Table 4 Relationship between Mental Status Measures and Outcome Criteria

Mental status measures	Outcome			
	Average functional status at discharge		Discharge destination	
	Tau	р	Tau	р
Orientation	.35**	.001	.23**	.001
Other cognitive measures	.48**	.001	.26**	.001
Aphasia	25 <b>**</b>	.006	.03	.360

<sup>\*\*</sup>Significant at the .01 level.

presence of aphasia is negatively correlated to functional ability but bears no relation to discharge destination. From these data it is apparent that a series of simple mental status tests are better predictors of functional status at discharge than are reflexes. While the setting to which a patient is discharged is probably roughly associated with his or her physical and mental condition, other factors such as family support also influence placement.

#### Discussion

Stroke patients make up the largest category of patients seen in rehabilitation hospitals, comprising 31% of all discharges in a recent study involving 20 hospitals.10 A major consideration in the selection of such patients for treatment directed at regaining lost functions or learning new activities is the extent to which these individuals are able to profit from treatment. Traditionally, the neurological examination has been used to establish lesion site and diagnosis, and to associate examination findings with function. In the present study the attempt was made to relate neurological abnormality to future functional expectations. More specifically, an inquiry was directed at the relationship of developmental reflexes at admission to performance of selfcare and mobility tasks after a course of rehabilitation

Several studies have found developmental reflexes in older populations. While some of these groups exhibited severe cerebral pathology, others were composed of individuals without other evidence of neurological abnormality. The present investigation demonstrates that such reflexes are common in those stroke patients who are selected for inpatient rehabilitation. The presence of developmental reflexes was found to be negatively related to favorable treatment outcome. The strength of the relationship was too small to be useful in the selection of patients or the prediction of outcome. A research design which does more than examine the presence of reflexes at one time might better establish their predictive nature in relation to directed motor activity.

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### **Disappearing Carotid Defects**

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SUMMARY Large intraluminal cervical carotid artery filling defects consistent with mural thrombi were angiographically demonstrated during acute hemispheric neurologic episodes. These thrombi disappeared benignly as shown by serial angiography in 2 patients treated with intravenous heparin and spontaneously in 1 patient

treated surgically. Thus, partially obstructing cervical carotid artery thrombi may lyse either with the use of anticoagulant therapy or else spontaneously.

The etiology of the thrombi may partly be related to underlying atheromatous disease.

CAROTID ARTERY DISEASE is a common cause of cerebral dysfunction. Pathophysiologically, carotid stenosis, occlusion, ulceration, dissection, inflammation, thrombus and trauma have all been implicated.1-4

A large intraluminal carotid artery filling defect shown by

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arteriography suggests a thrombus.<sup>5</sup> The imminent danger of such a clot is either distal embolization or progression to occlusion. The neurologic symptoms and signs may be related to either regional flow insufficiency or embolization or both. Discovery of an intraluminal lesion requires immediate decisions about treatment.

We have recently treated 3 patients with large intraluminal carotid defects and probable distal embolization. Two were treated with anticoagulation and the third had surgical exploration. In all 3 cases the presumed carotid clot disappeared. The findings and course in these patients help to define the natural history of some of the carotid intraluminal lesions.

#### Reports

#### Patient No. 1

A 46-year-old Caucasian, right-handed man with chronic obstructive pulmonary disease, bronchiectasis, mild hypertension, angina, and gout developed an acute right temporal headache, speech difficulty and visual blurring. After an hour, he noted left body weakness. He specifically denied neck trauma.

Examination revealed a moderate left hemiparesis, hemisensory loss and homonymous hemianopsia. Over the first 2 hospital days his deficits worsened.

Laboratory examination showed normal spinal fluid; non-contributory cardiovascular evaluation included electro- and echocardiography and there was negative collagen vascular disease evaluation. Clotting studies revealed a decreased activated partial thromboplastin time with normal prothrombin times and platelet counts. Without obvious cause he developed anemia with his hematocrit falling from 35 to 28 but this responded to folic acid.

Beginning on his fifth hospital day, he was treated with oral sulfinpyrazone and intravenous heparin until day 18. He also received steroids (dexamethasone).

Serial radionuclide studies revealed persistent decreased right-sided perfusion with an evolving right parieto-occipital static uptake.

