Light Touch Thresholds in Normal Persons and Cerebral Vascular Disease Patients: Bilateral Deficit After Unilateral Lesion

JUNE PERRY ESSING, M.S., R.P.T., JEROME W. GERSTEN, M.D., AND PHILIP YARNELL, M.D.

SUMMARY Sensory thresholds, using the 2-alternative forced choice technique, were studied in normal subjects and in patients with unilateral cerebral vascular disease, as documented by clinical-radiological correlation. In normal persons the absolute threshold for light touch increased with age. Bilateral impairment of sensation with unilateral cerebral vascular lesions occurred in 30% of the patients. Tactile deficit was always greater on the side contralateral to infarction or hemorrhage. An ipsilateral deficit did not exist independent of contralateral deficit, and was more likely to occur with complete sensory loss contralaterally. There was a tendency for persons with larger lesions as determined clinically and by CAT scan to have ipsilateral as well as contralateral deficit. The incidence or severity of ipsilateral deficit was not related to lesion laterality.

LOSS OR DECREASE of cutaneous sensory perception frequently accompanies cerebral infarction and hemorrhage. The assumption is usually made that the patient who has an infarction of a cerebral hemisphere has a sensory/motor deficit limited to the contralateral side. The concept of contralateral involvement resulting from a unilateral hemisphere lesion was mentioned early in the nineteenth century and a unilateral cerebral lesion was believed to cause only a contralateral tactile sensory deficit. This idea has persisted to the present, with the clinical literature identifying a “normal,” “uninvolved,” or “sound” side.

During the early nineteen hundreds there were several reports describing patients with left hemisphere lesions who had bilateral impairment in stereognosis. It was concluded that the left hemisphere mediated higher sensory performances, such as stereognosis, bilaterally, while more simple sensory processes were controlled contralaterally. Bychowsky and Eidinow, and Korner, described a bilateral deficit in light touch thresholds and pin prick in patients with unilateral cerebral lesions. Semmes et al. found that patients with unilateral brain injury at times had bilateral sensory defect. This finding has been supported by Vaughn and Costa, Corkin et al., Carmon, Boll, Gainotti, and Fontenot and Benton. Semmes finding that left hemisphere lesions produced bilateral sensory deficits more frequently than did right hemisphere lesions has been confirmed in some studies, but not in the others. In studies by Korner, Corkin et al., Carmon, Gainotti, and the present one, no significant difference in the prevalence of an ipsilateral deficit was found in comparing lesions of the right and left cerebral hemispheres. Some investigators report that a right hemisphere lesion was more apt to produce a bilateral sensory deficit.

The possibility of a bilateral sensory deficit for light touch following a unilateral hemisphere lesion has important prognostic and treatment implications. Experimental and neurophysiological evidence for bilateral somatosensory representation, possibly in SII area of the cerebral cortex, exist. More definitive information was sought to define tactile deficits in patients with clinically and radiologically determined unilateral hemispheric infarction or hemorrhage. In order to minimize the effects of motivational variables the 2 Alternative Forced Choice technique (2AFC), using 2 auditory signals and a tactile stimulus, was employed. This technique is believed to be superior to former psychophysiological techniques in obtaining thresholds. Light touch thresholds were studied in normal subjects of varying ages to provide a comparison with patients who had cerebrovascular disease.

Method

Normal Subjects

Subject Selection

Fifty-five normal human subjects were studied. There were 5 men and 5 women in each 10-year age range from 20 to 70, and 2 men and 3 women in the 70 to 80-year range. Only persons without history of neurological deficit were included. Nine of the subjects were left-handed for writing, while only 2 of the 55 persons were strongly left-handed for all activities.

