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The Cortical Control of Cycling Exercise in Stroke Patients: an fNIRS Study*

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Abstract: Stroke survivors suffering from deficits in motor control typically have limited functional abilities, which could result in poor quality of life. Cycling exercise is a common training paradigm for restoring locomotion rhythm in patients. The provision of speed feedback has been used to facilitate the learning of controlled cycling performance and the neuromuscular control of the affected leg. However, the central mechanism for motor relearning of active and passive pedaling motions in stroke patients has not been investigated as extensively. The aim of this study was to measure the cortical activation patterns during active cycling with and without speed feedback and during power-assisted (passive) cycling in stroke patients. A frequency-domain near-infrared spectroscopy (FD-NIRS) system was used to detect the hemodynamic changes resulting from neuronal activity during the pedaling exercise from the bilateral sensorimotor cortices (SMCs), supplementary motor areas (SMAs), and premotor cortices (PMCs). The variation in cycling speed and the level of symmetry of muscle activation of bilateral rectus femoris were used to evaluate cycling performance. The results showed that passive cycling had a similar cortical activation pattern to that observed during active cycling without feedback but with a smaller intensity of the SMC of the unaffected hemisphere. Enhanced PMC activation of the unaffected side with improved cycling performance was observed during active cycling with feedback, with respect to that observed without feedback. This suggests that the speed feedback enhanced the PMC activation and improved cycling performance in stroke patients. *Hum Brain Mapp* 34:2381–2390, 2013.

Key words: stroke; near-infrared spectroscopy; cycling; brain activation; rehabilitation

INTRODUCTION

Stroke is one of the leading causes of disability in the United States and other developed countries [Jemal et al., 2005; Rothwell et al., 2004]. For stroke patients, ambulation is often limited by muscle weakness, abnormal muscle synergies, and spasticity that could inhibit the selective activation of muscles during walking. One of the rehabilitation programs to improve the function of ambulation in stroke patients is leg cycling exercise [Hwang et al., 2003]. The muscle activation pattern during leg cycling is similar to that during walking, and has been proposed to be generated by the same central neural network [Christensen et al., 2000; Raasch and Zajac, 1999]. The rhythmic and reciprocal nature of cycling motion also allows patients to

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generate timely symmetrical and reciprocal forces from both limbs, a type of control also required for locomotion. In addition to improving the neuromuscular control for ambulation, cycling training can also help to maintain the functional range of motion of hip and knee joints required for walking [Brown and Kautz, 1998]. Furthermore, cycling exercise has been found to improve several aspects of cycling performance, strength gain, cardiopulmonary function, and the use of the affected leg in stroke patients and reduce muscle spasticity [Brown and Kautz, 1998; Potempa et al., 1995; Sibley et al., 2008].

The known effects of cycling exercise have been mainly attributed to active contraction of the leg muscles. However, passive cycling motion, even when it is conducted without active muscle contraction, can elicit sensory inputs that can be beneficial for stroke recovery [Dechaumont-Palacin et al., 2008a; Lindberg et al., 2004]. Passive exercise is a potential alternative program for patients who are too weak or medically unstable to repeatedly practice active movements to regain motor function. Cycling motion itself can activate somatosensory receptors and generate ascending inputs to the central nervous system. Studies in healthy subjects and stroke patients have demonstrated that proprioceptive inflow from upper limb movements can lead to increased activation not only in the sensory cortex but also in the sensorimotor cortex (SMC), supplementary motor area (SMA), premotor cortex (PMC), and secondary sensory cortex, with resultant improvement in motor performance [Carel et al., 2000; Dechaumont-Palacin et al., 2008b; Lindberg et al., 2004]. Although the neural networks in the spinal cord, referred to as the central pattern generator, are reported to govern locomotion, supraspinal neural structures also affect the control of human locomotion [Miyai et al., 2001; Suzuki et al., 2004]. Specifically for stroke patients, activation of the supraspinal neural structures, such as the SMC and PMC, has also been noted during walking [Miyai et al., 2002, 2003]. It is therefore possible that passive cycling motion could elicit sensory inputs to activate cortical structures and induce cortical changes leading to improved lower limb motor performance.

