

# Hemispheric Dominance for Stereognosis in a Patient With an Infarct of the Left Postcentral Sensory Hand Area

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**Abstract:** The concept of left hemispheric dominance for praxis, speech, and language has been one of the pillars of neurology since the mid-19th century. In 1906, Hermann Oppenheim reported a patient with bilateral stereoagnosia (astereognosis) caused by a left parietal lobe tumor and proposed that the left hemisphere was also dominant for stereognosis. Surprisingly, few cases of bilateral stereoagnosia caused by a unilateral cerebral lesion have been documented in the literature since then. Here we report a 75-year-old right-handed man who developed bilateral stereoagnosia after suffering a small infarct in the crown of the left postcentral gyrus. He could not recognize objects with either hand, but retained the ability to localize stimuli applied to the palm of his left (ipsilesional) hand. He was severely disabled in ordinary activities requiring the use of his hands. The lesion corresponded to Brodmann area 1, where probabilistic anatomic, functional, and electrophysiologic studies have located one of the multiple somatosensory representations of the hand. The lesion was in a strategic position to interrupt both the processing of afferent tactile information issuing from the primary somatosensory cortex (areas 3a and 3b) and the forward higher-order processing in area 2, the secondary sensory cortex, and the contralateral area 1. The lesion also deprived the motor hand area of its afferent regulation from the sensory hand area (grasping), while leaving intact the visuomotor projections from the occipital cortex (reaching). Our patient supports Oppenheim's proposal that the left postcentral gyrus of some individuals is dominant for stereognosis.

**Key Words:** astereognosis, sensory hand area, hemispheric dominance, tactile object recognition, tactile agnosia

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**BA** = Brodmann area. **S1** = primary sensory cortex. **S2** = secondary sensory cortex.

**A** brain function is said to be *dominant* if a unilateral lesion produces a behavioral deficit that extends across both sides of the body or a personal space. To be strongly *lateralized*, the behavioral deficit must be shown *not* to occur after lesions to the opposite hemisphere (Ross, 1997).

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The concepts of left hemispheric dominance and laterality for praxis (performing an action) and for the propositional aspects (intending to convey ideas) of language and communication have stood as pillars of neurology and neuroscience since the mid-19th century (Broca, 1861; Liepmann, 1905; Wernicke, 1874). The modality-specific sensory and motor systems, in contrast, are usually thought to have a symmetric cortical representation. However, sporadic exceptions to this rule of symmetry have been documented in the somatosensory system.

At its simplest, *stereognosis* is the perception of the three-dimensional form of objects through active touch. The loss of the stereognostic sense, with preservation of the elementary somatosensory submodalities (such as proprioceptive and thermal), is called *astereognosis*. For etymological reasons (Littré and Gilbert, 1905, p. 1584), in this report we have chosen instead to use the term *stereoagnosia* (from the Greek *stereo* = solid + *a* = deprived of + *gnosia* = knowledge). The same principle of word formation applies, eg, to *prosopagnosia* (not *aprosopagnosia*).

In the sense that we are using the term *stereognosis* in this paper, we must differentiate it from the term *tactile object recognition* both phenomenologically and neurologically. Patients with tactile agnosia lose the ability to recognize an object by active touch, but their ability to perceive form (stereognosis) and texture is by definition spared (Gerstmann, 1918).

In his seminal work on tactile perception, Jean Delay (1935) distinguished the ability to perceive the tridimensional form of objects, which he called *morphognosis* (from the Greek *morphē* = form)—thus avoiding the term *stereognosis*, which by that time was no longer precise—from *hylognosis* (from the Greek *hyle* = matter, substance), which is the ability to feel the texture of objects. These respectively spatial and molecular dimensions of the sense of touch provide the grounds for tactile object recognition (Johnson and Hsiao, 1992).

That tactile object recognition has distinct neural correlates has been demonstrated by patients in whom stereognosis and hylognosis are spared but tactile recognition is lost (Nakamura et al, 1998). By the same token, tactile object recognition must be distinguished from tactile object naming (Endo et al, 1992). It would be beyond the scope of this report for us to try to cover the broad literature on tactile object recognition and naming.

Hermann Oppenheim (1906) was the first to report a case of bilateral stereoagnosia resulting from a unilateral hemispheric lesion (Table 1). Oppenheim's patient complained of the gradual onset of numbness in the right hemibody, culminating in right-side weakness and bilateral stereoagnosia;

**TABLE 1.** Reported Clinicoanatomic Cases of Bilateral Stereognosis Caused by a Unilateral Hemispheric Lesion

Authors, Date	Sex, Age	Lesion Characteristics
Oppenheim (1906)	Man, not reported	Tumor of left postcentral gyrus and parietal lobe extending into subcortical white matter
Goldstein (1915)	Man, 53	Cystic tumor of left postcentral gyrus and inferior parietal lobule
Lhermitte and Ajuriaguerra (1938)	Woman, 75	Cystic tumor of left thalamus and white matter of postcentral gyrus, inferior parietal lobule, insula, and superior temporal gyrus
Okuda et al (1995) Case 1	Not reported	Infarct of left supramarginal gyrus and centrum semiovale
Okuda et al (1995) Case 2	Not reported	Infarct of left postcentral gyrus extending to supramarginal gyrus
Moll and <i>de Oliveira-Souza</i> (2017) (this patient)	Man, 75	Small infarct in left postcentral gyrus (Brodmann area 1)

his language and visual object recognition were intact. Surgical removal of a slowly growing tumor involving the left postcentral gyrus and parietal lobe led within a few days to a complete resolution of the stereognosis of the patient's left hand. Invoking Hugo Liepmann's then recently advanced concept of a left parietal dominance for praxis (Liepmann, 1905), Oppenheim proposed that the left hemisphere might likewise be dominant for stereognosis.

For some reason that we still do not understand, few case studies of bilateral stereognosis caused by a unilateral hemispheric lesion have been published since Oppenheim's original paper. Among those reports, Goldstein (1915) described a 53-year-old man with a slowly progressive syndrome of bilateral stereognosis, apraxia, mild right hemiparesis, and aphasia. Autopsy revealed a large cystic cavity occupying the left postcentral and supramarginal gyri and extending into the subcortical white matter, but without involvement of the corpus callosum or the right hemisphere (Goldstein, 1915).

Foix (1922) reported four patients with a similar syndrome consisting of somatosensory impairments in both hands, most severe in the proprioceptive and stereognostic domains, associated with varying degrees of aphasia, ideomotor apraxia, and right hemiparesis. Unfortunately, the author did not obtain postmortem confirmation of the site of damage in his patients.

