

# Motor recovery by improvement of limb-kinetic apraxia in a chronic stroke patient

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## Abstract.

**OBJECTIVES:** We report on a chronic stroke patient who showed motor recovery by improvement of limb-kinetic apraxia (LKA) after undergoing intensive rehabilitation for a period of one month, which was demonstrated by diffusion tensor tractography (DTT) and transcranial magnetic stimulation (TMS).

**METHODS:** A 50-year-old male patient presented with severe paralysis of the left extremities at the onset of thalamic hemorrhage. At thirty months after onset, the patient exhibited moderate weakness of his left upper and lower extremities. In addition, he exhibited a slow, clumsy, and mutilated movement pattern during grasp-release movements of his left hand. During a one-month period of intensive rehabilitation, which was started at thirty months after onset, the patient showed 22% motor recovery of the left extremities. The slow, clumsy, and mutilated movement pattern of the left hand almost disappeared.

**RESULTS:** DTTs of the corticospinal tract (CST) in both hemispheres originated from the cerebral cortex, including the primary motor cortex, and passed along the known CST pathway. The DTT of the right CST was located anterior to the old hemorrhagic lesion. TMS study performed at thirty and thirty-one months after onset showed normal and similar findings for motor evoked potential in terms of latency and amplitude of the left hand muscle.

**CONCLUSIONS:** We think that the motor weakness of the left extremities in this patient was mainly ascribed to LKA and that most of the motor recovery during a one-month period of rehabilitation was attributed to improvement of LKA.

Keywords: Motor recovery, stroke, diffusion tensor tractography, transcranial magnetic stimulation, apraxia, limb-kinetic apraxia

## 1. Introduction

Stroke is a leading cause of major disability in adults. Motor weakness is one of the most serious disabling sequelae of stroke, with more than 50% of stroke patients experiencing a residual motor deficit [6]. Injury of neural tracts affecting motor function is accompanied by motor weakness, however, apraxia can also accompany motor weakness like symptoms.

Apraxia is defined as the inability to perform learned skilled movements *in spite* of preservation of power, sensation, and comprehension for execution of movements [9–11]. Limb-kinetic apraxia (LKA), one of the apraxias, is defined as the execution disorder of movements, resulting from injury of the premotor cortex (PMC) or the corticofugal tract from the PMC with preservation of the corticospinal tract (CST) [12, 19, 21, 26].

Diagnosis of LKA can be difficult because it is made by clinical observation of movements with confirmation of the CST state. Diffusion tensor tractography (DTT), derived from diffusion tensor imaging (DTI), provides a unique advantage for three-dimensional visualization and estimation of the CST [13, 16, 17, 24]. Transcranial magnetic stimulation (TMS) stimulates the neurons of the CST and provides a unique advantage for estimating the amount of CST fibers by measuring amplitude of motor evoked potential (MEP) [27]. Therefore, TMS could be useful for demonstrating change in the amount of CST fibers.

In the current study, we report on a chronic stroke patient who showed motor recovery by improvement of LKA after undergoing intensive rehabilitation for a period of one month, which was demonstrated by DTT and TMS.

## 2. Case report

The standardized Motricity Index (MI) and Medical Research Council (MRC) were used for determination of motor function [2, 4]. The MI, with a maximum score of 100, is a measure of the integrity of extremity motor function. Reliability and validity of the MI are well-established [4]. The MRC score was used for evaluation of motor function: 0, no contraction; 1, palpable contraction but no visible movement; 2, movement without gravity; 3, movement against gravity; 4, movement against a resistance lower than the resistance overcome by the healthy side; and 5, movement against a resistance equal to the maximum resistance overcome by the healthy side [2].

