Somatosensory Evoked Potentials In Pure Sensory Stroke And Related Conditions

R. Kent Robinson, M.D.,* E.T. Richey, M.D.,† Carlos S. Kase, M.D.,* and J.P. Mohr, M.D.‡

SUMMARY Somatosensory evoked potentials were reviewed for their correlation with CT scan and clinical features of ischemic "pure sensory stroke" and "pure sensorimotor stroke". Somatosensory evoked potentials were normal in all 11 cases of pure sensory stroke, and CT was normal in ten. The N2-P2 components of the somatosensory evoked potentials were abnormal in all 4 cases of pure sensorimotor stroke, and all had low density CT lesions in the lateral thalamus and/or posterior limb of the internal capsule. It is concluded that somatosensory evoked potentials and CT scans are routinely abnormal in pure sensorimotor stroke, but they are consistently normal in pure sensory stroke, and should not be interpreted as evidence against a clinical diagnosis of pure sensory stroke.

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The Syndrome of "Pure Sensory Stroke" (PSS), first described by Fisher1 in 1965, is characterized by numbness and paresthesias of the face, arm, and trunk on one side, in the absence of weakness, homonymous hemianopia, aphasia, or apraxia. Autopsy studies of three cases have shown lacunar infarcts in the ventral-posterior (VP) nucleus of the contralateral thalamus.1,2 CT scan, EEG, and angiography have been normal, making PSS a purely clinical diagnosis.4 A related condition, "pure sensorimotor stroke" (PSMS),4 is characterized by a similar thalamic-type sensory disturbance in association with a mild and transient hemiparesis, without other neurologic deficits. One autopsy case reported by Mohr et al5 had a lacunar infarction in the contralateral VP nucleus of the thalamus and an ischemic area in the adjacent posterior limb of the internal capsule, and two cases reported by Lapresle and Haguenau5 had infarcts restricted to the contralateral thalamic nuclei.

The N2 and P2 components of the somatosensory evoked potentials (SEPs) have been reported to be abnormal in numerous conditions producing sensory abnormalities and lesions involving the thalamus and its cortical projections,6-13 but have not been reported in series of PSS or PSMS cases. The present study was undertaken to evaluate the SEP findings in such cases in an attempt to ascertain their value in providing support to the clinical diagnosis of PSS or PSMS.

Subjects and Methods

All subjects were in-patients on the Neurology service at the University of South Alabama Medical Center between September 1980 and March 1984. Eleven cases of PSS were studied. Partial syndromes, sensory TIAs, or patients with CT or EEG evidence of cortical involvement were excluded. SEP studies were performed after an average of 4.7 days (range: 1-13 days) from stroke onset. There were seven males and four females, mean age 52 years (range: 36-85). Eight were hypertensive (73%) and four (36%) were diabetic. All patients were evaluated clinically within forty-eight hours of the test, and all had sensory abnormalities involving dorsal column modalities at that time. Five patients had primarily loss of light touch, while six had lost light touch sensation and position sense and/or vibratory sense. A group of four cases of PSMS was also studied. The SEPs were performed in average 4 days (range: 1-12 days) from onset of the stroke. Three patients were males and 1 female, and their mean age was 62 (range: 50-71). Three were hypertensive (75%) and the fourth patient was diabetic. One patient in this group came to autopsy three years after the acute event. A group of ten cases of "pure motor hemiparesis" (PMH) was included for the purpose of comparing results of SEP determination in cases with and without sensory abnormalities. The SEP studies were done in average 4.2 days (range: 1-9 days) after stroke onset. There were five males and five females, mean age 61 years (range: 49-71). Nine were hypertensive (90%) and four (40%) were diabetic.