Lhermitte and Ajuriaguerra (1938) reported an elderly nondemented woman with a constant and uncomfortable feeling of numbness in her hands. These symptoms began subacutely at the same time that she lost the ability to recognize objects with either or both hands. All of her elementary sensations were normal—proprioception, exteroception, and topognosis—and she could easily describe the physical properties of items that she examined by active touch. Postmortem exam revealed a cystic tumor of the left hemisphere occupying the thalamus and white matter of the subcortex of the postcentral gyrus, inferior parietal lobule, insula, and superior temporal gyrus, all of which were atrophic. Notably, the basal ganglia and internal capsule were spared. Although this case supports the conclusion that the patient's stereognosis was strongly lateralized to the left hemisphere, the size of her tumor precluded an exact clinicopathologic correlation.

A report by Okuda et al (1995) provides the best-controlled investigation of Ross's (1997) argument for the possibility of a left hemisphere dominance and laterality for stereognosis. The authors studied four patients whose

hands had become clumsy because of nonprogressive vascular lesions of the postcentral gyrus. The two patients with a left hemisphere lesion showed bilateral sensorimotor impairments that were more severe in the right hand than the left, while the two patients with a right hemisphere lesion had impairments in only the left hand.

This finding, however, has not been the rule in studies of hand function in normal volunteers (Reed and Caselli, 1994) and patients with monohemispheric stroke (Rossini et al, 2001), or in studies of the sensory organization of the hand in nonhuman primates (Iwamura et al, 1994). These studies have established that *each* hand projects to *both* primary sensory cortices, but much more massively to the contralateral hemisphere.

This pattern of projections is reflected in the hemisensory deficit that follows an injury to the central somatosensory pathways in human patients (Dejerine, 1893; Rossini et al, 2001). These patients have a reduction of sensation in the contralateral hemibody, but not hemianesthesia, meaning that the ipsilesional pathways have some residual function. These studies do not mention the possibility of hemispheric dominance or laterality for sensory hand function unless the stereognosis is accompanied by extraneous symptoms such as left hemineglect (Caselli, 1997). Moreover, no case of bilateral stereognosis from a right hemisphere lesion has ever been reported (Bauer and Demery, 2003).

Here we describe a patient with bilateral stereognosis caused by a discrete lesion in the crown of the left postcentral gyrus. To the best of our knowledge, this is the smallest reported lesion to cause bilateral stereognosis without hemineglect or other associated cognitive impairments. Our case also illustrates how much meaningful information can be obtained on functional neuroanatomy from the observation of selected patients using the methods of inquiry developed by the founders of neurology (Freeman and Okun, 2002).

## CASE REPORT

A 75-year-old right-handed man with “numbness in the hands” was referred to us at the outpatient neurology service of the Federal University of Rio de Janeiro, Brazil, in February 1998.

The patient had diabetes mellitus. At around age 50 he had lost the sight in his right eye from diabetic retinopathy, but he could still see well with his left eye. He had quit

smoking several years before the onset of his hand symptoms. He was a retired clerk who had 5 years of formal education.

On initial evaluation, he told us that his numbness had started suddenly 1 year earlier and had always been more severe in his right hand than his left. The symptoms had never improved.

He said that he had great difficulty performing daily instrumental activities that depended on unimanual actions or bimanual cooperation, such as putting his wallet in the rear pocket of his pants, tying his shoelaces, buttoning and unbuttoning his shirt, fixing small household items such as electrical sockets, and handling and using a hammer or a screwdriver. He had difficulty in writing even short notes because the pen would fall from his hand as he pressed it against the table. He increasingly avoided social events because he felt embarrassed about his problems using tableware and holding soft plastic cups without crumpling them.

On closer inquiry, what he called “numbness” was really a cramp-like sensation provoked by active hand movements, making his manual actions clumsy and stiff. He said that he had no unprovoked sensory symptoms such as tingling, pricking, burning, pain, true numbness, or pins-and-needles sensations.

We confirmed the patient’s complaints by observing how he performed several ordinary tasks. He could not adjust his hands to the shapes of common manipulable objects because he was unable to grade the strength of his grasp in response to their physical properties. As a result, he would let objects fall from his hands or would crush them by applying excessive force while handling them. His hand and finger movements were slow and arrhythmic, contributing to the clumsiness, stiffness, and hesitation of his manual actions.

Hand movements that did not depend on proximal support from his arms and shoulders (eg, twisting the cap off a soda bottle or using a bottle opener) were slow and of high amplitude, as if he were executing them in an atmosphere of high gravity. Conversely, movements of tactile exploration without the aid of vision were of low amplitude and made up of small, hesitating displacements. When blindfolded, he often made palpatory movements in a vacuum, sometimes expressing doubts about whether any object was in his hand at all; at other times, he insisted that he still held an object that he had just dropped.

With his eyes closed, he was unable to name or identify any of our 12 experimental objects by active touch with his right hand, but he recognized four of them with his left. He seemed to be guessing at his responses, which he gave slowly and tentatively.

He did not recognize numbers drawn with a sharp pencil on the palms of his hands. He had normal sensation to passive contact (superficial and deep), vibration (tested with a 128-Hz tuning fork), joint position, cold/warm, and pin-prick in his head, legs, arms, trunk, and, notably, his hands. He found passive repetitive light stimulation of the palm and dorsum of his hands to be quite unpleasant (allodynia). He felt no discomfort, however, when he actively rubbed his hands together or against rigid or coarse surfaces.

He was fully oriented to time and place (Brucki et al, 2003) (Table 2). He communicated well by language and gesture and he displayed keen insight into his symptoms and disabilities. On both verbal command and imitation, he correctly pantomimed both transitive gestures (mediated movements such as using tools) and intransitive gestures (movements intended to communicate or symbolize rather than perform an action using an object). He performed

**TABLE 2.** This Patient’s Neuropsychological Test Results

	Score
<b>Background Inventories and Tests</b>	
Oldfield Handedness Inventory <sup>1</sup>	100
Mini-Mental State Examination <sup>2</sup>	29/30
Florida Apraxia Screening Test <sup>3</sup>	14/15
Right-Left Orientation <sup>4</sup>	20/20
Enhanced Cued Recall Test (total score) <sup>5</sup>	48/48
Clock Drawing <sup>6</sup>	10/10
3D Block Construction <sup>4*</sup>	<b>12/29</b>
<b>Tactile Sensory Processing</b>	
Tactile Object Recognition <sup>7</sup>	
Right hand	<b>0/12</b>
Left hand	<b>6/12</b>
Tactile Form Perception <sup>4†</sup>	
Right hand	<b>0/10</b>
Left hand	<b>5/10</b>
Finger Localization <sup>4‡</sup>	
Hand visible: single fingers	
Right hand	10/10
Left hand	10/10
Hand hidden: single fingers	
Right hand	9/10
Left hand	10/10
Hand hidden: simultaneously touched finger pairs	
Right hand	<b>5/10</b>
Left hand	9/10
Total right hand	<b>24/30</b>
Total left hand	29/30
<b>Two-Point Discrimination<sup>8§</sup> (mm)</b>	
Right hand fingertips	
1	2
3	5
<b>30</b>	<b>22</b>
Left hand fingertips	
1	2
3	5
<b>9</b>	<b>3</b>
<b>8</b>	
<b>6</b>	
<b>Sensory Thresholds<sup>8  </sup> (monofilament area in mm)</b>	
Right hand fingertips	
1	2
3	3
<b>5.07</b>	<b>4.31</b>
Left hand fingertips	
1	2
3	3
<b>4.93</b>	<b>4.17</b>
<b>4.17</b>	<b>4.17</b>