A 50-year-old, right-handed male patient presented with severe paralysis of the left upper and lower extremities at the onset of thalamic hemorrhage (MI: 0). The patient received conservative management at the neurosurgery department of a university hospital and then underwent rehabilitation at a local rehabilitation hospital. However, he could not perform most daily activities using his left hand and could not walk independently without a cane. At thirty months after stroke onset, the patient was admitted to the rehabilitation department of a university hospital in order to undergo intensive rehabilitation. At the time of admission, the patient's scores on the Mini-Mental State Exam and the ideomotor apraxia test were 28 (cut-off score <25) and 40 (cut-off score <32), respectively [3, 7]. According to the Nottingham Sensory Assessment (The subscales for tactile sensation (16, full mark: 20) and kinesthetic sen-

Table 1  
Changes of motor function and the results of transcranial magnetic stimulation in the patient

Duration from onset		Onset	30 months	31 months
MRC	Shoulder abductor	0	3 <sup>+</sup>	4 <sup>+</sup>
	Elbow flexor	0	4	5
	Finger flexor	0	3 <sup>+</sup>	4 <sup>+</sup>
	Finger extensor	0	3	4
	Hip flexor	0	3 <sup>+</sup>	4 <sup>+</sup>
	Knee extensor	0	4	5
	Ankle dorsiflexor	0	2 <sup>+</sup>	4
MI	Upper extremity	0	65	84
	Lower extremity	0	57	83
	Total	0	61	83
TMS	Latency (msec)		22.3	22.7
	Amplitude ( $\mu$ V)		4000	4000

MRC: Medical Research Council, MI: Motricity Index, TMS: Transcranial magnetic stimulation.

sation (20: full mark: 24), he showed mild impairment of somatosensory function [23]. The patient exhibited moderate weakness of his left upper and lower extremities (MI: 61) (Table 1). In addition, he exhibited slow, clumsy, and mutilated movements when performing grasp-release movements of his left hand. He walked with a pattern of spastic circumduction and severe associated reaction of the left elbow. T2-weighted MR images taken at thirty months after onset showed an old hemorrhagic lesion in the right thalamus, posterior limb of the internal capsule, and midbrain (Fig. 1). The patient provided signed, informed consent and our institutional review board approved the study protocol.

The patient participated in a comprehensive rehabilitative management program, including movement therapy, dopaminergic drugs for improvement of apraxia (ropinorole, 3 mg; bromocriptine, 10 mg; levodopa, 375 mg), antispastic drugs (baclofen 45 mg), and neuromuscular electrical stimulation therapy of the left elbow extensors, finger extensors, knee extensors, and ankle dorsiflexors (two times, 20 minutes/time, seven days/week). Movement therapy was performed primarily for improvement of motor function, movement pattern of the left extremities, and postural control, and was performed six days (Monday through Friday: 2.5 hours/day, Saturday: one hour/day) per week. During a one-month period of intensive rehabilitation, the patient showed motor recovery of the left upper and lower extremities of as much as 22% on MI (61→83). In addition, his slow, clumsy, and mutilated movement pattern when performing grasp-release movements of his left hand almost disappeared. He was able to walk independently, showing marked improvement of spastic circumduction gait pattern and severe associated reaction of the left elbow.