The SEP studies were performed in a dimly lit room with the patient in a supine position with eyes closed. The median nerve was stimulated at the wrist with surface electrodes placed 3 cm apart. Stimulus intensity was adjusted to the visible motor threshold of the opponens pollicis muscle with a pulse duration of 0.1 msec. Stimuli were delivered at a rate of 1.08 per second. The recording montage for left median nerve stimulation was C4'-Fz, C3'-Fz, C7-Fz, Fz-Erb's ipsilateral. For right median nerve stimulation, C4'-Fz, C3'-Fz, C7-Fz, Fz-Erb's ipsilateral (C1 and C4 are 2 cm posterior to Cz and Cz respectively in the 10-20 system). Several studies done prior to 1981 used two-channel recording: C4'-Fz and Fz-Erb's ipsilateral for right median nerve stimulation, and C3'-Fz and Fz-Erb's ipsilateral for left median nerve stimulation. A total of 256 repetitions were averaged per trial.

Results

Pure Sensory Stroke (table 1)

In all twelve cases the early SEP components (Erb's point potential from 9 to 12 msec, cervico-medullary...
negative potential from 11 to 15 msec) were normal, as were the N2 (N20) and P2 (P23) potentials. Although no patients were completely hemianesthetic, all had one or more dorsal column modalities (touch, joint position sense, vibration) impaired, in addition to subjective feelings of numbness and decreased sensation to pin-prick. EEG was done in nine cases, being normal in seven (78%), and mildly and diffusely slow in two (22%). CT scan was normal in ten (91%), and in one patient showed a low density area in the thalamus contralateral to the sensory deficit. Angiography was performed in one patient and was normal. On follow-up examinations three patients (27%) developed a "thalamic pain syndrome."14

Pure Sensorimotor Stroke (table 2)

The early SEPs were normal, but subsequent components were abnormal contralateral to the sensorimotor deficit in all four cases. In two cases the N2 and P3 components were absent (fig. 1), and in one case their latencies were prolonged beyond 3 SD from the norm (although the N2-P3 interpeak latency remained within normal limits). In the other case the latencies were normal, but the N2-P3 amplitude was reduced to 32% of the normal side (fig. 2): a mean of 5.25 μV (5 trials) compared to a mean of 16.25 μV. EEG was done in two cases, being normal in one and diffusely slow in the other. CT scans in all four cases showed low density areas in the lateral thalamus and/or the posterior limb of the internal capsule contralateral to the sensorimotor deficit (fig. 3). In one case the SEP abnormality was present before the documentation of thalamic infarction by CT scan. Angiography was performed in three cases, showing bilateral extracranial internal carotid artery (ICA) plaques and diffuse intracranial atherosclerotic changes in two, and a nonstenotic ICA plaque contralateral to the deficit in the other. On follow-up examinations two of the four patients developed a "thalamic pain syndrome". One case came to autopsy, showing a 10 × 8 × 12 mm infarct in the posterolateral thalamus contralaterally (fig. 4).

Pure Motor Hemiparesis (table 3)

The early SEPs were normal in all ten cases. The N2 potential was normal in nine cases, and the other showed absence of the N2 and P3 potentials contralateral to a severe hemiplegia. This patient was found at angiography to have an occluded vertebral artery ipsilateral to the SEP abnormality, but never developed sensory findings or clinical evidence of brainstem involvement. EEG was done in eight cases, being normal in seven, and showing mild focal slowing ipsilateral to the infarct in the other. Angiography was performed in six cases, and showed bilateral ICA plaques with irregularities in the carotid siphon (2 cases), occluded vertebral artery ipsilateral and contralateral to the infarct (one case each), and diffuse atherosclerotic changes and irregular cavernous portion of the ICA ipsilateral to the infarct (one case each). CT scan revealed a low density area in the corresponding internal capsule in four cases (40%), being normal in the other six patients.

