Ataxic Hemiparesis
Critical Appraisal of a Lacunar Syndrome

Mark J. Gorman, MD; Rima Dafer, MD; Steven R. Levine, MD

Background and Purpose—Ataxic hemiparesis is a well-recognized lacunar syndrome involving homolateral ataxia with accompanying corticospinal tract impairment. Despite 30 years of clinical experience there continues to be some doubt as to the defining clinical characteristics, precise neuroanatomic localization of the syndrome, and etiologic mechanisms.

Methods—We now present 45 new cases that have been analyzed for clinico-radiologic correlation and etiology. Also, all published cases from the English literature known to the authors are reviewed.

Results—We found that the clinical syndrome of ataxic hemiparesis accurately predicts a small deep infarction, generally in the pons or internal capsule. Sensory loss is highly associated with a capsular localization. We found that 47% of the cases were attributed to small-vessel disease, 11% to cardioembolism, and only 7% to artery-to-artery embolism (all in the basilar artery); 1 case was attributed to thrombocytosis, 1 to multiple sclerosis, and the rest either had negative or incomplete evaluation. Approximately two thirds of the infarctions occurred in patients with neuroimaging evidence of other ischemic brain lesions.

Conclusions—Ataxic hemiparesis is a distinct clinical syndrome that accurately predicts a small deep infarction, most commonly in the pons or internal capsule. Only sensory loss accurately predicts a capsular localization. Etiology in nearly half of the cases can be attributed to small-vessel disease. Furthermore, ataxic hemiparesis appears to be a good marker for generalized asymptomatic cerebrovascular disease. (Stroke. 1998;29:2549-2555.)

Key Words: ataxia ■ cerebrovascular disorders ■ lacunar infarction

Lacunar syndromes are a commonplace and well-accepted part of clinical stroke teaching. That “lacune” is defined as a small deep cerebral infarct is a synthesized reflection of the early literature in conjunction with the definitive articles published by Fisher and associates.1–6 In these articles the groundwork was laid for a group of recognizable clinical syndromes associated with small infarcts in the distribution of the deep penetrating cerebral arteries. Perhaps the most controversial of these syndromes, ataxic hemiparesis (AH) has generated considerable interest concerning its anatomic specificity, spectrum of associated clinical characteristics, and etiologic mechanisms. We present data from 45 new cases of AH from our institution and review all known previously published cases (in the English literature) to help clarify the range of etiologies, to further refine the diagnostic evaluation of these patients, and to determine whether specific clinical findings can predict the precise location of the lesion.

Subjects and Methods

Inclusion Criteria

We reviewed the discharge summaries of 1468 consecutive patients admitted to our institution or seen in the outpatient neurology clinic with the primary diagnosis of stroke between January 1989 and March 1995. Data from 1989 were obtained from the Henry Ford Hospital stroke database.7 From 1990 to 1995, the charts of all stroke admissions seen by the neurology service were systematically reviewed.

We found 110 patients with recent onset ataxia and pyramidal weakness. All patients were examined within 24 hours of onset of symptoms. Of these 110 patients, 65 did not fit the study criteria defined as (1) new onset ipsilateral ataxia and pyramidal signs, (2) dysmetria out of proportion to the weakness, (3) absent or minimal cortical signs, and (4) all signs documented by a neurologist. Forty-five patients fulfilled all inclusion criteria and comprise our study cohort.

Neuroimaging

CT brain scans were performed on GE 8800 or 9800 models. MRI on a GE Horizon 1.5-Tesla system. Carotid duplex Doppler ultrasonography was performed on Acuson 128 XT. Transcranial Doppler ultrasonography (TCD) studies were performed on a Medasonics model #VSC21.

Definitions of Stroke Risk Factors

Risk factors for stroke were identified, and the etiology was determined after reviewing all the diagnostic investigations. Stroke risk factors were defined as (1) hypertension—a systolic blood pressure of >140 mm Hg and diastolic blood pressure of >90 mm Hg on 2 different readings prior to the stroke or evidence

Received June 12, 1998; final revision received August 12, 1998; accepted August 31, 1998.

From the Department of Neurology, Wayne State University School of Medicine, Detroit, Mich (M.J.G., S.R.L.); and the Division of Neurology, Medical College of Ohio, Toledo, Ohio (R.D.).

Address correspondence to Dr. Mark J. Gorman, Department of Neurology, Wayne State University School of Medicine, 4201 St. Antoine 6E UHC, Detroit, MI 48201. E-mail mgorman@moose.med.wayne.edu

© 1998 American Heart Association, Inc.

