Sensory Symptoms and Signs and Results of Quantitative Sensory Thermal Testing in Patients With Lacunar Infarct Syndromes

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Background and Purpose
Quantitative data on sensory impairment in stroke patients are limited. We measured the perception thresholds for temperature and thermal pain in patients with different lacunar syndromes, correlated the results with clinical and magnetic resonance imaging (MRI) findings, and studied the long-term prognosis of sensory dysfunction.

Methods
Quantitative thermal testing was performed by means of the Marstock method in 39 patients with lacunar syndromes (pure motor, sensorimotor, or pure sensory stroke) and MRI findings compatible with occlusion of a single perforating artery. Thresholds for cold, warm, and heat pain were obtained bilaterally from the cheek, hand, and leg. The unaffected side was used as control. Follow-up included clinical assessments and repeated quantitative thermal testing (in 17 patients) up to 1 year after stroke onset.

Results
Patients with pure sensory stroke and sensorimotor stroke (n=22) had a significant thermal hypoesthesia on the affected side for all modalities and test locations. Patients with pure motor stroke (n=17) exhibited thermal hypoesthesia for cold and heat pain in the hand and for cold perception in the leg. On MRI, infarcts causing pure motor and sensorimotor stroke were predominantly lenticulocapsular, while a thalamic site of infarction was found in pure sensory stroke. The prognosis of sensory impairment was favorable, except for poststroke pain syndromes in three patients.

Conclusions
Quantitative thermal testing confirmed an involvement of spinothalamic pathways in lacunar infarcts causing pure sensory and sensorimotor stroke and revealed a subclinical sensory impairment in patients with pure motor stroke. Infarction sites were similar in patients with pure motor and sensorimotor stroke. (Stroke. 1994;25:2165-2170.)

Key Words
lacunar infarction • magnetic resonance imaging • sensory testing, quantitative • temperature sense

The term lacunar infarction has been used to indicate a small brain lesion resulting from occlusion of a single, small penetrating artery arising from large intracerebral arteries. Lacunar infarcts, which constitute up to 25% of all ischemic strokes,1-5 are associated with lacunar syndromes, which are defined clinically from findings on the neurological examination. Of the five generally accepted lacunar syndromes, pure motor stroke (PMS) and sensorimotor stroke (SMS) are the most common, whereas pure sensory stroke (PSS), ataxic hemiparesis, and dysarthria/clumsy hand are less frequently encountered.

There has been some reluctance to accept SMS as a lacunar syndrome, mainly because of limited pathological confirmation. The clinical separation of SMS from PMS depends on the presence or absence of sensory signs, and the conventional sensory examination is probably the least reliable part of the neurological examination, with considerable interobserver variations.5-7 Quantitative data on sensory function would therefore be of interest, but available data are limited. Involvement of lemniscal pathways has been studied with somatosensory evoked potentials (SEPs).8-11 To assess spinothalamic-mediated sensory function, Borrie et al12 and Leijon et al13 used quantitative estimation of thermal thresholds (quantitative thermal testing [QTT]) in their studies on central poststroke pain. They found this technique well applicable to patients with brain lesions and also reported a good correlation between quantitative and clinical findings.

In this report we describe QTT findings in 39 patients with a lacunar syndrome (PMS, SMS, or PSS) and magnetic resonance imaging (MRI) findings compatible with occlusion of a single perforating artery; we describe the correlation between quantitative data, clinical symptoms, and infarct sites on MRI. We also report the long-term prognosis of sensory dysfunction and its contribution to late disability in these patients.

Subjects and Methods
The patients were collected from a larger series of 100 consecutive patients admitted between August 1989 and February 1992 to the emergency care unit at Örebro Medical Center Hospital with a first-ever stroke and presenting with a clinical lacunar syndrome. On enhanced MRI, 81 of the patients had findings compatible with an infarct in the territories of the deep penetrating arteries, as previously reported.14 Thirty-nine of these patients were examined with QTT and form the basis of the present report. We included all patients with PSS (n=10) and 12 of 13 patients with SMS (1 patient had her stroke before QTT was available). Among patients with PMS, only those admitted between February and December 1991 were examined (n=17).