Discussion

Lacunar infarcts are small (.2 to 20 cu mm) ischemic lesions occurring in the basal ganglia, internal capsule, thalamus, and brainstem. They are caused by occlusion of small (100-400 microns) penetrating arteries,16 which are terminal vessels with no collateral circulation.17 This hundredfold range of size in lacunar infarcts has been thought to relate to the site of the penetrating artery occlusion, with proximal occlusions causing large infarcts and more distal ones causing smaller infarcts.18 Thus the clinical syndromes caused by lacunar infarcts (PMH, PSS, PSMS, dysarthria-clumsy hand, ataxic hemiparesis) may be a function of the infarct size, the precise location of the infarct, or

### Table 1 Pure Sensory Stroke

<table>
<thead>
<tr>
<th>Pt/age/sex</th>
<th>CT</th>
<th>SEP (N2)</th>
<th>Thalamic pain syndrome</th>
<th>↑ BP</th>
<th>DM</th>
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<tbody>
<tr>
<td>CA 85 F</td>
<td>neg</td>
<td>nl</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>SC 42 F</td>
<td>neg</td>
<td>nl</td>
<td>–</td>
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<td>+</td>
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<td>BD 41 F</td>
<td>neg</td>
<td>nl</td>
<td>–</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>CE 51 M</td>
<td>neg</td>
<td>nl</td>
<td>+</td>
<td>+</td>
<td>–</td>
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<tr>
<td>EG 50 M</td>
<td>neg</td>
<td>nl</td>
<td>–</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>JG 36 M</td>
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<td>nl</td>
<td>–</td>
<td>–</td>
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<td>EO 40 M</td>
<td>neg</td>
<td>nl</td>
<td>–</td>
<td>+</td>
<td>–</td>
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<tr>
<td>WR 48 M</td>
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<td>nl</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>FS 39 M</td>
<td>neg</td>
<td>nl</td>
<td>–</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>MA 68 F</td>
<td>R. thal.</td>
<td>nl</td>
<td>–</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

N = 11. Abbreviations: ↑ BP = hypertension; DM = diabetes mellitus.

### Table 2 Pure Sensorimotor Stroke

<table>
<thead>
<tr>
<th>Pt/age/sex</th>
<th>CT</th>
<th>SEP(N2)</th>
<th>Thalamic pain syndrome</th>
<th>↑ BP</th>
<th>DM</th>
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</thead>
<tbody>
<tr>
<td>MO 50 F</td>
<td>L. thalamus</td>
<td>delayed</td>
<td>+</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>DD 63 M</td>
<td>R. thal-int. cap.</td>
<td>absent</td>
<td>+</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>WT 65 M</td>
<td>L. thal-int. cap.</td>
<td>low amplitude</td>
<td>–</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>JO 71 M</td>
<td>L. thal-int. cap.</td>
<td>absent</td>
<td>–</td>
<td>–</td>
<td>+</td>
</tr>
</tbody>
</table>

N = 4. Abbreviations: ↑ BP = hypertension; DM = diabetes mellitus.
FIGURE 1. Median nerve SEPs (patient JO in table 2) showing normal response from left median nerve stimulation, with absent N2 and subsequent potentials from right median nerve stimulation.

SEP-Left Median Nerve

SEP-Right Median Nerve

FIGURE 2. Median nerve SEPs (patient WT in table 2) showing normal response from left median nerve stimulation, with significant reduction in amplitude on right median nerve stimulation.

SEP-Left Median Nerve

SEP-Right Median Nerve

both. A very small lacune located in a strategic area may cause significant clinical symptoms without abnormalities on CT scan, arteriography, or EEG. Such is the case in PSS in which dysesthesias with unusual qualities (“pressing”, “burning”, “cold”, “hot”, and “tingling” feelings) develop over one entire half of the body, as if a plumb-bob line had been drawn bisecting face, trunk, and genitalia. These symptoms may either resolve, persist, or develop into a “thalamic pain syndrome.”19 This syndrome occurred in 3 of our 11 cases of PSS (27%) and in 2 of the 4 cases of PSMS (50%). Fisher reported a lower incidence of this complication (2 of 48 cases, or 4%) in his series of PSS.19 There have been only two patients with PSS from lacunar infarction reported with a CT low density in the contralateral thalamus.20-21 We had a single case showing such a lesion.

