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Research paper

Different cerebral plasticity of intrinsic and extrinsic hand muscles after peripheral neurotization in a patient with brachial plexus injury: A TMS and fMRI study

Tie Li[a,1], Xu-Yun Hua[a,1], Mou-Xiong Zheng[a], Wei-Wei Wang[b], Jian-Guang Xu[a], Yu-Dong Gu[a], Wen-Dong Xu[a,*]

[a] Department of Hand Surgery, Huashan Hospital, Fudan University, 12 Wulumuqi Middle Road, Shanghai 200040, China
[b] Department of Radiology, Huashan Hospital, Fudan University, 12 Wulumuqi Middle Road, Shanghai 200040, China

HIGHLIGHTS

- Cerebral plasticity following BPI and neurotization varies in different functions.
- We combined TMS and fMRI to evaluate the brain plasticity after neurotization.
- The reorganization of proximal extrinsic hand muscles is relatively complete.
- The adaptive cerebral plasticity pattern is crucial for well clinical recovery.

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ABSTRACT

Contralateral C7 (CC7) neurotization has been an important approach for brachial plexus injury (BPI). Patients can achieve relatively good grasping function driven by the proximal extrinsic hand muscle (flexor digitorum, FD) after CC7 neurotization, whereas the thumb opposition function driven by the distal intrinsic muscle (abductor pollicis brevis, APB) is poor. The present study aimed to investigate the brain reorganization patterns of the recovery processes of intrinsic and extrinsic hand functions after repairing the median nerve by CC7 neurotization. Transcranial magnetic stimulation (TMS) and functional magnetic resonance imaging (fMRI) were used to evaluate the cerebral plasticity in one BPI patient after CC7 neurotization. After the CC7 neurotization, the patient showed improvements in the paralyzed hand. Combination of TMS and fMRI investigations demonstrated different cortical reshaping patterns of APB and FD. It was also found that the activated cortical areas of FD were located in bilateral motor cortices, but the area of APB was only located in ipsilateral motor cortex. The cerebral plasticity procedure appeared to be different in the gross and fine motor function recovery processes. It provided a new perspective into the cerebral plasticity induced by CC7 neurotization.

1. Introduction

Brachial plexus injury (BPI) is a severe peripheral nerve denervation. Several neurotization procedures have been employed to reconstruct the motor and sensory function of the paralyzed upper extremity in clinic. Contralateral C7 (CC7) neurotization is a special cross peripheral nerve transfer procedure, in which the seventh cervical spinal nerve root (C7) of the intact side is rearranged to innervate the median nerve (or other injured nerves) of the paralyzed upper extremity [1,2]. This surgery procedure is usually used to restore the motor and sensory function of the hand [1].

For BPI patients, it is crucial but difficult to restore the function of hand, especially the fine motor functions controlled by the intrinsic muscles [3,4]. The flexor digitorum (FD) and abductor pollicis brevis (APB) represent gross and fine motor functions of hand, respectively. Motions controlled by the thenar muscles are considered fine motor functions. Correspondingly, fingers flexion driven by FD is considered gross motor function [5,6]. The APB is the most impor-

Abbreviations: TMS, transcranial magnetic stimulation; fMRI, functional magnetic resonance imaging; MT, motor threshold; MEP, motor evoke potential; MI, primary motor cortex; CC7, contralateral cervical 7; FD, flexor digitorum muscle; APB, abductor pollicis brevis muscle; NCS, nerve conduction studies; BPI, brachial plexus injury; MSO, maximal stimulator output; SPM, statistical parametric mapping; FWE, family wise error.

* Corresponding author. Fax: +86 21 52888735.
E-mail address: wendongxu@fudan.edu.cn (W.-D. Xu).

[1] These authors contributed equally to this work.