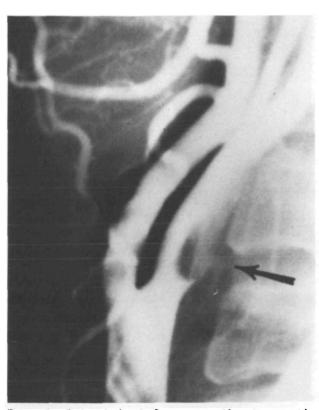


FIGURE 1. Patient 1, day 4: Common carotid angiogram with arrow demonstrating the large intraluminal filling defect in the cervical internal carotid artery.

Retrograde right brachial angiography on day 1 showed a right middle cerebral artery posterior trunk occlusion by an intraluminal filling defect. The extracranial vasculature was not studied. On day 4 a transfemoral right carotid angiogram showed a large smooth sausage-shaped intraluminal mass in the proximal internal carotid artery (fig. 1). The right middle cerebral artery was occluded more proximally than on day 1 and there was suprasylvian mass effect. Repeat transfemoral right carotid angiogram on day 19 no longer showed an intraluminal filling defect. There was only a small smooth plaque in the proximal internal carotid artery (fig. 2).

Seventeen months after his initial infarction, he had evidence of further right hemisphere injury. He developed a severe right temporal headache followed by left visual field hallucinations. Radionuclide images show a new area of right posterior parietal infarction. He complained of increased spasticity. Laboratory evaluation again failed to demonstrate a cardiac, hematologic or collagen vascular etiology. A computerized tomographic (CT) scan showed extensive right parietal infarction. Angiography showed slight increase in the size of the right internal carotid plaque plus persistence of the paucity of the right posterior parietal circulation.

He has continued to have intermittent seizures despite anticonvulsants. He has a dense, spastic, left hemiplegia, sensory loss and homonymous hemianopsia.

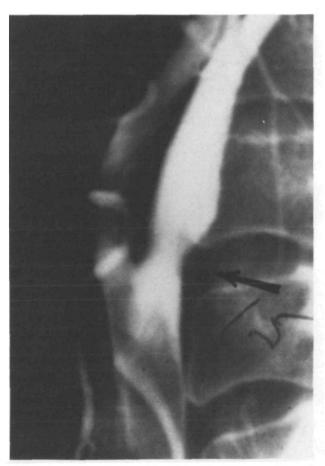


FIGURE 2. Patient 1, day 19: Slight atheromata at base of formerly present thrombus.

#### Patient No. 2

A 49-year-old right-handed Caucasian housewife developed nausea, vomiting and mild headache. She awoke early the next morning with a left arm paresis, rightward gaze preference and headache. For the preceding months, she had had episodes of transient blurring of vision. She specifically denied neck trauma.

She had been taking birth control pills 3 years because of menometrorrhagia. She also had been taking 1 grain of thyroid extract daily, chronically.

Her initially flaccid, paretic left arm evolved within a day to a spastic paresis. Her gaze preference resolved after admission, but she still maintained left-sided inattention to visual and tactile stimuli.

Pertinent laboratory findings included: a hypochronic, microcytic, anemia with a hematocrit of 30, a 608,000 platelet count and a low antithrombin III level. Initial, normal or noncontributory findings included: chest x-ray, echoand electrocardiograms, blood chemistry panels, spinal fluid, prothrombin and activated partial thromboplastin times.

Radionuclide images showed initial symmetrical perfusion later evolving into a right-sided decrease, and an evolving right fronto-parietal static uptake. CT scans evolved from normal on day 1 to a right posterio-frontal infarction picture. Transfemoral right carotid angiography on day 2 showed a large, irregular, intraluminal mass attached to the bifurcation of the common carotid, (fig. 3). There was also



FIGURE 3. Patient 2, day 2: Common carotid angiogram demonstrating intraluminal thrombus (arrow) at the common carotid bifurcation, subtraction view.

occlusion of some middle and anterior cerebral artery branches with retrograde flow into the angular branch of the middle cerebral artery. Repeat angiography at 5 weeks showed only small, smooth indentations of the right internal and external carotids at the bifurcation (fig. 4). The intracranial vasculature was normal.

Birth control medication was discontinued on admission and she was treated initially with sulfinpyrazone. On day 3, sulfinpyrazone was stopped and she was started on intravenous heparin. Over the subsequent 2 weeks, she was gradually switched to oral anticoagulation with warfarin.

At 5 weeks, anticoagulation was discontinued and her antithrombin III level had become normal. She continued with severe left hand weakness and inattention to stimuli on her left side. There was continued improvement over the next 9 months with only mild residual left hand clumsiness.

