Spinal Cord Control of Movement: 
Implications for Locomotor 
Rehabilitation Following 
Spinal Cord Injury

In recent years, our understanding of the spinal cord’s role in movement control has been greatly advanced. Research suggests that body weight support (BWS) walking and functional electrical stimulation (FES), techniques that are used by physical therapists, have potential to improve walking function in individuals with spinal cord injury (SCI), perhaps long after the stage of spontaneous recovery. Walking is one of the most desired goals of people with SCI; however, we are obligated to be judicious in our claims of locomotor recovery. There are few controlled studies that compare outcomes of BWS training or FES with those of conventional interventions, and access to services using BWS training or FES may be restricted under managed care. [Field-Fote EC. Spinal cord control of movement: implications for locomotor rehabilitation following spinal cord injury. Phys Ther. 2000;80:477–484.].

Key Words: Body weight support, Central pattern generator, Gait training, Locomotion, Spinal cord injury.

Edelle Carmen Field-Fote
An almost universal statement of individuals following spinal cord injury (SCI) is: “I will walk again.” Estimates suggest that more than 80% of individuals with motor incomplete SCI regain some locomotor function. Although motor abilities may improve throughout the first post-injury year, the quality and efficiency of walking can be adversely affected by a myriad of factors. These factors manifest in the resulting gait pattern as aberrant timing of muscle group activity, insufficient muscle activity to achieve stepping, inadequate weight-bearing capacity, and balance deficits. Traditionally, rehabilitation of locomotor deficits in individuals with SCI has entailed a regimen of stretching, strengthening, gait training, and prescription of appropriate assistive devices to compensate for motor deficits. Following this course of intervention, gait performance typically plateaus and then there is negligible improvement.

Our evolving understanding of the role of the spinal cord in the control of movement suggests that it may be time to rethink our views on locomotor training of individuals with SCI. Recent evidence suggests that, rather than teaching compensatory strategies, it may be in order to focus our interventions on retraining the spinal motor output. The focus of this perspective will be to review recent findings regarding the role of the spinal cord in the control of movement and evidence that the spinal cord can undergo activity-dependent plasticity. Along with the enthusiasm that accompanies these advances, I believe there also should be prudence in its interpretation. Although the emphasis of this review will be on the potential for the development of interventions to improve walking, the need to critically evaluate the literature will also be addressed.

Spinal Cord Control of Movement

For decades, physical therapists have recognized the contribution of spinal cord circuitry to the control of movement. The result has been the development of interventions to either inhibit this output (eg, neurodevelopmental therapy) or promote and use it (eg, Brunnström’s techniques, Rood’s techniques, proprioceptive neuromuscular facilitation). Spinal reflexes, although evoked by sensory stimuli, can be modified by supraspinal centers in animals or humans with intact spinal cords. Rather than being a hard-wired response to sensory input, recent evidence suggests that, if the sensory stimulus is applied during the course of an ongoing movement, the motor output associated with the stimulus will vary depending on its timing with respect to that movement. In neurologically intact humans during walking, for example, lower-extremity H-reflexes (the electrically evoked analogue of the stretch reflex) are inhibited during late swing and early stance phases of the gait cycle. This modulation appears to be important for normal walking. In individuals with SCI, many mechanisms that modulate segmental reflex activity are deficient. This impaired reflex modulation disrupts the motor output and results in gait abnormalities.

