Ipsilateral hemiplegia caused by right internal capsule and thalamic hemorrhage: Demonstration of predominant ipsilateral innervation of motor and sensory systems by MRI, MEP, and SEP

Article abstract—A patient with a right internal capsule and thalamic hemorrhage showed ipsilateral hemiplegia. MRI at 10 months after the cerebral hemorrhage demonstrated Wallerian degeneration, which could be traced to the ipsilateral anterior funiculus at the cervical level. The findings of motor evoked potentials and somatosensory evoked potentials indicate a predominantly ipsilateral innervation of motor and sensory systems in this particular patient.

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Ipsilateral hemiplegia is occasionally caused by medullary lesions or by secondary damage of the cerebral peduncle contralateral to the lesion (Kernohan's notch). There are few reports of ipsilateral hemiplegia or hemiparesis caused by a supratentorial cerebral lesion.<sup>1,2</sup> We report a patient with ipsilateral hemiplegia caused by a right internal capsule and thalamic hemorrhage and evaluate the findings of MRI, motor evoked potentials (MEPs), and somatosensory evoked potentials (SEPs) to investigate the cause of the ipsilateral hemiplegia.

**Methods.** *MRI study.* MRI studies were performed 10 months after the cerebral hemorrhage, using a high field strength (1.0 tesla), superconducting MAGNETOME Impact (Siemens) MR imaging system.

Motor evoked potentials. MEPs were recorded using surface electrodes placed over the right and left biceps, triceps, thenar, and hypothenar muscles. Transcranial stimulation of the cerebral motor cortex was performed with a magnetic stimulator (Nihon Kohden), using a twin coil with an inside diameter of 5 cm and an outside diameter of 10 cm in each loop (10,000 A = 900 V at maximal output), which provided a focal magnetic stimulation at its intersection. Magnetic stimulation was given in the stimulus intensity range of 800 to 900 V. For the stimulation of the motor cortex hand area, the intersection of a twin coil was placed at 5 cm lateral to Cz.

Somatosensory evoked potentials. Recording electrodes were placed on the bilateral scalp (F3, F4, C3, C4, P3, and P4), and linked ears served as a reference. Electrical stimulation with square-wave pulses (duration: 0.1 msec) was applied to the median nerves at the wrist at a rate of 5/sec. Stimulus intensity was a voltage 20% above the motor threshold of the thenar muscle. A total of 256 responses were summated using an analysis time of 80 msec. The frequency response of the recording system was 8 to 1,200 c/sec.

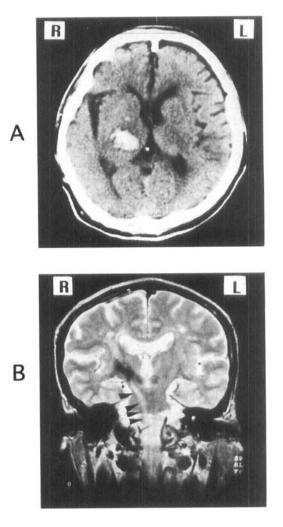
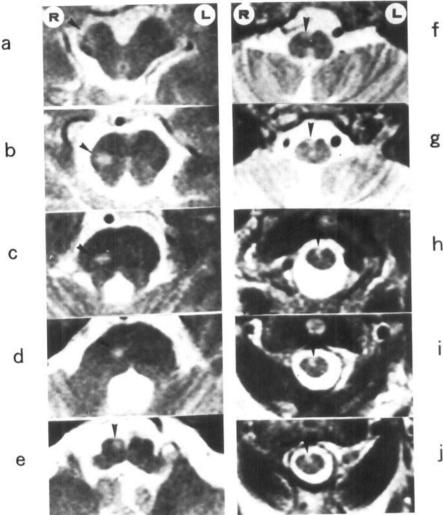


Figure 1. (A) CT at the time of onset showing the cerebral hemorrhage in the right internal capsule and thalamus. (B) T2-weighted coronal MR image (TR 3,700 msec, TE 90 msec) at 10 months after the onset demonstrated resolution of the hemorrhage, necrosis of the right internal capsule and thalamus, and Wallerian degeneration (arrowheads).



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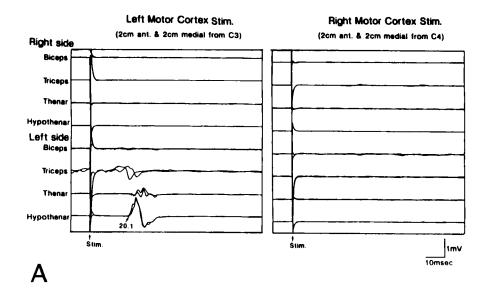
Figure 2. T2-weighted axial MR images (TR 3,700 msec, TE 90 msec) at 10 months after the onset. Wallerian degeneration (arrowheads) extends through the right cerebral peduncle (a, b), pons (c, d), and medulla (e, f, g). At the cervical level (h, i, j), Wallerian degeneration can be seen in the ipsilateral anterior funiculus, which includes the anterior corticospinal tract.

**Patient history.** A 60-year-old, right-handed man suddenly developed a right hemiplegia. His history revealed moderate hypertension without medication. A noncontrast CT scan showed an intracerebral hemorrhage in the right internal capsule and thalamus (figure 1A). Ten months later we investigated the cause of the ipsilateral hemiplegia. He had a marked congenital scoliosis. He was alert without speech disturbance. He had a moderate spastic right hemiplegia including the face. The deep tendon reflexes were exaggerated on the right with positive Babinski's response. He felt a tingling sensation with paresthesia and impaired deep sensation on the right side of his body, including the face.