PMS, PSS, and SMS were defined according to the original descriptions by Fisher15-17 and later definitions for study...
purposes by Bamford et al. Consequently, patients with visual field defects and evidence of higher cerebral dysfunction such as dysphasia, visuospatial disturbance, and predominantly preclinical sensory loss on clinical examination were not included. We accepted patients with partial (ie, faciobrachial and brachiocephalic) syndromes but not patients with monoparesis. The clinical diagnosis of a lacunar syndrome was made by the first author (M.S.) within the first week after the onset of stroke.

The clinical examination of sensory modalities included touch, pinprick, vibration, kinesthesia, and graphesthesia. Touch and pinprick were tested by strokes of cotton wool and light pricks with a pin. Vibration was tested with a tuning fork, and kinesthesia and graphesthesia were tested by asking the patient to identify passive joint movements and digits written in the palm, respectively. The nonaffected side was used for comparison. An abnormal finding was said to be present if the patient reported or the examiner noted differences in reactions between the two sides on repeated testing. Clinical follow-up examinations were performed at 3 to 4 weeks and 3, 6, and 12 months after the onset of stroke.

QTTh was performed for temperature and pain by determining thresholds for cold, warmth, and heat pain with a modified Marstock thermostimulator operating on the Peltier principle (Thermotest, Somedic AB). The thermode, with a stimulating surface of 2.5×5 cm², can be heated or cooled at a rate of 1°C to 2°C per second with the stimulating temperature continuously measured by a thermocouple and recorded by a pen recorder. When the stimulator is applied in good contact with skin, the patient is asked to reverse a switch as soon as the sensation of cold, warmth, or heat pain is experienced. The examinations were all performed by the same person (L.S.). After a trial test on abdominal skin to introduce the patient to the test technique, thresholds for cold, warmth, and heat pain were determined from the cheek (maxillary region), hand (thenar or hypothenar eminence), and leg (L5 dermatome). The perception threshold was, with few exceptions, calculated as the mean value of at least three consecutive stimulations. A baseline adapting temperature of 32°C was used in all tests. The outer temperature limits were set at 10°C for cold and 50°C for heat to avoid tissue damage. The first registration was performed within the first week after stroke onset in all patients. Of the patients with PSS and SMS (n=22), 14 patients were reexamined after 6 months and 17 after 1 year. In the 17 patients with PMS, only the initial examination was performed.

We used the Wilcoxon rank sum test to statistically compare thermal thresholds on the affected and nonaffected sides. A level of P<.05 was considered statistically significant. In patients with a partial (ie, faciobrachial or brachiocephalic) distribution of symptoms, we included only registrations from body parts with sensory dysfunction on clinical examination. Registrations in patients who did not perceive warmth or heat pain at 50°C or cold at 10°C were assigned the value of the upper and lower temperature limit, respectively. In individual patients, registrations of temperature thresholds were compared with the corresponding mean values (+1 or 2 SDs) obtained from the unaffected body parts of all patients.

All patients were examined with an enhanced MRI (1.0-T Siemens Magnetom) at a mean time of 18 days (range, 8 to 30 days) after onset and a second MRI without enhancement 1 year later, as previously reported. The MRI examination included T1-weighted images (repetition time [TR], 600 milliseconds; echo time [TE], 10 milliseconds) in the sagittal plane with 5-mm slice thickness and proton density-weighted (TR, 2500 milliseconds; TE, 20 milliseconds) and T2-weighted (TR, 2500 milliseconds; TE, 90 milliseconds) images in the axial planes with 8-mm slice thickness and 2-mm interslice gaps. The matrix size was 256×256 cm²; field of view was 30 cm. For the enhanced MRI, gadopentetate dimeglumine (Magnevist, Schering AG) was administered by intravenous infusion at a standard dose of 0.1 mmol/kg, followed within 5 minutes by postcontrast T1-weighted images in the axial plane with 8-mm slice thickness and 2-mm interslice gaps. The MRI findings were independently assessed by a neuroradiologist (D.L.) who was at that time blinded to clinical data.