#### Patient No. 3

A 66-year-old Caucasian woman had recurrent bouts of left hand weakness and numbness. Her symptoms had begun 2 years previously with a severe episode lasting 10 days. Subsequent brief episodes occurred 1 year, 3 weeks and immediately before admission.

Examination revealed a slightly weak left hand with diminished sensation to pin and touch. There was a harsh right carotid bruit. Preliminary laboratory tests were normal and included: CBC, biochemical survey, coagulation panel, ECG, EEG and CT scan.

A transfemoral right carotid arteriogram showed an estimated 95% stenosis of the proximal internal carotid artery



FIGURE 4. Patient 2, 5 weeks: Small, smooth, indentation at carotid artery bifurcation, site of former thrombus.

with a large, poststenotic, intraluminal filling defect (fig. 5). The intracranial vessels were normal.

Within 5 hours she underwent right carotid artery exploration. There was no palpable thrill. Great care was exercised to avoid possible embolization of loose clot or atheroma. After systemic heparinization, the mean internal carotid artery stump pressure was measured (110 mm Hg) and an arteriotomy performed without the use of a temporary shunt. There was a thick atheromatous plaque involving the carotid bifurcation. The proximal one cm of internal carotid artery contained a 95% stenosis and a 1 mm ulcer on its posterior wall. No evidence of intraluminal thrombus was found at or above the atheroma. Neither back flushing nor Fogarty catheter exploration of the cervical portion of the internal carotid artery produced any thrombus. A standard endarterectomy was performed.

Postoperatively the patient made an uneventful recovery. Her left hand became stronger; and she and her family reported that her cognitive function improved.

#### Discussion

An intraluminal filling defect seen on angiography fulfills the definition of a thrombus if it is at least partly surrounded by contrast material.<sup>5</sup> This was the finding in patients #2 and #3. In patient #1, the defect was demonstrated as bulging into the lumen. The differential diagnosis of that defect was

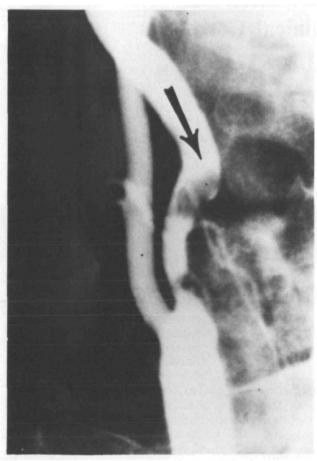


FIGURE 5. Patient 3: Carotid angiogram demonstrating intraluminal thrombus (arrow) just distal to internal carotid artery origin high grade stenosis.

a mural lesion or an intraluminal clot. The absence of a history of trauma, the angiographic finding of probable distal embolization and the eventual course, and subsequent disappearance of the filling defect led to a diagnosis of intraluminal thrombus in this case also.

The natural history of large, intraluminal, carotid clots is still unclear. Possibilities include progressive thrombosis, distal embolization of fragments or the entire clot, stabilization or disappearance. In each of our patients the clot disappeared, 2 with anticoagulation therapy and the third spontaneously. Distal embolization remains a remote possibility but this was not shown in the follow up angiography in patients #1 and #2 so studied; nor was there any clinical suggestion of embolization after the carotid clot was discovered and treated in any of the patients.

Although in patients #1 and #2 the carotid clot was no longer demonstrated on follow up angiography, there were small, smooth plagues at the base of the former filling defects. In patient #1 this plaque had further increased in thickness when he had arteriography at 17 months following another right cerebral infarction. This suggests that a banalappearing atheroma may, in combination with as yet undefined other factors, serve as a nidus for the formation of a large superimposed thrombus. Patient #2 had the known thrombogenic stimulus associated with birth control medication4, 6 and thrombocytosis. Both patients #1 and #2 had evidence on their earliest angiograms of intracranial embolization with branch filling defects as described by Ring.7 These defects presumably originate from or are simultaneous with their carotid lesions. Patient #3 had her carotid clot in association with an atheromatous carotid stenosis. All these findings suggest some relationship between atheromata and the thrombus, a previously suggested association.5

Intravenous heparin infusion was the major therapy in patients #1 and #2. The anticoagulation was started on day 5 in patient #1 and on day 3, in patient #2 following the method described by Genton,8 and using the activated partial thromboplastin time (PTT) to monitor the therapy. We aimed for a PTT of once and one-half to twice control.