In the clinical setting, extrinsic feedback can increase the effectiveness of skill training and enhance motivation in subjects [Wulf et al., 2002]. The provision of goal-related extrinsic feedback for movement correction has been shown to be effective in improving motor performance and the retention of learned skills in both healthy subjects and patients [Subramanian et al., 2010; Wulf et al., 2002], and therefore is frequently used clinically to facilitate motor learning. Previous studies have shown that extrinsic feedback is useful for improving motor learning in upper limb functions [Durham et al., 2009], postural control [Van Peppen et al., 2006], balance [Wulf et al., 2009], and functional performance such as sit-to-stand [Van Vliet and Wulf, 2006] in stroke and Parkinsonism patients. A few studies have examined the effect of extrinsic feedback on the facilitation of cortical plasticity during bimanual coordination tasks in healthy subjects [Debaere et al., 2003] and during upper limb functional tasks in stroke patients [Jang et al., 2005]. However, whether changes in cortical activation patterns accompany improvements in cycling performance is not well investigated in stroke patients.

A neuroimaging technique, near-infrared spectroscopy (NIRS), has been utilized to observe the brain activity that results from changes in concentrations of oxygenated (HbO) and deoxygenated hemoglobin (HbR) during human locomotion [Miyai et al., 2001]. NIRS detects changes in the number of absorbed photons versus those scattered back to the surface of the scalp during event-evoked neural activity, such as finger tapping and walking [Arenth et al., 2007; Strangman et al., 2006]. Notably, NIRS is a highly portable neuroimaging system and is virtually silent and less affected by subtle movements, compared to the enclosed chamber and restriction of body movement in the fMRI setting [Strangman et al., 2002]. In addition, NIRS is more suitable to measuring cortical activity during motor tasks, making it particularly applicable to neurorehabilitation studies of motor performance [Strangman et al., 2006].

The purpose of this study was to investigate cortical control during self-paced active cycling without and with extrinsic speed feedback, and during motor-driven passive cycling in patients with subcortical stroke. We hypothesized that passive and active cycling would activate similar cortical regions and that feedback of cycling speed could lead to additional cortical activations and hence improvement in the cycling performance in stroke patients.

METHODS

Subjects

Seventeen patients with hemiparesis resulting from a first-time subcortical stroke were recruited from the rehabilitation department in the National Cheng Kung University Hospital (NCKUH). The subjects all met the criteria of first-time unilateral stroke, medically stable conditions, no previous histories of other neurological or orthopedic problems known to affect pedaling performance, and able to follow verbal commands. The clinical characteristics of the patients are listed in Table I. The average Fugl-Meyer motor scale [Fugl-Meyer et al., 1975] for the lower extremity (mean $\pm$ SD) was 22.88 $\pm$ 6.79. The study protocol was approved by the Institutional Review Board of the NCKUH. All subjects were informed of the experimental procedures and gave written consent.

Experimental Paradigm of Cycling Tasks

Figure 1a illustrates the experimental setup. To standardize the condition under which the cycling test was conducted, the leg position relative to the crank axis was arranged to allow for the knee joint a maximal range of

TABLE I. Clinical characteristics of stroke patients

	Age (years)	Gender (M/F)	Duration from onset (months)	Hemi side (R/L)	Location of stroke	Stroke subtype (H/I)	FM (L/E)
Pt. 1	51	M	20	L	pons	I	18
Pt. 2	67	F	12	R	corona radiata/nuclucus	I	29
Pt. 3	40	M	5	R	putamine	H	17
Pt. 4	43	M	5	R	putamine	H	14
Pt. 5	80	M	90	L	putamine	I	30
Pt. 6	59	M	1	R	basal ganglion	I	34
Pt. 7	74	M	5	L	putamine	H	12
Pt. 8	66	M	96	L	thalamus	H	20
Pt. 9	62	M	2	R	thalamus	H	31
Pt. 10	42	M	3	R	putamine	I	34
Pt. 11	65	M	1	L	internal capsule	I	17
Pt. 12	37	M	42	R	putamine	H	22
Pt. 13	55	M	2	R	basal ganglion	I	22
Pt. 14	53	M	7	L	putamine	H	18
Pt. 15	58	M	17	R	putamine	H	25
Pt. 16	44	M	9	R	putamine	H	25
Pt. 17	48	M	1.5	R	basal ganglion	I	21
Mean ± SD	55.53 ± 12.06		18.74 ± 29.76				22.88 ± 6.79

F: female; FM: Fugl-Meyer motor scale; H: hemorrhage; I: infarction; L: left; L/E: lower extremity; M: male; R: right.