In addition to spinal reflexes, which by definition are single-phased motor responses to sensory input, the spinal cord is able to generate complex, rhythmic behaviors in the absence of both supraspinal and movement-related (proprioceptive) information. Almost a century ago, Sherrington demonstrated that “spinal animals” (animals with a complete transection of the spinal cord) are able to produce reciprocal, alternating patterns of hind-limb movements following spinalization. By cutting the dorsal roots following spinalization, Brown further demonstrated that the deafferented spinal cord, deprived of both supraspinal control and proprioceptive sensory input, can still produce complex motor output. Since that time, animals with transected spinal cords have been shown to be capable of executing a variety of complex innate behaviors (ie, walking, scratching, swimming, wiping, and paw-shaking) in the absence of supraspinal and proprioceptive sensory input. This finding suggests an organizational scheme in which spinal central pattern generators (CPGs) generate the basic motor pattern, with the roles of the higher centers being to activate the appropriate set of spinal CPGs and to impose modifications on this pattern. Spinal CPGs are also influenced by sensory input such that the behavioral output responds to the environmental demands. This parsimonious arrangement reduces the need for higher centers to issue complex commands and for descending pathways to transmit the intricate details of the intended movement.
The spinal cord is able to generate not only rhythmic behaviors, but novel forms of behavior as well. Turtles with intact and transected spinal cords perform scratching behaviors in response to tonic sensory input (tactile stimulus to the carapace, below the level of transection in turtles with transected spinal cords). During these movements, characteristic intralimb relationships can be used to define 3 different forms of scratching. When there are bilateral stimuli, the intralimb coordination of the evoked behavior is well preserved in both limbs. In addition, the interlimb relationship is well coordinated and resembles the form of scratching that turtles with intact spinal cords generate in the performance of other rhythmic behaviors, such as swimming and walking. Most interestingly, during this novel bilateral scratching behavior, the spinal cord is able to generate and coordinate 2 different forms of scratching and control interlimb coordination—in effect, generating massaging the head while rubbing the tummy.

**Spinal Cord Motor Output in Humans**

In humans, the role of CPGs has been appreciated in many forms of rhythmic movement such as respiration, mastication, and speech. The pattern-generating neural circuitry for these activities is thought to exist at supraspinal levels of the central nervous system, primarily in the brain stem. Although it has long been known that the human spinal cord is able to generate rhythmic movements such as clonus, this type of movement differs from CPG-generated movement. Clonus is perpetuated by phasic proprioceptive input (ie, cyclic reactivation of the unmodulated stretch reflex). Until recently because of the absence of locomotor-like behavior in spinal primates and in humans with complete SCI, it was inferred that spinal CPGs, which by definition have the capacity to produce movement in the absence of phasic, movement-related sensory input and in the absence of supraspinal input, existed only in animals phylogenetically below the level of primates. Although accounts of “self-propagated” stepping movements in adult humans with SCI appear in the literature, they are rare. Part of the reason for this dearth of accounts may be that individuals with SCI had not been tested under conditions, such as those used in studies of animals, that encourage the manifestation of such activity. In the last few years, evidence obtained under such conditions suggests that spinal CPGs may, indeed, be present in primates, including humans.

Calancie et al documented a case of an individual with incomplete SCI who complained of being awakened at night by his “walking” legs. This individual exhibited involuntary stepping movements of the lower extremities when he was positioned supine with hips extended. The strength, rate, and rhythmicity of these movements exceeded those that the subject was able to produce voluntarily. The temporal relationships of the electromyographic (EMG) patterns were consistent both within and between testing sessions. The investigators contended that afferent input to the spinal cord due to active osteoarthritis at the hip was a primary factor allowing the central pattern-generated movement to be manifest in this individual with SCI. This hypothesis was supported by a reduction of movement after infusion of lidocaine into the hip joint capsule. The idea that noxious sensory input can facilitate central pattern-generated movement is consistent with observations of researchers working with experimentally spinalized animals who reported that the strength and rate of the central pattern-generated locomotion in these animals is increased, and the pattern is more consistent, in the presence of tonic afferent input (eg, squeezing the tail, stimulating the perianal area). The incomplete spinal cord lesion present in this individual’s injury indicates that this case does not provide unequivocal evidence that the observed movements were solely generated by the spinal cord. However, studies of individuals with neurologically complete SCI offer evidence for the existence of spinal CPGs in humans. Under favorable conditions of body weight support (BWS) and treadmill speed, subjects with complete SCI exhibit appropriately timed lower-extremity EMG activity. The timing of this motor response cannot be explained simply on the basis of muscle stretch reflexes.

**“Training” the Spinal Cord**

Physical therapists have long practiced techniques that utilize our understanding of spinal motor output, but the concept that this spinal reflex circuitry exhibits activity-dependent plasticity is a more recent idea. The concept should not be surprising, however, because as a familiar brain-stem reflex, the vestibulo-ocular reflex has been used as a model of the plasticity of neuronal circuitry. Recent studies investigating modulation of stretch reflex magnitude have revealed evidence that spinal reflexes, and by extension spinal neural circuitry, have the capacity for activity-dependent plasticity. Wolpaw and colleagues have demonstrated that spinal reflexes are amenable to training. Monkeys can be operantly conditioned either to increase or to decrease stretch reflex and H-reflex output to a given level of stimulus. The training effects remain even following spinal cord transection, evidence that the change occurs at the level of the spinal cord. Wolf and Segal have shown similar results in individuals with SCI. Evidence suggests that these training effects persist over time. Additional support for the role of activity in modifying spinal motor output comes from the work of Nielsen et al, who demonstrated that professional ballet dancers have lower levels of proprioceptively mediated reflex activity in the lower extremities than do
untrained individuals (as measured by H-reflex amplitude).

