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Discrete Cortical Infarction With Prominent Impairment of Thumb Flexion

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Background: Intracortical efferent zones in the primary motor cortex for thumb movements, namely thumb flexion, extension, adduction, and abduction, have been described in *Maccaca mullata* monkeys but not in humans. Even recent cortical mapping based on intraoperative monitoring does not provide information about intracortical efferent zones as it is not ethically possible to search the human motor cortex extensively by punctate electrodes.

Case Description: A 78-year-old man with discrete cortical embolism over the left central sulcus is described. Only a mild weakness of his right thumb was observed. Thumb flexion was affected to a greater degree than thumb extension, adduction, and abduction. The lesion ran along the bottom of the central sulcus and affected part of the precentral and postcentral gyri on both sides of it. Animal studies in *Maccaca mullata* monkeys have shown that the intracortical efferent zones for thumb movement, especially for thumb flexion, are located in the part of the motor cortex just adjoining the central sulcus that folds down from the "bank" to the depth of the central sulcus. It was possible to explain the prominent weakness of thumb flexion if we applied the results of the animal studies.

Conclusions: Our case suggests that the same arrangement of intracortical efferent zones found in monkeys may also exist in human beings. (*Stroke*. 1993;24:2118-2120.)

KEY WORDS • cerebral cortex • embolism • motor activity

P revious studies^{1,2} using intracortical stimulation in Maccaca mullata monkeys have shown that intracortical efferent zones exist in the primary motor cortex for each of the four thumb movements, namely, thumb flexion, extension, adduction, and abduction. Recent developments in cortical mapping techniques based on intraoperative monitoring cannot be applied to humans because it is not ethically possible to search the human motor cortex extensively by punctate intracortical electrodes. We describe a case of discrete cerebral embolism in which prominent weakness of thumb extension was observed and suggest that the same cortical organization that has been reported in monkeys may also exist in human beings.

Case Report

A 78-year-old man without hypertension, hyperlipidemia, diabetes mellitus, atrial fibrillation, or previous history of myocardial infarction was admitted because of right hemiplegia and sensory disturbance. On admission he could not raise his right arm from bed. A milder weakness was noted in his right leg and he could barely walk with assistance. There were no neuropsychological signs such as aphasia, impairment of calculating ability, or disturbance of right-left discrimination. The symptoms gradually subsided during his stay in the hospital and at the time of discharge only a mild weakness in his

thumb remained. Thumb flexion was affected to a greater degree (muscle power was 2 by manual muscle testing [MMT]) compared with thumb extension, adduction, and abduction (muscle power -4 to ≈ 4 by MMT). Weakness was slightly more prominent for thumb extension (-4 by MMT) than for thumb adduction and abduction (4 by MMT). Because the power of extension exceeded that of flexion, his right thumb was kept slightly extended. The power of the other fingers and extremities was completely normal (only a slight weakness was noted in the flexion of the index finger). Dysesthesia was confined to his right thumb and the skin around the corner of the right side of his mouth, showing a distribution of cheiro-oral syndrome. Mild hypesthesia for pin prick and light touch was noted in his right thumb and the tip of his index finger. He found great difficulty in identifying items rubbed across the thumb. The thumb position sense was mildly impaired. He experienced great difficulty in picking things up because his fingers felt "slippery" after the attack, and he thought that this awkwardness could not be explained by the weakness of thumb flexion. Echocardiogram showed no clot in the left atrium.

