

with changes in the reported phantom limb condition and EMG activity, suggesting that the wrist motion of the intact limb adequately reflected that of the phantom limb.

Phantom limb movement

In the first test session, A.S. performed wrist motions with his phantom limb very slowly (0.25 ± 0.02 Hz), and he said he could not perform wrist flexion because of the existence of the metal bar.

Interestingly, after several months of mirror

Figure 2. (A) Waveforms of the wrist joint motion and EMG activity of the FCR and EDL muscles during rhythmic synchronous wrist

flexion-extension movement. (B) The difference in wrist joint motion (frequency and range of motion) and EMG activity of the FCR

and EDL muscles among before (white) and after mirror therapy (dot) and follow-up (gray). The error bars indicate the standard deviation

of the mean value. *Significant difference ($p < .05$).

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therapy, A.S. said that the metal bar gradually disappeared

and that by the end of therapy he finally

did not feel the existence of the metal bar, even

without the mirror. Following therapy, A.S. was

able to move the wrist of the phantom limb faster

and smoother than before (0.56 ± 0.19 Hz), and the

range of wrist joint motion was significantly

increased (before vs. after: 21.2 ± 0.23 vs. $32.4 \pm$

0.26 degrees, $p < .05$). In the follow-up experiment,

A.S. no longer felt the metal bar, and the improved

wrist motion was well preserved. Awareness of the

phantom limb motion recorded by VAS was increased from 4 at the beginning to 10 at the end of therapy, and it remained at the same level in the follow-up experiment. A.S. reported that the phantom limb awareness remained after the metal bar disappeared.

EMG activity

As clearly shown in Figure 2, the wrist muscles showed profound changes during the course of therapy: namely, after mirror therapy, flexor FCR and extensor EDL muscles showed clear alternate modulation that was not observed at the beginning of therapy. For the FCR muscle, the ANOVA results [$F(1,9) = 17.43, p < .05$] revealed a significant effect of time course of therapy (before, after, and follow-up), and a *post-hoc* test identified significant differences between before and after (2.45 ± 0.83 vs. $28.74 \pm 6.45, p < .05$) and before and follow-up (2.45 ± 0.83 vs. $35.03 \pm 16.43, p < .05$). For the EDL muscle, the ANOVA results [$F(1,9) = 13.12, p < .05$] revealed a significant effect of time course of therapy (before, after, and follow-up), and a *post-hoc* test identified significant differences between before and after (20.37 ± 4.89 vs. $58.11 \pm 12.99, p < .05$), before and follow-up (20.37 ± 4.89 vs. $27.24 \pm 5.01, p < .05$), and after and follow-up. Contrary to the phantom limb side, the magnitude of the EMG activity in the FCR muscle on the right side was significantly decreased after therapy as compared to before therapy, and again recovery.

to the baseline level in the follow-up experiment ($[F(1,9) = 9.93, p < .05]$). For the EDL muscle, no significant effect of therapy was identified by ANOVA [$F(1,9) = 2.12, ns$].

Phantom limb pain

In the first test session, A.S. had much pain feeling, which he described as being like an electric shock. However, such unpleasant sensations were profoundly decreased at the end of therapy. Although A.S. did not participate in any mirror therapy after the second experiment, the pain had not returned at the follow-up session and he no longer felt the existence of the metal bar.

DISCUSSION

In the first testing session, the patient, A.S., could not move his phantom limb. According to his comments, the wrist flexion motion of his phantom limb was prevented by a metal bar grasped by the hand. More interestingly, A.S. reported that the metal bar previously grasped by his hand was released from the phantom limb and that the phantom limb pain had disappeared during the course of mirror therapy. As a result, A.S. had a vivid awareness of the phantom limb motion. This observation was supported by changes in EMG modulation in the residual wrist muscles. In the following section, the possible mechanisms underlying the present results will be discussed.

Why was the metal bar grasped by the phantom limb?

