Letters To The Editor

Angiogram is Read
As Pseudostenosis

To the Editor:

I would like to comment on the angiographic findings in the article "Resolving Siphon Stenosis Following Carotid Endarterectomy," Stroke 11: 278-281, 1980. The authors indicated that there was resolution of severe stenosis and vascular irregularity in the supraclinoid segment of the internal carotid artery in 2 patients following internal carotid endarterectomy for severe occlusive disease at the origin of the internal carotid arteries. The authors do not specifically indicate or discuss the nature of the carotid siphon narrowings in these 2 patients.

It is my interpretation that the intracranial carotid narrowing in their first patient is a pseudostenosis. In the first patient's angiogram (Figure 1B) there is evidence of considerable dilution of the contrast material at and beyond the point that they indicate is stenotic and irregular. This change appears to me to be located at the carotid bifurcation and in the proximal middle cerebral artery. I therefore interpret the apparent narrowing and irregularity to be due to dilution and streaming of the dye column as it enters the middle cerebral artery where it is being mixed with non-opacified collateral blood flow coming across the anterior communicating artery from the opposite carotid artery circulation. This figure demonstrates dilute faint filling of the middle cerebral artery branches and no filling of the anterior cerebral artery which does fill in an antegrade fashion from this carotid after endarterectomy (Figure 1C).

In the second patient (Figure 2B) besides the narrowing and irregularity in the distal half of the supraclinoid carotid artery as indicated, I note that only the orbital-frontal and anterior supra Sylvian branches of the middle cerebral artery fill. There is no filling of the inferior division of the middle cerebral artery. On the post-endarterectomy angiogram (Figure 2C) the inferior middle cerebral division and its branches to the temporal, posterior frontal, parietal and angular regions are well demonstrated. This combination of findings strongly suggests that there was embolic occlusion of the inferior division of the middle cerebral artery on the pre-endarterectomy angiogram which had lysed before the postoperative study. The supraclinoid carotid irregularity and stenosis in this patient is, therefore, probably related to residual thrombus adherent to the wall from the embolus which initially lodged in the distal carotid artery.

This second case strongly supports the authors' contention that embolic occlusions will usually lyse and the vessel will return to its normal caliber and contour. The first case, however, I believe, represents a pseudo-occlusion due to low perfusion pressure with partial collateral filling of the middle cerebral artery across from the anterior communicating artery. The dilution and streaming of the dye column at the carotid bifurcation and proximal middle cerebral artery resulting from the collateral circulation will produce an appearance of irregularity and narrowing of the vessel wall. The angiographic changes, I believe, support this interpretation.

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The Author Replies:

To the Editor:

I agree with Dr. Goldberg's suggestions that apparent distal carotid siphon stenosis may be due to collapse of this vessel from reduced flow beyond a high grade cervical lesion. This phenomenon has recently been reported by Little, et al, using the term "pseudotandem" stenosis. Our cases, however, differ from those in this report for several reasons: first, flow beyond the level of the sinus stenosis, although high-grade, rapidly reached the carotid siphon, indicating that stagnation within this vessel is not to the degree usually seen in the high-grade cervical carotid stenosis with distal cervical carotid collapse. Secondly, and more importantly, a collapse of the distal carotid siphon secondary to low pressure might allow filling of the middle cerebral territory from other collateral sources, as demonstrated in both of Little's cases. Such cross-filling was not present in our cases, although it was not mentioned in the text. We think it most likely, therefore, that distal reactive spasm secondary to an embolism, probably extending into the proximal middle cerebral artery, was responsible for the stenosis rather than a low pressure phenomenon. Dr. Goldberg's comments emphasize the difficulty in distinguishing pathologic diagnoses on an individual arteriogram, especially when proximal carotid disease is present.

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Reference

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