MOVING THE ARMS TO ACTIVATE THE LEGS

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FERRIS, D.P., H.J. HUANG, and P.-C. KAO. Moving the arms to activate the legs. Exerc. Sport Sci. Rev., Vol. 34, No. 3, pp. 00–00, 2006. Recent studies on neurologically intact individuals and individuals with spinal cord injury indicate that rhythmic upper limb muscle activation has an excitatory effect on lower limb muscle activation during locomotor-like tasks. This finding suggests that gait rehabilitation therapy after neurological injury should incorporate simultaneous upper limb and lower limb rhythmic exercise to take advantage of neural coupling. Key Words: gait, locomotion, rehabilitation, interlimb coordination, electromyography

INTRODUCTION

Humans naturally swing their arms when they walk and run. Although arm swing has often been compared with pendular motion, it is not a purely passive phenomenon. Muscle activity controls arm swing magnitude and timing during human locomotion (5). During running, humans recruit upper limb muscles to swing their arms at a much faster rate than the arms’ natural frequency. During slow walking, humans control arm swing motion via low-level phasic muscle activity. As humans change walking speed, their nervous systems adapt muscle activation patterns to modify arm swinging for the appropriate frequency.

Humans have neural connections between their upper limbs and lower limbs that coordinate muscle activation patterns during locomotor tasks. During swimming, crawling, and walking, the upper limbs and lower limbs move at integral frequency ratios that preserve interlimb coordination (12). Because the arm motion used in swimming is not pendular-like, it requires more active nervous system control compared with walking. However, even with increased neural control and decreased passive dynamics, the basic coordination pattern between upper limbs and lower limbs is preserved. Many other studies have identified interlimb reflex responses during walking and combined arm-leg cycling (14,15). These interlimb reflex studies also point toward a common neural origin for upper limb and lower limb movement during rhythmic activities based on the similarity in reflex modulation patterns.

Mechanical analyses indicate that arm swing during human locomotion helps stabilize rotational body motion. Elftman first proposed that arm swing during walking balances torso torques caused by swinging of the lower limbs (4). This idea has been studied further by others with the same general conclusions (5,9). The primary mechanical effect of arm swing during walking is that it reduces body twisting torque along the vertical axis. This happens because the upper limb moves forward as the contralateral lower limb moves forward. The angular momentum of contralateral upper and lower limbs partially balances each other, reducing the rotational moment between the foot and the ground.

Recent studies indicate that arm swing may also facilitate lower limb muscle activation via neural coupling. Clinical observations of individuals with spinal cord injury first suggested that rhythmic upper limb movement improved lower limb muscle recruitment during stepping. More recently, studies on neurologically intact subjects have demonstrated an increase in lower limb muscle activation that is proportional to upper limb muscle recruitment during seated recumbent stepping (6,7). This observation has clear implications for gait rehabilitation after neurological injury. If active upper limb movement provides facilitation of lower limb motor neurons during rhythmic movements, then neurorehabilitation therapies should engage patients in activities that require simultaneous upper limb and lower limb rhythmic exercise.

The aim of this review is to summarize evidence for a direct proportional link from upper limb motor neuron recruitment to lower limb motor neuron recruitment during human locomotor tasks. Other recent reviews have focused...
on anatomical and reflex evidence indicating the existence of neural pathways linking upper limb and lower limb motor neurons during rhythmic movement (3,15). The present review will not duplicate those works, but instead will focus on evidence showing a facilitation of lower limb muscle recruitment from upper limb muscle activation. In addition, implications for gait rehabilitation will be discussed.

**OBSERVATIONS FROM CLINICAL RESEARCH**

Locomotor training with partial body weight support is an effective gait rehabilitation therapy after neurological injury (1). The therapy consists of using a harness to reduce lower limb loading, enabling a patient to practice stepping with or without manual assistance (Fig. 1). Repetitive exposure to sensory feedback related to walking helps stimulate the neural networks responsible for locomotor muscle activation patterns and promotes neural plasticity. The idea for body weight–supported locomotor training came from animal experiments examining the effect of treadmill training on cats with spinal transections (1). Those studies demonstrated that spinal cats could relearn to step with their hind limbs on a treadmill if given sufficient stepping practice. Traditional gait rehabilitation therapy usually involved patients stepping between two parallel bars as they used their upper limbs to support some of their body weight. The parallel bar method is successful in reducing the load on the lower limbs, but it results in a compensatory gait pattern rather than a natural gait pattern. This occurs because patients have to activate their upper limb muscles tonically to support themselves and are unable to swing their upper limbs naturally. Locomotor training with partial body weight support from a harness allows patients to move their upper limbs freely, leading to a more natural gait pattern.

