Motor Recovery After Early Brain Damage
A Case of Brain Plasticity

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Background Motor recovery is remarkable when the brain is damaged early in life. We describe a case of early damage to the right hemisphere with remarkable reorganization and plastic functional changes, studied by computed neuroimaging.

Case Description A 31-year-old man had a left-sided hemiplegia at the age of 12, followed by good motor recovery despite a large right cortical-subcortical lesion. Single-photon emission computed tomography with motor activation study showed cerebral blood flow increase in the left premotor and sensorimotor cortices irrespective of the hand he was moving, without flow changes in the right hemisphere.

Conclusions A remarkable reorganization and plastic brain functional changes occurred in a patient after early diffuse damage of the right hemisphere. The study points to a potential role of ipsilateral cortical efferent pathways in subserving hand movements after early cerebral damage. (Stroke. 1994;25:514-517.)

Key Words • motor activity • tomography, emission computed • tomography, x-ray computed • young adults

Recovery of function is remarkable when the brain is damaged early in life. However, the mechanisms by which patients regain reasonable motor functions in contralateral muscle groups after hemispherectomy or severe brain infarcts are still unclear. Emission tomography techniques have shown that cortical areas in the unaffected hemisphere play a role in the recovery of motor function after stroke. Using these techniques, however, the role of ipsilateral pathways in motor recovery has thus far been described only in aged ischemic stroke patients.

We report the case of a young man who had exhibited left-sided hemiplegia at the age of 12, followed by good motor recovery despite a widespread loss of neural tissue in the right hemisphere. Data obtained by x-ray computed tomography (CT) and motor activation with single-photon emission computed tomography (SPECT) show a remarkable reorganization of the ipsilateral sensorimotor and premotor cortices.

Case Report

A 31-year-old right-handed white man was admitted to our ward because of a fainting episode followed by left limb weakness. By the time of his hospital admission the deficit was almost completely resolved. The neurological examination showed a slight strength deficit of the left hand and leg. He was able to move his left fingers individually. Tendon reflexes of left limbs were very brisk, and an extensor plantar reflex of the left foot was evident. No sensory deficits were detected. This neurological picture did not show any change during the entire hospitalization.

In reviewing past medical history we found that at the age of 12 our patient had been hospitalized because of fever, headache, vomiting, and neck stiffness. During that hospitalization, he had undergone a ventriculography suggested by the evidence of large cranial diameters. A diagnosis of chronic right subdural hygroma had been made, and three drainings of xanthochromic fluid had been performed. Moreover, during that hospitalization the patient had exhibited a sudden sensorimotor deficit of left limbs, which recovered in approximately 2 months with the slight residua observed at the aforementioned neurological assessment. Afterward, the patient had presented episodes of myoclonic jerks of the upper left limb, accompanied by left oculogyric crises and followed by Todd’s paralysis. The electroencephalograms showed an epileptogenic focus in the central posterior areas of the right hemisphere, and the patient had been under anticonvulsant therapy since then. No seizures had been recorded in the last 5 years until the present episode, which we interpreted as a partial seizure.

At hospital admission a CT scan (Siemens-Somatom CR high-resolution scanner) showed a large right frontotemporal opercular cortical-subcortical porencephalic area with a density similar to cerebrospinal fluid, enlargement of both frontal horns, and partial agenesia of the corpus callosum. The cortex of the right hemisphere was reduced to a very thin layer, whereas the left hemisphere was normal. A right frontoparietal craniectomy was also evident (Figs 1A and 2A).

Cerebral blood flow (CBF) was measured using a dedicated tomograph (Tomomatic 564, Medimatic) and 133Xe by inhalation (2560 MBq, 80 mCi). Data were simultaneously collected from five transverse slices at 0, 20, 40, 60, and 80 mm above the orbitomeatal plane (slice thickness, 19 mm; spatial resolution, 16 mm). CBF values were corrected for Pco2 changes. Blood pressure and heart rate were measured after each scan. Three CBF measurements were obtained in the same study session: at rest and during the movements of the recovered and normal hands.

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Fig 1. Integrated single-photon emission computed tomography (SPECT)/computed tomography (CT) Images. Right side in the images corresponds to the right in the patient. A, Transaxial CT section after spatial SPECT/CT alignment. The position of CT slice corresponds to the center of the SPECT section (80 mm above the orbitomeatal plane). Arrow indicates the central sulcus. SPECT images were obtained by subtracting pixel-by-pixel cerebral blood flow (CBF) resting scan from those during the motor task with the normal right (B) and recovered left (C) hand. The CBF increases were superimposed on CT images (B and C). Mean CBF increases during the motor task are displayed in color scale (expressed in milliliters per 100 g per minute). They occur in the cortical walls of the central sulcus, corresponding to the premotor and primary sensorimotor cortices. During the motor task with the normal hand, mean CBF increases in the contralateral premotor cortex were 76% and in the contralateral primary sensorimotor cortex were 65%. During the motor task with the recovered hand, mean CBF increases in the ipsilateral premotor cortex were 71% and in the ipsilateral primary motor cortex were 57% (C).