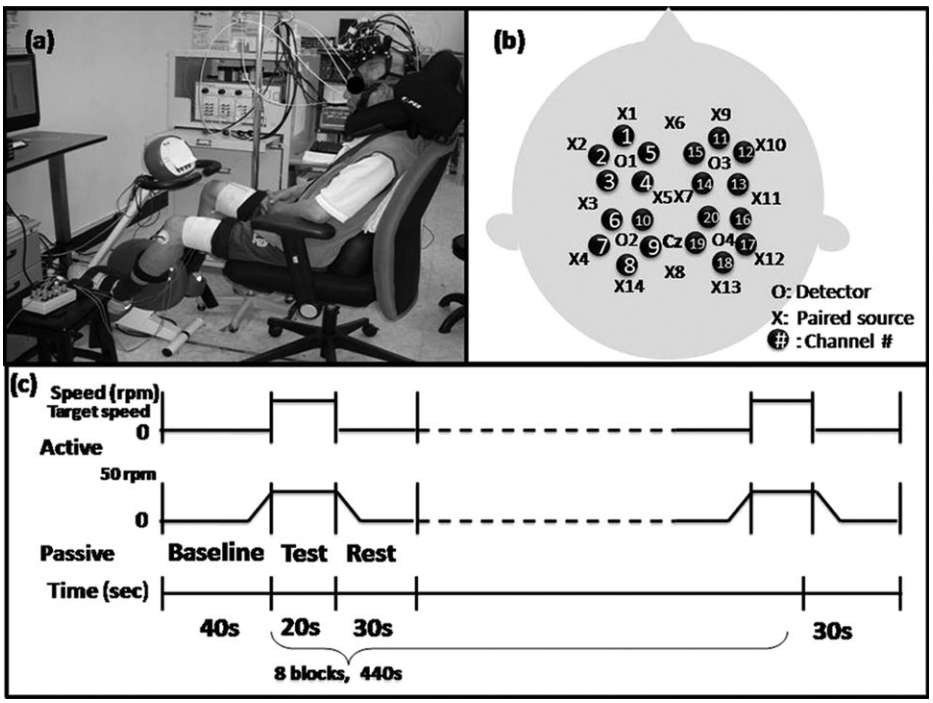

Figure 1.

(**a**) The experimental setup and (**b**) arrangement of the optode locations for the task design in (**c**). The subject was sitting in a comfortable chair with back support. The NIRS signals and EMG activities were measured simultaneously during cycling. A total of 20 channels of measurement was performed in the parietal and parietal-frontal lobes. The optode channels covered the SMC, SMA, and PMC. Patients performed 3 cycling tasks, each starting with a 40 s rest period and a 20 s cycling period followed by 30 s of rest, repeated 8 times. During the passive cycling condition, the ergometer begins automatically 5 s before the cycling period because of the acceleration time needed to reach the target speed of 50 rpm.

motion of 110°–120° of flexion throughout the entire cycling cycle. The zero degree of the crank angle was defined as when the right crank was perpendicular to the ground surface [Fujiwara et al., 2003]. The cycling speed and the crank angle could be transmitted via RS232 to a PC while subjects pedaled on a cycling ergometer (MOTOmed viva 2, RECK-Technik GmbH & Co. KG, Betzenweiler, Germany). The computer monitor connected to the PC in front of the subject showed the visual bars of target speed and the actual speed. There were three cycling conditions: active with no visual feedback (active_nVF), active with visual feedback (active_VF), and passive. Prior to active_nVF, a practice session was conducted to allow the subject to pedal at a steady speed of 50 rpm with the cycling speed continuously displayed on a computer monitor in front of him or her. When the subject was able to follow the pace consistently, the visual feedback was removed and formal data collection of active_nVF begun. In active_VF, visual feedback about the target and self-cycling speeds were provided continuously during data collection. The subjects were asked to adjust their cycling speeds to match the target speed. In the passive cycling task, the pedals were driven by the motor embedded in the ergometer and the subjects were asked not to intervene, only to "allow the pedals to move their legs." The speed of the passive ergometer motion was also set to 50 rpm. For data collection, each cycling task began with a 40-s baseline followed by eight cycles of a 20-s cycling period and a 30-s rest period, resulting in 440-s test sessions, as depicted in Figure 1c. These time frames were required for the measurement of cortical activation from the NIRS signals.