Stroke is available at http://www.strokeaha.org

2549
of left ventricular hypertrophy (LVH) on echocardiography (without evidence for aortic stenosis), (2) diabetes mellitus with 2 random blood sugars of >140 mg/dL or elevated glycosylated hemoglobin, (3) cigarette smoking (>10 cigarettes/d), and (4) hypercholesterolemia with a cholesterol level of >200 mg/dL.

Etiologic Classification

The etiology of ischemic infarction was considered cardioembolic when the patient had a high-risk potential cardiacogenic source (atrial fibrillation, mural thrombus, valvular vegetation, prosthetic valve, severe hypokinesis or akinetic segment, or acute myocardial infarction) or a lower-risk potential source (patent foramen ovale) without other potential sources. Etiology was deemed artery-to-artery embolus when there was unequivocal evidence of large cerebral vessel (extra- or intracranial) disease (>49% stenosis) with infarct distal to the arterial stenosis.

If 2 or more potential causes were found, cause was listed as mixed. Patients with a nondiagnostic evaluation and 2 or more stroke risk factors were put in the category of presumed small perforating vessel disease with local arteriolar occlusion. Laboratory abnormalities presumed to have caused or contributed to the stroke were listed separately. In patients with negative, incomplete, or inadequate diagnostic evaluation, the etiology was considered unknown. We then summarized the different clinical findings and correlated them with the lesions on the neuroradiological imaging.

Results

Demographics and Prior History

The mean age of the 45 patients was 69 years (±12.4 years [SD]; range, 33 to 89 years). Thirty-one (69%) patients were men, and 14 (31%) were women (gender ratio, 2.2:1). Thirty (67%) patients were black and 15 were white (a ratio of 2:1, which reflects the population seen overall by our stroke service).

Thirty-seven (82%) of our patients had hypertension, 27 (60%) were diabetic. Twenty-five (56%) had both diabetes and hypertension, whereas 6 (13%) had neither. Elevated serum cholesterol levels were detected in 23 (51%). Thirty-five (78%) had a history of cigarette smoking, 24 (53%) of whom were still smoking at the time of the stroke. Two (4%) patients had a history of heavy alcohol intake (>3 drinks/d).

Other pertinent medical conditions included the following: a history of myocardial infarction (n=5), prior stroke (n=5), peripheral vascular occlusive disease (n=3), prior transient ischemic attack (n=2), Parkinson’s disease (n=2), epilepsy (n=2), sarcoidosis (n=1), treated syphilis (n=1), “crack” cocaine use (n=1), and anticardiolipin antibody syndrome (n=1).

History and Examination Findings

The temporal onset of symptoms varied from acute (minutes) in 14 (31%) to subacute (hours and days) in 11 (24%) and 5 (11%), respectively; 15 (33%) awoke with the symptoms. Although a neurologist examined all patients, not all the findings were recorded for each patient; therefore we list only those findings recorded, either present or absent, and in tables denoted as “(n) number of patients tested.”

Many relevant clinical findings are summarized in Table 1. Dysarthria was mild in 17 patients, moderate in 4, and severe in 2. Regarding strength, the upper extremity was weak and ataxic in all but 1 patient, whereas the lower extremity was predominantly weaker in 7 patients. Regarding sensation, 1 patient showed extinction to double simultaneous somatosensory stimulation, 1 had a contralateral sensory deficit, and 4 had bilateral deficits. Sensory examination was unreliable in 1 patient. Dysmetria was more prominent in the upper extremity in 18 of 45 (40%) patients, in the lower extremity in 1 of 45 (2%), and equally present in the remainder. Gait was mildly ataxic in 28 patients, severely ataxic in 7, normal in 4, and not tested in 6. Deep tendon reflexes were brisker ipsilaterally in 13 patients, ipsilaterally depressed in 11, symmetrical in 20, and absent in 1. Ipsilateral plantar reflexes were extensor in 19, flexor in 13, equivocal in 7, bilaterally extensor in 4, and withdrawal in 1 patient.

Laboratory Evaluation

Laboratory abnormalities included the following (the denominator indicates number tested): anemia 12/45 (27%), thrombocytosis 1/45 (2%), positive VDRL and fluorotroponemal antibody 4/39 (10%), elevated angiotensin-converting enzyme level 1/1, antinuclear antibody 3/21 (14%), and antiphospholipid antibodies IgM 3/36 (8%) positive, IgA 1/36 (3%) positive, and 3/36 (8%) indeterminate; and IgG 3/36 (8%) positive, and 4/36 (11%) indeterminate.