A comparison of muscle activation patterns during harness-supported stepping and parallel bar stepping in patients with incomplete spinal cord injury suggests that upper limb movement improves lower limb motor patterns. Visintin and Barbeau (11) found that patients with spastic paresis displayed greater and more symmetric muscle activation when stepping with partial body weight support from a harness compared with stepping with support from parallel bars (Fig. 2). They also noted that the amount of spastic muscle activation was less with harness support than with parallel bar support. These results may be attributed to the fact that harness-supported stepping allows patients to swing their upper limbs. Based on this qualitative evidence, partial body weight support with a harness seems to be a much more appropriate method to train neurologically impaired patients to walk again.

More recently, Behrman and Harkema (2) suggested that facilitating arm swing could be beneficial during locomotor training for individuals with spinal cord injury. Describing a series of case studies, these authors discuss the importance of different sensory cues to obtain good stepping during therapy. Although many of the sensory cues presented are similar to those determined from experiments on spinal cats, Behrman and Harkema also make a point of commenting on arm swing. Figure 1 shows an example of how arm swing could be incorporated during locomotor training in patients with neurological injuries. The patient grips a pole that a therapist moves forward and backward with each hand to help guide the patient’s arm swing to be coordinated to the patient’s lower limbs. Although results
from upper limb involvement have varied from patient to patient, they could greatly facilitate lower limb stepping improvements in some patients.

OBSERVATIONS IN NEUROLOGICALLY INTACT HUMANS DURING RECUMBENT STEPPING

Based on the clinical observations described above, we have tested the influence of upper limb exertion on lower limb muscle recruitment in neurologically intact subjects (6,7). Instead of making measurements during walking or running, we concentrated initially on recumbent stepping (Fig. 3). The recumbent stepping machine has two pedals and two handles that are contralaterally coupled so that users can drive the stepping motion with their lower limbs and/or upper limbs. Recumbent stepping has the experimental advantage of the lower limbs moving through a fixed range of motion every step. In addition, the seated position allows less torso movement and postural shifts. This makes it easier to compare when the lower limbs are passively moved through the stepping motion and when the lower limbs are actively pushing against the pedals. For active recumbent stepping, a subject can push or pull on the handles and/or pedals using normal locomotion coordination (i.e., right handle and left pedal move forward, whereas left handle and right pedal move backward). Alternatively, a subject could push and pull both handles to drive the lower limbs through the stepping motion, allowing their lower limbs to simply go along for the ride (self-driven stepping). It is also possible to have a second person move a handle to drive the lower limbs through the stepping motion so that both the upper and lower limbs are driven passively through the stepping motion (externally driven stepping). Externally driven stepping during treadmill walking would be difficult to control because of torso movement and...
postural stability. Recumbent stepping has a simplified motion compared with walking, but it likely activates similar neural substrates as walking. There is a growing body of evidence indicating that the neural networks responsible for controlling walking also provide basic muscle activation patterns for many other rhythmic limb movements (13). As a result, it seemed likely that any neural coupling between upper limb muscle activation and lower limb muscle activation during locomotion would also be present during recumbent stepping.

Our first study tested if rhythmic upper limb exertion during recumbent stepping enhanced lower limb muscle activation in a proportional manner (6). We collected electromyography (EMG), joint kinematics, and pedal forces in neurologically intact subjects during several different recumbent stepping conditions. Conditions included active stepping (using both upper limbs and lower limbs actively), self-driven stepping (using upper limbs actively to drive lower limb stepping motion), and externally driven stepping (upper limbs and lower limbs both driven passively by another person or just lower limbs driven passively by another person). A key hypothesis that we wanted to test was that lower limb excitation would be dependent on the magnitude of upper limb muscle recruitment via neural coupling. As such, we examined subjects performing self-driven stepping at three different levels of upper limb resistance: easy, medium, and hard.