Patient #3 underwent carotid endarterectomy just 5 hours after demonstration of her carotid filling defect near the site of her ulcerated and highly stenotic atheromatous plaque. Despite a careful and vigorous search at surgery no intraluminal clot was detected. It was feared that intraoperative distal embolization had occurred. However, the patient had no change in her neurologic status. The failure to find an angiographically well seen carotid thrombus at subsequent carotid surgery was noted in 1 of 8 operated patients described by Roberson, Scott and Rosenbaum,<sup>5</sup> but not explained. Siebert and Swanson<sup>9</sup> have also described such an occurrence and pointed out the similarity to radiologically defined disappearing pulmonary artery thrombi.

Internal carotid artery occlusion with early spontaneous restoration of flow has been angiographically documented.<sup>10, 11</sup> Thromboembolus migration, fragmentation or complete lysis have been offered as possible explanations. Many of these cases have shown sanguinous spinal fluid as indication of hemorrhagic infarction.<sup>11-13</sup> Hemorrhagic infarction has also been a complication of the early surgical embolectomy on patients with infarction.<sup>3</sup> Fortunately, our patients did not have this complication.

The incompleteness of the carotid occlusion may have helped avoid this. Other factors favorable to their outcome may have been the absence of bleeding as evidenced by spinal fluid and CT examinations. Also, the patient treated surgically had only mild and transient neurologic signs.

The carotid filling defects were symptomatic in all of our patients; i.e., completed embolic infarctions in patients #1 and #2 and reversing neurologic events in patient #3. Unlike other reports where persistent mural clots were found at surgery or post mortem, 1. 2. 8 our 3 vanished. This disappearance was temporally linked to neurologic stabilization or improvement without any further embolic phenomena angiographically or clinically. Both heparin anticoagulation and carotid surgery have been associated with successful outcome. The etiology of these carotid thrombi may be related in part to underlying atheromatous disease. A search for all factors associated with thromboembolic disease will help to clarify the need for eventual carotid endarterectomy.

#### Acknowledgment

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# Leptomeningeal Artery Atherosclerosis Visualized by Angiography: Clinical Correlates

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SUMMARY Circumscribed atherosclerotic involvement of secondary and tertiary branches of major cerebral arteries is a common angiographic finding whose nature is rarely in question. However, widespread and severe changes are unusual, and radiologic interpretation may be more difficult. We recently cared for a patient whose angiogram demonstrated extensive involvement of leptomeningeal vessels and were prompted to review the clinical courses

and autopsy findings of a number of other patients with similar angiographic findings. Our observations suggest that the radiological appearance of leptomeningeal artery atherosclerosis can be confused with that of an arteritis. Atherosclerosis of leptomeningeal arteries is strongly associated with a history of arterial hypertension and seems to parallel arterial lesions thought responsible for lacunar infarction and intraparenchymal hemorrhage.

ATHEROSCLEROTIC INVOLVEMENT of the secondary and tertiary branches of the major cerebral arteries is common.<sup>1, 2</sup> That more extensive involvement may cause small vessel angiographic arteriopathy similar to that produced by various inflammatory diseases is, however, poorly documented in the literature.<sup>2, 3</sup> In contrast to atherosclerosis of the cervical vessels or circle of Willis, and in contrast to the intraparenchymal arteriolar sclerosis not seen angiographically, leptomeningeal artery atherosclerosis visualized at angiography has incited few efforts to describe clinical correlates.<sup>4, 5</sup> We recently cared for a middle-aged patient whose clinical course and angiographic findings

were, we thought, compatible with inflammatory arteriopathy. Postmortem studies, however, disclosed the presence of marked atherosclerotic involvement of the smaller surface arteries, while the larger vessels were spared. The case emphasizes the necessity, we believe, for histologic confirmation prior to initiating anti-inflammatory therapy for cerebral "vasculitis" suspected on angiographic and clinical grounds. The case, furthermore, stimulated an examination of the literature and of our previous clinical experience with angiographically demonstrable small-vessel atherosclerosis with the purpose of defining characteristic clinical and pathological correlates of the radiologic entity. Our findings suggest that such leptomeningeal artery disease occurs most commonly in hypertensive patients and perhaps in those with diabetes mellitus. It appears that the pathogenetic mechanisms responsible for the production of leptomeningeal artery atherosclerosis may be more akin to

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