Neurovascular Imaging and Cardiac Evaluation

Neurovascular imaging and cardiac evaluation are summarized in Table 2. CT brain scan was performed on all patients.

### Table 1. Clinical Findings and Associated Anatomic Locations of Infarcts

<table>
<thead>
<tr>
<th>T/C</th>
<th>Pons</th>
<th>IC/BG</th>
<th>Multiple</th>
<th>Others</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysarthria (40)</td>
<td>5 [20%]</td>
<td>10 [40%]</td>
<td>2 [8%]</td>
<td>8 [32%]</td>
<td>0</td>
</tr>
<tr>
<td>Ipsilateral facial weakness* (45)</td>
<td>7 [22%]</td>
<td>10 [31%]</td>
<td>4 [13%]</td>
<td>0</td>
<td>11 [34%]</td>
</tr>
<tr>
<td>Ipsilateral trigeminal sensory loss† (42)</td>
<td>3 [60%]</td>
<td>1 [20%]</td>
<td>0</td>
<td>0</td>
<td>1 [20%]</td>
</tr>
<tr>
<td>Ipsilateral sensory deficit (45)</td>
<td>8 [89%]</td>
<td>1 [11%]</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Severe gait ataxia (39)</td>
<td>0</td>
<td>2 [29%]</td>
<td>1 [14%]</td>
<td>0</td>
<td>4 [57%]</td>
</tr>
</tbody>
</table>

T/C indicates thalamocapsular; IC/BG, internal capsule/basal ganglia; [%], percentage of total with that finding; {%}, percentage with that finding of total tested; *contralateral facial weakness was noted in 9/45 [20%]; †contralateral facial sensory loss was noted in 2 of 42 [5%] patients. Laterality noted is in relation to the ataxia and hemiparesis; (n)=number of patients tested.
and revealed abnormalities in 20 (44%). MRI revealed abnormalities in 24 of 30 (80%). Both CT and MRI revealed abnormalities in 10 of 30 (33%) patients, with both studies normal in 4 (13%). CT was abnormal in 2 patients with internal capsular lesions who had normal MRIs. MRI was abnormal in 13 of 30 (43%) patients who had normal CT scans, 8 located in the pons and 5 in the internal capsular region. No single lesion consistent with the clinical picture could be identified in 15 patients, 9 of whom had MRI. Asymptomatic ischemic lesions were identified in 28 of 47 (60%) patients.

**Lesion Location**
The distribution of lesion locations is summarized in Table 3. A single lesion was identified as the probable cause of the syndrome in 30 (67%) patients; in 16 of these patients the identified lesion was accompanied by other small deep infarcts, only 5 of which could have reasonably led to some of the signs (the latter infarcts were excluded on the basis of the age of the lesions, prior scans were used when available for comparison). In 3 patients more than 1 potential lesion was visualized, and a clear decision as to the causative lesion could not be made. In 2 patients a lesion could not be identified (neither had MRI), in 8 patients several lesions were seen but could not be correlated with the clinical examination, and in 2 patients it was felt that 2 separate lesions together appeared to result in the AH.

**Etiologic Mechanisms**
Forty-three patients had ischemic stroke, 1 had a hemorrhage, and another had demyelinating lesions. A breakdown of the ischemic causes is shown in Table 4. The most common presumed cause was small vessel occlusive disease based on an otherwise nondiagnostic evaluation and 2 or more risk factors for cerebral arterial disease.

**Treatment and Outcome**
The majority of our patients (n=32) were treated with antiplatelet agents. Of the 26 patients in whom follow-up information was available, 7 (27%) had complete recovery, and 13 (50%) had only mild residual abnormalities. Two (8%) were the same or only minimally improved; 3 (12%) patients developed recurrent stroke, 1 (4%) of whom died; and 1 developed poststroke epilepsy. If improvement occurred, it was generally rapid, often within days or weeks.

