



: fMRI (fMRI)
 fMRI
 : 6 2 7 1
 61 (grade of muscle power by the Medical Research
 Council) II (2), III (3), IV (3) . 1.5 T
 4 . SPM99

: 6 II
 가 , III - IV
 가 4 가 III 40
 II 가 가 가
 . IV 가 가 가
 : 가 III - IV
 가

가 (8, 9).
 가 가 fMRI
 (1-4).

(Functional Magnetic Resonance Imaging; fMRI) (grade of muscle power by
 the Medical Research Council)
 (5-7).

PET

가

가

가

1

6

45

75

2, 7

61

¹
²

(grade of muscle power by the Medical Research Council)

II (2), III (3), IV (3) . 8

(10) 7 - 10 fMRI 960 (spin
 fMRI 1.5 T (Vision, Si- echo) 4 mm 26 (level)
 emens, Erlangen, Germany) (head T1 (TR/TE 500/14 ms, 70,
 coil) fMRI , 256 x 256, 4 mm, 220 x 220 mm)
 (noise) (artifact)
 (foam pad)
 (turbo - FLASH) (scout (screen)
 image) (central 가
 sulcus)가 (localizer) 10

. blood oxygen level - dependent (BOLD)
 (echo planar imaging, EPI)
 TR/TE 3599/40 ms, 90°, 64 x 64,
 6 mm, 220 x 220 mm

(A) 6

(B) 6

A - B - A - B - A - B - A - B).

4 (

24 , 20

(simple motor)

가

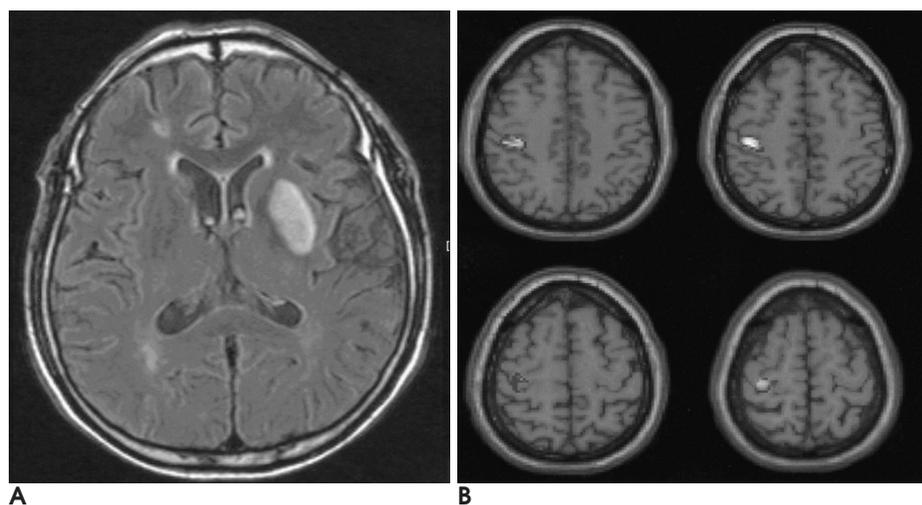


Fig. 1. Axial FLAIR image show left lentiform nucleus infarction (A). The muscle power by the Medical Research Council was Grade II. Right (non-lesion side) sensorimotor cortex is activated in the axial fMRI (B).

