Directional Effect on Post-Stroke Motor Overflow Characteristics

Li-Cheng Tung 3, Jeng-Feng Yang 1, Chun-Hou Wang 4, 5, and Ing-Shiou Hwang 1, 2

1 School of Physical Therapy, National Cheng Kung University, Tainan 70101
2 Institute of Allied Health Sciences, National Cheng Kung University, Tainan 70101
3 Department of Physical Medicine & Rehabilitation, Chi Mei Medical Center, Tainan 71001
4 School of Physical Therapy, College of Medical Technology, Chung Shan Medical University, Taichung 40201
and
5 Department of Physical Therapy, Chung Shan Medical University Rehabilitation Hospital, Taichung 40661, Taiwan, Republic of China

Abstract

Motor overflow (MO) is an involuntary muscle activation associated with strenuous contralateral movement and may become manifested after stroke. The study was undertaken to investigate physiological correlation underlying atypical directional effect of joint movement on post-stroke MO in the affected upper limb. Thirty patients with unilateral post-stroke hemiparesis and fifteen age-matched healthy controls participated in this study. According to motor function assessed with the Fugl-Meyer arm scale, the patients were categorized into two groups of equal number with better (CVA_G; n = 15) or poorer motor functions (CVA_P; n = 15). Surface electromyography (EMG) was used to record irradiated muscle activation from eight muscles of the affected upper limb when the subjects performed maximal isometric contractions in different directions with the unaffected shoulder, elbow and wrist joints. The results showed that only MO amplitude of the CVA_G and the control groups was more sensitive to variations in direction of joint movement in the unaffected arm than the CVA_P group. The CVA_G group exhibited larger amplitudes of MO than the control analog, whereas this tendency was reversed for the CVA_P group. In terms of EMG polar plots, spatial representations of post-stroke MO were insensitive to direction of contralateral movement. The spatial representations of the CVA_G and CVA_P groups were predominated by potent flexion-abduction synergy, contrary to the typical extension-adduction synergy seen in the control analog. In conclusion, post-stroke MO amplitude was subject to contralateral movement direction for healthy controls and stroke patients with better motor recovery. However, alterations in MO spatial pattern due to directional effect were not strictly related to the degree of motor deficits of the stroke victims.

Key Words: motor overflow, stroke, electromyography, movement direction

Introduction

Motor overflow (MO) is vaguely defined as an unintended muscle coactivation accompanied by production of the contralateral movements (1, 37). It is generally believed that intricate transcallosal processes are crucial for bilateral activation during unilateral movement (14). A unilateral movement could bring about a very early transcallosal facilitation and subsequent transcallosal inhibition of the contralateral motor cortex (14, 29). The facilitatory and inhibitory transcallosal effects normally reconcile to an optimal level; however, damages in supraspinal structures alter inter-cortical connectivity of the affected hemisphere and balance between the two transcallosal effects. The facilitation effect often prevails leading to increase in
size of MO (1, 6), and pathological overflow is commonly found in patients with neurological or psychiatric disorders (14, 15, 22, 32). In patients with post-stroke hemiparesis, MO can be seen in either the affected or unaffected limbs (10, 13). Cross-excitation due to MO can spread over several non-homologous muscles without being confined to that utilizes to produce the target movements in the opposite limb (1, 2). Although MO has been considered as reflexively stereotyped and functionally non-purposive (4, 25), yet this viewpoint is being challenged, considering the fact that MO is modifiable to attentional focus (1), task characteristics (11, 16, 27) and exertion level (17, 24). Also, post-stroke MO seemingly evolves with the stroke-recovery process and/or restorative cortical reorganization (18, 21, 26). As MO is a sensorimotor consequence of inter-hemispheric disinhibition (14), it has been used for designing rehabilitation strategies to facilitate feebile muscles in the paralyzed body portions (4, 33, 35). Despite that overflow characteristics of healthy adults seemingly varies with movement pattern of the target joint (17, 19), it is debatable whether cross-excitation by engaging in contralateral movement yields patterned activations that are beneficial to stroke patients (28). At least a part of the doubt can be resolved by confirming [1] whether remodeling of a stereotypical post-stroke overflow is possible by placing a selected contralateral movement, and [2] how spatial representation of MO in stroke victims of various recovery levels deviates from that of healthy individuals.

