lysed. With a little lower certainty we can also exclude the carotid siphon and carotid bifurcation as sources of recurrent emboli to the same location. The most reasonable interpretation is that the whole process was due to occlusion of one (or a few) lenticulo-striate vessels which the DSA was not sensitive enough to visualize. In the second case, the increased middle cerebral arterial flow velocity might be consistent with stenosis of the artery. However the art of intracranial vascular diagnosis by Doppler ultrasound is in its infancy, and reliable only as a qualitative index of the presence of flow. But this simple fact is the fundamental point of the presentation: That flow through the full length of the horizontal portion of the middle cerebral artery was repeatedly demonstrated during the clinical progression of disability.

What could have been the mechanism of the clinical progression? Of course we cannot know if the postulated lenticulo-striate arterial thrombus propagated or was static during the period of clinical progression. Edema would be a very unattractive hypothesis: The ultimate lesion in each case was so small that it is difficult to envision enough change in local tissue pressure to affect local flow. Moreover both patients remained completely alert during the whole process. No ventricular distortion was evident on the second CT scan in either case. Though a definitive elucidation of the mechanism of progressive lacunar stroke is not yet possible, the negative conclusion is now clear: It is not necessary to postulate occlusion of the parent middle cerebral artery.

**References**


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**Horizontal Gaze Paresis in Hemispheric Stroke**

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**SUMMARY** Of 156 stroke patients prospectively and consecutively evaluated, one-third had a homonymous hemianopia. Of those 52 patients, 46% had a horizontal conjugate gaze paresis at the time of presentation. This gaze paresis was most commonly seen with large hemispheric stroke. The overall prognosis in patients with a gaze paresis was poor. The 30-day case fatality rate was 49% which was significantly higher than for stroke patients presenting with homonymous hemianopia without a gaze paresis.

We prospectively and consecutively assessed 156 patients with completed stroke admitted to Temple University Hospital over an eight month period. All patients were personally examined by the authors within 24 hours of presentation and computed tomographic (CT) brain scan was performed in all patients with a followup scan in one-third. Each patient had an assessment of visual acuity and visual fields when neurological status allowed this. Confrontational visual field techniques included finger counting and color comparison with each eye tested separately. Response to threat in each eye was utilized in obtunded patients. We also assessed pupillary size, pupillary response to light and accomodation, direction of gaze, extraocular motility, and optokinetic response. Evaluation of oculocephalic reflex and cold caloric response was performed when appropriate.

Patients were followed an average of 76 days for survivors and 23 days for nonsurvivors. Serial medical...
and neurological assessment was performed over this time period. The size of the stroke was classified as small, moderate or large based upon CT scan findings and neurological examination and corresponded with previously published criteria. Small strokes included those associated with lesions of 1 cm or less in size by CT scan. These correlated with minor motor or sensory deficit or with isolated speech deficit such as a Wernicke’s aphasia. Moderate size strokes were those associated with lesions greater than 1 cm and up to 3 cm in size by CT scan and in which moderate neurological impairment was observed clinically. These lesions were not accompanied by significant obtundation of consciousness. Large strokes consisted of lesions greater than 3 cm in size by CT scan. These were typically associated with significant disturbance of higher cortical function including at least some degree of obtundation.

Results

Fifty-two patients were found to have a HH. Of these, 24 (46%) had a conjugate horizontal eye deviation toward the involved cerebral hemisphere. This gaze palsy was on a supranuclear basis with intact intraocular motility by pursuit or by oculocephalic reflex testing. Three additional patients had conjugate horizontal eye deviation secondary to a hemispheric lesion but depressed level of consciousness did not allow visual field assessment and at least one of these patients had adverse seizure activity related to the eye deviation. In addition, there were two patients with a homonymous superior quadrantanopia secondary to a small calcareous cortex infarction.