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tant component of the thenar muscle group, functioning palmar abduction and pronation of the thumb [5]. Although shared with the same peripheral nerve supply, the functional recovery of APB was reported different with FD [7,8]. Muscle atrophy after denervation was supposed to be one of the major reasons of poor function recovery of intrinsic muscles [9]. However, in a long-term follow-up by Wang et al. only unsatisfactory motor recovery of APB was obtained even EMG implied successful reinnervation in 5 of 32 BPI patients [7]. Fingers flexion driven by FD regained relatively better recovery as long as successful reinnervation. This discrepancy suggested that peripheral causes such as muscle atrophy might not be the mere attribution to the function outcomes after CC7 neurotization. The central control could also be an important explanation.

Recent studies have shown that peripheral nerve injury and repair would induce plastic changes at different levels of central nervous system [10,11]. It has also been proved that CC7 neurotization, a complicated rearrangement of peripheral pathway, was able to induce dramatic cerebral plasticity between bilateral hemispheres [12–17]. Specifically, we found that after CC7 neurotization, the paralyzed forearm flexor was first controlled by the ipsilateral motor cortex, and then the contralateral motor cortex gradually regained its control [16,17]. However, the precise cerebral plasticity patterns in APB and FD recovery after CC7 neurotization is still unknown.

In the present study, the combination of TMS-related MEP and fMRI was used to explore the cerebral plasticity patterns of FD and APB in a BPI patient at 7 years after CC7 neurotization. To our knowledge, this is the first report of different patterns of gross and fine motor function recovery after cross nerve transfer in BPI.

2. Materials and methods

2.1. Case report and surgery

A right-handed patient was injured in a traffic accident while riding a motorcycle. The 5 roots of his left brachial plexus nerves, from C5 to T1, were all avulsed from the spinal. Peripheral neurotization surgeries were performed between 1 and 11 months after accident. The patient was examined at 7 years follow-up. Transcranial magnetic stimulation (TMS) and functional magnetic resonance imaging (fMRI) were used to evaluate the cerebral plasticity. The study was approved by the local ethical committee, and the patient provided his written informed consent.

The patient received two-stage procedures of the CC7 neurotization to repair the median nerve, which artificially established the connection between the contralateral C7 nerve root and the paralyzed upper extremity. At the first stage, the ulnar nerve of paralyzed side was cut at the wrist level and freed proximally to the upper arm. Then a cross-chest subcutaneous tunnel was made to bring the ulnar nerve to the divided contralateral C7 for a tension-free nerve suture. Approximately 10 months after the first-stage surgery when the contralateral C7 nerve has regenerated to the axilla of the paralyzed side as judged by the Tinel's sign and nerve conduction studies, the proximal ulnar nerve of the paralyzed side was divided in the upper arm and transferred to the median nerve [18]. In the subsequent follow-up, reinnervation of FD and APB was confirmed.

2.2. Functional recovery and nerve conduction study (NCS)

After a postoperative follow-up period of 7 years, this patient showed an improvement in clinical status, with better fingers flexion recovery than thumb opposition. At the final visit, the active range of motion (ROM) of the wrist reached 45° of active flexion, as the metacarpophalangeal joint was 25–60° of active flexion. The strength of the affected APB was grade M2 (muscle grade refers to the medical research council scale), while the strength of the finger flexors reached to grade M3. However, no nascent motor units were observed in other intrinsic muscles, such as the opponens pollicis muscle. The results of the postoperative NCS for the median nerve and FD and APB muscles were shown in Table 1.

Table 1
Results of postoperative NCS (nerve conduction study) tests for median nerve.