Surface electromyography (EMG) has been widely used to characterize MO. However, a vast portion of published evidences just focused on contralateral irradiation of the homologous muscle or mirror reversal of the intended movements (9, 10, 24). Physiologically, paretic limb overflow is underestimated for overlooking contralateral irradiation of the non-homologous muscles. The objective of the present study is to systematically investigate how movement direction influences MO characteristics in stroke survivors of different motor functions, with special focuses on amplitude and spatial representation of MO in the whole paretic limb. Our findings may provide a better insight into underlying mechanisms of MO and the potential of overflow in motor rehabilitation.

Materials and Methods

Thirty patients with unilateral post-stroke hemiparesis (18 men, 12 women, 18 left hemiparesis; mean age = 52.8 ± 6.4 years, range = 27~73) and 15 age-matched healthy subjects (9 men, 6 women; mean age = 49.2 ± 7.8 years, range = 24~70) without any history of neuromuscular disease were recruited. Inclusion criteria for volunteers with hemiparesis were [1] diagnosis of a single, unilateral chronic cortical stroke (six months to three years) confirmed by neuroimaging examination (CT/MRI), and [2] sufficient cognitive and language abilities to follow instructions properly. All subjects were right-handed, and eighteen of them were affected in the dominant limb. The study was approved by the local ethic committee, and informed consents were obtained from all subjects in accordance with the institutional guidelines.

None of the participants was aware of the focus of our research. In different anatomical directions, they performed isometric contractions with the shoulder, elbow and wrist joints at maximum exertion levels [100% maximal voluntary contraction (MVC)]. The stroke subjects utilized the unaffected limb to complete the assigned tasks. Nine of the control subjects performed the same tasks using the right upper limb to balance potential laterality effect on MO characteristics. A Biodex dynamometer (Multi-Joint System 3 Pro; Biodex Medical Systems, Inc., Shirley, NY, USA) was used to provide the resistance for the isometric contraction. In total, each subject performed eight randomized isometric tasks (shoulder flexion/extension/abduction/adduction, elbow flexion/extension and wrist flexion/extension) according to standardized procedures documented in the user’s manual of the Biodex system. The exertion level was ensured by matching a line for true dynamometer torque output with a line representing the required level as displayed on an oscilloscope. Each subject was strongly motivated to perform steady isometric contractions of 100% MVC, while irradiated muscle activities were recorded continuously for 3 sec EMGs from eight primary muscles in the unexercised limb [biceps brachii (Bi), brachioradialis (Bra), flexor carpi radialis (Fcr), deltoid (Del), triceps brachii (Tri), pronator teres (Pro), extensor carpi radialis (Ecr), and pectoris major (Pec)] using preamplified bipolar surface electrodes ([Iomed, Inc.; Salt Lake City, UT, USA; electrode spacing 2.5 cm; diameter 1.1 cm, gain of 365 and a common mode rejection ratio (CMRR) of 104 dB]. The subjects performed three repetitions of each isometric paradigm with rest intervals of 2 min between trials. Background EMG was recorded for 3-second in the resting period. The recorded myosignals of each muscle were digitized at 1 KHz using a computer program constructed on the Labview 6.0 platform (National Instruments; Austin, TX, USA). Stroke patients were evaluated with the Fugl-Meyer arm motor score (FMA) (range 0 - 66; normal score = 66) by a licensed physical therapist before the dynamometer experiments. The patients were divided into two groups according to FMA, CVA_G group (better motor function with FMA score 34-66, n = 15); mean score and standard deviation of FMA: 53.0 ± 6.1) and CVA_P group (poorer motor function with FMA score: 0 - 33, n = 15). The demographic data of the two patient subgroups are

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Score</th>
<th>Standard Deviation</th>
</tr>
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<tbody>
<tr>
<td>CVA_G</td>
<td>53.0</td>
<td>6.1</td>
</tr>
<tr>
<td>CVA_P</td>
<td>17.5</td>
<td>8.2</td>
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shown in Table 1.

Raw EMG was conditioned off-line to remove linear trend from the raw EMG and possible artifacts using a digital Butterworth bandpass filter (cut-off frequency = 40-400 Hz). For each resistance paradigm, the root mean square (RMS) from the conditioned EMG signals of three trials was determined and averaged. Net excitation of an irradiated muscle was derived via subtracting the EMG RMS for the background activity from the RMS of the irradiated muscle activity. This net excitation was further normalized with the EMG background activity to deserve standardized net excitation of the individual muscle (SNEi) with the SNE value. Namely, RE of each muscle accounted for its relative contribution to overall MO amplitude in percentage. Methodologically, we expressed MO using SNE rather than a normalization with EMG values of MVC because maximal voluntary contraction of a joint movement for stroke patients involves several pathological synergists rather than the typical primary movers of the target movement. Another important reason for this preference is that muscle strength of the affected limb is differentially impaired among stroke victims. It is not possible to record EMG from a paretic muscle with severe dysfunction.