Patients with Stroke

Thirty patients who had a unilateral hemisphere lesion resulting in a hemiparesis from a cerebral vascular accident (CVA) — either thrombus, embolus, or hemorrhage — took part in this study. The patients had their most recent stroke one month to 4 years prior to the test. A computerized axial tomography (CAT) scan, radionuclide scan or arteriogram was used to establish that there was only a unilateral hemisphere lesion. Patients were excluded from the...
study if the brain scans were normal (thus not confirming a unilateral hemispheric lesion) or if there was clinical or neuroradiologic evidence of bilateral hemisphere and/or brain stem involvement. Subjects who had sustained a prior cerebral vascular event in the same hemisphere as the episode under study were eligible. Subjects with a history of alcohol or drug abuse, head injury or other illness associated with diffuse neurological disease, were excluded. Diffuse or local upper extremity symptoms and signs of preexisting sensory dysfunction, as with symptomatic diabetes or nerve compression syndromes, disqualified patients. Those patients who could not understand and follow directions, or who could not hear well, were also excluded.

Persons with aphasia were included if they could understand and respond, even if only with gestures. There were patients with dysphasia who met all of the above criteria, but in the course of testing proved unable to perform and were excluded. Patients were excluded who had too short an attention span, who could not sequence the auditory signal with the tactile stimulus, who had severe apraxic-aphasic deficits, and who extinguished the tactile stimulus with the auditory one.

Instrumentation

The instruments used for determining thresholds were designed to be portable. They included a battery powered speaker, modified von Frey hairs (fig. 1) and a cylinder device (fig. 2). Two clicks were presented in the midline from a speaker mounted on a stand or on top of the bed behind the subject's head. The examiner controlled the first click by a foot pedal. The second click followed automatically 4 seconds (± 0.04 sec) later.

The light touch stimulus was presented using a modified von Frey device (fig. 1) which consisted of 4 wires, each of a different diameter and degree of stiffness. The wires were mounted separately in 4 plastic handles. Each of the von Frey wires was calibrated and covered the following ranges: 0–50 mg with 5 mg intervals, 0–200 mg with 20 mg intervals, 0–1.5 grams with 100 mg intervals, and 0–5 grams with 500 mg intervals. A plastic resin drop was placed on the end of each wire to assure that each stimulator had the same diameter and configuration. An adjustable metal arm on the device was used to bend the wire and set the pressure. The stimulus was applied until the wire was lifted slightly off the metal arm thus assuring that the desired amount of pressure was delivered to the skin. An effort was made to keep the wire perpendicular to the skin.

Another device was used to apply a touch stimulus in the 3–30 gram range. This consisted of a cylinder with a spring loaded plastic rod (fig. 2). The rod was rounded on the end and the pressure was calibrated from 3 to 30 grams with one gram intervals. The stimulus was applied to the hand with the probe perpendicular to the skin. The examiner practiced applying the stimulus on a strain-gauge force transducer, in order to control duration and rate of application.

Test Procedure

The threshold detection of a light touch was determined on the dorsum web space between the first and second digits of both hands in a region innervated by the radial nerve, using a randomized 2-alternative forced-choice test. The area to be tested was shaved one-half hour prior to testing. Subjects were either seated in a comfortable chair or were supine on a bed in a quiet, normal temperature room. The patient was carefully instructed in the procedure to be followed, and trial tests were given until responses were reliable.

The first touch was a force of approximately 5 grams. The force was decreased until the subject no longer felt the touch. The force was increased one interval (either 500 mg, 100 mg, 20 mg, or 5 mg, depending upon which instrument was used), until the touch was felt again. The 2-alternative forced-choice method was then introduced. The stimulus was applied either after the 1st or 2nd of 2 clicks. The subject was required to state after which click the touch was felt. Language deficient subjects responded by raising one or 2 fingers.

A series consisted of 10 touches using one of 4 sets of randomized tests. The touch was presented at the force previously established as being slightly above the subject's threshold. If the subject got 6, 7 or 8 correct then 15 more touches were applied at the same force. If fewer than 6 touches were correctly identified then a greater force was applied for another 10 trials. If more than 8 were correct then a lighter force was used for 10 more trials. The series of touches was continued until 60 to 88 percent of 25 touches were correct. The threshold was defined at 75 percent correct responses and was corrected to this value for statistical use.