Table 1 summarizes the hemispheric lesion responsible for the HH in patients with and without a gaze paresis. Ischemic infarction was responsible for HH in 89% of patients without gaze paresis and in 67% of patients with gaze paresis. Patients with middle cerebral artery involvement most often had a frontoparietal infarction which extended to involve the deeper subcortical region by CT scan. No patient with an isolated frontal lobe infarction in the anterior cerebral artery distribution (four patients) was observed to have a gaze paresis or a homonymous visual field defect. One patient did have an asymmetric optokinetic nystagmus response. Furthermore, no patient with an isolated ocipital lobe infarction in the posterior cerebral artery distribution (4 patients) was observed to have a gaze paresis associated with their homonymous field defect.

Of the 24 patients with horizontal gaze paresis, 16 stabilized to the degree that they could be serially assessed for resolution of gaze paresis. Three had resolution within 1 to 2 days, five within 3 to 5 days, five within 6 to 8 days and the remaining three had resolution within 2 weeks.

The clinical features of patients with HH in our series are summarized in table 2. The mean age, incidence of previous stroke, presence of significant cardiac disease and length of followup was quite similar in the two groups. Patients with a gaze paresis had a 3-fold higher incidence of large stroke. As would be expected, their prognosis was considerably worse. The 30-day case fatality rate was 15% for patients with HH without gaze preference and 49% for patients with gaze preference (χ² = 11.8, p < 0.001). Exclusion of patients with intracerebral hemorrhage resulted in a 30-day case fatality rate of 23% for ischemic infarct patients without gaze paresis versus 50% for those with gaze paresis (χ² = 8.1, p < 0.01). The cause of death in our series of patients was confirmed by autopsy in 2 of the 19 who died and was based on clinical features in the others. Death was attributed to brain herniation in 1 patient without gaze paresis and in 5 patients with gaze paresis. This is undoubtedly related to the higher incidence of large stroke in the latter group. The remaining patients died of cardiopulmonary and infectious processes which were presumably related to prolonged immobility. Functional status at the completion of followup was better in patients without a gaze paresis (table 3).

Discussion

Gaze paresis secondary to hemispheric stroke has been associated with obtundation but not with an adverse effect on outcome. The horizontal gaze paresis associated with cerebrovascular disease is usually at-
Contributed to a lesion of the FEF which corresponds to the prearcuate gyrus, Brodmann area 8, of the frontal lobe. This area is responsible for the generation of horizontal saccadic eye movements. The fibers originating in the FEF project caudally in the anterior limb of the internal capsule. These fibers reach the premotor areas of the brainstem by three different pathways: a dorsal transthalamic pathway, a ventral pedunculotegmental pathway and an intermediate pathway. The superior colliculus also contains neurons which discharge in relation to saccadic eye movements and may be an integral part of this pathway.  

Selective removal of the frontal or occipital cortex of the monkey had little effect on spontaneous eye movement in one study. It appears that more massive damage is required to produce contralateral horizontal gaze palsy. Furthermore, the deeper the subcortical lesions are, the greater the degree and duration of the gaze paresis. Very restricted lesions within the reticular formation of the midbrain can result in a severe longer lasting contralateral gaze palsy. Thus, it is not surprising to find a relationship between the extent of a hemispheric stroke and the presence of a horizontal gaze paresis. A lesion may either involve the FEF, the projected pathways of the FEF, or cause pressure upon the midbrain tegmentum to result in a contralateral gaze palsy.

Our results support the concept that horizontal gaze paresis, on a hemispheric basis, is usually associated with massive stroke involving the deeper subcortical structures. This appears to be especially true if there is a concomitant HH. Dr. C. Miller Fisher’s neuro-ophthalmological observations in large supratentorial hemorrhage are in agreement with this. Of note, he reports that a conjugate, horizontal eye deviation away from the lesion can occur of there is significant distension of the third ventricle by blood. Thus, involvement of the FEF projection pathways, as they pass in the deep subcortical region, appears to be the most common mechanism of horizontal gaze paresis in hemispheric stroke.

### Acknowledgment
The authors wish to thank Dr. Jonathan Trobe for his critical review of this manuscript.

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Stroke. 1986;17:1030-1032
doi: 10.1161/01.STR.17.5.1030

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http://stroke.ahajournals.org/content/17/5/1030