Table 1. The characteristics of patients with stroke in this study

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>FMA Score</th>
<th>Onset Time</th>
<th>Affected Side</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVA_G</td>
<td>51.9 ± 6.1</td>
<td>Female: 7</td>
<td>53.0 ± 6.1</td>
<td>3 months – 1 year</td>
<td>Right: 9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male: 8</td>
<td></td>
<td>11 months</td>
<td>Left: 9</td>
</tr>
<tr>
<td>CVA_P</td>
<td>53.7 ± 6.1</td>
<td>Female: 5</td>
<td>17.0 ± 7.9</td>
<td>2 months – 2 years</td>
<td>Right: 9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male: 10</td>
<td></td>
<td></td>
<td>Left: 9</td>
</tr>
</tbody>
</table>

Means ± Standard deviation. CVA_G = stroke victims with better motor function, CVA_P = stroke victims with poorer motor function.

Spatial representation of MO was featured by relative excitation (RE), and RE of an individual muscle was deserved by dividing the standardized net excitation of the muscle (SNEi) with the SNE value. Namely, RE of each muscle accounted for its relative contribution to overall MO amplitude in percentage. Methodologically, we expressed MO using SNE rather than a normalization with EMG values of MVC because maximal voluntary contraction of a joint movement for stroke patients involves several pathological synergists rather than the typical primary movers of the target movement. Another important reason for this preference is that muscle strength of the affected limb is differentially impaired among stroke victims. It is not possible to record EMG from a paretic muscle with severe dysfunction.
The non-parametric approach was used for the statistical analysis. The Kruskal-Wallis one-way analysis of variance by rank was used to examine differences in SNE level comparing the CVA_G, CVA_P and control groups for all target movements; and between-group differences in the RE for each recorded muscle for all target movements. The Friedman two-way analysis of variance by rank or Wilcoxon matched-pairs signed-ranks test was used to contrast the differences in SNE levels for various joint movements within a population. The level of significance for all the non-parametric tests and post-hoc testing was set at \( P = 0.05 \). Signal processing and statistical analyses were completed using Matlab v. 6.0 (Mathworks Inc.; Natick, MA, USA) and SPSS for Windows v.10.0 (SPSS Inc.; Chicago, IL, USA).

**Results**

**MO Characteristics for Contralateral Shoulder Movements**

In terms of the SNE and RE levels, Figs. 2A and 2B show amplitude and spatial representation of MO among the CVA_G, CVA_P and the control groups due to shoulder movement in various directions, respectively. The results of the Friedman test indicated a significant effect of movement direction on SNE level for all groups [control: \( \chi^2(3) = 8.84, P = 0.031 \); CVA_G: \( \chi^2(3) = 17.80, P < 0.001 \); CVA_P: \( \chi^2(3) = 12.28, P = 0.006 \)], with the lowest SNE level for shoulder adduction \( (P < 0.05) \) (Fig. 2A). However, SNE level of the CVA_P group was less sensitive to variations in movement direction. For both the CVA_G and the control groups, the SNE levels of shoulder abduction and flexion were significantly greater than that of shoulder extension \( (P < 0.05) \), but such a directional modulation on SNE was absent in the CVA_P group \( (P > 0.05) \). In addition to directional effect, the SNE level of shoulder flexion and abduction differed among the control and the CVA_G and CVA_P groups [flexion: \( \chi^2(2) = 8.50, P = 0.014 \); extension: \( \chi^2(2) = 3.27, P = 0.195 \); abduction: \( \chi^2(2) = 6.37, P = 0.041 \); adduction: \( \chi^2(2) = 4.55, P = 0.103 \)]. The SNE level of shoulder abduction and flexion was the greatest for the CVA_G group but was the smallest for the CVA_P group \( (P < 0.05) \). Next, MO spatial representation was illustrated by schematizing the RE for each recorded muscle in eight distinct azimuths of a polar plot (Fig. 2B). For all shoulder movements of various directions, MO spatial representation for the patient groups was visibly differed from that of the control group. The most noteworthy finding was that RE of the pectoris major (Pec) for the healthy controls was greater than that of the CVA_G and CVA_P groups \( (P < 0.05) \). Furthermore, the healthy group produced a greater RE of the triceps (Tri) than those of the patient groups in the cases of shoulder extension and adduction \( (P < 0.05) \). Conversely, for the two patient groups, shoulder movements were associated with greater RE of either the Bra, Bi or Del muscle \( (P < 0.05) \), depending on the movement
direction. Spatial representations of MO of the CVA_G and CVA_P groups were comparably similar. MO of both groups of the patients was governed by flexion-abduction synergy, contrary to normal extension-adduction synergy.

