Motor Recovery Mechanisms in Patients with Middle Cerebral Artery Infarct: A Mini-Review

Sung Ho Jang

Department of Physical Medicine and Rehabilitation, College of Medicine, Yeungnam University, Taegu, Republic of Korea

Key Words

Stroke · Diffusion tensor imaging · Diffusion tensor tractography · Middle cerebral artery · Motor recovery · Brain plasticity

Abstract

Previous studies on the mechanisms of motor recovery in patients with middle cerebral artery (MCA) infarct except for the studies on cortical peri-infarct reorganization after primary motor cortex infarct were reviewed. Eight studies were classified and reviewed by the following four motor recovery mechanisms: contribution of premotor cortex, subcortical peri-infarct reorganization, ipsilateral motor pathway, and contribution of aberrant motor pathway. MCA infarct is the most common type among the arterial territory infarcts. In addition, MCA territory is important for motor function because it comprises the corticospinal tract, the corticoreticulospinal tract and the neural tracts which are related with apraxia (superior longitudinal fasciculus and corticofugal fibers from the premotor cortex). In spite of the significance of the MCA territory for motor function, fewer studies have reported on the mechanisms of motor recovery in MCA infarct compared to total number of studies about cerebral infarct. In addition, the majority of these studies have focused on the corticospinal tract. Therefore, the total number of studies on this topic should be increased. We believe that

further studies comprising other neural tracts except for the corticospinal tract including the extrapyramidal tracts such as the corticoreticulospinal and neural tracts which are related with apraxia, are necessary.

Copyright © 2012 S. Karger AG, Basel

Introduction

Middle cerebral artery (MCA) infarct is the most common type among the cerebral vascular territory infarcts [1]. In addition, patients with MCA infarct are known to become highly dependent for daily activities [1–3]. That seems to be related to the characteristics of the MCA territory for motor function. The MCA territory comprises the corticospinal tract (CST) which is mandatory for fine motor activity of hand, the corticoreticulospinal tract which is involved in postural control and locomotor function, the corticofugal fibers which are responsible for limb-kinetic apraxia, and the anterior portion of superior longitudinal fasciculus which is related with ideomotor apraxia [4–16].

Elucidation of the mechanisms of motor recovery in stroke is important because such information could provide the scientific information for stroke rehabilitation and for prediction of prognosis. Therefore, many studies have tried to clarify the motor recovery mechanisms in

stroke and several recovery mechanisms have been suggested: the ipsilateral motor pathway from the unaffected motor cortex to the affected extremities, perilesional reorganization, recovery of a damaged lateral CST and contribution of the secondary motor area [17–19]. However, as for the patients with MCA infarct, relatively fewer studies have reported on the mechanisms of motor recovery compared to total number of studies about cerebral infarct, consequently, the mechanisms of motor recovery in MCA infarct have not been clearly elucidated so far [20–27].

In the current study, we reviewed previous studies with regard to the mechanisms of recovery in patients with MCA infarct. Relevant studies were identified using the following electronic databases: PubMed and MEDLINE, from 1966 to 2012. The following key words were used: stroke, MCA, cerebral infarct, CST, corticoreticulospinal tract, brain plasticity, rehabilitation, motor recovery, functional recovery, hemiparesis, diffusion tensor imaging (DTI), diffusion tensor tractography (DTT), functional MRI (fMRI) and transcranial magnetic stimulation (TMS). We confined to the pure motor recovery mechanism excluding apraxia-related or intervention-induced motor recovery. In addition, we excluded the studies on cortical peri-infarct reorganization after primary motor cortex infarct because this topic was already reviewed in detail by a previous review paper [28]. Eight studies on the mechanisms of motor recovery in MCA infarct were selected for this review [20–27]. These studies were classified according to the following four motor recovery mechanisms: the contribution of premotor cortex (PMC), subcortical peri-infarct reorganization, ipsilateral motor pathway and contribution of aberrant motor pathway.

Contribution of the Premotor Cortex

The PMC is an important area in terms of motor recovery for the following reasons. First, the corticoreticulospinal tract, which innervates the proximal muscles of extremities and axial muscles as one of the extrapyramidal motor pathways, originates mainly from the PMC [7, 9, 12, 13, 29]. Second, the CST originates from a variety of areas in the cerebral cortex including the PMC [4, 16, 30, 31]. It was reported in monkeys that 31% of the descending fibers in the pyramid come from the primary motor cortex, 29% from the PMC, and 40% from the parietal lobe [31]. Finally, one of the important roles of the PMC is planning of motor execution [32–34].