**Discussion**

**Clinical Features**
The clinical syndrome of AH, first defined by Fisher and Cole and later modified by Fisher, involves cerebellar-type

---

**TABLE 2. Diagnostic Information**

<table>
<thead>
<tr>
<th>Source Identified</th>
<th>Abnormal/Not Relevant to Infarct Location</th>
<th>Nondiagnostic/Inadequate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>24 [63%]</td>
<td>0</td>
</tr>
<tr>
<td>Abnormal/Potential</td>
<td>9 [41%]</td>
<td>7 [32%]a</td>
</tr>
<tr>
<td></td>
<td>8 [40%]</td>
<td>1 [5%]b</td>
</tr>
<tr>
<td>Conventional angiography (9)</td>
<td>3 [33%]</td>
<td>3 [33%]c</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source Identified</th>
<th>Abnormal/Potential</th>
<th>Normal/Abnormal/Potential</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid duplex Doppler (38)</td>
<td>24 [63%]</td>
<td>0</td>
</tr>
<tr>
<td>Transcranial Doppler (22)</td>
<td>9 [41%]</td>
<td>7 [32%]</td>
</tr>
<tr>
<td>Magnetic resonance angiography* (20)</td>
<td>8 [40%]</td>
<td>1 [5%]</td>
</tr>
<tr>
<td>Conventional angiography (9)</td>
<td>3 [33%]</td>
<td>3 [33%]</td>
</tr>
</tbody>
</table>

Cardioembolic (5)
- High risk: vegetation (1), thrombus* (1), atrial fibrillation* (3)
- Low risk: patent foramen ovale (1)
Artery-to-artery embolism (3)
Thrombocytosis (1)
Mixed (3)
Negative evaluation with 2 or more stroke risk factors (21)
Negative evaluation (10)
No risk factor (1)
Single risk factor (1)
Incomplete evaluation (8)

*One patient had both thrombus and atrial fibrillation.
Ataxic Hemiparesis

ataxic on the same side as pyramidal distribution weakness (mainly of the foot, hence “crural”) and corticospinal tract signs in the absence of loss of sensation, typical brain stem (asymmetrical nystagmus was allowed), or cortical signs. A contemporary article described a similar entity, the dysarthria–clumsy hand syndrome,5–9 the findings of which (dysarthria, dysphagia, prominent facial weakness homolateral to mild weakness and clumsiness of the hand, and corticospinal tract signs) were later subsumed by Fisher under the auspices of AH10,11 but then separated again in a subsequent review.12

Since that time numerous reports have expanded the spectrum of clinical symptoms and signs associated with AH. Included are accompanying contralateral trigeminal weakness (motor only,13 sensorimotor13) and partial or complete ipsilateral hemihypesthesia. There are frequently reported patients with subjective numbness, usually without sensory loss, and 1 patient13 had accompanying pain without sensory loss. Review of the published cases reveal that also noted but not emphasized by the authors were 2 cases of contralateral Horner’s syndrome,14 ipsilateral facial hypesthesia,15–19 and ipsilateral tongue deviation8–19,23 (presumably considered part of the hemiparesis). A more recent report of 2 patients,17 1 with fleeting symptoms of weakness and corticospinal tract signs and the other with mild weakness, both accompanied by ataxia and prominent hemisensory loss was coined “hypesthetic-ataxic-hemiparesis” and likened to the Dejerine-Roussy (thalamic) syndrome. Indeed, the most consistent clinical findings of AH, besides some degree of clumsiness and homolateral corticospinal tract signs, appear to be what is excluded (visual field deficit, aphasia, diplia) rather than specific inclusions.

Accompanying a debate on the origin of the “cerebellar” component of ataxic hemiparesis is skepticism regarding its actual existence. A report of 2 cases of capsular infarct24 included 1 in which the patient actually fit the criteria of pure motor hemiparesis (with a slight sensory component), where only as the hemiparesis improved did signs of hemiataxia appear. The authors suggest that cortiocerebellar fibers are possibly intermingled within the corticospinal tract and speculate that cerebellar-type signs may be hidden by the severity of the weakness only to be found when the weakness abates, leading to underdiagnosis because of hesitancy on the part of some physicians to interpret cerebellar-type signs in the context of weakness. Landau25 asserts that some degree of clumsiness will be found in virtually any patient with hemiparesis and that it is nearly impossible to differentiate the 2 types of clumsiness clinically. Impaired position sense was thought to be the mechanism behind the ataxic component of the syndrome14,26 in 5 patients14 and was dubbed “sensory ataxic hemiparesis” (all of whom had thalamic lesions). Other reports27 argue for cerebellar-type findings in patients with cortical infarction and postulate that the mechanism is intrinsic to the role of the lateral cerebellar hemisphere in integrating and communicating information from the cortical sensory association areas (superior parietal lobule) to the motor and premotor cortices. Based on our collective personal experience, we were unable to discern different neuroanatomical localizations for ataxic signs by neurological examination alone.