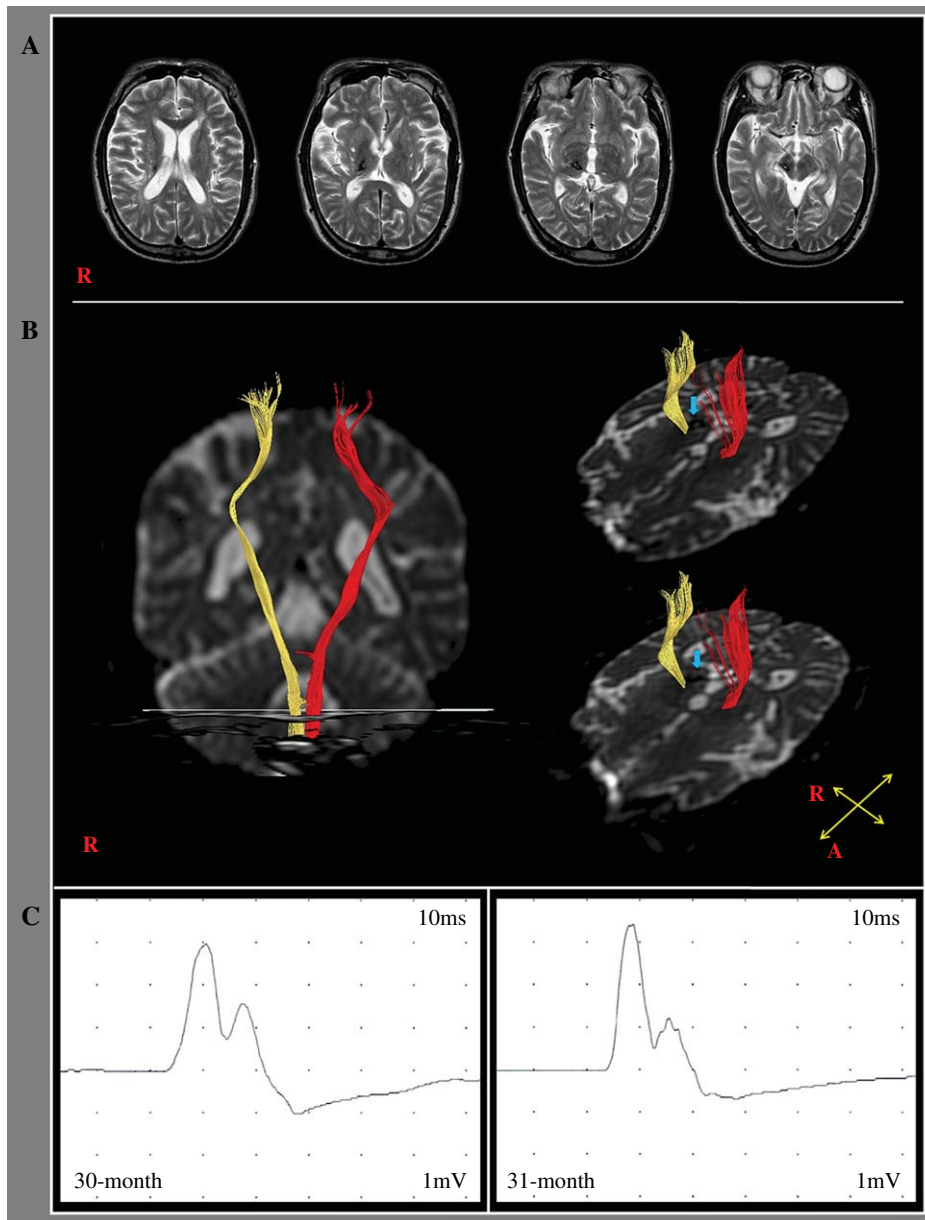


Fig. 1. A. T2-weighted brain MR images show an old hemorrhagic lesion in the right thalamus, posterior limb of the internal capsule, and midbrain. B. Diffusion tensor tractography for the corticospinal tract of the affected hemisphere at thirty months after onset was similar to that of the hemisphere of the unaffected side. The old hemorrhagic lesion (arrow) was located posterior to the right corticospinal tract. C. Motor-evoked potentials obtained from the left abductor pollicis brevis muscle at thirty and thirty-one months after onset showed similar latency and amplitude.

### 2.1. Diffusion tensor tractography

A 6-channel head coil on a 1.5 T Philips Gyroscan Intera (Philips, Ltd., Best, the Netherlands) with single-shot echo-planar imaging was used for acquisition of DTI data. For each of the 32 non-collinear diffusion sensitizing gradients, we acquired 70

contiguous slices parallel to the anterior commissure-posterior commissure line. Scanning was performed from the cortex to the middle of the second cervical vertebral body. Imaging parameters were as follows: acquisition matrix =  $96 \times 96$ ; reconstructed to matrix =  $192 \times 192$  matrix; field of view =  $240 \text{ mm} \times 240 \text{ mm}$ ; TR = 10,398 ms; TE = 72 ms;