In the training paradigms mentioned so far, it could be argued that, even if these changes are retained at the level of the spinal cord, supraspinal mechanisms are necessary to mediate the change. There is additional evidence, however, that supraspinal input may not be necessary. This research suggests that spinal reflex mechanisms can be modulated by sensory input that is applied regularly over time. In individuals with hyperreflexia, Crone et al. observed changes in reflex modulation with routine use of functional electrical stimulation (FES), as shown by the following: In neurologically intact individuals, the soleus muscle H-reflex was inhibited by a conditioning stimulus to the common peroneal nerve that precedes the test stimulus. This conditioning effect was not observed in most individuals with hyperreflexia. However, in individuals with hyperreflexia who were regular users of FES at the peroneal nerve for walking, the conditioning stimulus inhibited the response to the test stimulus in the same manner as observed in the neurologically intact individuals. Functional electrical stimulation has also been used with some success to restore the normal phase-dependent modulation of the soleus muscle H-reflex in individuals with SCI. Fung and Barbeau applied stimulation to the medial plantar nerve at the sole of the foot to modulate the H-reflex during walking and reported success in partially restoring the normal reflex modulatory mechanisms and eliciting a more normal gait. These investigations suggest that appropriate sensory input can be used to normalize the reflex output of the spinal cord that is disrupted with SCI. Along these lines, one could reason that the rhythmic input supplied by a moving treadmill is another form of sensory input that may be used to train spinal neural mechanisms.

Beyond evidence that spinal reflexes are amenable to training are studies indicating that spinal CPGs respond to training as well. Spinal CPGs exhibit “learning” effects that have heretofore been associated only with higher neural centers in that this learning is task-specific. The type of training used and its specificity to the task of interest are important factors in the resulting motor performance. For example, cats with transected spinal cords trained to step with the hind limbs on a treadmill step proficiently, producing kinematic and EMG responses that are consistent with those of neurologically intact animals, whereas those trained in stationary standing are able to bear body weight in a static position. The stepping-trained animals do not stand well, and the standing-trained animals do not step well on the treadmill. However, the animals can be cross-trained over time, a further indication that the capacity of the spinal cord for motor output is plastic and amenable to training. Treadmill step training does not transfer to overground walking in these animals, for a number of reasons. Among these is the loss of neural connections, and therefore coordination, between forelimbs and hind limbs as a result of the transection, as well as the loss of balance and postural responses that are controlled by supraspinal centers.

The evidence that spinal neuronal organization is amenable to influence from sources other than supraspinal centers is important in the rehabilitation of individuals with SCI. Preliminary studies to investigate training effects in individuals with SCI have yielded results that can be interpreted with cautious optimism. Following a program of BWS treadmill training, individuals with incomplete SCI have demonstrated changes in gait variables consistent with improved neural control of locomotor output (eg, increased walking speed, decreased co-contraction, more normal limb kinematics). This training consists of an interactive program of BWS walking on a treadmill and can be used in conjunction with FES. Although improvements such as increased endurance and muscle force can be explained on the basis of peripheral neuromuscular effects of training, other improvements, such as more normal gait kinematics and muscle recruitment patterns and decreases in inappropriate coactivation, suggest changes at the level of the central nervous system. In a study of individuals with acute SCI who were trained to step on a treadmill with BWS, Wernig et al. found improvement in overground walking ability in this group (as measured by increased walking speed and endurance) compared with those in a conventional therapy group, even though there were no between-group differences in voluntary motor recruitment. Evidence of locomotor-like EMG output has also been noted during BWS treadmill walking in individuals with complete SCI. For this population, however, there is as yet no evidence that this activity leads to useful, overground walking.