The motor task consisted of sequential finger-to-thumb opposition movements in turn (rate, 60/min). Methodological details of the motor activation study are described elsewhere.7

The threshold for identifying the scalp surface was set at 30% of the maximal gray value on resting scan images and then copied onto the corresponding slices of activated scans. We calculated the global cerebral and normal hemispheric mean flow by summing the CBF values of all slices.

The brain areas activated by the motor task were determined by subtracting pixel by pixel the resting state CBF images from the images obtained during motor tasks. The 30% isocontour, defined on the rest image and copied onto the subtracted image, was used to exclude artifactual areas of increased signal lying outside the brain. To reduce the signal-noise ratio, we considered only the "spots" of increased activity that were more than 30% of the maximum pixel value of the subtracted image. After this procedure, "activated spots" were visualized on slices 4 and 5.

Regions of interest were defined on the spots of increased activity and then automatically transferred onto the corresponding slices of both the rest and
activated scans. CBF was measured in each region of interest for each condition, and the CBF responses to stimulation were expressed as CBF percent change (CBF%Δ) from the initial unstimulated scan.

SPECT and CT images were then scanned and overlapped with an image processing software on a microcomputer (Macintosh Quadra) to obtain anatomic localization of the CBF spots on the corresponding CT images. Identification of the precentral and postcentral gyri on CT images was obtained using a method proposed by Iwasaki et al.8

At rest, global and hemispheric CBF were 29 mL/100 g^-1 • min^-1 and 46 mL/100 g^-1 • min^-1, respectively. These values did not change during motor activation of either the normal or recovered hand.

During the motor task with the normal hand, CBF increased in the brain regions corresponding to the contralateral premotor (46 mL/100 g^-1 • min^-1 versus 81 mL/100 g^-1 • min^-1, %Δ=76, slice 5 [Fig 1B]) and primary sensorimotor cortices (51 mL/100 g^-1 • min^-1 versus 84 mL/100 g^-1 • min^-1, %Δ=65, slice 5 [Fig 1B]; 62 mL/100 g^-1 • min^-1 versus 95 mL/100 g^-1 • min^-1, %Δ=53, slice 4 [Fig 2B]). During the motor task with the recovered hand, CBF increased in the ipsilateral premotor (41 mL/100 g^-1 • min^-1 versus 70 mL/100 g^-1 • min^-1, %Δ=57, slice 5) and ipsilateral primary motor cortices (46 mL/100 g^-1 • min^-1 versus 72 mL/100 g^-1 • min^-1, %Δ=53, slice 5) (Fig 1C).

No CBF changes were observed during motor task performances on the right hemisphere.

Discussion

We described a patient with a putative congenital cerebral malformation, suggested by large cranial diameters since infancy and CT evidence of a poroencephalic area, who at the age of 12 developed a left-sided hemiplegia of undetermined origin. Despite severe damage of the entire right cerebral hemisphere, the patient was able to move both hands normally. Thus, we aimed to determine which cerebral areas were involved in movement execution in this patient.

Motor activation is a well-known method that allows the active brain areas involved in the voluntary movements to be mapped by measuring CBF changes using either SPECT6 or positron emission tomography.7 In normal subjects, the performance of this motor task induces a significant CBF increase in the contralateral sensorimotor cortex.7,8 In normal conditions a bilateral control of motor areas is described only for axial and proximal muscles.9 In our patient, the motor task induced a CBF increase in the left premotor and sensorimotor cortices irrespective of the hand he was moving. No CBF changes were observed on the right (affected) hemisphere. However, the electroencephalographic examination recorded an epileptogenic focus located on this hemisphere. These observations suggest that the motor function of the left hand is predominantly controlled by the ipsilateral motor areas and related pathways, although a partial participation of the contralateral hemisphere cannot be excluded.

Glucose metabolism in the intact cerebral cortex of children after hemispherectomy,10 as well as in the cortex of normal children,11 slowly increases, exceeding adult values at the age of 9, and then returns to adult values at the end of the second decade. In particular, between the ages of 9 and 15, glucose metabolism in sensorimotor cortices of normal children is still as much as twice that at the end of the second decade.12 These metabolic changes are consistent with anatomic data showing a vast excess of neurons, synapses, and dendritic spines in childhood and their subsequent regression in the developing brain.13 Brain plasticity after early brain damage could take place because of this maturation process, as suggested by a remarkable recovery observed after hemispherectomy in children.14 Neurophysiological studies with magnetic stimulation of the intact motor cortex have shown that ipsilateral compound muscle action potentials are present in patients with early brain damage; in contrast, they are not present in patients with later-acquired brain damage or in normal subjects.14-15 The ipsilateral uncrossed pyramidal tract is presumed to mediate the recovery after brain damage in young subjects. Our observation is in agreement with this hypothesis.

In our case, the anatomic localization of activated cortical areas was not exactly similar for the normal and recovered hand. Our findings suggest a greater capacity of recruitment of the ipsilateral premotor cortex in motor recovery, according to recent positron emission tomography studies.4

Despite the difficulties of analyzing CBF changes in a single case, our findings are suggestive of remarkable reorganization and plastic functional changes that have occurred because of early damage to the right hemisphere and, in agreement with other clinical observations2,12 and with neurophysiological studies,14-15 point to a potential role of ipsilateral cortical efferent pathways in subserving hand movements after early cerebral damage.

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