The inclusion of the sensory signs in the syndrome is somewhat controversial. Of the original 14 patients reported by Fisher and Cole (5 in analyzable detail),9 only 1 patient had impairment of sensation associated with the stroke. Subsequently, Fisher8 presented 3 cases without sensory loss, and he noted this absence as part of the typical clinical syndrome. Of the next 58 cases (either single reports or small series), 48 (83%) reported the sensory examination findings: 23 (48%) were abnormal, all attributed to the lesion in question and homolateral to the AH. Six had impairment of pinprick and light touch sensation, 2 with only vibratory loss, a single patient had solely proprioceptive loss, and 13 had multimodality sensory impairments (all involving at least some impairment of position sense). There were 4 cases reported with “cortical” sensory findings. Another group found sensory loss accompanying AH to be “fairly frequent.”28 Helgason and Wilbur29 reported 23 patients with AH and homolateral sensory loss. All of the patients had involvement of the posterior one third of the posterior limb of the internal capsule extending superiorly and paraventricularly in 15 (one third also involved the lateral thalamus).

Of the 91 cases reported in the literature in sufficient detail concerning sensation, 57 (63%) of the patients had sensory loss and all but 1 of them had lesions in the posterior limb of the internal capsule or overlying the corona radiata (20 also had some thalamic involvement). Huang and Lui30 first suggested sensory loss as localizing to the internal capsule or more rostral. It has also been suggested from data in which somatosensory evoked potentials were used in cases of AH that sensory loss may be subclinical in some cases involving the internal capsule.18,26 This information would tend to implicate involvement of either the ascending cerebellar efferent pathways or the lemniscal/proprrioceptive pathways as they synapse in the thalamus or pass through the posterior limb of the internal capsule on their way to the cortex as the main cause of the ataxia in capsular lesions, especially when hypesthesia is involved. Our series found 20% of patients had sensory loss attributable to the infarct. Of those with a clear lesion, all involved the internal capsule and thalamus except for 1 pontine lesion. We therefore confirm sensory loss in a high percentage of otherwise typical AH patients and a clear predilection for lesions of the posterior limb of the internal capsule, commonly with thalamic involvement.

Dysarthria may be explained either by interruption of corticobulbar tracts or cerebellar connections. In the latter case, the speech disturbance is more typical of scanning or ataxic descriptions and may be lateralized with the left cerebellar tracts (right brain stem, after their decussation) more frequently affected.20,31 A review of the reported cases finds 32 of 88 (36%) with dysarthria, 12 with localization to the brain stem, and the rest supratentorially, primarily to the internal capsule (2 thalamic lesions). Our findings suggest that although dysarthria is not a consistent localizing sign in this syndrome, a pontine lesion or multiple lesions were more likely to be identified than a capsular lesion.

Bamford and Warlow32 reemphasized the central tenants of the lacunar hypothesis in which certain distinct clinical syndromes are associated with small, deep infarcts and that these infarcts are generally the final result of a particular type
of cerebral vascular arteriolar lesion. In a CT study of patients presenting with lacunar syndromes, of the 7 who had either dysarthria–clumsy hand syndrome or AH, 6 had a small deep infarct, and 1 had a normal scan. The other “classic” lacunar syndromes were variably associated with larger lesions. The usefulness of clinical lacunar syndromes to predict small, deep cerebral infarcts was evaluated and found to be quite accurate (88%), using all 4 classic lacunar syndromes (pure motor stroke, pure sensory stroke, sensorimotor stroke, and AH). Ataxic hemiparesis was the most accurate of the 4 syndromes, with a predictive ability of 100%.

**Etiologic Mechanisms**

An association of lacunar syndromes and of small, deep infarcts with hypertension was postulated early on by the very nature of the underlying vascular lesions. Many studies have concurred with this, yet others have shown a surprisingly low association (44% to 53%) with known hypertensives, which correlates with some pathological studies. Our series included 82% hypertensives, 49% of those with presumed small-vessel disease, for an overall frequency of 40% hypertensive small-vessel disease. Because the presence of a potential cardiac or arterial embolic source does not exclude small-vessel disease as the actual mechanism of infarction, our findings most likely underestimate the true amount of small-vessel disease.