Perhaps the metal bar was invisible, but as shown in the picture and the project drawing in Figure 1, A.S. was able to clearly explain his phantom limb condition. A.S. suffered an injury in which his hand was crushed by a machine at work, and he had experienced motor paralysis and had strong feeling of pain before amputation. Such experiences may have influenced his perception of the existence of the metal bar because the pre-amputation condition is known to be an important factor determining the extent of phantom limb sensation and pain (Nikolajsen, Ilkjær, Kroner, Christensen, & Jensen, 1997). At the first mirror therapy, A.S. was extremely surprised to see his hand in the mirror. He attempted to move the wrist of his phantom limb while watching the mirror, but after several minutes of trying, he felt very bad and finally vomited. This may be attributed to the conflict between actual perception of the phantom limb and visual information. A.S. reported that the metal bar was still perceived even when he saw the mirror. He also said that visual information provided through the mirror did not match his actual phantom limb condition. Such a discrepancy could have caused A.S. to become confused. With regard to this

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point, a previous study has revealed that a mismatch between intention, proprioception, and visual feedback increases activity in the right lateral

prefrontal cortex that could represent central monitoring of the induced cognitive conflict (Fink et al., 1999).

It is not easy to determine the mechanism underlying the patient's perception that the phantom limb was grasping a metal bar, but the present showing changes in the patient's phantom limb condition induced by the mirror therapy provide us with useful information.

What happened with the phantom limb?

A.S. did not show EMG activity in the wrist flexor muscle in the first session. At that time, A.S. said that he could not move the wrist of his phantom hand well because the perceived metal bar restricted wrist motion. However, as shown in Figure 2, the wrist flexor and extensor muscles showed clear alternate EMG activity, which was not seen in the first session. Awareness of the phantom limb and the metal bar deduced by A.S.'s report was altered during the course of mirror therapy. He told us that the first time he felt a release of the metal bar was 1 month after initiation of the therapy. Then, he gradually became able to move the wrist and finally could relatively easily move his phantom limb even when he did not utilize the mirror in the second session. This result implies that A.S. was able to reactivate motor commands to the phantom limb. With regard to this point, Mercier et al. (2006) have reported that phantom movement can be re-awakened

through transcranial magnetic stimulation, even when the patient has a 'paralyzed' phantom limb. This finding is also supported by previous results demonstrating that the activated regions of the motor cortex when upper-arm amputees try to move their phantom hand were similar to those during actual movements of the intact hand (Lotze, Flor, Grodd, Larbig, & Birbaumer, 2001). Taken together with these points, we deduce that A.S. might have had an actual motor command for wrist movement during the first session, but that the perceived metal bar prevented the neural circuit from functioning properly. It is likely that the visual feedback induced by the mirror worked as a trigger to re-activate the motor command to the phantom limb, and that A.S. was then gradually able to match motor commands to the phantom limb and visual feedback. Finally, A.S. gained an awareness that the metal bar no longer existed in his hand.

It should be noted that the phantom limb pain that accompanied the presence of the metal bar disappeared during the course of therapy. Based on a previous study, Lotze et al. reported that enhanced use of myoelectric prosthesis in upperextremity amputees is associated with reduced phantom limb pain, which is related to their reduced cortical reorganization (Lotze et al., 1999). They suggested that muscle training of the stump and visual feedback from the prosthesis

might have a beneficial effect on phantom limb pain. This explanation may be applicable to our results.

Implications for rehabilitation

The present results have important implications regarding the use of prosthetic limbs. Rehabilitation for re-activating the phantom limb might be a necessary process for the enhancement of motor cortex activity. If amputees are able to generate proper descending motor commands related to different phantom movements, the induced EMG patterns could be used in an artificial system. Such a strategy could enhance the effectiveness of prosthetic limbs, and thus the ability of amputees to interact with their environment.

CONCLUSION

While the existence of a phantom limb is reported by almost all amputees, to the best of our knowledge, it is very rare that an artificial object is perceived to be in the hand of the phantom limb.

Interestingly, the patient reported that the metal bar previously grasped by his hand was released from the phantom during the course of therapy.

The main finding of the present study was that the wrist flexor and extensor muscles around the stump showed clear alternate EMG activity which was not seen prior to therapy. Considering that A.S. was able to reactivate the motor command to the phantom limb, it is likely that A.S. might have had an actual motor command for wrist movement

during the first experiment, but that the metal bar prevented the neural circuit from functioning properly. The present results suggest that even if a phantom limb is clenched and/or paralyzed, the motor command to the phantom limb can be

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re-activated by an appropriate therapeutic strategy such as mirror-induced visual feedback.

Original manuscript received 30 October 2008

Revised manuscript accepted 26 March 2009

First published online

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