Our results demonstrated a clear facilitation of lower limb muscle activation when subjects actively used their upper limbs to drive the lower limb stepping motion (Fig. 4). Importantly, the timing of the lower limb muscle recruitment was the same as that demonstrated during stepping with active upper limbs and lower limbs (Fig. 5). The similarity in timing was verified with cross-correlation analysis (6). This indicates that the rhythmic lower limb muscle activation observed during self-driven stepping originates from similar neural pathways as the rhythmic lower limb muscle activation during active stepping. Furthermore, as hypothesized, the magnitude of lower limb muscle recruitment increased for conditions when subjects pushed against greater mechanical resistance with their upper limbs (Fig. 6).

Figure 5. Averaged EMG bursts of neurologically intact subjects (n = 20) for Active Arms & Legs (blue), Self-Driven (hard) (green), and Externally Driven (red). The timing of the Self-Driven (hard) passive leg EMG patterns is similar to the timing of the active leg EMG patterns in the Active Arms & Legs condition for the six lower limb muscles shown (VL, vastus lateralis; VM, vastus medialis; MH, medial hamstrings; MG, medial gastrocnemius; SO, soleus; TA, tibialis anterior).
Our results support the existence of an excitatory neural pathway connecting upper limb motor neurons and lower limb motor neurons. This pathway could be in the spinal cord, linking upper limb interneurons to lower limb interneurons or motor neurons (Fig. 7). Alternatively, it is possible that descending signals from the motor cortex that activate upper limb interneurons or motor neurons also branch off to lower limb interneurons or motor neurons (Fig. 7). There is no clear way to differentiate these possibilities with the evidence at hand.

It would be interesting to examine the relative effects of transcranial magnetic stimulation on lower limb muscle recruitment during self-driven recumbent stepping. If the enhanced lower limb muscle recruitment originates from spinal neural networks responsible for locomotor pattern generation, then motor potentials evoked from transcranial stimulation would likely be attenuated during self-driven stepping compared with tonic, voluntary contraction at the same level of background muscle activation. This difference in corticospinal excitability would occur because cortical involvement with lower limb muscle activation would be less during self-driven stepping compared with the tonic, voluntary contraction (15).

An assumption of the aforementioned results is that the subjects were not cheating by consciously activating their lower limb muscles during self-driven stepping. Cheating in this manner could have benefited subjects because it would have reduced the mechanical work required of the upper limbs. To control for this possibility, we mechanically decoupled the handles and pedals for another experiment (6). With the handles and pedals decoupled, there was no longer any possible mechanical benefit to the subjects for activating their lower limb muscles to lessen the work of the upper limbs. Results from this second experiment were identical to the first experiment with mechanically coupled handles and pedals. For the decoupled setup, we only examined handle and pedal coordination phasing that matched the phasing of the mechanically coupled setup (6). It is not clear how the results would have been affected by other phase relationships (e.g., ipsilateral coordination instead of contralateral coordination).

Another possible confounding factor is the need to stabilize the torso. At greater resistances, it is possible that subjects relied on greater tonic excitation of lower limb muscles for postural stabilization. This could have then amplified any rhythmic motor neuron excitation that
occurred as a result of passive motion. This possibility does not seem likely, as we found similar facilitation effects with different degrees of external torso stabilization. When subjects had two belts constraining movement of their hips and torso, the results were similar to when they just had a lap belt.

We conducted pilot studies on other conditions and found that lower limb motion was critical to the neural coupling between upper and lower limbs. Upper limb exercise with passive stationary lower limbs did not demonstrate any rhythmic lower limb muscle activation. It seems likely that the passive motion of the lower limb triggers afferent depolarization, bringing the motor neurons closer to threshold. More sensitive neurophysiological techniques (e.g., single motor unit recording, Hoffmann reflex testing) might be able to detect some subthreshold excitation without lower limb movement. Future studies should examine this possibility in greater detail to determine the importance of sensory pathways related to passive movement.