**Bold type** indicates an abnormal result.  
 \*Normal  $\geq 19$  ( $t = 2.48$ ,  $P > 0.05$ , two-tailed).<sup>9</sup>  
 †Pattern D: bilateral asymmetric right hand defect, indicating left hemisphere damage.<sup>4</sup>  
 ‡Pattern F: unilateral right hand defect, indicating left hemisphere damage.<sup>4</sup>  
 §Normal  $< 3$  mm.<sup>9</sup>  
 ||1.65 mm  $\leq$  normal  $\leq 2.83$  mm (normative data provided by Lafayette Instrument Company, Lafayette, Indiana).  
<sup>1</sup>Oldfield, 1971. <sup>2</sup>Brucki et al, 2003. <sup>3</sup>Ochipa et al, 1989. <sup>4</sup>Benton et al, 1994. <sup>5</sup>Grober et al, 1988. <sup>6</sup>Mendez et al, 1992. <sup>7</sup>Caplan and Hollander, 2011. <sup>8</sup>Spren and Strauss, 1998. <sup>9</sup>Crawford and Garthwaite, 2002.

normally on the Florida Apraxia Screening (Ochipa et al, 1989), Right-Left Orientation (Benton et al, 1994), and Enhanced Cued Recall (Grober et al, 1988) tests. He did not show extinction on double simultaneous stimulation or evidence of neglect on the Clock Drawing test (Mendez et al, 1992).

On neuropsychological testing, the patient's only abnormal score was on the Three-Dimensional Block Construction test (Benton et al, 1994).

He had a restriction of upgaze and a slight bilateral hesitation in the final third of the finger-to-nose test, with his eyes closed. His posture and gait were consistent with his age. He did not have a Romberg sign, and he adjusted his posture normally on the push test maneuver. His knee jerks were symmetric (2+/4+), his Achilles reflexes were hypoactive (1+/4+), and his finger flexors were hyperactive and symmetric; his plantar reflexes were bilaterally flexor.

Magnetic resonance imaging (1.5T Philips Achieva, Philips Healthcare, Best, The Netherlands) revealed a focal lesion in the upper convexity (crown) of the postcentral gyrus with a low signal in T1-weighted spin-echo images (Figure 1, Panel A) and a high signal in T2-weighted turbo spin-echo images. In fluid-attenuated inversion recovery sequences, this region appeared hyperintense, indicating gliosis encompassing the cortex and underlying white matter of the crown of the postcentral gyrus (Figure 1, Panel B). The lesion was not visible one slice higher (Figure 1, Panel C), meaning that it had a height of at most 5 mm. Punctate foci of subcortical gliosis were also visible in the precentral and middle frontal gyri and in the left inferior parietal lobule.

The postcentral lesion had an irregular shape that roughly followed the contour of the postcentral gyrus, its long axis measuring 10.3 mm. Spared were the cortices of the anterior and posterior bank of the postcentral gyrus abutting the lesion. The injured cortex, which corresponds

to one of the sensory representations of the hand (Pizzella et al, 1999), is supplied by the anterior parietal artery, which is a leptomenigeal branch of the middle cerebral artery (Tatu et al, 1998).

Our patient's electromyogram and nerve conduction studies were normal (Appendix A). In particular, we found no clinical or electrophysiologic evidence of a carpal tunnel syndrome or a cervical radiculopathy (Appendix B).

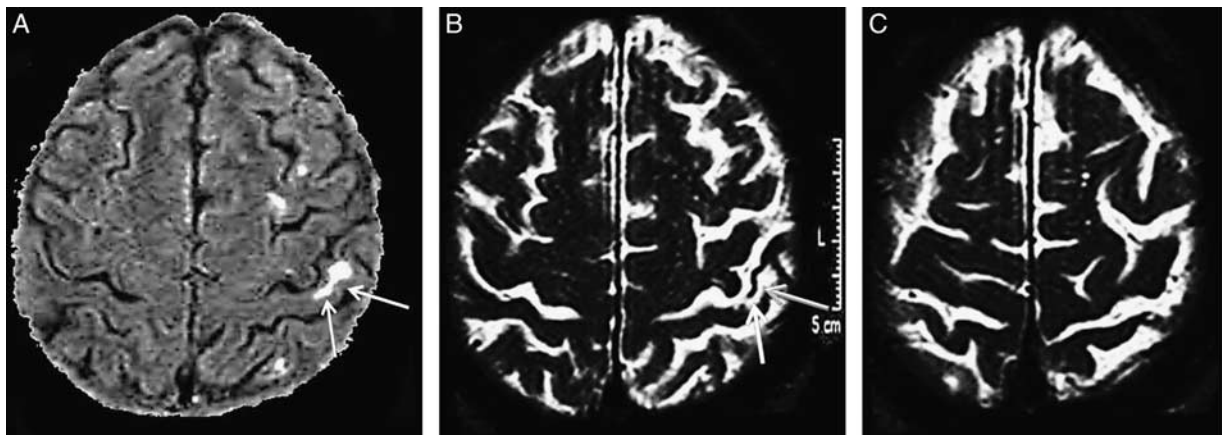
With the patient's written informed consent, we conducted the clinical assessment described in the next section to characterize his symptoms in greater detail. He also gave written informed consent for us to publish his medical information.

We followed him for the next few weeks, but, unfortunately, lost him to further follow-up.

### EXAMINATION OF THE PATIENT'S HANDS AND MANUAL ACTIONS

We conducted the clinical assessment in five sessions held about every 2 weeks over 2 months. We met with the patient in a quiet room during his routine visits to the outpatient service of the Federal University of Rio de Janeiro. Each of our sessions lasted about 4 hours, with as many rest periods as the patient needed to avoid fatigue. He was happy to cooperate in the testing, and most of the proposals for breaks came from us. The five sessions allowed us to examine him thoroughly, repeat tests as needed to document the reliability of the results, and clarify our remaining doubts.

For most of the tasks requiring that the patient's vision be blocked, we blindfolded him. For the others, we had him use a "cover box." This is a wooden box with a slanted top and open ends. The patient put his hand in one end and we inserted stimuli through the other end, so that he could touch the stimuli without being able to see them.