Two studies have reported on the contribution of the PMC for motor recovery in patients with MCA infarct [25, 26]. In 1998, Seitz et al. [26] recruited 7 patients with MCA infarct who showed marked recovery of hand function after severe hemiparesis. They found that the bilateral PMC was activated during affected finger movements without the activation of the primary sensorimotor cortex of either hemisphere by positron emission tomography. The motor-evoked potentials in the hand and leg muscles contralateral to the infarcts were normal. Therefore, they suggested that motor recovery in these patients appeared to rely on activation of the PMC of both cerebral hemispheres. Subsequently, Miyai et al. [25] compared the characteristics of motor recovery according to the presence of PMC lesions in 31 patients with MCA infarct. They found that patients with intact PMC showed better recovery in terms of mobility and motor function of the hip than patients with PMC lesion. As a result, they concluded that the presence of PMC involvement could affect the recovery of locomotor function in patients with MCA infarct.

Subcortical Peri-Infarct Reorganization

Peri-infarct reorganization indicates the transfer of motor function into adjacent areas of an infarct. There have been many studies on peri-infarct reorganization of affected motor function in stroke patients [28, 35–40]. However, the majority of these studies have been focused on peri-infarct reorganization at the cortical level [28, 35, 36, 38, 40]. The recent development of DTI allows analysis of motor pathways at the subcortical level and several studies have reported on peri-infarct reorganization at the subcortical level [20–22, 35, 39]. To the best of our knowledge, three studies have reported on peri-infarct reorganization in the subcortical white matter after MCA infarct [20–22].

In 2009, Jang [22] reported on a patient whose leg motor function appeared to be reorganized into the region of the wall of the lateral ventricle following a MCA infarct, using DTT, fMRI and TMS. The patient presented with complete paralysis of the right extremities at the onset of left MCA infarct, and recovered slowly, to the point of being able to extend the affected knee against resistance by 9 months without recovery of the affected hand. The affected CST descended through the medial corona radiata at or around the wall of the lateral ventricle on DTT. TMS (the motor-evoked potential obtained from the affected leg muscle showed the charac-

Table 1. Previous studies on mechanisms of motor recovery in patients with MCA infarct

Recovery mechanism	Study group	Publication year	Patients n	Evaluation method
Contribution of premotor cortex	Seitz et al. [26]	1998	7	PET, TMS
	Miyai et al. [25]	1999	31	MRI
Subcortical peri-infarct reorganization	Jang SH [22]	2009	1	DTT, fMRI, TMS
	Hong et al. [20]	2010	1	DTT, fMRI, TMS
	Hong and Jang [21]	2011	1	DTT, fMRI
Ipsilateral motor pathway	Kato et al. [23]	2001	1	fMRI, TMS, near infrared spectroscopy
	Yeo and Jang [27]	2012	1	DTT, fMRI
Aberrant motor pathway	Lindenberg et al. [24]	2010	35	DTT

PET = Positron emission tomography; TMS = transcranial magnetic stimulation; MRI = magnetic resonance imaging; DTT = diffusion tensor tractography; fMRI = functional magnetic resonance imaging (the motor tasks of all fMRI studies were grasp-release movements of hand except for Kato's study which employed hand grasping).

teristics of the CST in terms of latency (34.0 ms) and amplitude (2.0 mV)) and fMRI (the contralateral primary sensorimotor cortex was activated during the affected knee movements) for motor function of the affected leg showed results compatible with those for the lateral CST. Findings from this study indicated the importance of the subventricular zone, which is known to have potential for neurogenesis, even in the mature brain [41]. Subsequently, Hong and Jang [20] reported on a patient who recovered via the spared anterior choroidal artery territory following a MCA infarct, using DTT, TMS and fMRI. The patient presented with complete paralysis of the left extremities at the onset of MCA infarct. Motor function of the affected extremities recovered to a nearly normal state 6 months after onset. The 2-week DTT for the CST showed interruption below the infarct. However, the affected CST on 6-month DTT descended through the spared periventricular area (anterior choroidal artery territory) along the known CST pathway. TMS (the motor-evoked potential obtained from the affected hand muscle showed the characteristics of the CST in terms of latency (23.4 ms) and amplitude (1.3 mV)) and fMRI (the contralateral primary sensorimotor cortex was activated during the affected hand movements) revealed that the motor pathway for the affected hand was compatible with the lateral CST. As a result, they concluded that motor function of the affected hand seemed to be recovered by the CST that passed through the spared anterior choroidal artery territory following a MCA infarct. Recently, Hong and Jang [21] demonstrated perilesional reorganization in a patient with a MCA infarct, using DTT ana-

