Ipsilateral hemiparesis after putaminal hemorrhage due to uncrossed pyramidal tract

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Article abstract—*Objective:* Previous case reports supported the presence of the uncrossed pyramidal tract in exceptional patients. However, most of these case reports have not fully discussed involvement of the motor cortex controlling the ipsilateral limbs. *Design and Method:* The authors investigated a 62-year-old man who developed right hemiparesis after right putaminal hemorrhage by using MRI, transcranial magnetic stimulation, functional MRI (fMRI), and sensory evoked potentials. He had moderate weakness including the face, spasticity with brisk deep tendon reflexes and Babinski sign, and impaired vibration and position sense, all on the right side. *Result:* A MRI study showed hemorrhage in the right putamen and the wedge-shaped medulla. A fMRI study during a sequential finger opposition task showed activation in the motor cortex ipsilateral to the finger movements, but not on the contralateral side. Sensory evoked potentials showed cortical response ipsilateral to the side of stimulation. *Conclusion:* The pyramidal tract and the dorsal column-medial lemniscus pathway did not cross in the medulla in this patient. In view of the presence of the abnormal shape in the medulla and congenital scoliosis, a congenital factor might be responsible for the uncrossed pyramidal tract and dorsal column-medial lemniscus in this patient. **Key words:** Ipsilateral hemiparesis—Functional MRI—Ipsilateral innervation.

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Previous neuroanatomic studies have firmly supported the notion that the primary motor cortex predominantly innervates the contralateral half of the body. Conversely, some case reports supported the existence of the uncrossed pyramidal tract in exceptional patients.[1-3] These case reports primarily discussed the uncrossed pyramidal tract alone and have not fully discussed the motor cortex controlling the ipsilateral limbs. We report a patient with right hemiparesis after hemorrhage in the right putamen in whom we demonstrated motor control by the ipsilateral motor cortex by using functional MRI (fMRI) and transcranial magnetic stimulation (TMS).

Case report. A 62-year-old, right-handed man with a history of hypertension suddenly developed right hemiparesis. He was taken to a hospital by ambulance. On neurologic examination, he was drowsy and had right hemiparesis. His muscle tone was diminished and deep tendon reflexes were absent, but the Babinski sign was present on the right side. A CT scan showed a hemorrhage in the right internal capsule and basal ganglia (figure 1). The lateral and third ventricles were of normal size without any deformity or deviation of the midbrain. On the day of admission, he underwent stereotaxic surgery for removal of the hematoma.

Ten months later, he was admitted to our hospital for further evaluation of residual right hemiparesis. He had a marked congenital scoliosis in the thoraco–lumbar spine. On neurologic examination, he was alert but had slight dysarthria. His pupils were 2.5 mm in diameter and equal. His pupillary responses were prompt bilaterally. Eye movement was mildly restricted in the horizontal direction bilaterally. He had facial weakness on the right side. The soft palate moved normally. He had moderate weakness, spasticity with brisk deep tendon reflexes, and Babinski sign on the right. He did not have mirror movements. He had subjective hypesthesia and impaired vibration and position sense on the right side, but impaired light touch and pain sensation on the left side.

MRI study was performed using a 1.5-T MR system (Magnetom, Siemens, Erlangen, Germany) 10 months after the hemorrhagic event, which revealed sequelae of old hemorrhage and surgical intervention in the right putamen. With the fluid-attenuated inversion recovery (FLAIR) image, high signal intensities were observed in the cerebral peduncle, pons, and medulla on the right side, considered to represent Wallerian degeneration following destruction of the corticospinal tract and neuronal death in the putamen.[4] However, we could not identify Wallerian degeneration in the cervical spinal cord due to the resolutional ability of MRI. The medulla had a wedge-shaped notch and appeared to be separated, but there was no abnormality in the upper cervical cord (figure 2).

fMRI was performed by the same device. The patient's head was positioned within the standard head coil and head movements were restricted by using foam rubber pads and Velcro band across the forehead. Body movements were also minimized by using the proximal arm

