LETTER TO THE EDITOR

13 December 2005
To the Editor:

The case series by Winchester et al. (“Changes in Supraspinal Activation Patterns following Robotic Locomotor Therapy in Motor-Incomplete Spinal Cord Injury,” Neurorehabil Neural Repair 2005 Dec;19(4):313-24) raises some very interesting questions about the role of supraspinal centers in the control and recovery of walking function in individuals with spinal cord injury (SCI). However, there are issues related to the subject sample and data collection methods that warrant closer examination as they bear on the interpretation of the results. There are meaningful differences in the control of ankle plantarflexion with voluntary activation compared to that which occurs during walking.1,2 During walking, there appears to be little cortical involvement in the activation of ankle plantarflexors; therefore, it is not certain that the activation pattern that the authors observed during voluntary plantarflexion in the MRI unit is comparable to what would be observed during walking. Furthermore, as the authors acknowledge, the 2 subjects who were within the subacute phase of their injury demonstrated the greatest improvement in their walking function. Spontaneous recovery of walking function within the 1st postinjury year is common in individuals with incomplete SCI,3 and therefore this may or may not be related to the training or to the change in cerebellar activation; control studies without training would be required to resolve this question. This is further complicated by the fact that there are significant spontaneous changes in cortical activation patterns that accompany early recovery after SCI.4 This suggests that change in cortical activation patterns may have little to do with the training in this early stage of injury. These points emphasize the need to interpret with great caution the results of small intervention studies of individuals with neurological deficits who are within the acute or subacute phase of injury.

Respectfully,

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References

27 December 2005
Authors’ Response

We appreciate the thoughtful comments made by Drs. Dietz and Field-Fote regarding our recent article (Neurorehabil Neural Repair 2005 Dec; 19(4):313-24). They bring up several important considerations regarding our study.

Drs. Dietz and Field-Fote point out that there may be differences in the cortical control of ankle plantarflexion with voluntary movement compared to walking and that fMRI changes in cortical activation for voluntary plantarflexion following Lokomat training may not be what would be observed during walking. In our report, we did not assert that the cortical activation patterns associated with voluntary ankle plantarflexion performed within the constraints of the MRI would necessarily relate to cortical activation patterns for ankle movement during walking. We studied ankle plantarflexion because it is a movement that occurs during walking (as opposed to inversion or eversion) and one that our ASIA C spinal cord injury (SCI) patients could perform (as opposed to dorsiflexion) both before and after training. Other imaging technologies may be able to determine brain activation patterns during the act of walking.

The Capaday article Drs. Dietz and Field-Fote referenced in their letter examined normal subjects. The mechanisms for cortical activation in SCI patients, especially with locomotor training, may be quite different. In the Capaday study, the motor
cortex associated with dorsiflexion of the ankle was somewhat more excitable during walking than during a tonic contraction, whereas the motor cortex representing the plantarflexors was a bit less excitable in walking vs. voluntary contraction; nevertheless, corticospinal input was present for both movement groups during walking and, of course, is present during a voluntary foot movement made during an fMRI study. Corticospinal input during ankle dorsiflexion and plantarflexion was also evident in the report from Petersen et al.1

Drs. Dietz and Field-Fote further point out that spontaneous recovery of gait in motor-incomplete SCI patients can occur in the subacute phase of injury and that cortical reorganization can also occur following SCI, observations that can confound distinguishing a training effect from spontaneous changes. The fMRI changes we reported in cortical activation for ankle plantarflexion following training were rather different in all our patients. They may represent a more global training effect of being able to access more of the cerebral cortex with voluntary effort, rather than changes specific to the control of ankle movement. On the other hand, changes in cerebellar activation patterns in these patients seemed to be related to changes in their locomotor status and gait speed following Lokomat body weight–supported training. We would reason that if spontaneous cortical reorganization accounted for a large part of the cortical activation changes we observed and attributed to a training effect, we would expect patients with more remote injuries to have larger or more variable areas of cortical activation for foot movement prior to training than those with more recent injuries. We did not observe this in our sample. Rather, all patients, regardless of time since injury, activated just the primary motor cortex in or near the foot area prior to training.

In regards to the effect of training on gait recovery in subacute patients, we would be in favor of a study to determine if the number of patients with motor incomplete SCI (especially ASIA C) that regain locomotion spontaneously is different from the numbers that do so with locomotor training and/or to determine whether locomotor training augments, hastens, or furthers the speed, magnitude or quality of gait recovery. This would require a large and lengthy trial, not unlike the Spinal Cord Injury Locomotor Motor Trial, which showed similar gait recovery by some measures in subacute incomplete SCI patients with therapist driven locomotor training and equally dosed traditional rehabilitation therapy.

Finally, Drs. Dietz and Field-Fote caution against overinterpreting our study due to its size and the lack of control subjects. We agreed in the article that a small case series can only suggest the possibility of causality between training, some supraspinal activation pattern changes, and functional recovery in gait. To prove causality will require very much larger studies that carefully match controls and subjects across a multitude of characteristics (age, sex, injury level, injury severity, and time since injury) and will most likely need multicenter efforts, again a process we favor.

Sincerely,

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REFERENCE