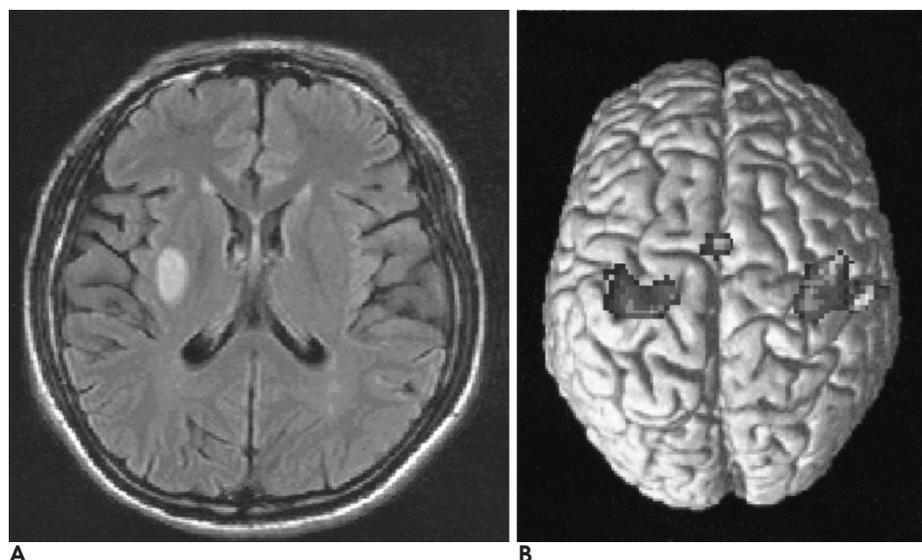


Fig. 2. Axial FLAIR image show right lentiform nucleus infarction (A). The muscle power by the Medical Research Council was Grade III. Volume rendering fMRI shows the extended activation in right (lesion side) sensorimotor cortex during the both motor tasks (B).

(11). head image file Statistical Parametric Mapping software (SPM99; Wellcome Department of Cognitive Neurology) (motion correction)

(reference curve) 48 (signal intensity) 가 (cross-correlation) (correlation threshold) $\rho = 0.005$

(activated pixel) 30vol (11). SPM99 (Talairach's coordinates) Talairach and Tournoux (primary sensorimotor cortex, Brodman's area 4 and 3) (region of interest) (Volume) (laterality index, LI)

(template) SPM99 (12).

(C:Contralateral) (I:Ipsilateral) , LI (C - I)/(C+I) LI

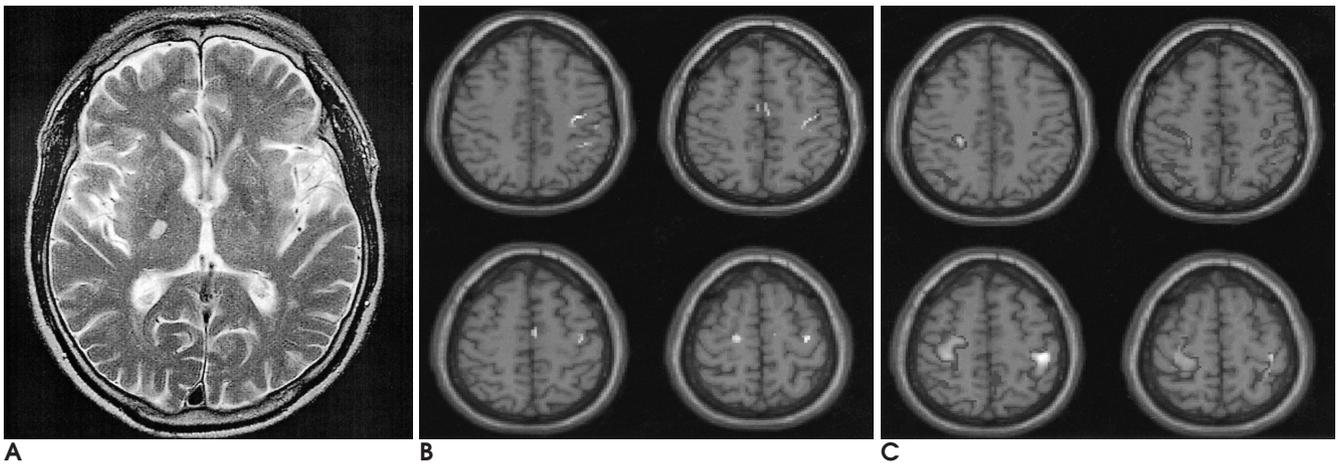


Fig. 3. Axial T2 weighted image show right thalamus infarction (A). The muscle power by the Medical Research Council was Grade II. Axial fMRI in the acute infarcted state shows the extended activation in left (non-lesion side) sensorimotor cortex during the both motor tasks (B). In the follow up fMRI after 40 days (MRC Grade: III at that time), the activated volumes of both sensorimotor cortex increase. The activated volume in lesion side sensorimotor cortex is more than that in non-lesion side sensorimotor cortex (C).

