Clinical and Arteriographic Comparison of Amaurosis Fugax with Hemispheric Transient Ischemic Attacks

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SUMMARY Eighty-seven patients with either amaurosis fugax (40 patients) or hemispheric transient ischemic attacks (47 patients) were studied to determine whether the two symptom groups could be differentiated clinically and arteriographically. Clinical data assessed were age of patient, incidence of cardiac disease, and presence of claudication, hypertension, diabetes, and carotid bruits. Contrary to a prior report, our results indicate no significant difference between the 2 patient groups based on clinical and arteriographic findings.

AMAUROSIS FUGAX and hemispheric transient ischemic attacks are the most common symptoms of cerebrovascular occlusive disease. Both reflect transient ischemic processes secondary to stenoses or embolization from ulcerated plaques of the carotid arteries. Slepyan et al. suggested that the contrasts are significant between patients with amaurosis fugax and those with hemispheric transient ischemic attacks with respect to age, cardiac status, and angiographic findings and that on the basis of these factors patients of one group can be discriminated from the other. Because the number of patients they studied was small, we undertook a review of a larger series to determine if any important clinical as well as angiographic differences exist between these groups.

Materials and Methods

Between January 1, 1972 and December 31, 1976, 121 consecutive patients were evaluated at one of our institutions and given the clinical diagnosis of either amaurosis fugax or hemispheric transient ischemic attacks. Amaurosis fugax was defined as transient monocular blindness; transient ischemic attack was defined as a focal neurologic deficit lasting less than 24 hours, probably attributable to ischemia in the distribution of the carotid arterial system and leaving no residua from the attack. Patients who had both amaurosis fugax and hemispheric transient ischemic attacks or whose symptoms persisted for more than 24 hours were eliminated from the study.

Clinical records and carotid arteriograms were available for 40 of the patients with amaurosis fugax and 47 with hemispheric transient ischemic attacks. The clinical records of each patient were reviewed for information regarding the patient's age, presence of carotid bruit, diabetes, and carotid bruits. Contrary to a prior report, our results indicate no significant difference between the 2 patient groups based on clinical and arteriographic findings.
The pattern of stenotic and ulcerative lesions in the two groups showed no statistically significant difference (table 2).

**Discussion**

There are two major mechanisms for the pathogenesis of both amaurosis fugax and hemispheric transient ischemic attacks. First, emboli originating from the surface of an atherosclerotic ulcerated plaque can cause temporary occlusion of retinal or cerebral vessels. These emboli consist of aggregates of platelets, small bits of thrombi, and degenerative atheromatous material and are rapidly lysed, resulting in the cessation of symptoms. Second, stenosis of the carotid vessels can lead to reduction in critical perfusion. In addition, tight stenoses predispose to formation of mural thrombus, fragments of which may subsequently embolize to the retinal or cerebral vessels.

Since these two mechanisms are similar, any lesion in the carotid circulation might result in embolization or decreased blood flow to either the retinal (amaurosis fugax) or cerebral (hemispheric transient ischemic attack) circulation. Therefore, as we have found, the overall clinical picture is the same in patients with amaurosis fugax and hemispheric transient ischemic attacks. Factors associated with cerebrovascular occlusive disease, such as we investigated (table 1), are equally frequent in both groups. In like manner, auscultatory and arteriographic findings are similar in both groups.

Our results differ markedly from those of Slepyan et al. who found a difference in average age of 5 years between the two groups (8 patients with amaurosis fugax and 23 with transient ischemic attacks). Their patients with amaurosis fugax had no evidence of cardiac disease; 60% of our patients with amaurosis fugax had confirmed myocardial infarction or congestive heart failure. Bruits were noted in only 45% of our patients with amaurosis fugax (rather than the 88% reported by Slepyan et al.) — the same frequency as patients with hemispheric transient ischemic attacks. Rather than the tight stenotic lesions of the carotid artery that uniformly occurred in the patients of Slepyan et al., almost half of the patients in our series had stenoses of less than 50%. Their group of patients with hemispheric transient ischemic attacks did not differ essentially from our group of patients with this disorder.

**References**

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*Stroke*. 1978;9:254-255
doi: 10.1161/01.STR.9.3.254

*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

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