To avoid unnecessary motion artifacts that would interfere with the NIRS signals, both the head and trunk were supported using a height-adjustable head and back rest, with the body well-secured by a soft belt above the belly as needed while testing. Moreover, the exercise-induced skin blood blow around the scalp [Kenney and Johnson, 1992] might lead to the overestimation of hemodynamic signals related to neural activity because the scalp is involved in the high probability area in banana-shape photon flux paths [Kohno et al., 2007]. For this reason, we measured the systolic and diastolic blood pressure (BP) and the heart rate (HR) before and after the pedaling exercise [Harada et al., 2009] to approximately take into account the possible effect of the skin blood flow induced by exercise on the fNIRS signals [Kohno et al., 2007]. The BP and HR were measured by an electronic blood pressure and heart rate monitor (UA-787, A&D, Saitama, Japan) as the patients rested on the ergometer before and after cycling.

fNIRS Recording of Cortical Activations

The optical system used for data collection was a multichannel frequency domain NIRS system (Imagent, ISS, Champaign, IL) with laser diodes with wavelengths of 690 and 830 nm. The NIRS source signals were modulated at 110 MHz with a cross-correlation frequency of 5000 Hz and recorded at 16 time-multiplexed switch mode. The sampling rate was 19.8 Hz. Each pair of sources was composed of two optical fibers, one for each wavelength. Each detector was surrounded by five pairs of sources with an interoptode distance of 3.0 cm. Each optode was defined as the midpoint of the corresponding source-detector pair. Four detectors and 14 pairs of sources accounted for 20 optodes (Fig. 1b).

The system could detect changes in the levels of HbO, HbR, and total hemoglobin (HbT) in the cortex. The detectors and sources were positioned in a custom-made cap and placed on the parietal skull surface; the center of the second row of detectors was placed at the subjects' vertex (Cz). The distance between Cz and the second row of detectors was 2.90 cm. To estimate the location of the NIRS channels on the cortex, the probabilistic estimation method [Collins et al., 1994] was used to register the NIRS signals to Montreal Neurological Institute (MNI) standard brain space. The optode locations with scalp landmarks were marked using a 3D digitizer (Microscribe G2LX; CNC services, Amherst, VA). After calculating the locations of the individual channels on the skull surface, the locations of the NIRS channels on the surface projecting to the cortex were estimated using the balloon-inflation method [Okamoto et al., 2006]. Then the estimated locations on the cortex were transformed to the MNI standard brain template using open source software (NFRI function, available at http://brain.job.affrc.go.jp) imported into MATLAB (MathWork). In the mapping of optodes, channels 6–10 and 16–20 covered the left and right SMC; Channels 4 and 5 in the left hemisphere and Channels 12 and 13 in the right hemisphere for SMA and Channels 1–3 and Channels 11, 14, and 15 for PMC, respectively [Picard and Strick, 2001]. Furthermore, the concentration changes of all channels within one area were averaged to represent the regional activation changes. For the SMC regions, Channels 6 and 7 as well as Channels 16 and 17 in the lateral SMC were excluded from statistical analyses because the lateral part of the SMC covers mainly the arm regions.

Data Analysis

The HbO concentration was used as a marker for the regional activation as research has shown its superior sensitivity to locomotion-related signal changes [Miyai et al., 2001; Suzuki et al., 2004, 2008]. The data were analyzed using the open source software Homer (available at http://www.nmr.mgh.harvard.edu/homer), [Huppert et al., 2009]. First, a 3rd IIR Butterworth band-pass-filter between 0.0016 and 0.80 Hz was used to eliminate slow drifts and cardiac pulsation [Franceschini et al., 2006]. A principle component analysis, removing the first one or two eigenvectors, accounting for 75–80% of the variance in the optical data, was then performed to further remove

large motion artifacts and filter the physiological noises. The data of ΔHbO for each cycling period were then averaged to obtain an average response to the cycling event. The average response was calculated for each cycling task. The optical brain image was then reconstructed using back-projection and tomographic algorithms in Homer [Boas et al., 2004].

To evaluate the interhemispheric asymmetry of regional activation, we calculated the laterality index (LI) of each cortical region [Miyai et al., 2002, 2003]. The values of LI were between -1 and 1, with positive LI indicating greater activation in the affected than in the unaffected hemisphere and vise versa.

LI was defined as

$$\mathrm{LI} = \frac{\Delta\mathrm{HbO\ in\ the\ affected\ hemisphere} - \Delta\mathrm{HbO\ in\ the\ unaffected\ hemisphere}}{\Delta\mathrm{HbO\ in\ the\ affected\ hemisphere} + \Delta\mathrm{HbO\ in\ the\ unaffected\ hemisphere}} \quad (1)$$