**Results.** MRI study. MRI at 10 months after the onset revealed sequelae of the cerebral hemorrhage. The MRI of

T2-weighted axial and coronal images also demonstrated Wallerian degeneration, which extended through the right cerebral peduncle, pons, and medulla (figures 1B, 2). At the cervical level, Wallerian degeneration could be seen in the ipsilateral anterior funiculus, which includes the anterior corticospinal tract. Moreover, the shape of the medulla was unusually flat, and each side looked separated.

Motor evoked potentials. Stimulation of the hand area of the left cerebral motor cortex produced MEPs in the ipsilateral triceps, biceps, thenar, and hypothenar muscles with latencies of 12.8 msec, 12.9 msec, 22.8 msec, and 20.1 msec, respectively (figure 3A). No MEPs were evoked in muscles contralateral to the stimulation. Stimulation of the hand area of the right motor cortex produced no MEPs in muscles on either side. Magnetic stimulations of the



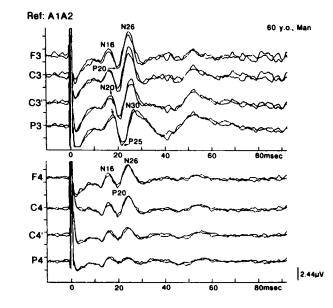


Figure 3. (A) Motor evoked potentials (MEPs) following transcranial magnetic stimulation of the hand area of the cerebral motor cortex. Stimulation of the left produced MEPs in the ipsilateral arm muscles, but not in the contralateral arm muscles. Stimulations of the right produced no MEPs in the arm muscles on either side. (B) Somatosensorv evoked potentials following the left median nerve stimulation. The primary cortical response, N20, appeared in the central (C3) and parietal (P3) regions, exclusively on the left side, ipsilateral to the side of stimulation.

posterior neck produced symmetric MEPs bilaterally in the muscles of the upper extremities.

Somatosensory evoked potentials. Following left median nerve stimulation, N16, P20, and N26 were recorded bilaterally in the frontal region. In contrast, the primary cortical response, N20 was localized in the left central and parietal regions, which are ipsilateral to the side of stimulation (figure 3B). Right median nerve stimulation evoked no cortical response over the entire scalp. **Discussion.** This is the first report of persistent ipsilateral hemiplegia and hemisensory disturbance caused by a supratentorial cerebral lesion in which the lesion site was confirmed by CT and MRI. Cuatico<sup>1</sup> reported a patient with ipsilateral hemiparesis due to surgical occlusion of the left anterior cerebral artery. He demonstrated the angiographic findings, but the lesion site was not confirmed by CT or necropsy. A patient with transient ipsilateral hemipare-

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sis caused by a subarachnoidal hemorrhage was also reported by Kudo and Uno.<sup>2</sup> They attributed the ipsilateral hemiparesis to compression of the secondary motor and sensory area by a hematoma in the sylvian fissure.

Transcranial stimulation of the hand area of the left cerebral motor cortex produced MEPs in the arm muscles exclusively on the ipsilateral side. These exclusive ipsilateral MEPs by magnetic cortical stimulation have never been reported. However, bilateral MEPs with large ipsilateral amplitudes have been reported in a patient with congenital mirror movement.<sup>3</sup> The ipsilateral response cannot be explained by a current spread of the stimulus to the contralateral motor cortex, since the use of a twin coil enabled focal discrete stimulation of the cerebral cortex.<sup>4</sup> The ipsilateral MEPs in our patient indicate that he has an anomaly in which the cerebral motor cortex predominantly innervates the ipsilateral half of the body.

MRI at 10 months after the onset of the cerebral hemorrhage demonstrated Wallerian degeneration in the ipsilateral cerebral peduncle, pons and, medulla, extending to the ipsilateral anterior funiculus at the cervical level, which includes the anterior corticospinal tract.

Neuroanatomic studies<sup>5.6</sup> have demonstrated that most of the pyramidal fibers cross to the contralateral lateral column and that about one fifth proceed caudally in the ipsilateral anterior funiculus. However, considerable variations with regard to the proportion of crossed and uncrossed corticospinal fibers are probable in man. Verhaart and Kramer<sup>5</sup> reported four human cases in which the pyramidal tracts are said not to decussate, all fibers descending down to the ipsilateral anterior funiculus of the spinal cord. Encephaloceles<sup>6</sup> and Dandy-Walker<sup>7</sup> syndrome were associated with an uncrossed pyramidal tract.

The MEP and MRI findings in our case suggest that the uncrossed ventral corticospinal tract in the anterior funiculus is predominant to the crossed lateral corticospinal tract in the lateral column in this particular case. The result of the SEP study indicated that the primary cortical response N20 appeared exclusively in the ipsilateral central and parietal areas after the left median nerve stimulation. N20 is believed to originate in the primary somatosensory cerebral cortex, and to be recorded from the central and parietal regions, contralateral to the side of stimulation. The predominant ipsilateral appearance of N20 has never been reported. Since the SEP response by electrical stimulation is believed to be mediated by the dorsal column-medial lemniscal pathway,<sup>8</sup> the present finding suggests that the dorsal column-medial lemniscal pathway mainly projects to the ipsilateral primary somatosensory cerebral cortex. Neuroanatomic study<sup>9</sup> demonstrated that all fibers of the medial lemniscus cross the midline in the medulla. The presence of sensory disturbance, ipsilateral to the side of the cerebral hemorrhage, and the SEP results suggest that this patient has an anomaly of the predominant ipsilateral innervation of the dorsal column-medial lemniscal pathway.

In conclusion, this is the first documentation that the motor and sensory cerebral cortices can predominantly innervate the ipsilateral half of the body as in this particular patient, and the results indicate the utility of MRI, MEP, and SEP for demonstrating the ipsilateral innervation of motor and sensory systems.

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