Although initially attributed to the intrinsic arteriolar diseases such as lipohyalinosis and microatheroma common to lacunar infarctions, it was clear early on that other etiologies might be implicated. Fisher acknowledged the possibility of cardiogenic sources for some of the lacunes, and in 1 case he demonstrated atheroma in the large artery overlying the origin of the penetrating artery, causing occlusion and subsequent infarction in the arteriolar distribution. In 1 case infarction related to cysticercosis-induced meningovascularitis was found. In abundant case reports other etiologic mechanisms, such as hemorrhage, and neoplastic infiltration have been well documented. Information from studies in which neuroimaging was used to diagnose small, deep infarcts provides some evidence for diffuse etiologic mechanisms, with 33% of those patients with small deep infarcts on CT scan having either a potential cardiac or carotid embolic source.

Substantial controversy exists concerning the role of carotid disease in lacunar infarction. A synthesis of the studies to date implies that, although potentially some lacunes are related to carotid embolic disease, the majority are probably unrelated and are most likely caused by intracranial small-vessel disease. Supporting this latter contention, our study found no high grade (>50%) carotid stenosis to explain the ischemic lesions in AH. We did, however, find 3 of 13 (23%) pontine lesions to be associated with basilar artery stenosis.

**Utility of Diagnostic Testing**

In our experience, blood laboratory testing, both routine and tailored to a stroke evaluation, led directly to a diagnosis in only 1 case (thrombocytosis). It did, however, lead to the identification of risk factors (diabetes mellitus or hypercholesterolemia) in 10 patients not previously identified with these problems and may have subsequently led to an improvement in their long-term health.

The ECG was abnormal in a high percentage of our patients, revealing a potential cardiogenic source in 7% (atrial fibrillation). Echocardiography, transthoracic and transesophageal together, found potential cardiogenic sources in 12% of those tested.

Neurovascular imaging was helpful in that carotid duplex Doppler served to exclude significant extracranial carotid disease, whereas TCD testing revealed evidence to support small vessel disease in seven patients, consistent with the clinical supposition. MRA led to a diagnosis of basilar stenosis in one patient. Conventional angiography in selected patients led to a diagnosis in nearly half, but none required surgery.

MRI is the current imaging test of choice, localizing the lesion in all lacunar infarcts 74% to 90% of the time, whereas CT scanning sensitivity is only 15% to 78%. We also found that MRI was more sensitive than CT for demonstrating the lesions, especially in brain stem infarction. Asymptomatic, small, deep infarcts were commonly encountered in our patients (60%), suggesting that AH may be a marker for more generalized asymptomatic cerebrovascular disease.

**Prognosis**

The reported recurrent stroke rate varies between 5% and 11.8% in the first year after a lacunar infarct with silent infarction noted in 10% at combined clinical and MRI follow-up. Most of the recurrent strokes were lacunar in type. Recently, progression over time of periventricular–deep white matter lesions and small deep infarcts (mainly asymptomatic) was correlated with clinical lacunar infarction. Although this same progression was generally not correlated with worsening disability scores on the Rankin scale, it has been correlated with diminished cerebral blood flow and may reflect an underlying state of diffuse cerebral arteriolar disease. A follow-up study of lacunar patients found an overall lower survival rate than in the general population. Reported case fatality rates of 1% to 2% at 1 month and 9.8% to 15% at 1 year include all lacunar syndromes (but very few
cases of AH).60,68 Although our follow-up data were limited, we were able to document only 1 death.

Conclusions
Approximately 3% of stroke populations studied fulfilled criteria for a diagnosis of AH. AH is a distinct clinical syndrome that accurately predicts a small deep infarction, most commonly in either the internal capsule or basis pons. Of its various clinical manifestations, only sensory loss accurately predicts a capsular localization. Although AH is an etiologically heterogeneous entity, nearly half of the cases may be attributable to small vessel disease. It generally occurs in the company of other small cerebral ischemic lesions and therefore may be a marker for more generalized asymptomatic cerebral vascular disease. AH appears to predict an overall good clinical recovery.

Acknowledgments
This study was supported in part by National Institutes of health, Bethesda, Md, Grants NS23393 and NS30896.

References
Ataxic Hemiparesis: Critical Appraisal of a Lacunar Syndrome
Mark J. Gorman, Rima Dafer and Steven R. Levine

Stroke. 1998;29:2549-2555
doi: 10.1161/01.STR.29.12.2549
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1998 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/29/12/2549

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/