Kinematical Evaluations of Cycling Performance

Cycling performance was evaluated using cycling speed, speed variation, and the symmetry index (SI) of muscle activations. The latter is a quantitative measure of the level of asymmetry during cycling [Chen et al., 2005]. The surface electromyography (EMG) of the bilateral rectus femoris (RF) was measured using active surface EMG electrodes (AMT-8; Brotec Biomedical Ltd, Canada), with the electrodes positioned over the distal half of the muscle belly, aligned longitudinally along the muscle fibers. The EMG signals were amplified at a gain of 375 and then sampled at 2 kHz for further processing. The raw EMG signals were first processed with a band-pass filter (Hamming window with cutoff frequency of 40 and 400 Hz), and then rectified and smoothed using a 10-Hz low-pass filter. Because the cycling motion was reciprocal, a phase delay between the EMG linear envelopes (LEs) of the two legs needed to be shifted about 180° in angle axis. The two EMG LEs can be used to determine the shape symmetry index (SSI) by calculating the maximal normalized crosscorrelation coefficient from the following equation:

$$\mathrm{SSI}_j = \frac{|C_{xy}(j)|}{\left[\sum_{n=0}^{N-1} x^2(n) \sum_{n=0}^{N-1} y^2(n)\right]^{1/2}}; j = 0...360 \quad (2)$$

where x and y are EMG LEs recorded from bilateral RF; $C_{xy}(j)$ denotes the circular crosscorrelation function with the lag j between two LEs of one cycle. The maximum of $\mathrm{SSI_j}$ measured between two EMG LEs is defined as the SSI. Because the SSI considers only the consistency of the shape of two LEs, we calculated Area SI (ASI) from the following equation to represent the symmetry of amplitudes of two EMG signals:

$$\mathrm{ASI} = 1 - \left|\frac{R_{\mathrm{area}} + L_{\mathrm{area}} - (R_{\mathrm{area}} \cap L_{\mathrm{area}})}{R_{\mathrm{area}} + L_{\mathrm{area}}}\right| \quad (3)$$

where R_{area} denotes the area of the underlying LE from the right RF and L_{area} represents that of the left RF.

The values of SSI and ASI are between 0 and 1. Higher values represented better symmetry in the pattern of EMG activities during cycling. To quantify the symmetry of cycling, the overall symmetry index (SI) is defined as the product of ASI and SSI.

Statistical Analysis

The paired t test was used to determine the effect of visual feedback on cycling performance (i.e., speed, speed variation, and the symmetry index) by comparing the two active conditions. The differences in heart rate and blood pressure before and after the cycling tasks were also tested using the paired t test to determine whether cycling tasks induced physiological changes. For the analysis of cortical activation, the changes in the HbO between rest and testing were used. To determine if the changes in the activation level during testing was significant, one sample t test was used. To compare the degree of regional activation with each of the three cycling conditions, we performed two-way repeated measures analysis of variance (ANOVA) with conditions (active_nVF, active_VF, and passive) as the within-subject factor and site of cortical regions (SMC, SMA, and PMC at affected and unaffected hemispheres) as the between-subject factor. Another two-way repeated ANOVA was used to identify the effect of different cycling conditions on LIs with conditions (active_nVF, active_VF, and passive) as the within-subject factor and different cortical areas in LI (SMC, SMA, and PMC) as the between-subject factor. The Fisher least significant difference test was used as a post hoc test. Statistical significance was set at $P < 0.05$.

RESULTS

All of the subjects were able to perform the cycling tasks well, without experiencing any discomfort. The physiological measures showed no significant changes before and after cycling tasks (Table II), indicating that the exercise intensity was within the subjects' comfort level. The average speeds in active cycling were 52.77 ± 3.51 and 51.07 ± 1.71 rpm without and with visual feedback, respectively. These were compatible with the target speed of 50 rpm.

TABLE II. Physiological changes before and after cycling tasks

	Before task	After task	*P* values
Heart rate (beats min^{-1})	73.88 ± 13.49	72.47 ± 12.96	0.193
Systolic BP (mm Hg)	141.88 ± 16.83	138.94 ± 17.02	0.319
Diastolic BP (mm Hg)	91.76 ± 9.88	90.35 ± 12.69	0.440

Data present as mean ± SD.

Cycling performance improved in active_VF, indicated by reduced speed variation ($P = 0.001$) and increased SI ($P = 0.008$) with respect to active_nVF (Fig. 2). Figure 3 shows an example of the EMG LEs during active cycling with and without speed feedback. For this representative subject, the SI in active_VF (0.71) was greater than in active_nVF (0.40) due to more synchronized RF activation of the affected side with feedback.