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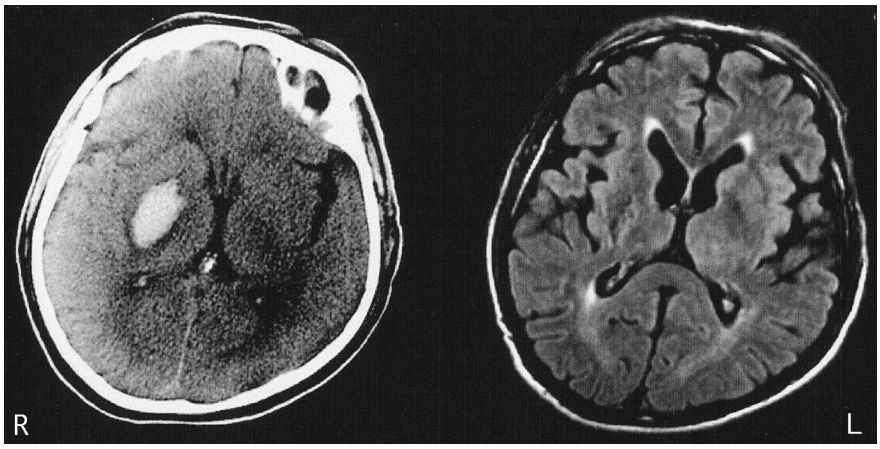

Figure 1. CT scan on the day of onset (left) and MRI 10 months after intracranial hemorrhage (right). A hematoma is visible in the right putamen with CT, which is also visible with MRI after stereotactic surgery.

strips. Conventional T1-weighted axial images were acquired parallel to the bicommissural plane. A single shot gradient recalled echoplanar imaging was used with interscan interval of 2 seconds, echo time of 66 msec, flip angle of 10 degrees, matrix of 128 × 128, field of view of 24×32 cm, and slice thickness of 5 mm. The patient was asked to perform a sequential finger opposition task, during which he repeatedly touched the left four fingers with the left thumb in a sequential order as quickly as possible with eyes closed. The cue to begin and cease the movement was a light tap on the knee. Pixels showing the stimulus-related signal increase were identified by calculating the correlation between the MR signal time course and time-shifted boxcar function. The threshold for significant activation was set at $p < 0.005$. The resulting binary map was then filtered by a 3 × 3 median filter. For visualization, the map was superimposed onto the corresponding MRI slices. During the motor task, the functional activity was observed in the motor cortex ipsilateral to the finger movements (figure 3).

SPECT with ^{99m}Tc-hexamethyl-propylene-amine-oxime demonstrated hypoperfusion in the right basal ganglia and the frontoparietal region of the right cerebral hemisphere.

An ultrasound examination of the carotid artery showed no plaque or stenosis on either side.

TMS was performed using a magnetic stimulator (Nihon Kohden, Tokyo, Japan) with a circular electromagnetic coil in loop with an inside diameter of 8 cm and an outside diameter of 14 cm (10,000 A = 900 V at maximal output). We used magnetic stimulation at the intensity ranging

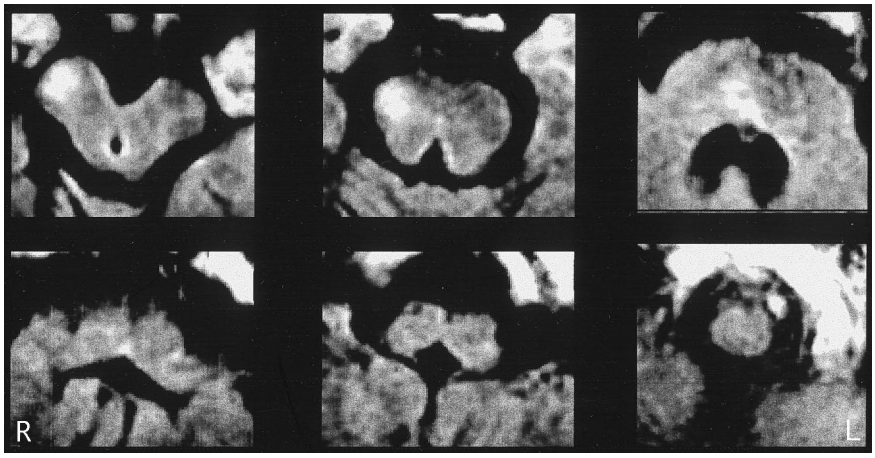

Figure 2. Fluid-attenuated inversion recovery MRI 10 months after onset. High-intensity lesions are visible in the right cerebral peduncle, pontine base, and medulla, corresponding to the location of the pyramidal tract. Note the shape of the medulla, which appeared to be separated in the midline, and the cervical cord, which was normal in shape.