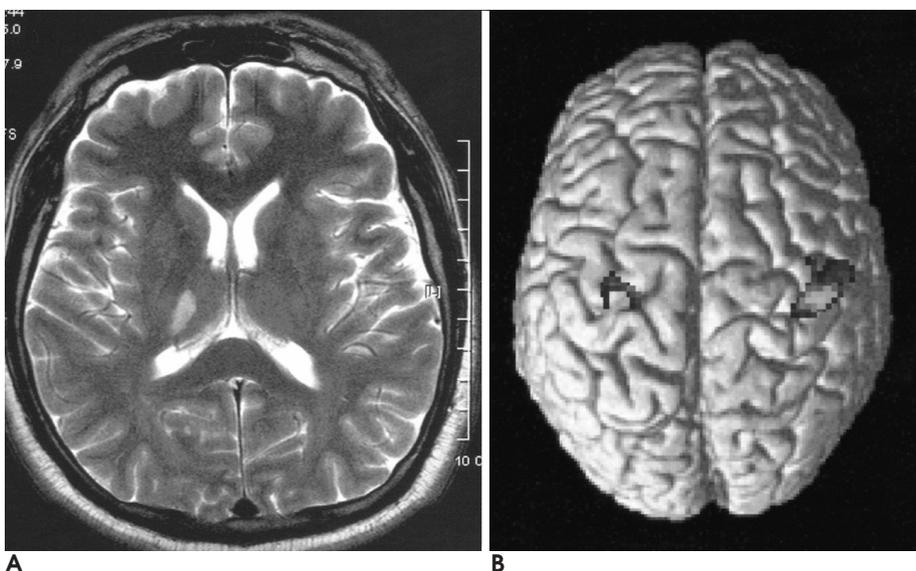


Fig. 4. Axial T2 weighted image shows right thalamus infarction (A). The muscle power by the Medical Research Council was Grade IV. Volume rendering fMRI shows the extended activation in right (lesion side) sensorimotor cortex during the both motor tasks (B).

Motor fMRI in Acute Infarction of Basal Ganglia and Thalamus¹

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Purpose: We wanted to assess the cerebral activation of the motor function after deep cortical (lentiform nucleus and thalamus) infarction.

Materials and Methods: We studied the motor function of eight right-handed deep cortical infarcted patients (mean age, 61 years; 7 men and 1 woman) who suffered a single unilateral deep cortical (lentiform nucleus or thalamus) infarction. The grade of muscle power by the grading system of the Medical Research Council was II in two patients, III in three patients and IV in three patients. All the MRI experiments were performed with a 1.5T scanner. The fMRI protocol consisted of eight alternating periods of task performance and rest. The activation tasks consisted of finger movements. Data analysis of activated area and calculation of the activated volumes in sensorimotor cortex were done.

Results: For the six lentiform nucleus acute infarction patients, one right hemiparetic patient (MRC Grade: II), and only the right sensorimotor cortex (the unilateral non-lesion side) were activated. In five (MRC Grade: III - IV) of the six lentiform nucleus infarcted patients, bilateral activations of the primary sensorimotor cortex were recorded. In four of the five bilaterally activated patients, extended activations in the lesion side sensorimotor cortex were observed. In the two right thalamic infarction patients, bilateral activations of the primary sensorimotor cortex were recorded. One patient (MRC Grade: II) was observed to have an extended activation in the non-lesion side sensorimotor cortex. On the follow up fMRI done on this patient after 40 days (MRC Grade: III at that time), the activated volumes of both sensorimotor cortexes were increased. The activated volume in the lesion side sensorimotor cortex was more than that in the non-lesion side sensorimotor cortex. The other patient (MRC Grade: IV) was observed to have extended activation in the lesion side sensorimotor cortex.

Conclusion: fMRI allows for the study of the motor function in deep cortical infarction. We were able to investigate the differences in motor activation according to the individual MRC Grades. fMRI may be a useful tool to monitor and study deep cortical infarction, and it may be important to help us understand the function of the deep cortical areas.

Index words : Brain

Magnetic resonance (MR)

Infarction

fMRI

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