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Role of primary motor cortex in gait: automatic-voluntary dissociation seen in paretic leg of a patient who had a stroke

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ABSTRACT

Objective To examine the role of primary motor cortex in gait through exploring the dissociation of impaired voluntary leg muscle contraction and preserved rhythmic activities during gait in a patient who had a stroke.

Subject and methods A 49-year-old man with an infarct in the primary motor cortex exhibited automatic-voluntary dissociation in the paretic leg. Functional studies were conducted using surface electromyography (EMG) and near-infrared spectroscopy (NIRS).

Results The patient was incapable of voluntary contraction of individual leg muscles on the paretic right side but was able to walk automatically while contracting those muscles rhythmically. Surface EMG confirmed the earlier findings objectively. The preserved automatic activities helped recovery of gait capability, but NIRS showed no functional recovery in the corresponding motor cortex during treadmill gait. We considered that the loss of voluntary leg muscle contraction and the preserved gait capacity in this patient represented a form of automatic-voluntary dissociation.

Conclusions The preserved gait capability suggests that the leg representation of the primary motor cortex may not play a major functional role in gait, but other components of the nervous system, including the spinal central pattern generator, would serve important functions to maintain gait capability.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ The dissociation of impaired voluntary leg muscle contraction and preserved rhythmic activities in a stroke patient was not described before.

WHAT THIS STUDY ADDS

⇒ The observation suggested that the role of primary motor cortex in gait was not indispensable.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE AND/OR POLICY

⇒ The findings proposed the new insight of stroke rehabilitation.

INTRODUCTION

The dissociation of voluntary and emotional facial innervation has been described in textbooks.[1 2] A hemiplegic patient with injury of the corticobulbar fibres and having complete haemifacial palsy of the central pattern may be able to smile spontaneously; for instance, when someone makes him/her laugh. The pathophysiological mechanisms are not fully known, but it is suggested that the fibres mediating the emotional and volitional inputs to the nuclei descend separately.[3] Such dissociations are seen in several other voluntary movements such as kinesié paradoxale in Parkinson's disease and apraxia seen in patients who had a stroke.

Human gait is supported by various neural, skeletal and muscular structures. As neural structures, the primary motor cortex, supplementary motor cortex, basal ganglia, brainstem nuclei, cerebellum and spinal cord are involved. Particularly, the primary motor cortex is considered to be the main structure for fine and fractionated movements.

We observed a patient who had a stroke who had localised ischaemic lesion in the left primary motor cortex. He was incapable of isolated leg muscle contraction on the paretic side but was able to walk. We considered this phenomenon to represent a form of automatic-voluntary dissociation and examined the role of the primary motor cortex in gait.

CASE PRESENTATION

A 49-year-old Japanese sweets craftsman who had no previous stroke episode noticed paresis in his right leg one morning, and was transported to a local acute care hospital. MRI studies at that hospital showed high-intensity lesion in the left paracentral lobule on fluid-attenuated inversion recovery and diffusion-weighted images (figure 1A,B). MR angiography revealed pearl and string sign in the right anterior cerebral artery at A2 segment, suggesting a dissecting aneurysm as the cause of infarction (figure 1C).