**FIGURE 1.** Anatomic magnetic resonance images showing an area of gliosis in the crown of the left postcentral gyrus, consistent with an infarct of the subcortical white matter of the somatosensory hand area. The images are T1-weighted (Panel A) and fluid-attenuated inverse recovery (Panels B and C). The lesion is marked by arrows in two views of the same slice (Panels A and B), but is not visible one slice higher (Panel C). Because it appears in only one slice, the infarct has a maximum possible vertical extent of 5 mm.

### Tactile Object Recognition (Caplan and Hollander, 2011)

For this task, we used 12 common objects: a cork, a coin, the patient's own wristwatch, a safety pin, a rubber ball, a paper cup, a metal spoon, a plunger, a washcloth, a soda bottle, a 30-cm plastic ruler, and a ballpoint pen. With the patient blindfolded, we put an object in one of his hands and then in the other, and asked him to identify the object. We considered him to make a positive identification if he either named the object or showed how to use it by words and/or gestures. We then uncovered his eyes and asked him to identify each object as we held it in front of him.

### Overlearned Routine Activities (Poeck, 1986)

We asked the patient to perform eight routine activities, using his vision. Each activity involved a sequence of actions that required him to manipulate tools, household objects, or his own clothes:

- Fasten his watch on his wrist
- Pick a cigarette from a pack, light it, and prepare to take a puff (we interrupted the action before the cigarette reached his lips)
- Lock and unlock a medium-sized padlock
- Twist off the cap of a bottle of soda, pour the soda into a plastic cup, and drink
- Unbutton and button his shirt
- Pull his wallet and other papers from the right pocket of his shirt
- Remove paper money from the left pocket of his pants
- Twist the cap off a small bottle of water with his left hand while holding the bottle with the right, and repeat the procedure switching hands

### Replication of Passive Poses in the Contralateral Hand (Dejerine, 1914)

We administered this task in two steps: first with the patient's eyes open and then after blindfolding him. The examiner manipulates one of the patient's hands (the "passive hand") into each of a sequence of five poses and asks the patient to imitate each pose with his other, "active" hand. We first tested his right hand and then repeated the sequence with his left hand.

Performed without the aid of vision, this maneuver probes an extended proprioceptive circuitry that departs from the segments passively manipulated by the examiner and crosses to the other hemisphere via callosal pathways. There the proprioceptive information ultimately informs the motor areas about the position of the manipulated parts. This allows the active hand to reproduce the poses imposed by the examiner. The test assesses proprioception in the *passive* hand, although, in principle, damage to any point in the circuitry can impair performance.

As with all tests of proprioception developed since Romberg (1853) described his famous maneuver, the elimination of visual feedback from the execution of motor actions leaves the correct performance of those actions exclusively dependent on patients' knowledge of the position of their

body parts in space, a position conveyed by proprioceptive afferents (Lanska and Goetz, 2000). When proprioception is impaired, as with our patient, the elimination of vision makes these manual actions difficult or impossible to perform.

### Thumb Localizing Test (Hirayama et al, 1999)

After blindfolding the patient, the examiner raises one of his arms (the *fixed* limb) and asks him to pinch the thumb of that hand with the opposite thumb and index finger (the *reaching* limb). We modified the task by starting with a practice session in which the patient performed the task with his eyes open.

This test is sensitive to deficits of localization of the fixed limb from lesions in the posterior column–medial lemniscal system at levels from the peripheral nerves, cervical cord, brainstem, and thalamus to the parietal lobe.

### Small Corks Test (Caplan, 1981)

The examiner blindfolds the patient and then places five cubic corks ranging in volume from 1 to 2 cm<sup>3</sup> first in one of his hands and then in the other. The patient is asked to hand the corks back to the examiner in order of size, the largest first. Because our patient was unable to handle the corks with either hand alone, we let him use both hands to perform the task. Over several testing sessions, we gave him this test at least seven times.

This test probes the ability to discriminate the relative sizes of small objects of equal texture and weight, thus requiring optimal function of the stereognostic sense.

### Direction of Scratch Test (Hankey and Edis, 1989)

With the sharp end of a wooden tongue depressor cut on its long axis, the examiner uses constant mild pressure to make 2-cm vertical scratches on the patient's skin. After blindfolding our patient, we made 10 scratches on each of seven regions of each hemibody: the anterior mid-tibia, halfway between the medial malleolus and the tibial tuberosity, mid-thigh, abdomen (8 cm lateral to the umbilicus), chest (4 cm above the nipple), arm (halfway between the ulnar styloid and the medial epicondyle), and cheek. Because of the involvement of our patient's hands, we extended the protocol to include 10 scratches on each of his palms.

As we made each scratch, we asked the patient to say whether the direction of the scratch was up or down. We recorded an error if he felt uncertain about the direction of the scratch or if he said the wrong direction.

Since we scratched each region 10 times, total hemibody scores could range from 0 to 80.

### Tactile Form Perception Test (Benton et al, 1994)

This nonverbal task tests the patient's ability to distinguish shapes by feeling changes in texture. The stimuli are two sets of 10 cards, sets A and B. Pasted on the surface of each card is a different flat geometric figure (eg, a circle, triangle, star) cut out of fine-grade sandpaper. From set A, we placed one card at a time in a cover box so the patient could not see it. We asked him to put his right hand inside the box, feel the figure, and determine the sandpaper shape.

Sitting on top of the cover box was a multiple-choice response card containing line drawings of 12 figures: the 10 figures shown on the stimulus cards plus an extra figure for practice before the test began and one more as a confounder. We asked the patient to look at the response card and use his left hand to point to the figure that matched the sandpaper shape he had felt inside the box.

After we tested his right hand with all 10 cards from set A, we tested his left hand with each of the 10 cards from set B and we asked him to point to the correct figure with his right hand.

One point is scored for each correct response, making a maximum score of 10 for each hand.

This test is sensitive to impairments in the spatial synthesis that underlies the perception of form through active touch (Dee and Benton, 1970).

### Finger Localization Test (Benton et al, 1994)

This nonverbal test assesses patients' ability to localize individual fingers on each hand in response to tactile stimulation. The examiner touches the tip of one of the patient's fingers with the sharp end of a pencil. On a card showing an outline drawing of the stimulated hand, the patient points to the finger being touched.

The test has three parts, each with 10 trials. In Part I, the patient can see his hand as the examiner touches it. In Part II, the hand is hidden from view inside a cover box. In Part III, the hand remains hidden from view and the examiner touches *two* of the patient's fingers simultaneously. The maximum score for each hand is 30.

A variety of abnormal patterns have been described in brain-damaged patients, depending on lesion location (see Table 2 footnotes for examples). The test is sensitive to impairments of perception of restricted body parts.