Results

Clinical Features and Demographic Data

Demographic data, distribution of symptoms, and severity of hemiparesis in the 39 patients are detailed in Table 1. The lacunar syndromes were complete (ie, affected face, arm, and leg) in 23 patients and partial (faciobrachial or brachiocephalic) in 16 patients. Among patients with SMS, weakness and sensory impairment involved the same body parts, except in 1 patient who had a brachiocephalic paresis but sensory symptoms from arm and hand only. Transitory sensory symptoms at the time of stroke onset were reported by 3 of the 17 patients with PMS.

On the initial examination all 22 patients with PSS and SMS had decreased sensation of pinprick on the affected side, whereas decreased sensation of touch was reported by all patients with PSS and 8 of the 12 patients with SMS. Four patients had decreased perception of vibration, and 1 patient with PSS also had decreased sensation of passive joint movements. Graphesthesia was normal in all patients. Generally, all sensory symptoms and signs were most pronounced in the hand.

All 12 patients with SMS regarded hemiparesis as their main handicap, whereas the sensory disturbances were considered of minor importance. The sensory symptoms resolved during the first week in 1 patient and during the first month in 2 patients. At 3 months only 6 patients had any persistent sensory disturbances, which remained at 12 months, although less pronounced. On clinical exam-
TABLE 2. Thermal Perception Thresholds on Symptomatic and Asymptomatic Sides in 22 Patients With Pure Sensory Stroke (n=10) and Sensorimotor Stroke (n=12)

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<th>Cold</th>
<th>Warmth</th>
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<td>Cheek</td>
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<td>(n=17) 29.7°C</td>
<td>5.1</td>
<td>31.3°C</td>
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<td>P*</td>
<td>.006</td>
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<td>Hand</td>
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<td>(n=22) 28.6°C</td>
<td>3.8</td>
<td>30.7°C</td>
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<td></td>
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<td>P*</td>
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<td>Leg</td>
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<td>(n=17) 25.8°C</td>
<td>6.2</td>
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Sympt indicates symptomatic; Asympt, asymptomatic.

Quantitative Assessment of Thermal Thresholds

Mean temperature thresholds for perception of cold, warmth, and heat pain on the affected versus the nonaffected side and the results of the Wilcoxon rank sum test in the 22 patients with PSS and SMS are given in Table 2. A statistically significant reduced sensibility was found on the affected side for all modalities and all locations.

Thermal perception generally improved over time, all 6 reported decreased sensibility to pinprick, whereas only 4 patients still reported decreased sensibility to touch; none demonstrated side differences in perception of vibration. No case of central poststroke pain was encountered among patients with SMS.

In 19 of the 22 patients with PSS and SMS, thermal thresholds in at least one body part of the affected side deviated >1 SD (in 11 patients >2 SDs) from the corresponding values on the asymptomatic side. Warmth and/or heat pain was not perceived at 50°C in the hand and in the leg in 2 patients, whereas cold perception at 10°C was lost in the cheek or in the leg in 2 patients. Thermal thresholds outside the range tested (10°C to 50°C) were recorded in all 3 patients with PSS who later developed painful dysesthesia.

Among the 9 patients with a faciobrachial or a brachio-crural distribution of the sensory loss on clinical testing, 5 demonstrated side differences (in 1 patient >2 SDs) in thermal thresholds in all three body parts on the affected side.

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QTT findings in the 17 patients with PMS (by definition without signs of sensory dysfunction on clinical examination) are provided in Table 3. Side asymmetries of thermal thresholds were found for all sensory modalities in all locations except for perception of warmth in the hand and were statistically significant for cold and heat pain in the hand and for cold perception in the leg. In 9 patients thermal thresholds in at least one body part on the affected side deviated >1 SD (in 6 patients >2 SDs) from the corresponding mean values on the asymptomatic side. Two patients had differences in thermal thresholds >2 SDs on all locations on the hemiparetic side. Thermal anesthesia within the temperature range limits was not recorded in any of the patients with PMS.