Nerve	Stim site	Record site	NCV (m/s)	Latency (ms)	Amplitude (mV)
MN	Wrist	APB	–	5.81	1.37
	Elbow	APB	22.0	14.9	0.59
MN	Elbow	FD	–	8.42	0.19

2.3. TMS–MEP tests

TMS was performed using a stimulator with a monophasic current waveform (*Magventure MagPro R30*, *Dantec*, *Denmark*) connected to a figure-of-eight-shape coil. The coil was held with a handle pointing backwards and laterally approximately 45° to the inter-hemispheric line to produce an anteriorly directed current in the brain. In the tests, the coil was optimally positioned to evoke motor evoked potentials (MEPs) in the right APB muscle. Intensities were expressed as a percentage of the maximal stimulator output (MSO). The resting motor threshold (RMT) was defined as a minimal stimulator output intensity that evoked a MEP of $\geq$50 μV in five out of ten consecutive trials [19]. The intensity was then adjusted to induce approximate a peak-to-peak amplitude of 1 mV in the resting FD and APB. Lateral hemisphere was tested for 4 attempts at a 10 s interval. Then the scalp was mapped systematically to explore the cortical representations of both FD and APB, using a standard protocol [20,21]. We utilized a 15 × 15 grid of points 1 cm apart for motor mapping in each hemisphere, located to encompass the majority of the motor cortical hand representation. Grid location was determined based on anatomical landmarks such that the top of the grid was placed 2 cm infero-laterally from point Cz [22,23]. Each point was stimulated 6 times at 130% of motor threshold with at least 6 s between stimulations. Recordings were simultaneously drawn from relaxed muscles bilaterally to evaluate the presence of ipsilateral MEPs (iMEPs) and contralateral MEPs (cMEPs). During this test process, the patient was instructed to relax and not to move any of his limbs.

2.4. fMRI study

The fMRI study was performed on a 3.0 Tesla (T) GE magnetic resonance scan system according to the block design paradigm, with alternating rest/control to motor task conditions of the paralyzed hand. The following gradient echo planar imaging sequence parameters were used for acquisition of the fMR images: TR 3000 ms; TE 35 ms; flip angle 90°; FOV 240 × 240 mm^2; and acquisition matrix 64 × 64, resulting in voxel resolution of 3 × 3 × 3 mm^3. The images taken in the first 12 s were discarded to ensure that the signal had achieved a steady state. For the structural images a 3D spoiled gradient-recalled acquisition sequence was used to acquire 1-mm-thick axial sections, and the parameters were as follows: TR 1000 ms; TE 5 ms; flip angle 200°; interslice space 0 mm; FOV 240 × 240 m^2; acquisition matrix 256 × 256.

The patient underwent a resting-state scan first, which will not be discussed in this study. After the resting-state scan, the patient was instructed to perform thumb opposition or fingers flexion of the left (paralyzed) upper extremity at a frequency of approximately 2 Hz for 30 s, alternating with a 30 s rest period. He was instructed to relax completely during the rest period. There were total 6 blocks of fMRI scan, each activation was calculated by the

Table 2
Neurophysiological data after bilateral motor cortex stimulation.

Affected hand	FD			APB		
	MEP latency (ms)	Volume (mV)	Area (ms × mV)	MEP latency (ms)	Volume (mV)	Area (ms × mV)
iMEP	16.4	0.32	2.8	23.9	2.5	17.2
cMEP	24.6	0.72	8.1	NR[†]	NR	NR

APB: abductor pollicis brevi; NCV: nerve conduction velocity; NCS: nerve conduction study; FD: flexor digitorum; [†]NR: not recorded.

comparison of three scans of task and three scans of rest. The patient was trained for 30 min prior to the formal procedures.

Preprocessing steps were carried out using custom routines as well as functionalities available in SPM8 (Wellcome Department of Imaging Neuroscience, University College, London, UK; http://www.fil.ion.ucl.ac.uk/spm). The functional images were realigned and unwarped, and coregistered to the mean functional image from the first session [24]. Individual dataset were excluded if excessive head motion was observed (translation >3 mm or rotation >3°) during this realignment pre-processing step. The 3D-dataset was segmented in native-space, using a unified segmentation approach. The segmented tissue maps were coregistered to the mean functional image from the first session. The normalization step was then performed. The segmentation parameters were used to normalize the functional series to a final resolution of $3 \times 3 \times 3\,mm^3$. Finally, the images were spatially smoothed 8 mm full-width-at-half-maximum (FWHM) Gaussian kernel. For statistical inference, *t*-contrasts MOTOR > REST were calculated for each subject and for each session using the framework of the general linear model. Data were analyzed using statistical parametric mapping (SPM8 software). The statistical significance threshold for activations on fMRI was $p = 0.05$, with FWE (family-wise error) correction.