### Two-Point Discrimination (Spreen and Strauss, 1998)

We used a simple sharp-pointed caliper to determine the patient's two-point sensory threshold in the tips of fingers 1, 2, 3, and 5 of each hand, starting with the less impaired (left) hand. After being shown the basic testing procedures, the patient placed his open hand under a cover box with the palm up. Starting with the widest caliper distance, we touched each of his fingertips with one or both points. His task was to say whether the caliper had touched his fingertip in one or two places. We repeated the procedure with progressively narrower widths until he could no longer discriminate whether he was being touched in one or two places. Then we tested the more impaired right hand following the same procedure.

The test score for each fingertip is the sensory discrimination threshold, ie, the minimum distance (in mm) at which the patient perceives two caliper points.

The fingertips are the somatosensory equivalent of the retinal macula. As such, they contain the densest concentration of neural receptors specialized for extracting certain physical attributes from stimuli, particularly contours, edges, shapes, depth, and texture.

### Sensory Thresholds of Fingertips (Spreen and Strauss, 1998)

We measured the patient's sensory thresholds with Semmes-Weinstein monofilaments (Lafayette Instrument Company, Lafayette, Indiana). We used the standard set of 20 nylon monofilaments of uniform length (3.8 cm) and different diameters. With the patient's hand under a cover box, we gently pressed the filament against the first phalanx of each of his first three fingers successively, with enough pressure to create a 45° bend in the filament. This angle ensures the adequacy of the tactile stimulus. The patient's task was to say whether he felt the stimulus. We tested each hand with all 20 filaments, and repeated the stimulations as needed to ensure that we had stable measures.

The sensory threshold is the mean of the first ascending response (the first stimulus that the patient can feel as he is tested with monofilaments of increasing diameter) and the last descending response (the last stimulus that he can feel as he is tested with monofilaments of decreasing diameter).

## RESULTS

We first tested our patient's Tactile Object Recognition with him blindfolded. By touch alone, he could not recognize any of the 12 objects using his right hand (Table 2). With his left hand, he was able to recognize half of the objects, but only after much effort and hesitation. When we uncovered his eyes, he recognized and named all of the same objects quickly and without error. Neurologically normal individuals perform both stages of this task flawlessly (Caplan and Hollander, 2011).

Our patient's performance on all eight overlearned routine activities was hampered by clumsiness, but he did normally at sequencing and purpose, showing that he did not have ideational (tool use) apraxia. Normal people do not make any mistakes on this task (Poock, 1986).

Our patient had a severe bilateral impairment in replicating passive poses in the contralateral hand when blindfolded. With his eyes uncovered, he promptly corrected his poses. He could reproduce passive poses normally at the wrist and elbow with his eyes closed as well as open. Normal people perform this task completely correctly (Caplan and Hollander, 2011).

Our patient's Thumb Localizing test was also grossly abnormal. He could pinch his right thumb on only about 25% of attempts, and his left thumb on about 40% of attempts. Normal people perform this test perfectly (Hirayama et al, 1986).

On the Small Corks test, our patient made at least one error per trial in determining the descending sizes of the corks, eg, 5→3→4→2→1. Normal people perform this task without error (Caplan, 1981).

On the Direction of Scratch test, our patient performed nearly perfectly. In particular, he correctly judged the directions of all the scratches on the palms of his hands. Normal controls make no more than one or two mistakes on each side of the body, usually from attentional slips (Hankey and Edis, 1989). The test is difficult for patients

with central nervous system diseases that impair proprioception and that slow conduction time in the posterior column–medial lemniscal pathway (Motoi et al, 1992).

On the Tactile Form Perception test, our patient missed all 10 trials with his right hand and five of the 10 with his left hand (Table 2). These results fit the “bilateral asymmetric right hand defect” (Pattern D) described by Benton and colleagues (1994) in their original normative series of selected neuropsychological tests. The authors found Pattern D in patients with left hemisphere damage (Table 2 footnote). Most normal people make a maximum of two errors with each hand (Benton et al, 1994).

On the Finger Localization test, Part III, in which the examiner touches pairs of the patient’s fingers simultaneously, our patient was again more impaired with the right hand than the left (Table 2). These results fit the “right unilateral hand defect” Pattern F in Benton et al’s (1994) normative series. The authors reported Pattern F in only one non-aphasic patient who had a left hemisphere lesion and no sensorimotor deficits (Table 2 footnote). On Part III of the Finger Localization test, most normal people make a maximum of two errors with each hand (Benton et al, 1994).

On Two-Point Discrimination, our patient had an obvious enlargement of at least several millimeters beyond normal in the sensory fields of the fingers on both hands, once again more severe in the right (Table 2). The overall difference between the hands was marginally significant (Wilcoxon test:  $Z=1.83$ ,  $P<0.07$ ). Normal people discriminate two points on the fingertips at distances of  $\leq 3$  mm (Spren and Straus, 1998).

Our patient’s sensory thresholds were heightened and roughly symmetric in all areas tested (Table 2). According to normative studies by Spren and Strauss (1998), sensory thresholds show great variation depending on the body area stimulated. In the fingertips they range between 1.65 mm and 2.83 mm, corresponding to a range of pressure between 0.005 g/mm and 0.028 g/mm.

## DISCUSSION

A small ischemic lesion in the cortex and subjacent white matter of the crown of our patient’s left postcentral gyrus caused a severe bilateral asymmetric impairment in his use of his hands in daily activities. The impairment was most severe in the contralesional hand. The injury was chiefly limited to Brodmann area (BA) 1 and its underlying subcortical white matter. His difficulties were obvious during exploratory and manipulative actions, reflecting a complex pattern of sensory deficits that ranged from subjective action stiffness and gross disturbances in the handling of objects to impaired recognition of objects by active touch.

His perception of object features was thus severely compromised in both hands, but his perception of spatial features was preserved in the ipsilesional (left) hand.

As we wrote in the Introduction, the diagnosis of stereognosis requires that the elementary somatosensory submodalities be spared. Accordingly, our patient had intact gross perception of *passive* contact, pressure,

vibration, joint position, temperature, and pain. Likewise, although he had an enlargement of his cutaneous receptive fields, he retained a normal ability to detect the direction of scratch and to localize individual fingers in both hands and pairs of fingers in the left hand.

The capricious dissociation between his tactile perception of spatial and object features shows that his left postcentral gyrus was dominant for the tactile perception of objects, but not of space. In this sense, his ipsilesional hand was similar to the “tactile analogue of blindsight” described by Paillard et al (1983) in a 52-year-old right-handed woman who had suffered an embolic infarction of the left postcentral gyrus and anterior parietal lobe. She was able to localize static stimuli applied to the right hand without being aware of being touched; her left hand was normal.