Seventeen of the 22 patients with PSS and SMS were followed up with QTT up to 1 year after the onset of stroke. Thermal perception generally improved over
time (Fig 1). However, 1 year after stroke onset there was still a statistically significant reduction for warmth and heat pain perception in the hand and leg (Fig 1). An improvement for all modalities was also seen in the 5 patients with the most severe abnormalities on the initial QTT examination. Thermal anesthesia had disappeared on all locations, although the threshold temperatures still exceeded 2 SDs on all locations in 2 patients and in the hand only in 1 patient. No concurrent relief of pain was reported by the 3 patients with central pain syndromes.

QTT follow-up was not performed in 5 patients who had SMS with sensory symptoms only initially and complete remission of symptoms and signs at 3 months after stroke onset. The initial QTT showed small (<2 SDs) but consistent side differences in thermal thresholds.

On the asymptomatic side, the initial examination revealed no statistically significant difference in thermal thresholds between patients with PSS, SMS, and PMS for the various sensory modalities or body parts tested. Thermal thresholds for all modalities on this side were also stable with time and did not differ by more than 0.6°C in any location in individual patients.

MRI Findings

MRI identified an infarct appropriate to the symptoms in 32 of the 39 patients (82.1%), who all developed characteristic changes in the T1- and T2-weighted sequences on the second MRI 1 year after stroke onset. Enhancement on MRI in the subacute stage was found in 25 of the 32 patients. In 7 patients a precise focal lesion to account for their symptoms could not be identified; 2 patients had multiple, nonenhancing lesions, 3 patients had confluent white matter lesions, and in 2 patients no infarct in possibly relevant areas was seen.

A relevant infarct was visualized in 9 of the 10 patients with PSS. They were all located in the postero-lateral part of the thalamus, in 1 case also extending into the posterior part of the internal capsule. In patients with SMS, a symptomatic infarct was identified in 9 of 12 patients; 8 patients had infarcts in the posterior part of the internal capsule (often also with involvement of the adjacent putamen, and in 2 cases also extending into the lateral part of the thalamus, probably due to edema in the acute phase), whereas 1 patient had a centrum ovale infarct. A symptomatic infarct was seen in 14 of 17 patients with PMS. There were 4 pontine infarcts and 1 centrum ovale infarct. The remaining 9 infarcts were located in the same area as the majority of the SMS infarcts, ie, in the posterior capsule and the putamen. Four of the larger PMS infarcts extended into the corona radiata and the periventricular white matter.

A schematic representation of the symptomatic infarcts for the different lacunar syndromes is provided in Fig 2. Whereas involvement of the thalamus was characteristic for patients with PSS, sites of the infarcts in patients with PMS and SMS were largely similar. Mean diameters of the lesions, measured on the initial MRI, were 9 mm for patients with PSS (n=9), 15 mm for patients with SMS (n=9), and 16 mm for patients with PMS (n=14).
Thalamic infarcts predominated among the patients with the most pronounced QTT abnormalities. All patients with side differences in thermal thresholds >2 SDs had infarcts located in the thalamocapsuloputaminal area. Among the 5 patients with thermal anesthesia within the temperature range limits, 3 had a thalamic infarct, 1 a capsular infarct, and 1 a capsuloputaminal infarct. Otherwise, we saw no correlation between the thalamic infarct site on MRI and the severity of QTT changes.

Discussion

This is the first study on QTT in patients with presumed lacunar infarcts. QTT has mostly been used in the assessment of metabolic and traumatic neuropathies, but the technique is also applicable to the study of central thermal pathways according to a few reports. However, data on thermal thresholds in healthy control subjects are limited in that they are based on small series of younger patients, and therefore they are not directly applicable to our material. Comparisons with previously published data may also be hampered by possible differences in the testing procedure and by the type of patients tested. For these reasons, we used the unaffected contralateral side as control and were careful to standardize the testing procedure. It is unlikely that cognitive and perceptual disturbances affected our results, since our patients were all alert and did not have any signs of higher cerebral dysfunction on clinical testing.

