recurrence rates are the same in patched and nonpatched arteries (data submitted for publication).

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References

The following is in reply:
To the Editor:
We apologize for the inadvertent omission of the landmark paper by Eikelboom et al1 in the discussion and references of our recent review article on patch grafting in carotid endarterectomy. We were familiar with Dr. Eikelboom’s excellent study as presented at the 41st Annual Meeting of the Society for Vascular Surgery in Toronto, Ontario, Canada, June 9–10, 1987. However, at the time we prepared our manuscript, we were unaware that the paper had appeared in print in February 1988. Appropriate credit must be given to Dr. Eikelboom and his colleagues for a well-designed and executed randomized study on this subject.

We will take this opportunity to comment on their study and its findings. Over a 2-year period, 129 carotid endarterectomies were prospectively randomized into primary and saphenous vein patch closure. In six of these cases, it was necessary to deviate from the randomized procedure, which the authors discussed clearly and included in their analysis. The patients were well-matched for sex, age, risk factors, and lesion characteristics. The study addressed the postoperative course during the first year after surgery and showed a significant decrease in the incidence of restenosis at 1 year in the patched group. Interestingly, the benefits of patch grafting were most evident in women. Recurrent stenosis occurred more frequently when residual lesions were seen on early postoperative digital subtraction angiography (DSA).

These latter observations are interesting but statistically questionable since the study was not actually designed to examine the impact of sex and early lesions on restenosis. In fact, the presence of residual lesions on postoperative DSA already weights this group heavily toward persistent lesions later on. Also, it must be emphasized that the study by Eikelboom et al only examined recurrent stenosis at one year postoperatively. Therefore, their findings that “we found no reason for reoperation in any of these cases” must be qualified as tentative pending further follow-up. Furthermore, as emphasized in the subsequent comments at the end of the paper by Drs. Hertzer and Archie, the issue of early postoperative morbidity (i.e., prevention of acute postoperative thrombosis) and the issue of graft rupture were probably not addressed adequately in the study, in view of the small number of patients and weak statistical power regarding these parameters.

The study of Eikelboom et al adds significant weight to the hypothesis that patch grafting is probably beneficial in carotid endarterectomy. Additional work still needs to be done regarding specific subgroups most likely to benefit from patch grafting and regarding delayed recurrent stenosis and the concerns about graft or suture-line disruption.

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Facial Spasm From Lacunar Infarction of the Thalamic Ventrolateral Nucleus
To the Editor:
In a recent issue of this journal, Ambrosetto et al reported an interesting case of pontine lacunar infarction producing hemifacial spasm.1 Here we report a case of lacunar infarction of the thalamic ventrolateral nucleus producing contralateral hemifacial spasm.

A 53-year-old poorly controlled, diabetic woman suddenly noticed “a floating sensation” of the right leg, soon followed by mild numbness of the right hand. On awakening the next morning, she noticed continuous muscular twitches on the right upper side of the mouth and irregular jerking movements of the right upper extremity, which she was not able to control by herself. The involuntary movements subsided gradually over a 4-day period, but recurred 2 weeks later.

Physical examination at that time revealed an obese woman 146 cm tall and weighing 72 kg. Neurological examination revealed irregular spasm of the right orbicularis oris and myoclonus of the right upper extremity dominantly. These two movements were mostly unsynchronized. In addition, she had mild hypalgesia of the right hand and very mild weakness of the right lower extremity. Computed tomography and magnetic resonance imaging disclosed a small, wedge-shaped infarction centered in the ventrolateral nucleus of the left thalamus (Figure 1).

Electroencephalography was normal. In the following 4 days, the involuntary movements subsided gradually, together with the weakness of the right lower extremity, subsided gradually.

Hemifacial spasm is commonly caused by compression of the root entry zone of the facial nerve.2 In the rare case of pontine lacunar infarction producing hemifacial spasm, Ambrosetto et al1

FIGURE 1. $T_2$-weighted magnetic resonance image of the brain. Note the wedge-shaped lacunar infarction in the ventrolateral nuclear region of the left thalamus.
speculated that it was caused by perifocal edema compressing the intrapontine root of the facial nerve.

As far as we know, hemifacial spasm due to supratentorial lesions has not been reported. In our case, two pathogenetic explanations seem possible: either the lacune itself or the perifocal edema surrounding it might have exerted a stimulative effect on the pyramidal tract fibers running in the adjacent internal capsule, or the lacune could have disturbed the cerebellothalamic cortical pathway running from the contralateral dentate nucleus to the motor cortex via the ventrolateral nucleus, thereby somehow causing the involuntary movements. It should be noted that innervations for the lower half of the face and the upper extremity are somatotopically close in both the internal capsule and the ventrolateral nucleus. Recurrence of the involuntary movements in our case could have been due to the lesion acquiring an epileptogenic nature.

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Facial spasm from lacunar infarction of the thalamic ventrolateral nucleus.
T Toda and K Matsumura

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