We followed up the initial study by examining the effect of stepping frequency on neural facilitation between upper and lower limbs (7). Conditions for these experiments were similar to the first study, with the exception that multiple stepping frequencies replaced multiple stepping resistances as a testing variable. Similar to the previous results, greater upper limb exertion (i.e., faster stepping frequencies) led to increased lower limb muscle recruitment (7). Figure 8 shows how muscle activation amplitude in the lower limbs increased with movement speed during self-driven recum-

**Figure 7.** Schematic of possible neural pathways responsible for lower limb muscle excitation from upper limb exertion. There are five potential pathways for lower limb muscle excitation pictured. Excitatory connections between the motor cortex and upper limb motor neurons could branch off to lower limb motor neurons (A). Excitatory connections between the motor cortex and upper limb interneurons could then excite connections to lower limb motor neurons (B) or interneurons (C). Alternatively, a group of common core interneurons (e.g., central pattern generator) could excite lower limb motor neurons (D) or interneurons (E). Unlabeled arrows show pathways for upper limb muscle excitation.

**Figure 8.** Averaged normalized RMS EMG during Self-driven and Externally driven conditions at four different frequencies. Self-driven EMG amplitudes were significantly higher than Externally driven EMG for all muscles (ANOVA with Tukey Honestly Significant Difference post hoc test, *P* < 0.05). In addition, Self-driven condition showed a significant increase in EMG amplitude with frequency (*P* < 0.05), but Externally driven condition did not (*P* > 0.05).
Subjects demonstrated rhythmic lower limb muscle activation (termed locomotor-like muscle activity) with externally driven lower limb movement (8). Passively imposed upper limb movement remarkedly affected the magnitude of the locomotor-like muscle activation in subjects with incomplete lesions at the cervical level. Although the effect of passive upper limb motion was variable from subject to subject, there was a clear excitatory effect of active upper limb motion in those subjects with sufficient upper limb motor control to swing their arms in a normal gait coordination pattern. Results in subjects with clinically complete lesions at the thoracic level were very different. Passively imposed upper limb movement had no effect on lower limb muscle activation in the subjects with clinically complete spinal cord injury.

Taken together, the results from these studies indicate that rhythmic upper limb movement results in an increase in lower limb muscle recruitment proportional to upper limb muscle activation. Although future studies are needed to better understand the basic neural mechanisms involved in this phenomenon, there are clinical implications of the existing findings that need to be considered.

**ARM AND LEG RHYTHMIC MOVEMENT FOR REHABILITATION**

Two common goals of rehabilitation after neurological injury are to increase volitional muscle activation and to produce more coordinated muscle recruitment within a limb. These goals are related to a certain extent. Improved muscular coordination requires modification of existing neural networks and creation of new neural networks (i.e., neural plasticity). To promote neural plasticity, it is necessary to activate neurons. Thus, it seems logical to concentrate on exercise therapies that maximize muscle recruitment and improve intermuscular coordination.

Upper limb rhythmic movement is likely to enhance and help coordinate lower limb muscle activation during neurological rehabilitation. Future studies could examine multiple patient populations, such as stroke, traumatic brain injury, and cerebral palsy patients, to determine whether similar motor facilitation occurs during simultaneous upper and lower limb rhythmic exercise. Interestingly, there has been an increase in the number of commercially available rehabilitation devices that allow for simultaneous upper and lower limb rhythmic movement (Fig. 9). Although there have been no randomized clinical trials examining the therapeutic efficacy of simultaneous rhythmic upper and lower limb exercise, there are some clinical case studies promoting simultaneous rhythmic upper and lower limb activity [c.f. (10)] as a means to promote neural plasticity in locomotor neural networks. Clearly, there is something to be said for task-specificity during motor relearning, but incorporating rhythmic upper limb movements with lower limb movements could easily be added into many therapy programs on a complementary basis. This would have the advantage of allowing patients to increase their therapy intensity with little cost in therapist labor or time. Obviously, clinical trials will need to be conducted to establish any real functional benefit of simultaneous rhythmic upper and lower limb exercise. However, based on the evidence presented in this review, we believe that simultaneous rhythmic upper and lower limb exercise holds strong promise for enhancing neurorehabilitation.

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**References**