Our findings confirm an involvement of spinothalamic-mediated sensory modalities in lacunar infarcts causing PSS and SMS. In the study by Leijon et al on 27 patients with central poststroke pain, which included two infarcts confined to the thalamus, all patients had clinical and QTT findings of reduced thermal sensibility. SEPs were investigated by Robinson et al in 15 PSS and SMS patients who had sensory abnormalities involving dorsal column modalities on clinical examination. SEPs were abnormal only in 4 of the patients with SMS who had larger infarcts on computed tomography (CT), which suggests that QTT may be a more sensitive test than SEPs for detection of sensory impairment in patients with lacunar infarction.

The fact that 5 of 9 patients with PSS or SMS and a partial distribution of sensory loss on clinical testing demonstrated reduced thermal perception on all three locations on QTT indicates that additional subclinical sensory impairment sometimes is present. Therefore, distinguishing between complete and partial syndromes based on sensory findings on clinical examination alone might be hazardous.

In addition, QTT revealed a sensory impairment of the spinothalamic type on the hemiparetic side in patients with PMS. Consistent side asymmetries of thermal thresholds were found for all sensory modalities in all locations except for perception of warmth in the hand; they were statistically significant for cold and heat pain in the hand and for cold perception in the leg. The less pronounced side asymmetries in warmth detection thresholds might be due to the low and variable distribution of warm receptors compared with cold and pain receptors in older healthy subjects. Involvement of lemniscal pathways in PMS has been demonstrated in previous studies by means of SEPs. An involvement of sensory modalities in PMS is also suggested by the experience of transient or even permanent sensory symptoms reported by many patients. Sensory symptoms were reported by 3 of our 17 patients with PMS; frequencies in previous studies have ranged from 9% to 42%.

On MRI, the infarcts causing both PMS and SMS were predominantly located in the posterior part of the internal capsule and the adjacent putamen. The only differences were that the larger PMS infarcts had a rostral extension and that a thalamic involvement was seen in some of the SMS infarcts. Similar topographic patterns have been reported in previous CT studies and are supported by the notion that corticospinal and thalamocortical pathways are thought to pass in close connection through the posterior limb of the internal capsule.

Contrary to other studies, we did not find that lesions in SMS were larger than in PMS. However, this may be due to the limited number of patients in our series. Neuropathological studies of lacunar infarcts are few. PMS infarcts due to occlusion of deep penetrating arteries have been demonstrated at different sites along the pyramidal tract, most frequently in the pons and the posterior capsule, in accordance with our findings, while mostly thalamocapsular lesions have been found in the few SMS infarcts investigated.

The nine PSS infarcts visualized on MRI were all located in the posterolateral part of the thalamus. A thalamic site of PSS infaracts has also been most frequently reported in previous CT series and in the few cases examined neuropathologically. However, CT often fails to visualize the lesion sites in patients with PSS. Kim has reported a series of 21 PSS patients in whom the lesion sites were identified by CT or MRI. Twenty of the patients had PSS due to a presumed lacunar infarct; 11 patients had thalamic infarcts, 2 had pontine infarcts, and 7 patients had infarcts located in the lenticulocapsular region or the corona radiata. Hommel et al have also described MRI-verified pontine lacunae causing PSS, and small capsular lesions have been demonstrated by autopsy and by MRI. Thus, PSS can be caused by small infarcts at different sites along the sensory pathways but predominantly in the thalamocapsular area.

The prognosis of sensory impairment in SMS patients was favorable in our series. At 12 months half of the SMS patients did not have any sensory residua, whereas half of the patients had only minor symptoms and signs. However, 3 of the 10 patients with PSS developed central poststroke pain, which was severe in 2 cases. On QTT thermal perception generally improved with time, although at 1 year after stroke onset a statistically significant reduction of warmth and heat pain perception in the hand and leg was still present.

Our study suggests a gradual transition from intact sensibility to subclinical and to clinically apparent sensory dysfunction in patients with lacunar infarct syndromes. Conceivably, small differences in extension of infarction or edema in the thalamocapsulolenticular area can cause variations in motor and sensory involvement. The timing and extent of the clinical examination also must be considered. We found QTT to be a useful technique to show sensory impairment of the spinothalamic type even in small lacunar infarcts and to assess the evolution of sensory dysfunction with time. The QTT abnormalities were most pronounced in the hand,
and registrations from this location are probably sufficient for most screening procedures.

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