**MO Characteristics for Contralateral Wrist Movements**

Fig. 3A compares means and standard errors of the SNE level among the two patient groups and the control analog due to elbow flexion and extension. A Wilcoxon test suggested no significant directional effect on the SNE level for all groups (control: \( z = -1.533, P = 0.125 \); CVA_G: \( z = -1.114, P = 0.910 \); CVA_P: \( z = -1.306, P = 0.191 \)). On the other hand, a significant group effect on the SNE level was noted in elbow flexion rather than elbow extension (flexion: \( \chi^2(2) = 12.23, P = 0.002 \); extension: \( \chi^2(2) = 5.735, P = 0.057 \)). Post-hoc analysis indicated that the SNE level for the CVA_P group was relatively smaller than that of the control group, and the CVA_G group exhibited the largest SNE level among the three groups (\( P < 0.05 \)). Fig. 3B contrasts schematic expressions of RE among the CVA_G, CVA_P, and the control groups due to elbow movements. In parallel with shoulder movements, we noted that elbow movements resulted in a similar group difference in MO spatial representation. For the elbow flexion and extension, both movements caused an atypical decline in Pec RE (\( P < 0.05 \)) but caused an enhancement of Bi RE for the CVA_G and CVA_P groups, as compared to those of the control group. Elbow flexion also further produced RE enhancement of the Del and Bra muscles for the patient groups (\( P < 0.05 \)).

**MO Characteristics for Contralateral Elbow Movements**

Fig. 4A summaries means and standard errors of the SNE level due to wrist flexion and extension for the patient and control groups. For the CVA_G and control groups, the SNE level depended on the direction of wrist movement (CVA_G: \( z = -2.897, P = 0.004 \); controls: \( z = -1.930, P = 0.057 \)). But, the SNE level of the CVA_P group was not subject to directional effect (CVA_P: \( z = -1.022, P = 0.307 \)). Besides, we found a significant group difference in the SNE level of both wrist movements [flexion: \( \chi^2(2) = 6.93, P = 0.031 \); extension: \( \chi^2(2) = 7.52, P = 0.023 \)]. The CVA_P group produced a smaller SNE level than the CVA_G and/or control analog (\( P < 0.05 \)). Fig. 4B schematically contrasts spatial representation of MO among the patient and control groups in the cases of wrist movements. The CVA_G and CVA_P groups both had a smaller Pec RE and Ecr RE than the control group (\( P < 0.05 \)). On account of enhanced RE of the Del, Bi or Bra muscle (\( P < 0.05 \)), flexor-abduction synergy accounted for the majority of MO spatial representation of the patient groups.

**Discussion**

One of the principal findings in this study was a significant group difference in relative MO production by manipulation of movement direction of the opposite limb. Stroke survivors with poorer motor recovery exhibited less overflow than those with better functional scores and control analogs, especially when directions of the joint movement were against the line of gravity (flexion/abduction of the shoulder, flexion of the elbow and extension of the wrist) (Figs. 2A, 3A and 4A). When the unaffected limb was active, MO involves with bilateral activations of large-scale
motor networks (8). Transcallosal projection from the lesioned hemisphere produced an initial facilitatory response followed by an inhibitory effect to mediate the ipsilateral corticospinal pathways of the undamaged hemisphere (5). A stronger initial facilitatory response and a weaker subsequent inhibitory effect (31) contribute to enhanced overflow in the paretic limb as a sign of compensatory restoration of distributed networks in the affected hemisphere and better inter-hemispheric communication in early recovery stage. However, a large-scale motor overflow does not necessarily affiliate with full motor recovery because the healthy controls exhibited a smaller amount of MO as compared with stroke survivors with better motor recovery. As a corollary, post-stroke MO is a neurological sequel analogous to spasticity the degree of which matches a bell-shaped pattern of a patient’s functional stages (4, 12). Excessive overflow in the affected limb that hampers bimanual coordination needs to be appropriately suppressed through proper functioning of the transcallosal inhibition for patients with full motor recovery. The evolitional changes in overflow-function relationship may partly explain previous criticisms (22, 36) against correlation between MO and recovery of hand function for selection of stroke patients at different recovery levels. On the other hand, all criticisms regarding insignificant overflow-function relationship were confined to the study of paretic fingers/hand. In fact, hand function recovery relies predominantly on the integrity of the contralateral corticospinal pathway of the affected hemisphere (20, 34) rather than the ipsilateral pathway of the undamaged hemisphere that is crucial for MO genesis in the upper arm and trunk.