A test-retest was done on each hand of the normal

![Image 1](http://stroke.ahajournals.org/)

**Figure 1.** Device used for applying up to 5 grams of force.

![Image 2](http://stroke.ahajournals.org/)

**Figure 2.** Device used to apply force between 3 and 30 grams.
subjects using 4 sets of randomized tests for each subject. Correlation coefficient for test-retest was 0.90. A retest was not done on the patients with cerebral vascular disease as the test itself lasted 45 minutes and most could not tolerate the additional 45 minute retest.

Results

Normal Subjects

Light touch thresholds of 55 normal subjects are depicted in figure 3, each point representing one subject's threshold. Paired t-tests were done for threshold values between right and left hands, test-retest, and between non-dominant and dominant hands. No significant differences were found and the data were therefore pooled. There was a tendency for mean touch threshold and variability on test-retest to increase with age. Although the mean touch threshold did increase from 20 to 60 years, this increase was not statistically significant. The mean threshold for the 60-70 age group was significantly higher than thresholds for the groups from 20 to 50 years, while the mean threshold for the 70-80 age group was significantly higher than mean thresholds of all younger groups.

When the thresholds for the total population of males vs females were compared (t-test), no significant sex difference was found. When comparisons were made within each decade there was no significant difference between the male and female subjects except for the 60 to 70 age group in which the males had higher thresholds. Within this decade the females were almost uniformly younger than the males.

Hemiparetic Patients

The data of the 30 patients with hemiparesis who had had a unilateral hemispheric lesion are summarized in figure 4. As expected, in most patients the side contralateral to the lesion (hemiparetic side) showed a dramatic increase in the threshold, with a mean of 16.64 grams and a standard deviation of 15.05 grams. A subject who was completely anesthetic was considered to have a threshold of 30 grams. If the 12 anesthetic patients are excluded, the mean threshold was 7.73 grams with a standard deviation of 13.29 grams. Six of the 30 hemiparetic patients had light touch sensitivity contralateral to the lesion comparable to that of the normals. There was no consistent location of lesion in those 6 subjects, but the lesions tended to be smaller as seen on the CAT scan.

Comparison of light touch thresholds for the non-paretic hand of patients with stroke and hands of normal subjects (table 1, fig. 5) shows that the thresholds of the non-paretic hand were significantly higher \( (p = 0.001) \). When examined by decades, differences between patients with stroke (non-paretic side) and normal subjects did not reach significance in the 20 to 29, 50 to 59, and 70 to 79 year-old age groups, though the mean threshold was always higher in the non-paretic hands of the patients.

There was no significant difference in thresholds for non-paretic hands between patients with dominant versus non-dominant hemisphere lesions. Patients who were tested from 1 month to 1 year post stroke had a mean threshold of 156 mg, while those tested from 1 year to 4 years post stroke had a mean threshold of 123 mg. This difference was not statistically significant and the relation between threshold and time-since-stroke was not explored systematically.

Nine of the 30 hemiparetic patients had thresholds on the non-paretic hand at least 2 standard deviations above the mean of the normal subjects for their age group. Of those 9 subjects, 6 had fronto-temporal-parietal lesions. One had a left parietal lobe lesion in the region from the superior margin of the lateral ventricle to the surface of the brain. One had a temporo-parietal lesion, while another had a moderate size frontal lesion with a smaller parietal lesion. A chi-square test was done comparing the severity of non-paretic hand deficit (light touch sensitivity loss either
FIGURE 4. Light touch thresholds in paretic hand of stroke patients (dots); non-paretic hand of stroke patients (solid line); and normal subjects (broken line).