3. Results

3.1. TMS–MEP data

No MEPs could be recorded from the left (paralyzed) APB while the right motor cortex was stimulated, even at an intensity of 100% of the MSO. In contrast, MEPs could be induced from the left (paralyzed) APB while stimulating the ipsilateral hemisphere, although the latency was prolonged (iMEPs: 23.9 ms). In addition, the MEPs could be produced from the left FD either the contralateral or the ipsilateral cortex was stimulated, although presenting different latencies (cMEPs: 24.6 ms; iMEPs: 16.4 ms) (Table 2). Thus, the APB and FD showed different representations in the contralateral and ipsilateral motor cortices (Fig. 1).

3.2. fMRI data

A complete cooperation of the patient was obtained in the block-design fMRI study. Activation was shown in bilateral M1 areas when the patient performed finger flexion tasks on the paralyzed side. However, only the ipsilateral M1 activation area was activated when the patient performed thumb opposition. Furthermore, activation of the corpus callosum was also observed when the patient performed FD flexion on the paralyzed side. And the supplementary motor area was activated in fMRI images when the patient performed either the fingers flexion or thumb opposition (Fig. 2).

4. Discussion

In the present study, the BPI patient showed motor function improvements in the paralyzed upper extremity after CC7 neurotization. The TMS–MEP and fMRI studies demonstrated different cortical reshaping patterns between APB and FD recoveries: the cortical representations of paralyzed FD were located in bilateral motor cortices; however, the representations of APB were only located in the ipsilateral motor cortex. Therefore, the cortical reorganization procedure appeared to be different in the recovery process of gross and fine motor functions following cross nerve transfer.

The CC7 neurotization is a procedure of cross rearranging the peripheral nerve pathway, which was first reported by Gu et al. in 1992 [1], has been performed in many clinics with rewording results [2,25,17]. This complicated peripheral change was reported to induce significantly adaptive cerebral plasticity in bilateral cortices. In particular, dynamic interhemispheric reorganization was observed both in the experimental and clinical studies [12–14].

In the animal experiments [26,12,15], the motor control of the paralyzed forepaw was located in the ipsilateral hemisphere in the relatively short-term after CC7 neurotization. Finally in the long-term follow-up, the motor center of the paralyzed forepaw gradually moved back to the contralateral hemisphere. Similar findings were also noted in the clinical studies [13,17]. The motor representation of the paralyzed upper extremity first appeared in the ipsilateral motor cortex after CC7 neurotization. Later on, the contralateral motor cortex gradually regained partial or total control over the paralyzed upper extremity, concurrent with further motor recovery [16,17]. In this sense, the brain always tries to restore the control of a paralyzed limb to its contralateral original cortex area. In a pioneering clinical study by Chantal et al. the combination of standardized scales and fMRI determined that gross motor activity in the proximal joints was more readily transferable than more complex activities such as fine hand movements. And the timing of cortical representation shifts of proximal joints was earlier than that of distal joints [27].

The CC7 neurotization anatomically connected the median nerve of paralyzed hand with ipsilateral hemisphere. Although, both innervated by the median nerve, the FD and APB take up different cortical representations. And they also showed varied plasticity processes following CC7 neurotization. According to the combination of TMS and fMRI results, the motor representation of FD partially returned to the original cortex area in the contralateral hemisphere, which was believed to be a better cortical control pattern and had a relative good recovery. The motor representation of APB was still located in the ipsilateral hemisphere and similar to the motor control pattern of FD in the relatively short-term follow-up.