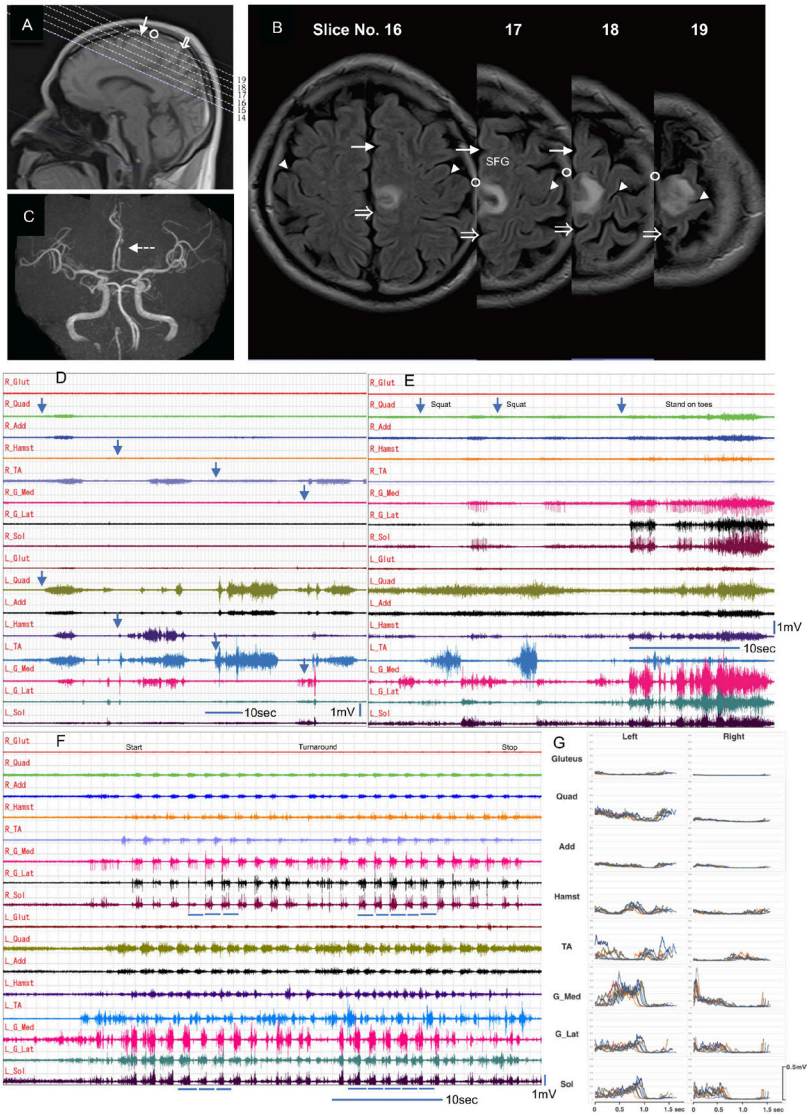

Figure 1 MRI obtained in the former acute care hospital (A–C), and surface electromyography (EMG) studies (D–G) conducted at our hospital. (A) MRI sagittal view 6 mm lateral from the midline; dashed lines indicate slice levels (slice thickness 6 mm) at which ischaemic lesions were revealed in axial view. (B) Fluid-attenuated inversion recovery (FLAIR) images depicted high-intensity lesion in slices No. 14–19, and four slices (No. 16–No. 19) are shown. Located in the paracentral lobule of the left hemisphere, the lesion has an irregular cone shape; 36 mm in height, with the apex directed towards the cingulate gyrus and the round-shaped base (around 20 mm in width) facing the cortical surface. SGF, superior frontal gyrus; triangle, central sulcus; arrow, paracentral sulcus; circle, precentral sulcus; double arrow, marginal ramus of cingulate sulcus. (C) MR angiogram showed pearl and string sign, tapering of the left anterior cerebral artery at A2 segment (arrow with dashed line), and irregular lumen thereafter. (D) Surface EMG recordings in sitting position. The patient was asked to contract each of the marked muscle simultaneously on the left and right (arrows). On the paretic (right) side, no apparent activities were observed in all the muscles, except weak contraction of quadriceps and co-contraction activities in tibialis anterior. (E) Recordings during squat and standing on toes. (F) Recordings when the patient walked to and fro for 10 m with minimal support by a therapist. Consistent activities are seen during standing on toes and walking. (G) To observe consistency of EMG activities during walking, EMG signals were rectified and eight steps with similar elapsed time for consecutive steps (average: 1.47 s on the right and 1.49 s on the left; indicated in F by blue dashed lines) were superimposed. A complete gait cycle was defined as the period between an initial heel floor contact and the next heel floor contact with the same foot. On the unaffected left side, not only reciprocal, but also coordinated activities are seen between tibialis anterior and triceps muscles. On the paretic right side, only reciprocal activities are observed. Amplitudes of all EMG channels are the same. (D–G) Surface electrodes were placed bilaterally on the gluteus maximus (Glut), quadriceps femoris (Quad), adductor femoris (ADD), hamstrings (Hamst), tibialis anterior (TA) and triceps surae [gastrocnemius lateralis (G_Lat), medialis (G_Med) and soleus (Sol)] muscles. R: right, L: left.

Low-density area observed on T2*WI image confirmed haemorrhage in the infarct area. No abnormal findings suggesting previous stroke were detected. The diagnosis was cerebral infarction in left frontal cortex caused by dissection of left anterior cerebral artery. Medical examination revealed risk factors of stroke including diabetes

mellitus, hypertension, dyslipidaemia and homocysteinaemia. After 3 weeks of conservative acute-phase treatment, he was transferred to our hospital for stroke rehabilitation.

Neurological examination at admission showed clear consciousness, no cognitive disturbances, no facial palsy and no upper limb weakness. However, paresis was found in his right leg. Manual muscle testing[4] score was 3 in the proximal muscles (iliopsoas, quadriceps and hamstrings), and 0 in the distal muscles (tibialis anterior, gastrocnemius, soleus, and toe extensors and flexors). No leg muscle atrophy was found. Deep tendon reflexes were normal in both arms and in the left leg. In the right leg, patella tendon reflex (PTR) and Achilles' tendon reflex (ATR) were diminished. Plantar reflex was flexor on the unaffected side, and no response on the paretic side. Sensation was normal to all modalities. Paralysis on the right side as assessed by Brunnstrom recovery stage[5] was stage VI in upper arm and fingers, and stage I–II in the leg.

