Somatosensory Evoked Potentials in Lacunar Syndromes of Pure Motor and Ataxic Hemiparesis

Michael A. Kelly, MD, Stuart J. Perlik, MD, and Morris A. Fisher, MD

Syndromes of hemiparetic lacunar infarction include pure motor hemiparesis and ataxic hemiparesis. Twelve such patients were evaluated with somatosensory evoked potentials. Potentials were delayed or absent in all 4 patients with ataxic hemiparesis and in 1 of 8 patients with pure motor hemiparesis. Unlike pure motor hemiparesis, ataxic hemiparesis appears to be associated with abnormal somatosensory evoked potentials. These abnormalities suggest that disturbance of afferent pathways are important in ataxic hemiparesis (Stroke 1987;18:1093-1097).

Pure motor hemiparesis (PMH) and ataxic hemiparesis (AHP) are lacunar stroke syndromes characterized by hemiparesis in the absence of objective sensory loss or evidence of cortical dysfunction. In AHP, the hemiparesis is combined with a "cerebellar" dysmetria of the affected limbs. It has been suggested that this is caused by injury to cortico-ponto-cerebellar pathways.1-6

Somatosensory evoked potentials (SSEPs) are thought to be generated by impulses traversing the posterior columns and the medial lemnisci reaching the cortex after relay through the thalamus. The more rostral projections of these pathways course through the internal capsule in areas frequently involved in lesions producing PMH and AHP. It has been shown that SSEPs are normal in PMH,7 presumably because afferent pathways are substantially spared. We sought to evaluate SSEPs in AHP. Abnormalities in SSEPs would suggest greater involvement of afferent pathways in AHP than in PMH and could provide an alternate explanation for the ataxia.

Subjects and Methods

SSEPs were performed on 12 patients with hemiparetic lacunar infarcts evaluated by the stroke service at Michael Reese Hospital during the period September 1984 to June 1985. SSEPs were evaluated on the average 12 (range 4-28) days after the ictus (Table 1). The clinical findings were consistent with a diagnosis of PMH in 8 patients or AHP in 4 patients without other evidence of cerebrovascular disease. All patients had computed tomography (CT) scans; an Elscint 2002 scanner (Haifa, Israel) was used with a 1-cm slice thickness. The CT scans showed either evidence consistent with lacunar infarction or no discrete abnormalities. Weakness was graded on a scale of mild to marked. Ataxia was evaluated except where weakness prohibited adequate voluntary movement. Ataxia was defined as dysmetria on finger-to-nose and heel-to-shin testing; dysarthria and gait instability may also have been present. The mean age of the patients was 67 (range 42-79) years (Table 1).

SSEPs were recorded with Ag-AgCl 9-mm disk electrodes applied with collodion and filled with electroencephalography gel. Resistances were < 5,000 Q. Electrodes were placed according to the International 10-20 System. Montages included Cz, C3, and C4 referred to Fpz, Cv (cervical spine 4 cm inferior to theinion) also referred to Fpz, bilateral Erb’s point (EP) recordings, and recordings between the lumbar spine at L1 and the iliac crest (L1-IC). Stimulation of the median nerves at the wrists and the tibial nerves at the ankles was percutaneous at 3 Hz, with nerves on the clinically involved and uninvolved sides stimulated separately. Stimulus intensity produced a minimal motor twitch, 512 responses were averaged, and at least 2 sets of responses were obtained to ensure reproducibility. Minimal montages for arm stimulation were contralateral C3 or C4 to Fpz, Cv to Fpz, and ipsilateral to contralateral EP; for leg stimulation, Cz to Fpz and L1 to IC. Latencies were measured with electronic cursors and labelled as follows: with tibial stimulation onset, P40, N50, P60 (scalp), and L1 (lumbar); with median stimulation EP, N14 (cervical), N20, P25, and N35 (scalp). Abnormality was defined as absent or delayed potentials (or interpeak latencies) differing by > 3 SD from laboratory controls (n = 40). For P40 and N20 scalp potentials, these values were 45.4 and 22.8 msec, respectively.

Results

The results are presented in Table 1, and representative CT scans as well as SSEPs are shown in Figures 1 and 2. Nerve conduction studies in the distribution of the median, posterior tibial, and sural nerves were unremarkable except for unobtainable sural nerve action potentials in 2 patients.

All patients demonstrated weakness. In general, this weakness was of mild to moderate degree and tended to be more pronounced in those with PMH compared with those with AHP. In most patients, weakness of...
Table 1. Clinical Characteristics of Patients With Lacunar Syndromes

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Weakness</th>
<th>Ataxia</th>
<th>CT</th>
<th>SSEP</th>
<th>Day</th>
</tr>
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<tbody>
<tr>
<td>Pure motor hemiparesis</td>
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<td>8</td>
<td>64</td>
<td>2-3</td>
<td>1-2</td>
<td>—</td>
<td>AC,BG</td>
<td>Delayed 28</td>
</tr>
</tbody>
</table>

Arm: 1, mild; 2, moderate; 3, marked. Ataxia: +, present; −, absent or negative. CT, lacunar infarct: −, negative; CR, corona radiata; AC, anterior capsule; BG, basal ganglia; PC, posterior capsule. SSEP, somatosensory evoked potential: N, within normal limits. Day, days from ictus to SSEP.