lyzed using fMRI activation results. The patient presented with complete paralysis of the right extremities at onset of the left MCA infarct. His hand function recovery was slow and he gained the ability to extend the affected fingers against gravity 3 years after stroke onset. The CST of the affected hemisphere originated from the primary motor cortex and descended along the anterior portion of the infarcted area at the centrum semiovale and spared the periventricular area at the middle corona radiata. At the centrum semiovale and corona radiata of the affected hemisphere, the affected CST was located anteriorly, compared with the CST of the unaffected hemisphere. Therefore, they suggested that the motor function of the affected hand seemed to have been reorganized into anterior areas of the infarcted centrum semiovale and corona radiata.

Ipsilateral Motor Pathway

The ipsilateral motor pathway is the neural pathway from a motor cortex to the same extremities. The ipsilateral motor pathway is a normal motor control pathway and has been accepted as one of the recovery mechanisms of stroke [42–44]. Among the mechanisms of motor recovery after stroke, the ipsilateral motor pathway from the unaffected motor cortex to the affected extremities has been the most actively researched [43]. It is important because it is related to poor motor outcome and can change with time or manipulated by various rehabilitative interventions [42, 43, 45–49]. However, only

a few studies have been conducted in MCA infarct [23, 27].

In 2001, Kato et al. [23] reported on a patient who showed ipsilateral motor pathway following a MCA infarct. The left hemiparesis of the patient recovered excellently following a right MCA infarct and fMRI was performed at 6 years after onset. During right (unaffected) hand grasping, activation was seen in the left (unaffected) sensorimotor cortex and supplementary motor areas. During the affected (left) hand grasping, activation was seen in the unaffected (left: ipsilateral) sensorimotor cortex, right parietal cortex and bilateral supplementary motor areas. Near infrared spectroscopy and TMS showed similar results. Therefore, the findings of the three techniques indicated that the recovery of the left hemiparesis of this patient had been recovered by motor cortical area reorganization including the ipsilateral motor cortex. Recently, Yeo and Jang [27] reported on a patient with a MCA infarct who showed an ipsilateral motor pathway without the contralateral motor pathway on fMRI and DTT. The patient revealed severe left hemiplegia following an infarct in the right MCA. During a 3-month period after onset, motor function of the affected (left) hand had recovered slowly to the extent that the patient was able to overcome gravity. fMRI showed that only the unaffected primary sensorimotor cortex was activated by movements of the unaffected hand or of the affected hand. On DTT, the CST in the unaffected hemisphere originated from the primary sensorimotor cortex and descended through the known CST pathway. By contrast, the CST in the affected hemisphere revealed an interruption with wallerian degeneration to the upper medulla. They concluded that motor function of the affected hand appeared to have been reorganized to the ipsilateral motor pathway from the unaffected motor cortex to the affected hand.

Contribution of Aberrant Motor Pathway

In 2010, Lindenberg et al. [24] suggested that the alternate motor pathway at the brainstem could contribute to the motor recovery in patients with MCA infarct. They recruited 35 chronic patients with varying degrees of recovery and classified them into three groups using DTT findings for motor tracts. The motor functions of the affected side were better in the following order: patients who had both pyramidal tract and alternate motor fibers in the posterior pons, patients who had alternate motor fibers in the posterior pons without the pyramidal tract

involvement, and patients who did not have any fibers of the pyramidal tract or alternate motor fibers in the posterior pons. These results seemed to suggest that the alternate motor fibers in the posterior pons could contribute to motor recovery in patients with MCA infarct. Although they did not declare the exact pathway of the alternate motor fibers in their study, the alternate motor fibers appeared to be the aberrant pyramidal tract which separates from the original pyramidal tract at the level of the midbrain and the pons and descends through the medial lemniscus [50–53]. It is well known that the pyramidal tract has collateral pathways [54, 55]. The aberrant pyramidal tract is a collateral pathway of the pyramidal tract and existence of the aberrant pyramidal tract has been demonstrated by various methods, including pathological, electrophysiological and radiological [50–53, 56]. In addition, recently, the aberrant pyramidal tract has been suggested as a motor recovery mechanism in patients with an infarct in the pons, midbrain and corona radiata [57–59].