METHODS AND RESULTS

Surface electromyography

Surface electromyography (EMG) was performed with electrodes placed on bilateral gluteus maximus, quadriceps femoris, adductor femoris, hamstrings, tibialis anterior, gastrocnemius medialis and lateralis, and soleus muscles. EMG activities were amplified by Bagnoli and recorded on PC simultaneously with video recording by TeraRevueMR. During sitting position, the patient was asked to contract each of the above muscles. While normal contraction was observed in all the muscles on the unaffected left side, no contraction was found in the distal muscles on the paretic right side, except weak contraction in the quadriceps and co-contraction in the tibialis anterior (figure 1D). The lack of contraction of isolated right leg muscles was seen also in supine and standing positions. Despite the lack of activities during volitional efforts, large amount of EMG activities and rhythmic activities were recorded from the distal leg muscles on the paretic side while the patient was standing on his toes and during floor walking (figure 1E,F). At the beginning of admission, his walking was unstable and supported by a therapist. To observe consistency of EMG activities, surface EMG was rectified and data of eight selected steps with relatively small variations in elapsed time per step were superimposed (figure 1G). Reciprocity between tibialis anterior and triceps muscle was clearly observed on the paretic right side, whereas variabilities and co-contractions among these muscles were depicted on the unaffected left side.

Functional condition of motor cortices

We conducted functional analysis of the cerebral motor cortex using near-infrared spectroscopy (NIRS). One set of 3×5 probe arrays was placed symmetrically on the vertex to cover the primary motor cortices. Oxygenation was measured by continuous-wave optical topography (ETG-4000, Hitachi Medical Corporation, Tokyo, Japan) with 28 channels using 3×5 probe arrays. Details of the methods have been reported by Ebihara *et al*.[6] Oxyhaemoglobin (Oxy-Hb) concentration was measured.

During treadmill walk, there was an increase in concentration of Oxy-Hb on the right side; that is, an increment of blood flow in the right motor cortex compared with the resting stage, but not on the left side, particularly in the parasagittal area.

Outcome of rehabilitation

Two months later, PTR on the right became more active than the left, but ATR remained weak. Finally, the patient was discharged from the hospital and returned to his previous work, using a T-cane and a small ankle-foot orthosis. At the time of discharge, NIRS showed no functional recovery in the left motor cortex during treadmill gait, although surface EMGs on the right side improved and the amplitudes of proximal muscles increased during isolated muscle contractions. During walking, however, lack of coordinated activities among flexors and extensors remained on the paretic side, and adapted changes of EMG activities were seen on the unaffected side.

DISCUSSION

We presented a 49-year-old man with an infarct in the left anterior cerebral artery, who was not able to contract his leg muscles individually on the paretic right side, whereas he was able to walk, dragging his paretic leg along and contracting the muscles of the paretic leg rhythmically. Combined with the neurological findings and functional recovery, this could be explained with the failure of central nervous system, but of the peripheral nervous system. We consider this phenomenon to represent a form of automatic-voluntary dissociation.

In our patient, the lesion was located in the terminal region of the anterior cerebral artery, and was interpreted as the paracentral lobule located between the precentral sulcus and the marginal part of cingulate sulcus. The lesion would correspond to the primary motor cortex of the right leg. Gait is realised by the coordinated works of the entire nervous system and body structures. The rhythmic activities of the lower limbs are speculated to be the results of CPGs. Although the existence of CPGs in humans remains a matter of debate, their functional role is increasing being supported by indirect evidence obtained in humans.[7 8] The spinal CPGs should be functioning in this case, because the reciprocal relation of antagonistic muscles in the leg shown in figure 1G was observed in CPGs of human spinal cord injury.[9] Besides the CPGs, the supraspinal structures comprising the brainstem, basal ganglia, cerebellum and cerebral cortex are engaged in many predictive actions.[10] In bipedally walking monkeys, microinjections of muscimol ($GABA_A$-receptor agonist) to inactivate the leg area of the primary motor cortex (M1) resulted in paresis of the contralateral

leg.[10] The monkey was able to walk while dragging the contralateral leg along.[11] This finding resembled the condition of our patient, implying that walking is possible even when the primary motor cortex is not functioning.

The automatic-voluntary dissociation seen in our patient could be interpreted as a symptom of apraxia which would explain the observation that he was able to move his leg automatically but just incapable of contracting the muscles intentionally. Even after acquiring the endurance in walking, the coordinated EMG activities between the tibialis anterior and triceps muscles in the stance phase seen on the unaffected side, presumably to compensate the unsteadiness of walking, were not seen on the paretic side, although there was clear reciprocity between flexors and extensors (not shown, but similar to figure 1G). With this finding, we speculate that EMG pattern of automatic-voluntary dissociation seen in this patient may reflect the features of paresis and its recovering process, and do not support apraxia.

When the supra-spinal innervation of the spinal cord is disrupted, the voluntary control and all the spinal reflexes will be lost, which is known as spinal shock.[12] Considering the partial recovery of isolated muscle movements and enhanced PTR during the rehabilitation period, the dissociation observed in our case might be due to transient loss of supra-spinal innervation, which differs from the automatic-voluntary dissociation of facial muscles.

In conclusion, the leg representation of the primary motor cortex may not play a major role in the generation of gait pattern but may control isolated contraction of leg muscles and coordinated actions among these muscles during walking.

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Data availability statement All data relevant to the study are included in the article. If required, any further data can be made available on reasonable request.

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