parallel imaging reduction factor (SENSE factor)=2; EPI factor=59;  $b = 1000 \text{ s/mm}^2$ ; NEX=1; and a slice thickness of 2.5 mm (acquired isotropic voxel size  $2.5 \text{ mm} \times 2.5 \text{ mm} \times 2.5 \text{ mm}$ ). The fiber assignment continuous tracking (FACT) algorithm implemented within the DTI task card software (Philips Extended MR WorkSpace 2.6.3) was used in performance of fiber tracking [24]. For CST analysis, a seed region of interest (ROI) was placed on the CST at the anterior pontomedullary junction on an axial slice and a target ROI was drawn in the CST area of the anterior medulla [14]. The termination criteria applied were fractional anisotropy (FA)  $<0.2$  and an angle change of  $>45$  degrees [17].

Results of DTT of the right and left CST showed that fiber tracts originated from the cerebral cortex, including the primary motor cortex, and passed along the known CST pathway (Fig. 1-B). DTT of the right CST was located anterior to the old hemorrhagic lesion.

## 2.2. Transcranial Magnetic Stimulation

A Magstim Novamatrix 200 magnetic stimulator with a 9-cm mean diameter circular coil (Novamatrix Inc.) was used in performance of TMS. Cortical stimulation was performed with the coil held tangentially over the vertex. Stimulation of the left hemisphere was provided by a counterclockwise current, and stimulation of the right hemisphere was provided by a clockwise current. Four motor-evoked potentials (MEPs) were obtained from both abductor pollicis brevis (APB) muscles in a relaxed state. Stimulation intensity was set at the maximum stimulator output. Each site was stimulated three times with inter-stimulus intervals of  $>10$  seconds, and MEP having the shortest latency and the average peak-to-peak amplitudes was adopted.

TMS study performed at thirty and thirty-one months after onset showed similar MEP findings at the left APB muscle (thirty months: latency- 22.3 msec; amplitude: 4000  $\mu\text{V}$ ), thirty-one months: latency- 22.7 msec; amplitude: 4000  $\mu\text{V}$ ).

## 3. Discussion

Diagnosis of LKA is difficult because there are no specific diagnostic tools; instead, it is dependent on clinical observation of movements. The nature of LKA has not been clearly elucidated, therefore, it has been controversial. Summarizing previous studies, the

characteristics of LKA are as follows [5, 9, 12, 21, 22]: 1) awkward, clumsy, coarse, mutilated pattern of execution of simple movements, confined mainly to movements of the affected hand, 2) resulting mainly from frontal lobe damage centered on the PMC or the corticofugal tract from the PMC with preservation of the CST, 3) no voluntary-automatic dissociation, 4) differentiation from concurrent limb weakness resulting from injury of the CST is difficult because it is similar to the motor weakness observed following CST injury, and 5) normal conceptual knowledge of motor performance.

In this patient, we observed 22% motor recovery for a period of one month from thirty months after onset. We think that the motor weakness of the left extremities in this patient was mainly ascribed to LKA and that most of the motor recovery during a one-month period of rehabilitation was attributed to improvement of LKA, for the following reasons. First, we confirmed that the CST in the affected (right) hemisphere showed normal findings in terms of MEP parameters (latency and amplitude) on TMS and configuration on DTT [15]. However, the patient exhibited moderate motor weakness in the left extremities until thirty months after onset. The clinical characteristics of left hand movements (slow, clumsy, and mutilated) were also compatible with those of LKA. However, because the patient showed normal cognition for motor performance and a normal result on the ideomotor apraxia test (20: cut-off score  $<32$ ), we were able to rule out ideational and ideomotor apraxia. Second, the patient had a lesion around the CST in the posterior limb of the internal capsule, which is known to related to the corticofugal tract from the PMC, which is responsible for LKA [5, 8, 9, 12, 21, 25]. Third, he showed rapid motor recovery for one month from thirty months after onset, which is well-known as the plateau stage of motor recovery in stroke patients. Considering the amplitudes of MEP, which indicate that the fiber numbers of the CST in the affected hemisphere were not changed between thirty and thirty-one months, we can assume that the motor recovery was mainly attributed to improvement of LKA. In particular, dopaminergic drugs, which have been reported to be effective for treatment of apraxia, appeared to have been effective for improvement of LKA [18, 20, 28, 29].