Next, the above-mentioned group difference in overflow amplitude was specific to contralateral movements in the directions against the line of gravity (Figs. 2A, 3A and 4A). Moreover, those stroke survivors with better motor recovery and control analogs demonstrated a more marked directional effect on overflow amplitude than the stroke survivors with poorer recovery. A previous study reported that the amount of motor overflow decreased progressively with involvement of more distal joints (18), but this patterned organization of MO was not seen in the stroke survivors with poor recovery. Together with gradient-change in MO with joint involvement (18), this suggests that another physiological watershed in the stroke recovery process. Although physiological mechanisms underlying the directional effect is not completely clear, it probably pertains to usage-dependent decline in transcallosal inhibition commonly found in professional musicians with intensive practices for years (29, 30). As there is a more frequent use of joint movements opposing the pull of gravity in daily activities, the acts of shoulder abduction/flexion, elbow flexion and wrist extension might preferentially well concur with more overflow than joint movements of the other directions. Once a large extent of motor network is damaged, stroke patients are expected to suffer from severe lost of functional activities as well as violation of direction-dependent MO attributable to usage-dependent adaptation of transcallosal projections.

In terms of RE and EMG polar plots, MO spatial representations for healthy controls were visibly different for variations in movement direction (Figs. 2B,
3B and 4B). Contralateral isometric contractions added to the contribution of irradiation of the Pec or Tri muscles to MO so that normal cross-excitation was governed by extension-adduction synergy. However, for both the CVA_G and CVA_P groups, the spatial pattern in healthy controls was replaced by uncharacteristic excitation of the Bi, Bra or Del muscles that skewed the excitation zone toward the right side of the polar plots in favor of flexion-abduction synergy. Flexion-abduction synergy of post-stroke overflow was similar to stereotyped mass activation of flexors during unilateral force-generation tasks using the paretic limb (3). Hence, we could not preclude that post-stroke MO obliged activation of spinal motoneurons influenced by primitive flexion-withdrawal reflex through terminal fiber blanching (23), though the details of the cortical mechanism have yet to be clarified. In spite of a marked difference in functional consequences, the CVA_G and CVA_P groups did not demonstrate a clear demarcation line of the EMG polar plots for all contralateral movements. This fact means that we cannot directly equate functional recovery with MO spatial representation of stroke victims. Besides, the use of resisted movements to therapeutically facilitate a desired activation pattern of the paretic musculatures through post-stroke MO should be reconsidered. In effect, there was no physiological evidence showing preferential cross-excitation of paretic adductors and extensors in the upper limb, irrespective of the direction selected for contralateral movement. The flexors and abductors of the upper limb were better excited by post-stroke MO of all movement directions.

In conclusion, MO amplitude in the paretic upper limb was related to arm motor function in our sample of patients with post-stroke hemiparesis. In spite of enhanced MO in stroke patients with better arm function, like spasticity evolution, this exaggerated overflow should finally be inhibited, considering relatively smaller MO amplitudes for all joint movements found in healthy controls. In addition, MO amplitude of the healthy controls and stroke patients with better arm functions was more susceptible to alterations in movement direction than that of stroke patients with poorer arm functions. For all joint movements, spatial representations of post-stroke MO governed by flexion-abduction synergy were neither sensitive to the direction of target movements nor reverent to degrees of motor deficits. Our findings suggest that overflow characteristics may be valuable to determining functional prognosis of stroke survivors. However, we did not find sufficient physiological evidences of cross-shaping desired movement patterns simply with post-stroke MO.

Acknowledgments

This research was supported by grants from the Chi Mei Medical Center, Taiwan (Grant no. CMNCKU9808) and in part from the National Science Council, R.O.C. (Grant no. NSC 93-2314-B006-002).

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