The episode was considered to be due to cerebral embolism because computed tomography of the brain performed 3 days after onset showed a discrete round area of low density over the left central sulcus with a spot of high density in the middle (Fig 1). In the transverse section of magnetic resonance imaging, which was taken a month after the stroke episode, the anterosuperior part of the lesion affected the posterior part of the precentral gyrus that folds down from the outer surface (or "bank") of the cortex to the bottom of the central sulcus, whereas the posteroinferior part

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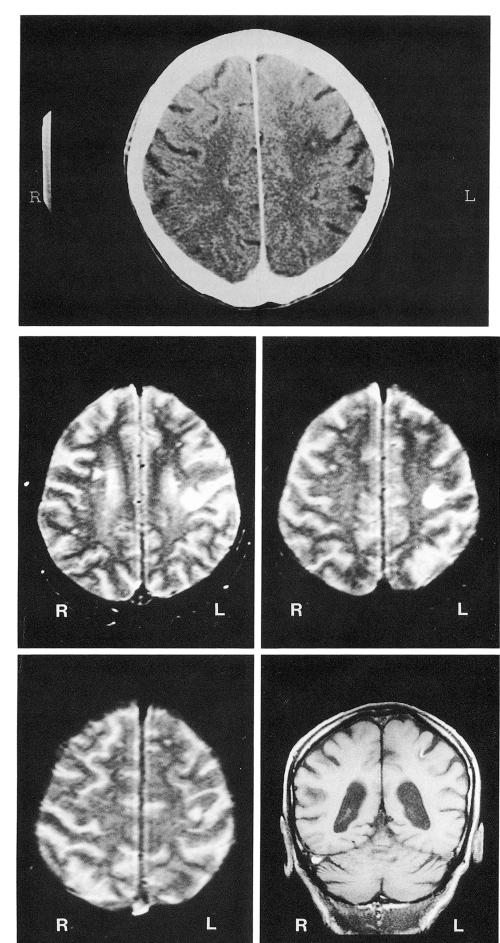
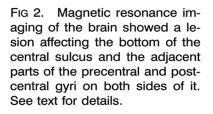


FIG 1. Computed tomographic scan taken 3 days after onset of discrete cortical embolism.



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involved the postcentral gyrus and the underlying subcortical area. The sagittal section showed the lesion running along the bottom of the central sulcus and affecting part of the precentral and postcentral gyri on both sides of it (Fig 2). Though other small lesions also existed in the bilateral centrum semiovale, the motor and sensory symptoms were considered to be due to this newly appeared lesion because the patient had no symptoms before the attack. Electromyographic studies showed no findings suggesting lower motor neuron or peripheral nerve lesion.

Discussion

Woolsey et al¹ mapped the precentral and supplementary motor cortex of the Maccaca mullata monkey by systemic punctate electrical stimulation and showed that the stimulation of the posterior part of the motor cortex that folds down to the bottom of the central sulcus elicited thumb movement. Though it was not possible by surface stimulation methods to produce all four thumb movements, ie, extension, flexion, abduction, and adduction in a single animal, Asanuma and Rosén² succeeded in eliciting these four movements with weak intracortical stimulation of 10 μ A or less. In the precentral gyrus the low-threshold efferent zones for thumb movement were located at the bank to the depth of the central sulcus; the zones for flexion, extension, adduction, and abduction were arranged in this order in the posteroinferior to anterosuperior direction according to the distance from the central sulcus.

The lesion in our case seemed to affect only part of the precentral gyrus near the bank to the depth of the central gyrus. If the results of the above-mentioned animal experiments are applied to this case, then the present lesion, which was just adjacent to the central sulcus, is presumed to affect the cortical zone for thumb flexion and its efferent fibers more than the zones and efferent fibers for the other three thumb movements. The lesion extended rather deeply, but not to the adjoining white matter, so the cortical efferent zones for the other three thumb movements and their efferent fibers were not affected. This may explain the predominant weakness of thumb flexion and the lesser weakness of thumb extension, adduction, and abduction. Our case suggested that the same arrangement of efferent zones found in monkeys may also exist in human beings.

Hypesthesia and impairment of two-point discrimination was also noted in the patient's right thumb in the same area where weakness was noted. This may reflect the damage to the adjacent part of the postcentral gyrus folding down to the bottom of the central gyrus. Again, according to experiments by Woolsey,³ the corresponding region in the left postcentral gyrus of a *Maccaca mullata* monkey shows somatotopic representation for its right thumb and the skin near the corner of the right side of its mouth.

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