To the best of our knowledge, only a few studies using DTI have reported on LKA [1, 12]. In 2008, in an investigation of changes in white matter in 21 patients with corticobasal degeneration syndrome, Borroni et al. reported correlation of limb apraxia with parietal atrophy and with FA reductions in parietofrontal associative

fibers [1]. In addition, the limb-kinetic component of apraxia showed correlation with reduction of hand sensorimotor connecting fibers. Recently, Hong et al. [2012] reported on a patient with ideomotor apraxia and LKA following a cerebral infarct in the left frontal cortex [12]. They demonstrated the partial injuries of the neural tracts from the PMC and superior longitudinal fasciculus in the affected hemisphere that were responsible for LKA and ideomotor apraxia, respectively.

In conclusion, we report on a patient who showed motor recovery during a one-month period between thirty and thirty-one months after stroke onset. Using DTT and TMS, we demonstrated that motor recovery was mainly ascribed to improvement of LKA. Because LKA can accompany motor weakness like symptoms, and it can be resolved by intensive rehabilitation, including dopaminergic drugs, diagnosis of LKA is important in stroke rehabilitation. Therefore, we believe that our results have important implications for stroke rehabilitation. However, because it is a case report, this study is limited. Conduct of further complementary studies involving larger numbers of cases is warranted.