Discussion

PMH is the most common of the clinical syndromes of lacunar infarction. Studies using CT and autopsied cases have shown that the lesion responsible for PMH is most often in the internal capsule or pons. AHP lesions may be similarly located. Fisher demonstrated pontine lesions in 3 pathologically studied cases. Since then, others have shown using CT scans that lesions of the internal capsule may produce an identical syndrome. Localization of lacunes by CT is limited, however, because lesions in the brainstem as well as those smaller than several millimeters in diameter may not be visualized.

In our study, 3 of the 4 patients with AHP had lacunar infarcts demonstrated by CT scan in the posterior limb of the internal capsule; the fourth patient had a normal CT scan. The location of these infarcts is similar to that of previous reports in patients with AHP and are therefore thought to be related. Two of the 8 patients with PMH had lacunes demonstrated radiographically: 1 was in the corona radiata, the other in the anterior capsule and basal ganglia. The former lacune was probably coincidental rather than causative, whereas the latter was similar to lesions previously reported in PMH. The remaining 6 patients with PMH presumably had unrevealing CT scans because of either lacunar location or size.

Robinson et al. have recently described their experience with SSEPs following median nerve stimulation in pure sensory, pure sensorimotor, and PMH lacunar syndromes. All 11 patients with pure sensory strokes had normal SSEPs; CT scans were normal in all but 1. Each of 4 patients with sensorimotor stroke had abnormal SSEPs; CT scans in these cases showed infarcts in either the thalamus or in the thalamus and internal capsule. Robinson et al. concluded that the size of the infarct was critical in generating abnormal SSEPs and in producing hemiparesis. In their 10 patients with PMH, 1 had abnormal SSEPs and 4 had abnormal CT scans.

In our 8 patients with PMH, only 1 had abnormal SSEPs confirming the observation of Robinson et al. that SSEP abnormalities are rare in PMH. In the 4 patients with AHP, all had delayed or absent SSEPs on stimulation of the involved limbs. These results suggest that abnormal SSEPs are related to ataxia (4 of 5 patients). They also suggest (Table 1) that abnormal SSEPs are associated with evidence of infarction on CT scan (4 of 5 patients) and therefore possibly with lacunar size.

Interruption of descending corticospinal tracts ex-
FIGURE 1. A: Lacune (arrow) involving posterior limb of the internal capsule (Patient 11, Table 1). B: Somatosensory evoked potentials stimulating left tibial and median nerves were within normal limits. When stimulating on the right, scalp potentials were absent while cervical (Cv), Erb's point (EP), and lumbar (L1) potentials were unremarkable.
FIGURE 2. A: Lacune (arrow) in region of the anterior limb of left internal capsule extending into the basal ganglia (Patient 8, Table 1). B: Scalp somatosensory evoked potentials were asymmetrically delayed when stimulating right median nerve, whereas subcortical potentials were unremarkable bilaterally. P40 potentials were also asymmetrically delayed when stimulating right tibial nerve (P40 = 47.6 msec) compared with left (P40 = 42.0 msec).
explains the hemiparesis in PMH and AHP. The apparent
cerebellar ataxia has been somewhat more difficult to
understand. Disruption of both cerebellar afferents and
efferents has been considered. Corticopontine fibers
traverse the internal capsule and pontine nuclei and
project to the contralateral cerebellum. Interruption of
these pathways has been proposed to explain capsular
and pontine AHP. Cerebellar efferents have received
less attention but do traverse the internal capsule in
passing to and from the ventral lateral nucleus of the
thalamus. Lemniscal pathways also deserve consider-
ation. Proprioceptive and kinesthetic information as-
cends in the dorsal columns of the spinal cord and in
the contralateral medial lemniscus of the brainstem to
enter the ventral posterolateral nucleus of the thala-
um. Thalamocortical fibers traverse the posterior
limb of the internal capsule en route to the sensory
cortex. Sensory loss has been described in AHP with
internal capsule lesions, and subclinical sensory
deficits could be present. It is possible that disruption
of thalamocortical fibers combined with weakness
may produce clinical ataxia.

SSEPs are thought to reflect the integrity of afferent
input, particularly of the large fiber sensory pathways
mediated through the posterior column—lemniscal sys-
tem. Lesions in the posterior limb of the internal cap-
sule might therefore alter SSEPs even if the deficits
were subclinical. At the same time, it is clear that the
issues are more complex, including the findings in our
patient with PMH, infarction in the anterior portion of
the internal capsule, and abnormal SSEPs. Although
the anatomy has not been defined, there is physiologic
evidence that primary afferents from muscle spindles
project to the cerebral cortex. These inputs may be the
dominant element in SSEPs, and it is of interest that
such projections are also prominent in mediating cere-
bellar function via the spinocerebellar tracts. SSEPs
were abnormal in each of our 4 patients with AHP, and
3 of those 4 patients had lesions of the posterior limb of
the internal capsule. If ataxia in these patients was due
only to interruption of corticopontine fibers, it is un-
likely that the SSEPs would have been abnormal. Dis-
ruption of afferent fibers would better explain both
the ataxia and the abnormal SSEPs.

We conclude that SSEPs are characteristically nor-
mal in PMH. Delayed or absent SSEPs are frequently
seen in AHP. The presence of abnormal SSEPs sug-
ests that the ataxia of AHP may be caused by disrup-
tion of afferent pathways.

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