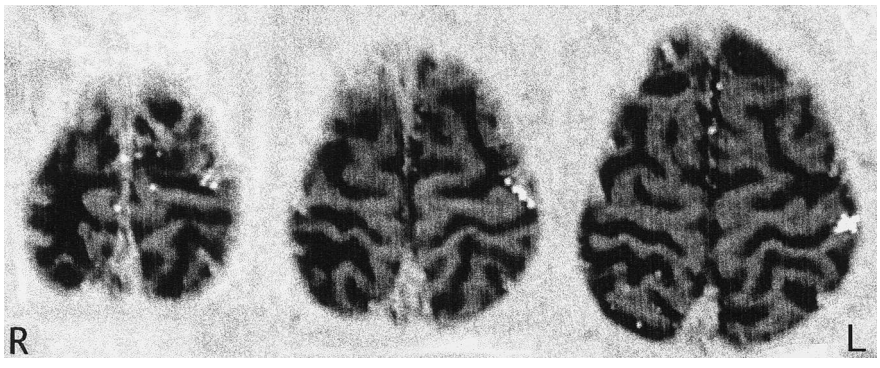

Figure 3. Functional MRI during motor activation. Three axial slices acquired parallel to the bicommissural plane at 4, 8, and 12 cm below the cerebral vertex are presented. Significantly activated pixels are visible on the left side during left finger movements.

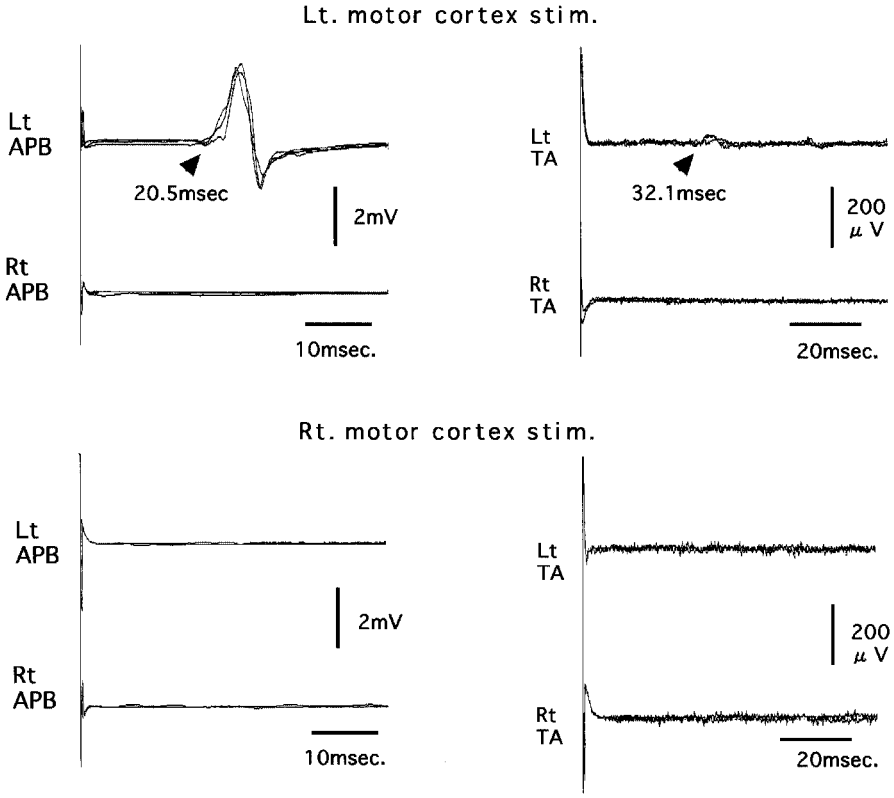

Figure 4. Results of the motor evoked potentials after stimulation of the primary motor cortex. The left abductor pollicis brevis and anterior tibialis muscles responded after stimulation of the left motor cortex (top), whereas the right abductor pollicis brevis and anterior tibialis muscles did not respond after stimulation of the right motor cortex (bottom).

from 800 to 900 V. We placed the center of the coil over the scalp in the vertex to stimulate the leg area and at 2 cm posterior to Cz to stimulate the finger area. We used counterclockwise coil current for preferential activation of the left motor cortex and clockwise coil current for preferential activation of the right motor cortex. EMG electrodes were placed on the belly tendons of the abductor pollicis brevis (APB) muscle and the anterior tibialis (TA) muscle on each side. Stimulation of the left motor cortex evoked responses in the left APB with a latency of 20.5 msec and in the left TA with a latency of 32.1 msec, both of which were ipsilateral to the side of stimuli. No response was evoked in the muscles on the right side. Stimulation of the right motor cortex evoked no response on either side (figure 4).