FIGURE 5. Light touch thresholds in normal subjects (broken line) and non-paretic hand of stroke patients (solid line). Bars represent ± 1 SE.

greater or less than 2 standard deviations above the mean for normal subjects) to degree of sensory loss in the paretic hand. The results showed that a patient who had no feeling on the contralateral (paretic) hand was more likely to have a high threshold on the ipsilateral (non-paretic) hand (table 2). All patients who had significant ipsilateral tactile loss also had greater contralateral deficit.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Normal Hands</th>
<th>Non-paretic Hand</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ave. Thresh. (mg)</td>
<td>S.E. (mg)</td>
</tr>
<tr>
<td>20-29</td>
<td>47</td>
<td>9</td>
</tr>
<tr>
<td>30-39</td>
<td>54</td>
<td>5</td>
</tr>
<tr>
<td>40-49</td>
<td>62</td>
<td>9</td>
</tr>
<tr>
<td>50-59</td>
<td>80</td>
<td>21</td>
</tr>
<tr>
<td>60-69</td>
<td>100</td>
<td>15</td>
</tr>
<tr>
<td>70-70</td>
<td>199</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>80</td>
<td>55</td>
</tr>
</tbody>
</table>
Table 2 Relation Between Sensation in Paretic Hand and Threshold in Non-Paretic Hand

<table>
<thead>
<tr>
<th>Threshold on Non-Paretic Hand</th>
<th>No Feeling at 30 g on Paretic Side</th>
<th>Some Feeling on Paretic Side</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>More than 2 S.D. above the mean threshold of normal subjects</td>
<td>7</td>
<td>2</td>
<td>9</td>
</tr>
<tr>
<td>Less than 2 S.D. above the mean threshold of normal subjects</td>
<td>7</td>
<td>14</td>
<td>21</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>16</td>
<td>30</td>
</tr>
</tbody>
</table>

\[ x^2 = 6, p < 0.05. \]

Discussion

This study demonstrates that a unilateral cerebral hemisphere lesion can result in an ipsilateral deficit in light touch thresholds and supports the results obtained by Corkin et al.,\textsuperscript{10-12} Carmon,\textsuperscript{13} and Gainotti.\textsuperscript{15} Corkin reported that 40 percent of the patients studied had an ipsilateral deficit in point localization. Carmon found that 39 percent of his patients had an ipsilateral defect in absolute pressure sensitivity. Gainotti found that 20 percent of his patients had an ipsilateral deficit for 2 point discrimination. In the present study 30 percent of the patients with a unilateral stroke had an ipsilateral deficit in light touch thresholds more than 2 standard deviations above the average threshold for the normal population of the same age group.

In these studies there were some differences in patient population. Corkin et al.\textsuperscript{10} tested patients with surgical incision in the treatment of epilepsy. Carmon\textsuperscript{13} and Gainotti\textsuperscript{15} included patients with tumor and vascular disease, and a few with brain trauma.\textsuperscript{15} The present study was limited to persons having a unilateral hemispheric stroke. All of our 30 patients had some brain damage in the distribution of the middle cerebral artery, both clinically and radiologically.

The present study did not support the findings of Semmes\textsuperscript{5} or Vaughn and Costa,\textsuperscript{6} who found ipsilateral involvement more frequently with a dominant hemisphere lesion. Nor does this study confirm Boll’s findings of ipsilateral involvement most frequently in right hemisphere lesions.\textsuperscript{14} We are in agreement with others\textsuperscript{10-12, 15} in finding no difference in lesion laterality for an ipsilateral tactile defect. This difference could be due to examination in our study of only a simple sensory process, compared to others who used a spatial quality in the test.\textsuperscript{14, 16}

Differences in patient population were also apparent in these and other studies. Semmes studied persons with penetrating brain wounds,\textsuperscript{5} and Vaughn and Costa\textsuperscript{6} patients with “unilateral focal lesions.” Boll\textsuperscript{14} studied patients with “unilateral cerebral disease,” but did not describe the method for eliminating patients with bilateral or brain stem damage. Clinical evidence alone does not seem sufficient for identification of site and numbers of lesions. In the present study, several patients who passed the clinical neurological screening for unilaterality had to be excluded from the study because the neuroradiologic scans showed bilateral brain damage or were normal. Vaughn and Costa used radiological evidence, but the CAT scan was not then available.

