have been prevented. 3

We have avoided immediate anticoagulation in patients with massive neurological deficits, uncontrolled hypertension and hemorrhagic infarction evident on initial CT. Of 59 patients with acute, nonseptic embolic strokes treated with immediate full dose heparinization, none had recurrent embolism or worsening of neurologic deficit secondary to anticoagulation. 5 We continue to advocate cautious immediate anticoagulation in selected patients with nonseptic cerebral embolism of cardiac origin.

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References

To the Editor:

We write with reference to the recent article by Shields et al concerning anticoagulant-related hemorrhage and acute cerebral embolism (May-June issue pp 426–437). The authors indicate that there have been only a few well documented examples of intracerebral hematoma following administration of anticoagulants to such patients. We would like to report a case which resembles those described by Shields et al and supports their contention that immediate anticoagulation therapy of

Anticoagulation-Related Hemorrhage in Acute Cerebral Embolism

To the Editor:

In their recent article, Shields et al 1 state their data indicate that immediate anticoagulation therapy of nonseptic cerebral embolism carries a "substantial" risk of "catastrophic hemorrhage" into the infarct. The majority of their five cases however, merely illustrate the pitfalls to be aware of in these patients and should not dissuade cautious immediate anticoagulation in selected patients. Earlier reports cited by the authors, recommend avoidance of anticoagulation in patients with massive deficits and alterations in consciousness such as Case 1 with spontaneous decerebrate posturing. Cases 3 and 4 illustrated the dangers of excessive anticoagulation. Case 5 does not really apply to immediate anticoagulation. The remaining patient, Case 2, although not benefiting from immediate anticoagulation did not appear to be an example of "catastrophic hemorrhage" either, since the focal neurologic deficit described did not worsen after the transient lethargy noted. This case also points out the potential benefits of prophylactic anticoagulation prior to elective cardioversion. Ideally, this patient’s embolic stroke could have been prevented. 3

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Middle cerebral artery stenosis.
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Stroke. 1985;16:331
doi: 10.1161/01.STR.16.2.331.a

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/16/2/331.1.citation