Sensory evoked potentials (SEP) were recorded with a standardized electrodiagnostic system (Neuropack4, Nihon Kohden). Electrical stimulation with square-wave pulses was delivered by a bipolar surface electrode with five electrical impulses per second for a duration of 0.2 msec to the right and left median nerve at the wrist, which produced a minimal thumb twitch. A total of 256 responses were summed and averaged. The recording electrodes were placed according to the international 10-20 system at 3 cm behind C3 and C4 (cortical N20 peak). Those active electrodes were referenced to a midfrontal Fz electrode. The ground electrode was placed on the side of stimulation 10 cm proximal to the stimulus electrode. After left median nerve stimulation, the primary cortical response N20 was elicited in the left central and parietal region, which was ipsilateral to the side of stimulation. Right median nerve stimulation elicited cortical response in the right central and parietal region, which was also ipsilateral to the side of stimulation (figure 5).

Discussion. The most notable clinical feature of this patient was the development of right hemiparesis following hemorrhage in the right putamen. Because this hematoma in the right putamen did not exert any signs of mass effect on CT, it could not compress the left pyramidal tract, primary motor area, or secondary motor area directly or indirectly.

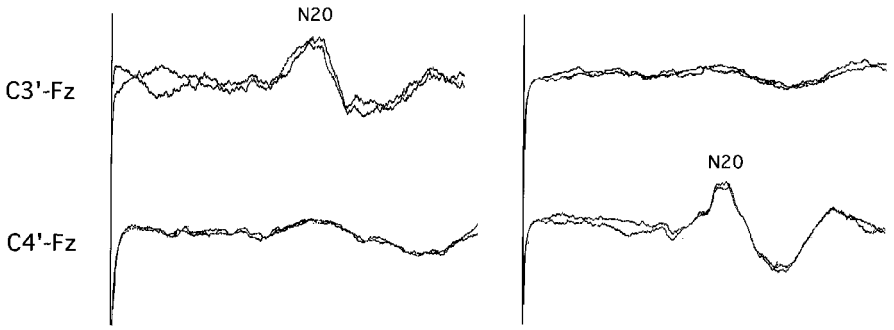

Figure 5. Results of the sensory evoked potentials after stimulation of the median nerve. A cortical potential (N20) was evoked in the left cerebral cortex (C3') after stimulation of the left median nerve (left), and in the right cerebral cortex (C4') after stimulation of the right median nerve (right).

Although dysfunction in the right secondary motor area is a possible reason for right hemiparesis, there was no lesion affecting the right secondary motor area on MRI. We conclude that dysfunction in the right pyramidal tract or motor cortex was the reason for right hemiparesis.

TMS allows a simple and painless evaluation of the human motor pathway.[5] Magnetic stimulation of the motor area usually evokes contralateral limb movement. However, our patient showed left limb movement after magnetic stimulation of the left motor cortex. This suggests that the pyramidal tract might not have crossed in this patient. fMRI is another way to gather information concerning cortical innervation for limb movements. Signals in fMRI represent changes in the deoxyhemoglobin concentration due to activation of neurons in the underlying cortical areas.[6] In our patient, the left primary motor cortex showed significant activation after finger movements on the left side, suggesting that the left motor cortex had control over finger movements on the left side. The results of TMS and fMRI are consistent with the presence of the uncrossed pyramidal tract and predominant control of movements by the ipsilateral motor cortex in this patient.

In addition to motor impairment, our patient also showed impairment of position and vibration sensation on the same side as that of hemiparesis, suggesting the presence of the uncrossed dorsal column–medial lamniscus pathway and of the dentatorubrothalamic pathway. The results of SEP support the presence of an aberrant dorsal column–medial lamniscus pathway. Because the light touch and pain sensation were impaired on the side opposite to the side of hemiparesis, the spinothalamic tract appeared to have run the ordinary course. We believe the most plausible explanation is that the pyramidal tract and the dorsal column–medial lamniscus pathway did not cross in the medulla in this patient. The abnormal shape of medulla, which appeared to be separated in the midline, supports this notion.