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## References

- [1] Borroni, B., Garibotto, V., Agosti, C., Brambati, S. M., Belli, G., Gasparotti, R., Padovani, A., & Perani, D. (2008). White matter changes in corticobasal degeneration syndrome and correlation with limb apraxia. *Arch Neurol*, *65*, 796-801.
- [2] Council, M. R. (1976). Aids to the Examination of the Peripheral Nervous System Her Majesty's Stationery Office London.
- [3] De Renzi, E., Motti, F., & Nichelli, P. (1980). Imitating gestures. A quantitative approach to ideomotor apraxia. *Arch Neurol*, *37*, 6-10.
- [4] Demeurisse, G., Demol, O., & Robaye, E. (1980). Motor evaluation in vascular hemiplegia. *Eur Neurol*, *19*, 382-389.
- [5] Denes, G., Mantovan, M. C., Gallana, A., & Cappelletti, J. Y. (1998). Limb-kinetic apraxia. *Mov Disord*, *13*, 468-476.
- [6] Duncan, P. W., Goldstein, L. B., Matchar, D., Divine, G. W., & Feussner, J. (1992). Measurement of motor recovery after stroke. Outcome assessment and sample size requirements. *Stroke*, *23*, 1084-1089.
- [7] Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state. A practical method for grading the cognitive state of patients for the clinician." *J Psychiatr Res*, *12*, 189-198.
- [8] Freund, H. J. (1990). Premotor area and preparation of movement. *Rev Neurol (Paris)*, *146*, 543-547.
- [9] Gross, R. G., & Grossman, M. (2008). Update on apraxia. *Curr Neurol Neurosci Rep*, *8*, 490-496.
- [10] Heilman, K. M. (2010). Apraxia. *Continuum (Minneapolis)*, *16*, 86-98.
- [11] Heilman, K. M., & Valenstein, E., (2003) *Clinical neuropsychology*, Oxford, New York: Oxford University Press, pp. 15, 716.
- [12] Hong, J. H., Lee, J., Cho, Y. W., Byun, W. M., Cho, H. K., Son, S. M., & Jang, S. H. (2012). Limb apraxia in a patient with cerebral infarct: Diffusion tensor tractography study. *NeuroRehabilitation*, *30*, 255-259.
- [13] Jang, S. H. (2011). Diffusion tensor imaging studies on corticospinal tract injury following traumatic brain injury: A review. *NeuroRehabilitation*, *29*, 339-345.
- [14] Jang, S. H. (2011). Somatotopic arrangement and location of the corticospinal tract in the brainstem of the human brain. *Yonsei Med J*, *52*, 553-557.
- [15] Jang, S. H., Cho, S. H., Kim, Y. H., You, S. H., Kim, S. H., Kim, O., Yang, D. S., & Son, S. (2005). Motor recovery mechanism of diffuse axonal injury: A combined study of transcranial magnetic stimulation and functional MRI. *Restor Neurol Neurosci*, *23*, 51-56.
- [16] Jung, Y. J., & Jang, S. H. (2012). The fate of injured corticospinal tracts in patients with intracerebral hemorrhage: Diffusion tensor imaging study. *AJNR Am J Neuroradiol*, *33*, 1775-1778.
- [17] Kunimatsu, A., Aoki, S., Masutani, Y., Abe, O., Hayashi, N., Mori, H., Masumoto, T., & Ohtomo, K. (2004). The optimal trackability threshold of fractional anisotropy for diffusion tensor tractography of the corticospinal tract. *Magn Reson Med Sci*, *3*, 11-17.
- [18] Kwon, H., & Jang, S. H. (2012). Delayed recovery of gait function in a patient with intracerebral haemorrhage. *J Rehabil Med*, *44*, 378-380.
- [19] Larner, A. J. (2001). *A dictionary of neurological signs: Clinical neurosemiology*, Dordrecht, Boston: Kluwer Academic Publishers, pp. 15, 226.
- [20] Lee, K. C., Finley, R., & Miller, B. (2004). Apraxia of lid opening: Dose-dependent response to carbidopa-levodopa. *Pharmacotherapy*, *24*, 401-403.
- [21] Leiguarda, R. C., & Marsden, C. D. (2000) Limb apraxias: Higher-order disorders of sensorimotor integration. *Brain*, *123*(Pt 5), 860-879.
- [22] Liepmann, H. (1920). Apraxie. *Ergebn ges Med*, *1*, 516-543.
- [23] Lincoln, N. B., Jackson, J. M., & Adams, S. A. (1998). Reliability and revision of the nottingham sensory assessment for stroke patients. *Physiotherapy*, *84*, 358-365.
- [24] Mori, S., Crain, B. J., Chacko, V. P., & van Zijl, P. C. (1999). Three-dimensional tracking of axonal projections in the brain by magnetic resonance imaging. *Ann Neurol*, *45*, 265-269.
- [25] Newton, J. M., Ward, N. S., Parker, G. J., Deichmann, R., Alexander, D. C., Friston, K. J., & Frackowiak, R. S. (2006). Non-invasive mapping of corticofugal fibres from multiple motor areas—relevance to stroke recovery. *Brain*, *129*, 1844-1858.
- [26] Ochipka, C., & Gonzalez Rothi, L. J. (2000). Limb apraxia. *Semin Neurol*, *20*, 471-478.
- [27] Rossini, P. M., Barker, A. T., Berardelli, A., Caramia, M. D., Caruso, G., Cracco, R. Q., Dimitrijevic, M. R., Hallett,

- M., Katayama, Y., Lucking, C.H., et al. (1994). Non-invasive electrical and magnetic stimulation of the brain, spinal cord and roots: Basic principles and procedures for routine clinical application. Report of an IFCN committee. *Electroencephalogr Clin Neurophysiol*, 91, 79-92.
- [28] Yamada, S., Matsuo, K., Hirayama, M., & Sobue, G. (2004). The effects of levodopa on apraxia of lid opening: A case report. *Neurology*, 62, 830-831.
- [29] Yasuoka, T., Ikeda, M., Maki, N., Hokoishi, K., Komori, K., & Tanabe, H. (2001). A case of corticobasal degeneration of which movemental disturbances were improved by administration of amantadine. *No To Shinkei*, 53, 781-785.

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