Regional activations during cycling are shown in Figure 4. One sampled *t* test showed that the changes in the cortical activation were significant during testing, compared to during rest. The results of two-way repeated measures ANOVA revealed a significant interaction effect between condition and region ($F(10,192) = 1.976$, $P = 0.042$, $\eta^2 = 0.061$). Post hoc tests to determine the between-condition differences within the same region showed that the SMC activation of the unaffected side was significantly greater than passive cycling condition in active_nVF ($P = 0.009$) and that the PMC of the unaffected side was greater in active_VF than in both active_nVF ($P = 0.011$) and passive ($P = 0.003$) cycling conditions. Figure 5 shows the representative optical images based on the cycling-related changes in HbO concentration in a stroke patient. During active_nVF, the activation was mostly centered in the SMC and the SMC activation appeared to be similar to that during active_VF. However, the activations of the SMA and PMC of the unaffected side were more apparent during

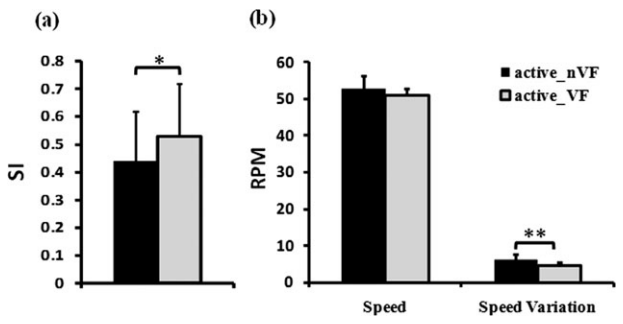

Figure 2.

The averaged (**a**) SI and (**b**) cycling speed with speed variation for active cycling with and without speed feedback in stroke patients. The speed variation was reduced and SI was improved when speed feedback was provided. Error bars represent one standard error. *$P < 0.05$, **$P < 0.001$ for significant difference from paired *t* test.

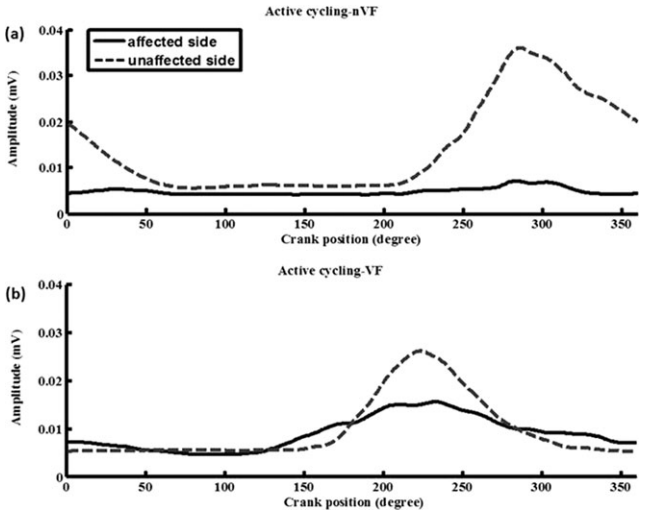

Figure 3.

Representative examples of EMG LEs of bilateral RFs for active cycling with and without speed feedback in one stroke patient. The improved SI represents a better symmetry of EMG activities during cycling with speed feedback.

active cycling with speed feedback than during that without feedback (SMA: 0.47/2.13 μM; PMC: 0.52/2.09 μM for nVF and VF, respectively). During passive cycling, the SMC activation was more apparent than the SMA and PMC (SMC: 2.70; SMA: 0.71; PMC: 1.03 μM) but the SMC of the affected side seemed slightly greater than that of the unaffected side (1.94 and 0.75 μM for the affected and unaffected sides, respectively).

For LI (Fig. 6), we observed a significant main effect for condition ($F(2,96) = 3.697$, $P = 0.028$, $\eta^2 = 0.048$). Post hoc analysis showed that the LIs during active_VF (mean ± SD: -0.264 ± 0.055) were significantly lower than those during active_nVF (-0.088 ± 0.051, $P = 0.025$) and passive (-0.049 ± 0.027, $P = 0.017$) cycling conditions.

DISCUSSION

This study applied fNIRS measurements to assess the cortical activation patterns during different cycling conditions in stroke patients. Our results showed that passive cycling activated cortical regions similar to those activated by active cycling and that a smaller intensity is evident in the SMC of the unaffected hemisphere. With active cycling, speed feedback led to improved cycling performance and greater PMC activation in the unaffected side.