The results of TMS–MEP mapping further identified the possible neural pathway between the motor representations and the target muscles. The latency of contralateral hemisphere induced MEP (cMEP, 24.6 ms) in the injured FD was much longer than the iMEP (16.4 ms), which was supposedly attributed to the transcallosal conduction [17]. The ipsilateral M1 served as a “transit point”, and magnetic stimulation over the contralateral hemisphere activated the “transit point” of ipsilateral M1 via corpus callosum conduction and then evoked the cMEP in the injured FD by the subsequent descending neural pathway. Furthermore, although thumb opposition is a more complex movement, the TMS–MEP mapping revealed a smaller representation area than that of the FD. This suggested that the brain reorganization process of complex movement was primary and incomplete.

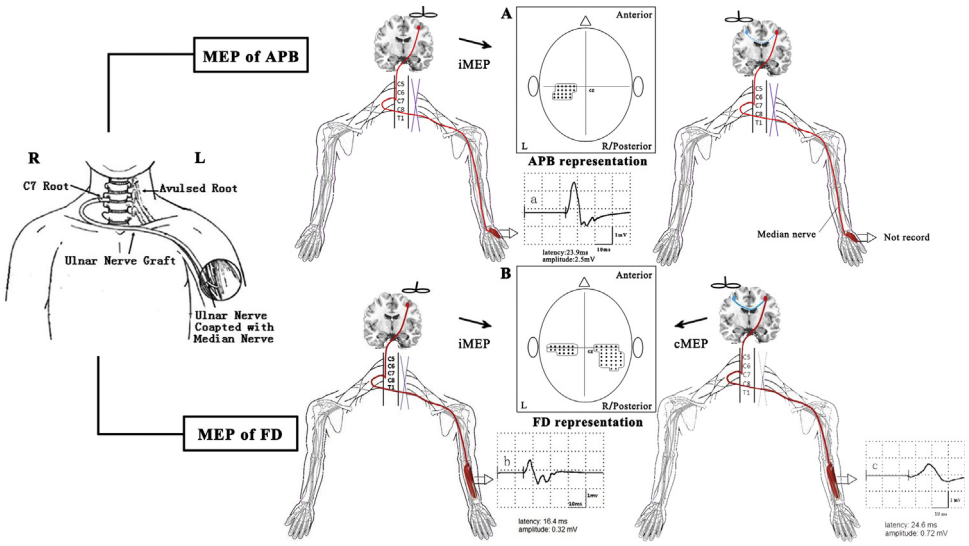

Fig. 1. The pathway and representation of the affected (left) abductor pollicis brevis (APB) muscle and flexor digitorum (FD) muscle evoking while stimulating the motor cortex of bilateral hemisphere. The APB (Fig. 1A) and FD (Fig. 1B) showed different representations in the contralateral and ipsilateral motor cortices. MEP could be induced from the left (affected) APB while stimulating the ipsilateral hemisphere, although the latency was relatively prolonged (a. iMEPs: 23.9 ms), while MEPs could be recorded from the left FD but exhibited a different latency (b. iMEPs: 16.4 ms; c. cMEPs: 24.6 ms).
L = left (affected side), R = right (unaffected side), FD = flexor digitorum muscle, APB = abductor pollicis brevis muscle.