Our patient differs in a number of ways from the few patients previously reported in the literature. The side, size, and location of his lesion were identical to those of Tomberg and Desmedt’s (1999) patient; however, our patient had bilateral stereognosis while their patient had stereognosis only in the contralesional (right) hand. Tomberg and Desmedt postulated that their patient’s injury was in BA 2, but their description instead suggests a lesion in BA 1. No image of the lesion appeared in their article, and so the location is impossible to confirm.

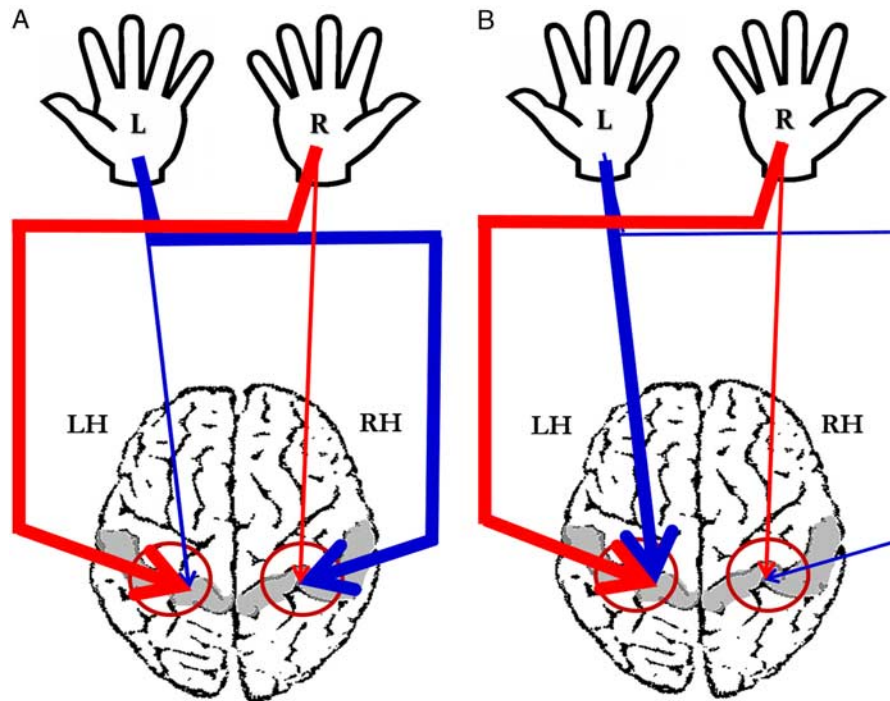
The bilaterality of the somatosensory hand symptoms and the unilaterality of the hemispheric injury of Lhermitte and Ajuriaguerra’s (1938) patient are identical to ours (Table 1). Their patient’s much larger injury, however, precludes accurate clinicopathologic inferences about the hemispheric areas that caused her hand symptoms.

To date, Okuda et al’s (1995) two patients, each with a left hemisphere injury, most closely resemble our patient. Even so, those patients’ lesions went far beyond the limits of the postcentral sensory hand area.

Our patient thus presents a distinctive clinicopathologic picture that combines characteristics of each of the previous patients without being identical to any.

One of the most intriguing aspects of our case and of those summarized in Table 1 is why left hemisphere dominance for stereognosis has not been reported more often. Several possibilities might, at least in part, explain this paradox. One is that many left hemisphere lesions cause aphasia, thus interfering with the patient’s ability to respond to questions on bedside examination or to formulate a verbal complaint of symptoms. A second possible explanation is that many left hemisphere lesions cause severe contralateral sensorimotor deficits; thus, clinicians may overlook problems with the left hand. A third possibility is that a diagnosis of stereognosis, at least in the right hand, would be hampered by concurrent sensorimotor deficits.

Notwithstanding the plausibility of these possibilities, we propose a fourth alternative, which is amenable to experimental verification. We note the considerable variability in the distribution of the pyramidal tracts in the human brainstem and spinal cord (reviewed in de Oliveira-Souza, 2015). We believe that the medial lemniscal fiber system may present similar variability, being totally crossed in some individuals and uncrossed in others. In principle, all



**FIGURE 2.** Diagrammatic representation of the sensory projections of the hands (inside the dark red circles) on the postcentral cortex (areas in gray). **Panel A:** In the common pattern, which is found in most normal individuals, each hand is asymmetrically represented in both cerebral hemispheres. **Panel B:** The clinicoanatomic findings in our patient indicate that the left hemisphere projections from both his contralesional (right) and ipsilesional (left) hands were much more massive than the right hemisphere projections. L = left. R = right. H = hemisphere.

intermediates between these extremes would be possible, including the pattern that we observed in our patient.

In current models of the neurosensory representation of the human hand (Tamè et al, 2016) (Figure 2, Panel A), each hand has multiple sensory representations in the postcentral cortex (BA 3a and 3b, 1, and 2). Areas 3a and 3b of the primary sensory cortex, which Kaas (2004) calls “S1 proper,” lie in the fundus and caudal bank of the central sulcus. BA 1 occupies the shoulder of the postcentral gyrus and BA 2 occupies the gyrus’ crown (Figure 3) (Geyer et al, 2000; Grefkes et al, 2001).

In most people, these representations are bilateral and strongly asymmetric because the somatosensory periphery projects more massively to the contralateral hemisphere (Marani and Schoen, 2005; Moll et al, 1997). Thalamocortical fibers that convey information from the contralateral half of the body project from the ventrolateral posterior nucleus to BA 3a and 3b, which, in turn, project to BA 1 and 2, respectively (Percheron, 2004). Callosal projections steadily increase from S1 to BA 1 and 2 (Innocenti, 1986). Neurons of areas 3a and 1 respond to contralateral proprioceptive stimulation, while areas 3b and 2 respond to exteroceptive stimuli. Moreover, neurons in area 1 respond maximally to simultaneous stimulation of different fingers on one or both hands (Iwamura, 2000).

BA 1 and 2 project to a limited region of the parietal operculum (BA 43), which is also known as the secondary sensory cortex, “S2” (Lüders et al, 1985). Removal of

small parts of the postcentral cortex of nonhuman primates results in deactivation of the corresponding body part in S2, but removal of the whole S2 has no effect on the physiologic responsiveness of postcentral neurons (Pons et al, 1987). Forward projections from postcentral and S2 neurons become progressively more complex and lose their modal specificity as they reach the association (heteromodal) cortices of the parietal, temporal, and frontal lobes (Friedman et al, 1986).