Numerous studies have looked at median nerve SEPs in patients with sensory disturbances and lesions of the thalamus and/or cortex.6-13 Though most authors agree that the P2 component of the SEP has its origin in the sensory cortex, there has been much debate over the exact origin of the N2 component — thalamus, thalamocortical radiations, or cortex.10, 22-27 Most studies correlating SEP findings and clinico-radiologic data have given little detailed clinical information when “strokes” have been involved, and most patients have had lesions large enough to be seen on CT scan. Such large, CT-positive thalamic lesions are not typically seen in cases of PSS.3 In our 11 cases of PSS presumably resulting from a small lacunar infarct of the ventral posterior thalamus, the N2 component was normal in every instance, as was the P2 component. This could imply either that the amount of tissue loss necessary to cause the PSS syndrome is not sufficient to disrupt the generation of the N2 potential, or that this potential is not generated entirely from the VP thalamic nucleus. In either case we did not find median nerve SEPs to be helpful in supporting a clinical diagnosis of PSS.

A less common lacunar syndrome is the PSMS described by Mohr et al4 and by Lapresle and Haguenau.5 The two cases reported by the latter authors showed infarcts in the VPL and ventral posteromedial (VPM) thalamic nuclei, whereas the case of Mohr et al.4 had a 4 × 2 × 4 mm infarct in the VPL nucleus with a zone of ischemia in the adjacent internal capsule. Our single autopsy case had a 10 × 8 × 12 mm infarct in the VPL and VPM nuclei. These lesions are larger than the more typical PSS thalamic infarcts and in our group of four cases all infarcts were large enough to be seen on CT scan. The early SEP components were normal in every instance, while the later (N2-P2) components were always abnormal: absent in two cases, delayed in one case (with a normal N2-P2 interpeak latency), and markedly reduced in amplitude (20-28% of the normal side) in one case. In one instance these SEP abnormalities occurred while the CT scan was still normal.
FIGURE 3. A (upper left) Large left thalamic lacunar infarct (arrow), patient MO in table 2. B (upper right) Large right capsular-supracapsular lacune (arrow), patient DD in table 2. C (lower left) Small lacune in left postero-lateral thalamus (arrow), patient WT in table 2. D (lower right) Large lacune in postero-lateral aspect of left thalamus (arrow), and old incidental smaller lacune at the level of the genu of the left internal capsule (arrowheads), patient JO in table 2.
believe that while the clinical sensory findings in these cases are identical to those of PSS, it is the larger size of these infarcts which is responsible for both the difference in SEP findings between the two groups, and the presence of hemiparesis, the latter related to extension of the infarct into the adjacent internal capsule.\(^4\)

![Figure 4. Large old cavitated lacunar infarct in the posterolateral aspect of the left thalamus (arrow), slightly involving the adjacent posterior limb of the internal capsule. Incidental finding of old large cavitated lacune in the genu of the right internal capsule and head of the caudate nucleus (arrows). Patient MO in table 2.](image)

The ten cases of PMH had the expected normal SEP studies in all cases but one, in whom a vertebral artery occlusion was found at arteriography. There may have been subclinical involvement of the sensory pathways in this case, but we were unable to document this possibility.

In conclusion, median nerve SEPs appear to be routinely normal in patients with PSS, a test result that should not be interpreted as speaking against the possibility of a thalamic lacune in the appropriate clinical setting. PSMS is caused by larger lacunes in the thalamus, usually large enough to be seen on CT, and frequently has abnormalities in the N2 and subsequent components of the median nerve SEPs. These abnormalities may be detectable early in the course of the stroke, before documentation of the lacunar infarct by CT scan. The "thalamic pain syndrome" is not infrequently a late complication of both PSS and PSMS.