Neuroradiologic clinical correlations were most helpful in defining the site and extent of the cerebral vascular lesions, and in selecting the patients for our study. CAT scans defined unilateral hemispheric disease in 77 percent of our patients. Eight of the 9 patients with ipsilateral touch deficits greater than 2 standard deviations had CAT scan localized lesions. The ninth was localized by radionuclide scan. Scans are a valuable investigative agent for clinical-anatomic correlation in cerebrovascular disease;\textsuperscript{24} however, the overall detection rate for ischemic infarction ranges from 58 percent in radionuclide scanning to 66 percent for the delayed CAT.\textsuperscript{25} Both the radionuclide and CAT scans show evolving pictures over time in terms of detection and size definition of cerebrovascular lesions.\textsuperscript{25-27} No definitive conclusion could be made as to lesion size and extent because of 1) the variable time period between the insult and the often evolving positive neuroradiologic studies, and 2), the variation in scanning technics in our patients, representing several different referral sources. Despite this lack of standardization, it appeared that patients with more extensive unilateral lesions had higher ipsilateral touch thresholds. Corkin\textsuperscript{10} had also suggested that lesion size was a determining factor in ipsilateral sensory deficit. This general correlation between scan lesion size and tactile deficit has been confirmed in studies of other cerebral defects.\textsuperscript{25, 28}

The CAT and radionuclide scans were useful in excluding patients with large bihemispheric lesions. Alternatively, some patients with clinically unilateral hemispheric vascular disease were not accepted into the study when subsequent neuroradiologic correlations were not definitive which may have excluded patients with smaller, deep, or scan resolved lesions. We hoped, by these criteria, to make the hemispheric unilateral component of the study most rigorous.

Another difference between this study and previous ones is the use of the 2-alternative forced choice technique for examination of sensation. This is a rigorous way of examining sensation and is believed to be superior to former psychophysical techniques.\textsuperscript{29} However, inherent in this method is the requirement for central processing. It is possible that spatial or temporal sequencing deficits could have affected our findings, though we tried to exclude patients with cognitive difficulty in performing the test. This may not be completely avoidable in this testing procedure. Whether this testing method could account for the observed differences from others’ results is not known.

Since tactile thresholds increased as a function of age in the normal subjects studied, it becomes important to compare brain damaged patients with controls in the same age bracket.
Clinical Implications

There are rehabilitation implications for a patient with a unilateral hemisphere lesion with an ipsilateral as well as a contralateral tactile deficit. Lack of accurate touch feedback can hamper the re-training of sensory motor function. Even if the ipsilateral deficit is only slight, light touch feedback possibly may be distorted enough to impair function. Thus it may decrease the effectiveness of a variety of facilitatory or inhibitory techniques used in rehabilitation, such as tapping, stroking, or pressure. Impaired function might appear as failure in fine skilled activities of the non-paretic hand, or as awkwardness in gross motor activities. Greater touch pressure, and/or other modalities involving touch stimulus on both sides of the body, or selective work in separate sessions with each side might be needed for rehabilitation training. Therefore, rehabilitation techniques should consider that there may not be a “normal” side for touch in some patients with unilateral vascular disease.

References
Light touch thresholds in normal persons and cerebral vascular disease patient: bilateral deficit after unilateral lesion.
J P Essing, J W Gersten and P Yarnell

Stroke. 1980;11:528-533
doi: 10.1161/01.STR.11.5.528
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1980 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/11/5/528

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/