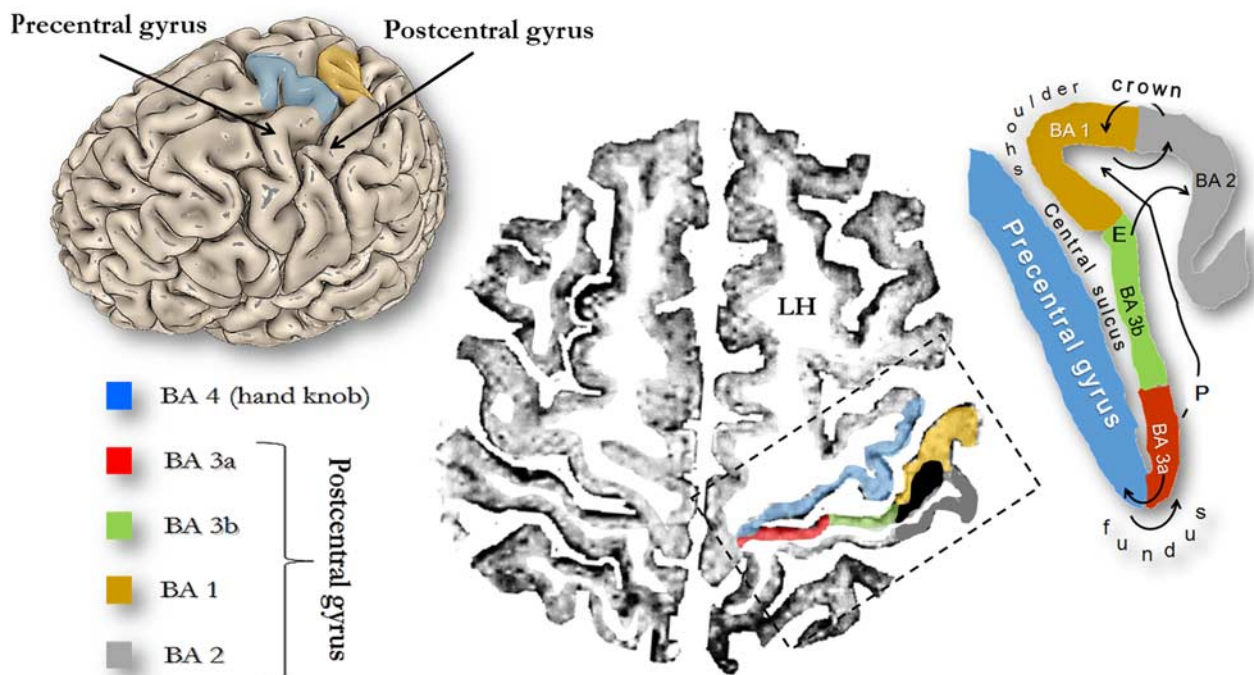
The proprioceptive role of BA 1 in somatosensory perception and the predominantly proprioceptive nature of our patient’s deficits support the classic view that proprioception is the fundamental submodality underlying somesthesia.

In 1899, Dejerine wrote, “C’est, par conséquent, presque exclusivement à la disparition de la sensibilité profonde qu’est due ici l’absence de sens stéréognostique” [Therefore, it is almost exclusively due to the loss of deep sensibility that we owe the abolition of the stereognostic sense] (Dejerine, 1899, p. 250).

Dejerine’s assertion has been largely confirmed by patients who have pure proprioceptive loss with stereognosia (Chesler et al, 2016; Jumentié and Ceillier, 1914; Sacco et al, 1987), as well as by patients with cutaneous hemianesthesia and preserved proprioception who easily recognize objects placed in the affected hand (Groothuis et al, 1977).

In our patient, a small infarction of the somatosensory hand area in the left postcentral gyrus gave rise to severe stereognosia in both hands, with relative sparing of the left





**FIGURE 3.** Schematic drawing of the central sulcus and neighboring precentral and postcentral gyri showing Brodmann areas (BA) 3a and 3b (primary sensory cortex in the narrow sense) and their respective first-order projections to BA 2 and 1. The lesion (shown in black in the lower right part of the central image) destroyed BA 1 as well as its afferent and efferent connections, and occupied a strategic position to deprive BA 2 of a major proprioceptive (P) input. LH = left hemisphere. E = exteroceptive.

hand's ability to localize static (Finger Localization test) and moving (Direction of Scratch test) stimuli. These findings show that, in sharp contrast to the typical pattern, his capacity to perceive objects was massively represented in the left postcentral gyrus, with the right postcentral gyrus playing a negligible role, if any (Figure 2, Panel B).

During the sensory exam, the relative sparing that we found when we stimulated his hands *passively*, eg, with pinprick or joint positioning, is consistent with the bilateral integrity of BA 3a and 3b (Chapman, 1994). In contrast, BA 1, which makes up the crown of the left postcentral gyrus, was destroyed by the lesion. The lesion was in a strategic position to compromise the earliest stages of somatosensory processing by depriving BA 1 of *afferents* originating in BA 3a and 3b (Zilles, 2004). On the *efferent* side, the BA 1 damage deafferented the association areas that are its primary targets, particularly BA 2, S2, and the contralateral homologous cortex through callosal projections (Zilles, 2004).

This anatomic organization suggests that the relative sparing of spatial cognition in the patient's left hand could be explained by projections of the left hand to the right hemisphere, which in most people is dominant for supramodal spatial cognition (Semmes, 1965), or by callosal connections to left BA 2 from its right hemisphere homologous region (Naito et al, 2005). These possibilities are not mutually exclusive and may be tested in both healthy people and patients with strategic cerebral lesions.

We believe that the nonparalytic clumsiness of our patient's hands was a symptom of a predominantly

proprioceptive deafferentation of the precentral cortex from BA 1 (Richardson et al, 2016; Shinoura et al, 2005). That his impairment was restricted to the somesthetic modality was shown by the improvement in his manual actions when he performed them under visual guidance.

The sensory regulation of the hands' grasping movements is mediated by a slender set of U-fibers with different orientations within the "hand knob" region (BA 4); these fibers have long been recognized in gross anatomic specimens (Rosett, 1933) and replicated by *in vivo* tractography (Catani et al, 2012). In our patient, these short cortico-cortical projections were also probably interrupted by his postcentral lesion. His intact reaching hand movements under visual guidance, in turn, are consistent with the integrity of the visual projections originating in the anterior parietal lobe (Rizzolatti and Luppino, 2001).

We conclude that our case lends support to Oppenheim's proposal that the left postcentral gyrus is dominant for stereognosis in at least a few individuals.