Conclusion

In the current study, 8 studies were classified by the following motor recovery mechanisms: contribution of the premotor cortex (2 studies), subcortical peri-infarct reorganization (3 studies), ipsilateral motor pathway (2 studies) and contribution of the aberrant motor pathway (1 study) [20–27]. The MCA territory is an important area for motor function because it is comprised of the CST, the corticoreticulospinal tract and neural tracts which are related with apraxia [4–16]. In addition, the MCA occupies the largest area among the cerebral artery territories and has many branches. This means that MCA infarct can cause various and serious motor dysfunction, consequently, there is a possibility that various mechanisms of motor recovery can occur after MCA infarct. Therefore, for the best outcome of the patients with MCA infarct, it seems that clinicians should understand the possible motor recovery mechanism which is most beneficial for a patient and facilitate that recovery mechanism through the rehabilitative interventions irrespective of motor outcome of each motor recovery mechanism. In spite of the characteristics of MCA territory for motor function, fewer studies have reported on the motor recovery mechanisms in MCA compared to total number of studies about cerebral infarct. In addition, the majority of these studies have focused on the CST. We believe that the total number of studies on this

topic should be increased. Furthermore, further studies comprising other neural tracts including the extrapyramidal tracts such as the corticoreticulospinal tract and the neural tracts which are related with apraxia, are necessary.

Acknowledgement

This work was supported by the DGIST R&D Program of the Ministry of Education, Science and Technology of Korea (11-IT-01).