In this study, we found the cortical activation pattern during passive cycling, including the activation of the SMC, SMA, and PMC, was similar to that observed during active cycling without visual feedback. This supports the first hypothesis that active and passive cycling motions will activate similar cortical regions and coincides with previous findings concerning the effect of passive motion

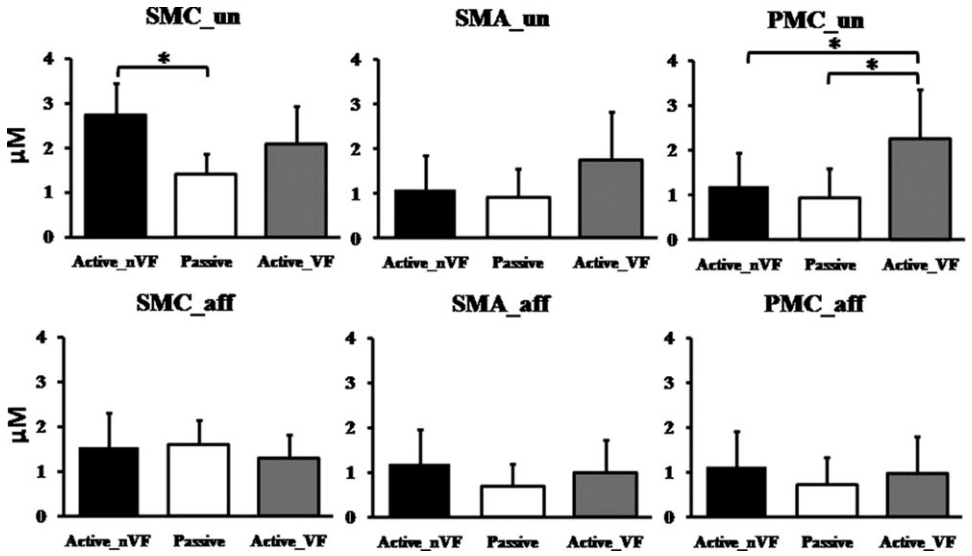

Figure 4.

Regional changes in HbO for SMC, SMA, and PMC of the unaffected and affected sides during different cycling conditions. ANOVA tests showed a significant interaction effect. Post hoc tests indicated that SMC activation of the unaffected hemisphere was greater during active_nVF than during passive cycling. Activation of PMC in the unaffected hemisphere is more prominent with active_VF than without speed feedback. Error bars represent 1 standard error. *$P < 0.05$ for significant difference between cycling conditions. aff, affected side; un, unaffected side.

of the upper limb on SMC activation in stroke patients [Carel et al., 2000; Dechaumont-Palacin et al., 2008b; Lindberg et al., 2004]. Our study first shows that passive cycling can gain access not only to the primary sensory cortex but also to the primary motor cortex, a phenomenon previously reported only in active cycling or other active lower limb joint motions in healthy adults [Christensen et al., 2000; Trinastic et al., 2010]. This finding provides evidence to support the use of passive cycling in stroke rehabilitation. Passive cycling may be used as an adjunct or a primary training program to allow for early or intensive exposure to somatosensory stimulations of the sensorimotor cortex after stroke to facilitate motor recovery. Further research examining the effect of passive cycling training is needed to confirm this.

Despite the similarities in cortical activation between passive and active cycling, activation of the SMC of the unaffected side was smaller in the passive cycling. The SMC is known to be activated in cycling in healthy adults from studies using single photon emission computed

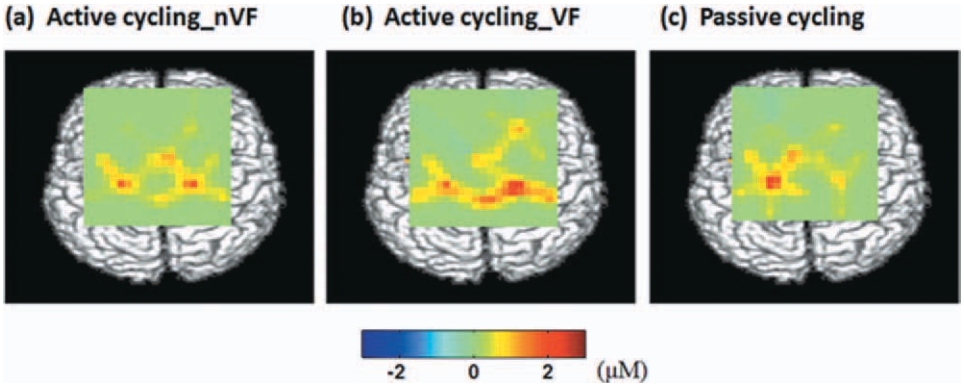

Figure 5.