#### ACKNOWLEDGMENTS

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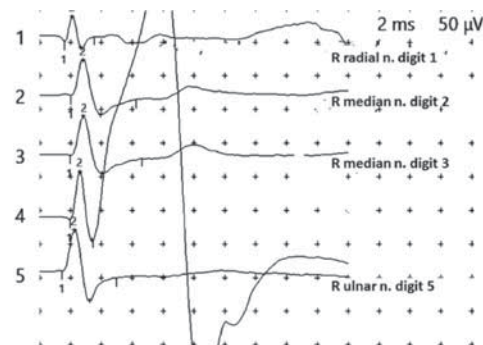
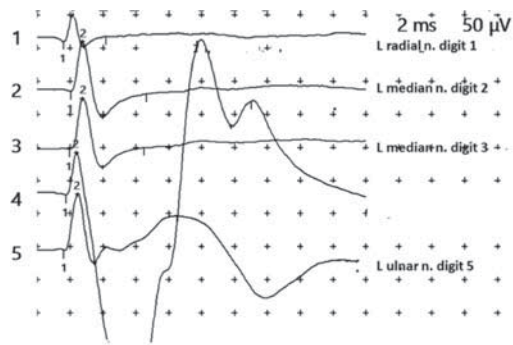
**APPENDIX A. Our Patient's Electromyographic and Nerve Conduction Studies**

**F Wave\***

Recording Site	Patient		Normal Values	Nerve	Roots
	Right	Left			
Abductor pollicis brevis	26.4	24.5	< 31.0	Median	C8-T1
Abductor hallucis	40.2	40.0	< 59.0	Tibial	L4-S1

\*The F wave probes the integrity of proximal motor nerves and ventral roots (results shown in milliseconds).

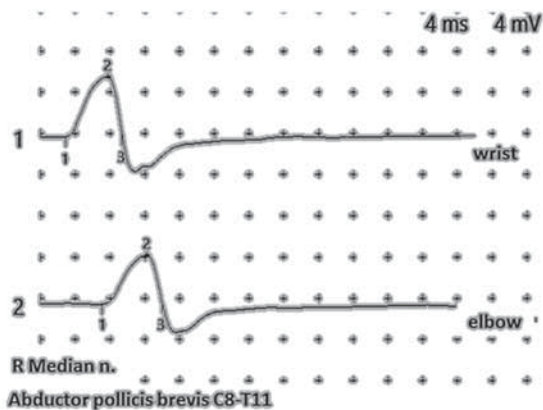
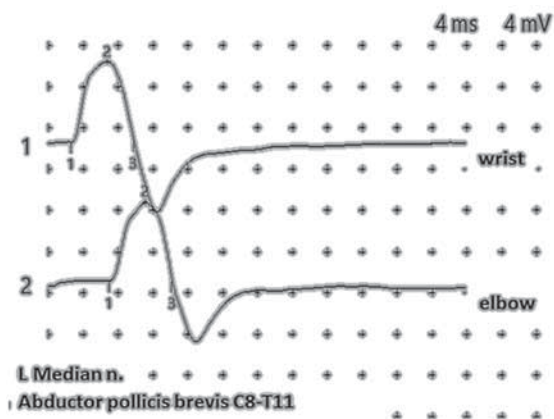
**Nerve Conduction Values: Somatosensory**



	Stimulation Site	Recording Site		Right	Left	Normal
<b>Radial (C5-C6)</b>	Forearm	Digit 1	Latency (ms)			
			Onset	1.6	1.6	< 1.9
			Peak	2.1	2.2	< 1.8
			Amplitude (µV)	37	41	> 20
			Distance (cm)	90	90	—
			Conduction velocity (m/s)	56	58	> 48
<b>Median (C7)</b>	Wrist	Digit 3	Latency (ms)			
			Onset	1.9	1.9	2.5
			Peak	2.8	2.7	3.5
			Amplitude (µV)	57	76	> 20
			Distance (cm)	113	116	—
			Conduction velocity (m/s)	58	59	> 52
<b>Ulnar (C8)</b>	Wrist	Digit 5	Latency (ms)			
			Onset	1.4	1.6	< 2.1
			Peak	2.2	2.5	< 3.0
			Amplitude (µV)	60	85	> 15
			Distance (cm)	91	97	—
			Conduction velocity (m/s)	63	59	> 52

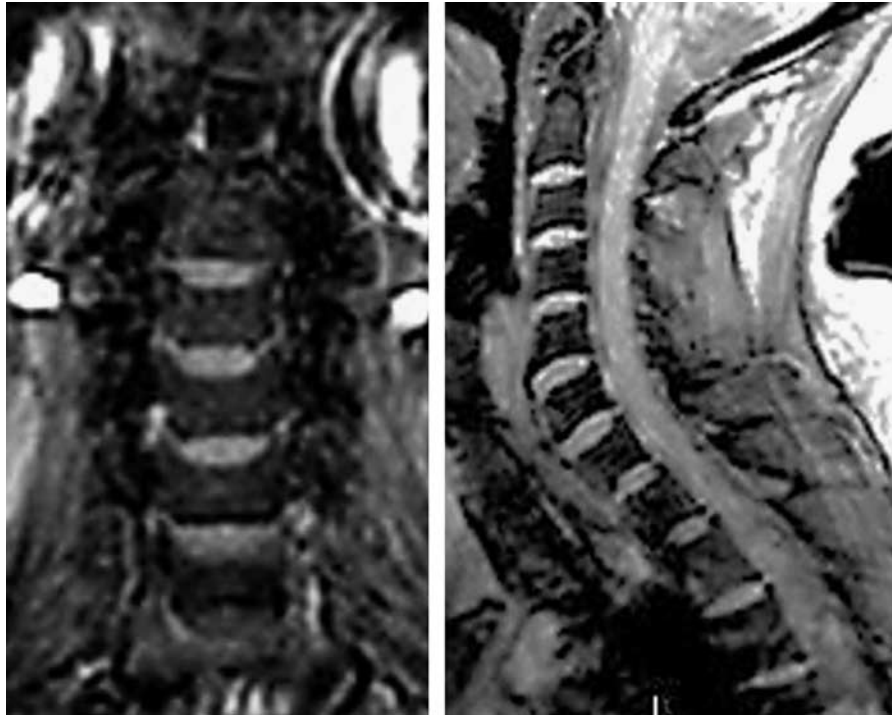
(Continued)

**Nerve Conduction Study: Motor**



Median (C8-T1)	Stimulation Site		Right	Left	Normal
			Wrist	Latency (ms)	2.88
		Amplitude (μV)	5.82	7.7	> 4.4
		Area (mV × ms)	22	36	
		Distance (mm)	70	70	55-75
		Time (ms)	4.28		
		Conduction velocity (m/s)	58.4		> 49
	Elbow	Latency (ms)	7.2	6.9	
		Amplitude (μV)	4.7	7.5	
		Area (mV × ms)	18.3	34.4	
		Distance (mm)	250	243	
		Time (ms)	4.3	4.3	
		Conduction velocity (m/s)	58.4	56	

**APPENDIX B.** Magnetic resonance imaging slices of our patient's cervical spinal cord, showing permeability of the cervical canal. No disc protrusions are visible.



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