References

- Ng YS, Stein J, Ning M, Black-Schaffer RM: Comparison of clinical characteristics and functional outcomes of ischemic stroke in different vascular territories. *Stroke* 2007;38:2309–2314.
- Balaban B, Tok F, Yavuz F, Yasar E, Alaca R: Early rehabilitation outcome in patients with middle cerebral artery stroke. *Neurosci Lett* 2011;498:204–207.
- Mills VM, DiGenio M: Functional differences in patients with left or right cerebrovascular accidents. *Phys Ther* 1983;63:481–488.
- Davidoff RA: The pyramidal tract. *Neurology* 1990;40:332–339.
- Gross RG, Grossman M: Update on apraxia. *Curr Neurol Neurosci Rep* 2008;8:490–496.
- Hong JH, Jang SH: Limb apraxia in a patient with cerebral infarct: diffusion tensor tractography study. *NeuroRehabilitation* 2012;30:255–259.
- Jang SH: The recovery of walking in stroke patients: a review. *Int J Rehabil Res* 2010;33:285–289.
- Jang SH: The role of the corticospinal tract in motor recovery in patients with a stroke: a review. *NeuroRehabilitation* 2009;24:285–290.
- Kably B, Drew T: Corticoreticular pathways in the cat. II. Discharge activity of neurons in area 4 during voluntary gait modifications. *J Neurophysiol* 1998;80:406–424.
- Kwon HG, Jang SH: Delayed recovery of gait function in a patient with intracerebral hemorrhage. *J Rehabil Med* 2012;44:378–380.
- Leiguarda RC, Marsden CD: Limb apraxias: higher-order disorders of sensorimotor integration. *Brain* 2000;123:860–879.
- Matsuyama K, Mori F, Nakajima K, Drew T, Aoki M, Mori S: Locomotor role of the corticoreticular-reticulospinal interneuronal system. *Prog Brain Res* 2004;143:239–249.
- Mendoza JE, Foundas AL: Clinical Neuroanatomy: A Neurobehavioral Approach. New York, Springer, 2007.
- Newton JM, Ward NS, Parker GJ, Deichmann R, Alexander DC, Friston KJ, Frackowiak RS: Non-invasive mapping of corticofugal fibres from multiple motor areas – relevance to stroke recovery. *Brain* 2006;129:1844–1858.
- Schmahmann JD, Smith EE, Eichler FS, Filley CM: Cerebral white matter: neuroanatomy, clinical neurology, and neurobehavioral correlates. *Ann NY Acad Sci* 2008;1142:266–309.
- York DH: Review of descending motor pathways involved with transcranial stimulation. *Neurosurgery* 1987;20:70–73.
- Binkofski F, Seitz RJ, Arnold S, Classen J, Bennecke R, Freund HJ: Thalamic metabolism and corticospinal tract integrity determine motor recovery in stroke. *Ann Neurol* 1996;39:460–470.
- Calautti C, Baron JC: Functional neuroimaging studies of motor recovery after stroke in adults: a review. *Stroke* 2003;34:1553–1566.
- Jang SH: A review of diffusion tensor imaging studies on motor recovery mechanisms in stroke patients. *NeuroRehabilitation* 2011;28:345–352.
- Hong JH, Jang SH: Motor recovery by anterior choroidal artery territory in a patient with middle cerebral artery infarct. *Neural Regen Res* 2010;5:1357–1360 (www.nrronline.org).
- Hong JH, Jang SH: Peri-infarct reorganization at the centrum semiovale and corona radiata: a diffusion tensor tractography and functional MRI study. *Neural Regen Res* 2011;6:826–829 (www.nrronline.org).
- Jang SH: Medial reorganization of motor function in corona radiata following middle cerebral artery infarction: a case report. *Neural Regen Res* 2009;4:668–671 (www.nrronline.org).
- Kato H, Izumiyama M, Shiga Y, Saito N, Koizumi H, Takahashi A, Itoyama Y: Hand motor cortical area reorganization following cerebral infarction evaluated with functional MRI, near infrared spectroscopic imaging, and transcranial magnetic stimulation. *No To Shinkei* 2001;53:869–874.
- Lindenberg R, Renga V, Zhu LL, Betzler F, Alsop D, Schlaug G: Structural integrity of corticospinal motor fibers predicts motor impairment in chronic stroke. *Neurology* 2010;74:280–287.
- Miyai I, Suzuki T, Kang J, Kubota K, Volpe BT: Middle cerebral artery stroke that includes the premotor cortex reduces mobility outcome. *Stroke* 1999;30:1380–1383.
- Seitz RJ, Hoflich P, Binkofski F, Tellmann L, Herzog H, Freund HJ: Role of the premotor cortex in recovery from middle cerebral artery infarction. *Arch Neurol* 1998;55:1081–1088.
- Yeo SS, Jang SH: Ipsilateral motor pathway without contralateral motor pathway in a stroke patient. *NeuroRehabilitation* 2012;30:303–306.
- Jang SH: Perilesional reorganization of motor function in stroke patients. *Neural Regen Res* 2010;5:1668–1672 (www.nrronline.org).
- Kably B, Drew T: Corticoreticular pathways in the cat. I. Projection patterns and collaterization. *J Neurophysiol* 1998;80:389–405.
- Jane JA, Yashon D, DeMyer W, Bucy PC: The contribution of the precentral gyrus to the pyramidal tract of man. *J Neurosurg* 1967;26:244–248.
- Russell JR, Demyer W: The quantitative corticoid origin of pyramidal axons of macaca rhesus. With some remarks on the slow rate of axolysis. *Neurology* 1961;11:96–108.
- Donoghue JP, Sanes JN: Motor areas of the cerebral cortex. *J Clin Neurophysiol* 1994;11:382–396.
- Freund HJ: Premotor area and preparation of movement. *Rev Neurol (Paris)* 1990;146:543–547.
- Passingham RE: Two cortical systems for directing movement. *Ciba Found Symp* 1987;132:151–164.
- Ahn YH, You SH, Randolph M, Kim SH, Ahn SH, Byun WM, Yang DS, Jang SH: Peri-infarct reorganization of motor function in patients with pontine infarct. *NeuroRehabilitation* 2006;21:233–237.
- Cramer SC, Moore CI, Finklestein SP, Rosen BR: A pilot study of somatotopic mapping after cortical infarct. *Stroke* 2000;31:668–671.
- Jaillard A, Martin CD, Garambois K, Lebas JF, Hommel M: Vicarious function within the human primary motor cortex? A longitudinal fMRI stroke study. *Brain* 2005;128:1122–1138.
- Jang SH, Ahn SH, Lee J, Cho YW, Son SM: Cortical reorganization of sensorimotor function in a patient with cortical infarct. *NeuroRehabilitation* 2010;26:163–166.
- Kwon YH, Lee CH, Ahn SH, Lee MY, Yang DS, Byun WM, Park JW, Jang SH: Motor recovery via the peri-infarct area in patients with corona radiata infarct. *NeuroRehabilitation* 2007;22:105–108.
- Rossini PM, Caltagirone C, Castriota-Scanderbeg A, Cicinelli P, Del Gratta C, Demartin M, Pizzella V, Traversa R, Romani GL: Hand motor cortical area reorganization in stroke: a study with fMRI, MEG and TCS maps. *Neuroreport* 1998;9:2141–2146.