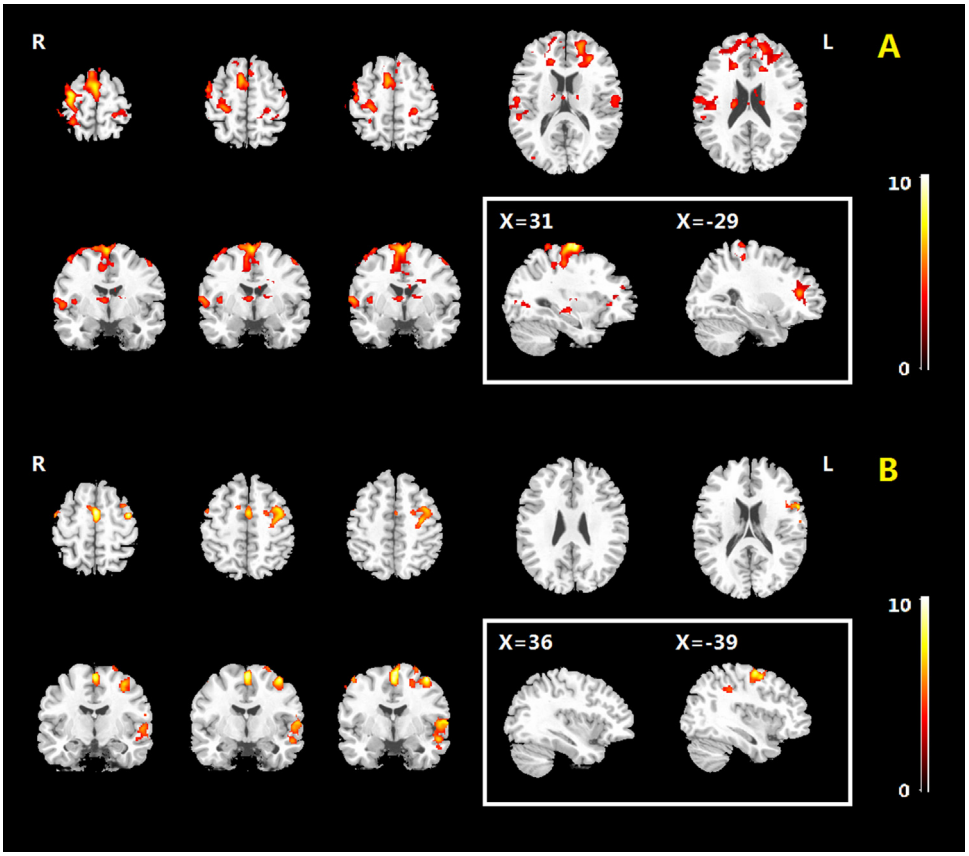

Fig. 2. The activation regions were present in the patients' bilateral hemispheres in the hand area. These results (Fig. 2A) showed that the bilateral motor cortices were activated when the patient performed the fingers flexion task; however, M1, supplementary motor areas (SMA), and the motor image (Fig. 2B) of the thumb opposition task in the premotor areas (PMA) resulted in unilateral activation, and this activation did not exhibit lateralization in the contralateral (right) motor cortex.

Additionally, we observed not only bilateral motor cortices activation during finger flexion of the paralyzed upper extremity in block-design fMRI, but also other activated areas, such as the corpus callosum. This might also suggest a potential involvement of corpus callosum in the gross motor task.

The present case first explained the central mechanisms in different outcomes between gross and fine motor functions of hand after CC7 neurotization, that adaptive cerebral plasticity pattern is crucial for well clinical recovery. Although, this was only a single case of motor cortex mapping, it provides a new insight into the

usefulness of the combination of TMS and fMRI is a feasible method to investigating central involvements in the functional recovery of peripheral neurotization. fRMI could be used for investigation of the functional correlation between the cortical representation and hand function in high spatial resolution while TMS is to explore the whole central-peripheral pathway.

5. Conclusion

The cortical remodeling procedure in restoring the gross and fine motor functions of the hand appeared to be different following contralateral C7 transfer to the median nerve. The adaptive cerebral plasticity pattern may be predictable of the neurological outcome in BPI patients. The absence of inter-hemispheric plasticity appeared to be related with the poorer recovery of fine motor function such as thumb opposition than that of gross motor function such as fingers flexion.

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