Representative cortical mappings during cycling movements in a 48-year-old patient with left basal ganglion infarction. These images are based on the changes in HbO levels during (**a**) active cycling without feedback, (**b**) active cycling with visual feedback, and (**c**) passive cycling. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

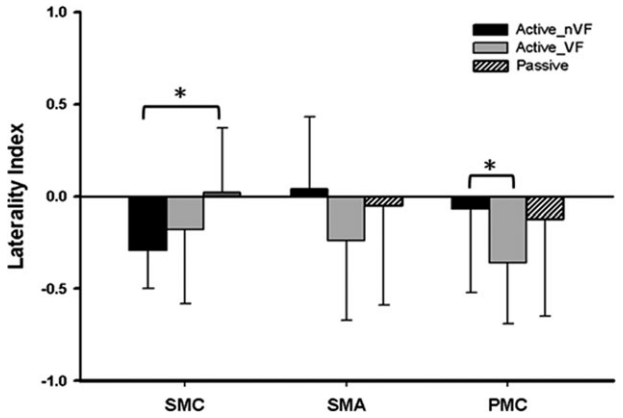

Figure 6.

Laterality index in SMC, SMA, and PMC during active_nVF, active_VF, and passive cycling. The repeated-measures ANOVA showed a significant main effect for condition. The LI in active_VF is generally lower than in active_nVF and passive cycling. Error bars represent one standard error. *$P < 0.05$ for significant difference from post hoc tests.

tomography [Williamson et al., 1997], positron emission tomography [Christensen et al., 2000], and NIRS [Lin et al., 2009]. The SMC is also reportedly involved in the control of locomotion, together with subcortical structures and putative central pattern generators in the spinal cord [Armstrong, 1988; Dietz et al., 2002]. The decreased SMC activation of the unaffected side during passive cycling might reflect the reduced demand of muscle contraction compared to the active condition [Miyai et al., 2006]. The insignificant difference in the SMC of the affected side between active_nVF and the passive condition may be attributed to the relatively low activation of the SMC of the affected side due to the asymmetrical cycling pattern during active_nVF.

Comparing active cycling with and without visual feedback, cycling with visual feedback was found to lead to a reduction in speed variation and to increases in the EMG symmetry index and cortical activation of the PMC. These findings support the second hypothesis that speed feedback will improve cycling performance with additional cortical activations in stroke patients. Furthermore, based on the EMG amplitudes, the increased SI originated from an increased activation of the affected leg, suggesting that the stroke subjects might have paid more attention to the control of the affected leg to match cycling speed when visual feedback was provided, thus leading to a more symmetrical activation pattern. The above findings, together with the improved cycling performance with speed feedback, suggest that the PMC activation might function to enable the patients to utilize external feedback cues for online adjustment of cycling movement.

The PMC has been proposed to be involved in information processing for the planning and execution of motor tasks [Grafton et al., 1998], and to mediate the complex motor skills, rather than the simple muscle force production [Ehrsson et al., 2000]. It has also been proposed that the PMC may be involved in mediating the proximal leg movements, the control of speed in locomotion in stroke patients [Miyai et al., 2002], and in adjusting locomotion speed in healthy adults [Suzuki et al., 2004]. Additionally, the PMC has been reported in rhythmic movements coordinated with external cues [Debaere et al., 2003; Halsband et al., 1993]. Thus, the recruitment of PMC might contribute to better timing control of the affected leg such that cycling speed was maintained more consistently and speed variation was reduced.

Our data showed that the Laterality Indices were generally lower during active_VF, favoring the unaffected side, with respect to to active_nVF and the passive condition. This may reflect the enhanced PMC and SMA activations of the unaffected side during active_VF, which could be a compensatory recruitment of additional motor-related cortices involved in the integration of external information about the cycling movements to counter the brain damage from stroke to achieve better movement control [Mihara et al., 2007]. It is interesting to note that, during active_VF, the increased cortical activations with visual feedback were primarily found in the unaffected hemisphere. These findings suggest that some of the motor recovery of the affected leg might be the result of cortical reorganization in the unaffected hemisphere, instead of in the affected hemisphere [Calautti and Baron, 2003; Schaechter, 2004]. However, to determine how cortical activation patterns are altered by different modalities of cycling training with improved clinical outcomes, a long-term follow-up is needed.

CONCLUSION

Our results showed that the cortical activation pattern during passive cycling was similar to that during active cycling without speed feedback, although the level of activation of the SMC of the unaffected side was smaller. Thus, passive cycling may be beneficial in activating cortical regions and facilitating motor recovery after stroke. When speed feedback was provided, both the cycling performance and the activation of the PMC of the unaffected side improved, suggesting that the PMC might be involved in controlling cycling performance to adapt to the usage of external feedback in stroke patients. Further study is needed to investigate the training effect of passive cycling on motor recovery and brain plasticity in stroke patients.

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