There are controversies over ipsilateral innervation of limb movements. In animals, ipsilateral innervation by the primary motor and premotor cortex has been reported.[7-9] In humans, Penfield and Boldrey conducted a classic study of somatotopic innervation of the motor function by the primary motor cortex and concluded that motor movements were predominantly innervated by the contralateral motor cortex.[10] More recently, an anatomic study showed ipsilateral projection from the primary motor cortex,[11] but it was considered to have a much weaker control over limb muscle movements than contralateral projection.[12] In addition, clinicopathologic studies,[13] TMS studies,[14,15] and PET studies[16,17] have emphasized the critical role of the ipsilateral pyramidal tract in the recovery from hemiparesis caused by cerebral damage.

Several case reports described ipsilateral hemiparesis following damage in the ipsilateral cerebrum.[18-24] Cuatico reported a case of ipsilateral hemiparesis but without further anatomic discussion.[1] Hosokawa et al. reported a patient with ipsilateral hemiparesis after putaminal hemorrhage and showed the presence of the uncrossed pyramidal tract from the putamen to the cervical cord by MRI, but without discussion of the cortical contribution.[3] Kudo and Uno reported a patient with transient ipsilateral hemiparesis following compression of the secondary motor area by a hematoma.[2] Schneider and Crosby reported a patient with ipsilateral motor impairment after rupture of an arteriovenous malformation in the island of Reil. They emphasized the role of the secondary motor area located in the precentral insular cortex bilaterally innervating the muscles in the face and limbs.[21] These previous case reports failed to show control of limb muscles by the ipsilateral motor cortex. In our patient, we were able to demonstrate that the motor cortex on the left side controlled the limbs on the left side by using TMS and fMRI.

Patients without decussation of the pyramidal tracts have been reported to have other cerebral malformations.[19,20,22] Krams and colleagues described mirror movements in Kallmann's syndrome as the result of abnormal development of the ipsilateral corticospinal tract.[25-27] Our patient did not have mirror movements. Our patient and a patient reported by Hosokawa et al. had congenital scoliosis.[3] These observations suggest the presence of a congenital factor for the uncrossed pyramidal tract in some of those patients.

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Poststroke depression and emotional incontinence

Correlation with lesion location

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Article abstract—*Objective:* To correlate the location of stroke with poststroke depression (PSD) and emotional incontinence (PSEI). *Methods:* The authors prospectively studied 148 patients (94 men and 54 women, mean age 62 years) with single, unilateral stroke (126 infarcts and 22 hemorrhages) at 2 to 4 months poststroke with regard to the presence of PSD (using Diagnostic and Statistical Manual of Mental Disorders IV criteria and Beck Depression Inventory) and PSEI. The lesion location was analyzed by CT or MRI. *Results:* Twenty-seven patients (18%) had PSD and 50 (34%) had PSEI. The presence of PSD and PSEI was not related to the nature, laterality, or size of the lesion. The frequency of PSEI, but not of PSD, was higher in women than in men and in ischemic rather than hemorrhagic stroke ($p < 0.05$). Although both PSD and PSEI were related to motor dysfunction and location (anterior versus posterior cortex) of the lesion, location was a stronger determinant for PSD ($p < 0.05$). The prevalence of PSD/PSEI in each location was 75%/100% in frontal lobe of anterior cerebral artery territory, 50%/0 in temporal lobe, 30%/40% in frontal–middle cerebral artery territory, 13%/0 in occipital lobe, 19%/45% in lenticulocapsular area, 11%/16% in thalamus, 16%/53% in pontine base, 36%/55% in medulla, and 0/22% in cerebellum. Parietal and dorsal pontine lesions were not associated with PSD or PSEI. PSEI was more closely associated with lenticulocapsular strokes than was PSD ($p < 0.01$). *Conclusion:* Development of PSD and PSEI is strongly influenced by lesion location, probably associated with the chemical neuroanatomy related to the frontal/temporal lobe–basal ganglia–ventral brainstem circuitry. Although the lesion distribution is similar, PSEI is more closely related to lenticulocapsular strokes than is PSD. **Key words:** Depression—Emotion—Laughing—Crying—Cerebrovascular disease.

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Many patients develop depression after the occurrence of stroke.[1,2] The prevalence of poststroke depression (PSD) ranges from 12% to 64%.[3-15] The wide variation in the frequency of PSD may be related to methodologic heterogeneity in items such as the criteria for depression, the time of assessment after stroke, and the sampled population. A main controversy regarding PSD has been the relationship between the location of stroke and PSD. Whereas some have reported that PSD occurred more often after

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