- 41 Ohab JJ, Carmichael ST: Poststroke neurogenesis: emerging principles of migration and localization of immature neurons. *Neuroscientist* 2008;14:369–380.
- 42 Chen R, Cohen LG, Hallett M: Role of the ipsilateral motor cortex in voluntary movement. *Can J Neurol Sci* 1997;24:284–291.
- 43 Jang SH: A review of the ipsilateral motor pathway as a recovery mechanism in patients with stroke. *NeuroRehabilitation* 2009;24:315–320.
- 44 Kim SG, Ashe J, Hendrich K, Ellermann JM, Merkle H, Ugurbil K, Georgopoulos AP: Functional magnetic resonance imaging of motor cortex: hemispheric asymmetry and handedness. *Science* 1993;261:615–617.
- 45 Chen R, Gerloff C, Hallett M, Cohen LG: Involvement of the ipsilateral motor cortex in finger movements of different complexities. *Ann Neurol* 1997;41:247–254.
- 46 Ehrsson HH, Fagergren A, Jonsson T, Westling G, Johansson RS, Forssberg H: Cortical activity in precision- versus power-grip tasks: an fMRI study. *J Neurophysiol* 2000;83:528–536.
- 47 Marshall RS, Perera GM, Lazar RM, Krakauer JW, Constantine RC, DeLaPaz RL: Evolution of cortical activation during recovery from corticospinal tract infarction. *Stroke* 2000;31:656–661.
- 48 Takeuchi N, Chuma T, Matsuo Y, Watanabe I, Ikoma K: Repetitive transcranial magnetic stimulation of contralesional primary motor cortex improves hand function after stroke. *Stroke* 2005;36:2681–2686.
- 49 Traversa R, Cicinelli P, Bassi A, Rossini PM, Bernardi G: Mapping of motor cortical reorganization after stroke. A brain stimulation study with focal magnetic pulses. *Stroke* 1997;28:110–117.
- 50 Hong JH, Son SM, Byun WM, Jang HW, Ahn SH, Jang SH: Aberrant pyramidal tract in medial lemniscus of brainstem in the human brain. *Neuroreport* 2009;20:695–697.
- 51 Kwon HG, Son SM, Chang MC, Kim S, Kwon YH, Jang SH: Characteristics of the aberrant pyramidal tract in comparison with the pyramidal tract in the human brain. *BMC Neurol* 2011;12:108.
- 52 Yamamoto T: Aberrant pyramidal tract: a clinicopathological review. *Neurol Med (Tokyo)* 1995;43:306–312.
- 53 Yamashita M, Yamamoto T: Aberrant pyramidal tract in the medial lemniscus of the human brainstem: normal distribution and pathological changes. *Eur Neurol* 2001;45:75–82.
- 54 Nathan PW, Smith MC: Long descending tracts in man. I. Review of present knowledge. *Brain* 1955;78:248–303.
- 55 Nyberg-Hansen R, Rinovik E: Some comments on the pyramidal tract, with special reference to its individual variations in man. *Acta Neurol Scand* 1963;39:1–30.
- 56 Yamamoto T, Yamasaki M, Imai T: Retrograde pyramidal tract degeneration in a patient with cervical haematomyelia. *J Neurol Neurosurg Psychiatry* 1989;52:382–386.
- 57 Hong JH, Jang SH: Aberrant pyramidal tract in a patient with corona radiata infarct: a diffusion tensor tractography study. *Neural Regen Res* 2011;6:1027–1030 (www.nrronline.org).
- 58 Jang SH: Aberrant pyramidal tract in the medial lemniscus of the brainstem in a patient with a pontine infarct: diffusion tensor tractography study. *J Neurol Neurosurg Psychiatry* 2009;80:243–244.
- 59 Yeo SS, Jang SH: Motor recovery via aberrant pyramidal tract in a patient with a cerebral peduncle infarct. *Neural Regen Res* 2011;6:1023–1026 (www.nrronline.org).