**Table 3**

<table>
<thead>
<tr>
<th>Name/sex</th>
<th>CT</th>
<th>SEP(N)</th>
<th>BP</th>
<th>DM</th>
</tr>
</thead>
<tbody>
<tr>
<td>AL 68 F</td>
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<td>nl</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>CM 71 F</td>
<td>neg</td>
<td>nl</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>WM 52 F</td>
<td>neg</td>
<td>nl</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>FM 58 F</td>
<td>neg</td>
<td>nl</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>LW 62 M</td>
<td>neg</td>
<td>nl</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>CM 49 M</td>
<td>neg</td>
<td>absent</td>
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<td>-</td>
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<td>L. int. cap.</td>
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<td>-</td>
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<tr>
<td>RB 63 F</td>
<td>L. int. cap.</td>
<td>nl</td>
<td>+</td>
<td>+</td>
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<td>JJ 56 M</td>
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<td>-</td>
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<tr>
<td>AW 66 M</td>
<td>R. int. cap.</td>
<td>nl</td>
<td>+</td>
<td>-</td>
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</table>

**References**

Amnestic Syndrome and Vertical Gaze Palsy: Early Detection of Bilateral Thalamic Infarction by CT and NMR

R. A. SWANSON, M.D., AND J. W. SCHMIDLEY, M.D.

SUMMARY A 27 year old woman with mitral valve prolapse presented with somnolence, bilateral Babinski signs, and grasp reflexes. As somnolence cleared, vertical gaze palsy and Korsakoffian memory deficit were apparent. Initial CT scan was normal, but NMR scan 24 hours after the onset of symptoms revealed prolonged T2 relaxation in medial thalami bilaterally, facilitating diagnosis of bithalamic infarction. Subsequent CT scans delineated infarction in the vascular territory of the paramedian thalamic arteries. Previous clinical reports and the neuro- and vascular anatomy underlying this syndrome are reviewed, including cases that suggest a relationship to the syndrome of transient global amnesia.

IN 1978 MILLS AND SWANSON described a patient with persistent vertical oculomotor apraxia, somnolence, and severe impairment of memory, presumably the result of bilateral medial thalamic infarction. Two and one-half years after the acute event a CT scan revealed a widened third ventricle, and small, approximately symmetric zones of low absorption in both dorsomedial thalami.

We have recently seen a patient with a nearly identical syndrome, and were able to use NMR imaging to detect bithalamic lesions within 24 hours of admission. High resolution CT scanning documented the evolution of bilateral dorsomedial thalamic infarctions over a ten day period.

Case Report

A 27 year old woman awoke on the day of admission complaining of headache, double vision, nausea and unsteadiness. She returned to bed and two hours later was barely arousable. She was taken to the emergency room where she was lethargic and vomited several times.

She did not use any medications, illicit drugs, or cigarettes. Blood pressure was 110/70, heart rate 80, respiratory rate 18, and temperature 36°C. General physical examination was unremarkable with the exception of a late systolic click with grade 3/6 apical systolic murmur. Peripheral pulses were full throughout. There were no bruits or cutaneous lesions. Fundi were normal.

The patient was stuporous but arousable with vigorous verbal or physical stimuli. When awakened she was able to follow simple commands. She did not speak spontaneously but was able to nod "yes" or "no". Ocular axes were skewed in the vertical plane, the left eye resting lower than the right, with full horizontal extraocular movements to doll's head maneuver. Pupils were 3 mm bilaterally and reactive. Corneal responses were brisk. Palate was midline, with brisk elevation to gag. The limbs were paratonic. She moved all limbs spontaneously, and withdrew from noxious stimuli appropriately. The tendon reflexes were brisk; bilateral extensor plantar responses and grasp reflexes were present. There were no meningeal signs.

Twenty-four hours later the patient was much less obtunded and was able to answer questions, although still tending to sleep if not stimulated. She knew her name, the year, her home address, and that she was in a hospital. The skew deviation had resolved. Lateral gaze was normal but she was unable to voluntarily move her eyes up or down to command or pursuit. However, full ocular excursions in the vertical plane were obtained with vertical doll's head maneuver. Bell's phenomenon was present on attempted eye clo-
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