In the pharmacological realm, new interventions using specific neural receptor agonists and antagonists are being examined. A number of these are directed at influencing the motor output of the lumbosacral spinal cord and at the recovery and maintenance of motor function. In many cases, the pharmacological agents that are used are the same as or similar to those used in studies of animals to elicit central pattern-generated locomotion. Fung et al. examined the combined effects of 2 pharmacological agents—clonidine, a noradrenergic agonist, and cyproheptadine, a serotonergic antagonist—in conjunction with an interactive gait training program using partial BWS. In this investigation of 2 individuals with chronic SCI who had previously been wheelchair-bound, both patients where able to...
perform overground locomotion with forearm crutches by the end of the study period.

Considerations for Improvement of Walking Function in People With SCI

Dramatic advances are being made in basic science research pertaining to SCI, and a “cure” for paralysis may be found in the foreseeable future. In the interim, however, the incidence of new SCIs (as estimated by state-based statistics) in the United States is between 30 and 40 individuals per million people in the population. This incidence results in a prevalence rate of 183,000 to 230,000 affected individuals70 who are required to manage with technology as it currently exists. The recent insights into the neural control of motor output offer direction for the development of effective rehabilitation strategies. Specificity of training can have an impact on acquisition of motor function in individuals with neurological impairment,71,72 and physical therapists are uniquely positioned to develop techniques that use this emerging model of motor control. With a knowledge of gait biomechanics and with tools such as FES,73–77 and with the increasingly prevalent use of BWS in the clinical setting,5,12,15,78,79 there are countless possibilities, but we must be judicious and critical in our choices. As yet, there have been few controlled studies to compare outcomes of training with BWS or FES with conventional physical therapy interventions in individuals with SCI. In the current health care system, I believe most individuals with SCI are discharged from physical therapy long before recovery of locomotion has been maximized, and most do not have access to these services beyond their acute stay. Although motor return is dramatic during the acute and subacute stages encompassing the first 6 to 12 months posttrauma,5,80–82 the individual may not be able to take advantage of this return without access to physical therapy for evaluation and intervention.83

Even though SCIs of greater than 1 year’s duration are generally considered to be chronic,14,28,83–85 some individuals with injuries of greater than one year’s duration have potential for functional improvement.14,83,86 This gives rise to a number of questions that must be asked when evaluating outcome studies of locomotor training in individuals with SCI. I argue that we must be able to answer these questions so that we can make the best decisions about what are the most appropriate interventions to use and to ensure that our claims can bear the scrutiny of the scientific community, the community of payors, and, most importantly, our patients.

1. Are changes due to the intervention? Motor function does not plateau until well into the first year postinjury. When interventions are applied prior to plateau of function, a control group is necessary to ensure that the observed changes are due to the intervention and are not due to the natural course of motor recovery.

2. How does any specific intervention compare with conventional locomotor training? Given that most individuals with chronic SCI typically do not receive locomotor training, I contend it is important to compare any “new” intervention with conventional techniques. Only then can we judge whether the effects were due to the particular intervention or simply due to the increased practice that would occur with any type of task-specific training. The answer to this question can make a difference in the selection of equipment.

3. What outcome measures are used? Although locomotor training has many benefits beyond improvement of walking ability,86–88 I believe the primary outcome measure must be capacity for overground walking if the primary goal of the intervention is to improve walking ability. The secondary benefits of locomotor training aside, it is not likely there will be recovery of independent overground walking6,14,48 solely as a result of training in individuals with chronic SCI whose injury, according to the American Spinal Injury Association (ASIA) Impairment Scale, is classified as ASIA A (no motor or sensory return below the level of the lesion) or ASIA B (sensory but no motor return below the level of the lesion).89

4. Is the type of lesion consistent with the idea that neurophysiologic changes leading to recovery of function are occurring at the level of spinal cord? I argue that discretion must be used in interpreting studies of individuals in which the injury is classified as ASIA D (sensory and motor function are preserved below the level of the lesion, and at least half of the key muscles have a grade of 3 or more). In these individuals, there remains considerable supraspinal input to the areas of the spinal cord that are below the level of the lesion. Although improved function is achieved no matter what the mechanism, in these individuals it is not judicious, in my opinion, to assert that changes observed are due to plasticity at the level of the spinal cord. Alternatively, in individuals with SCI below the level of T10, there may be considerable lower motor neuron damage from trauma to the nerve roots exiting the spinal cord, resulting in a mixed upper motor neuron and lower motor neuron lesion. In such cases, outcomes of training may be equivocal because it may be difficult to discern between disuse atrophy from upper motor neuron damage and denervation atrophy due to the lower motor neuron damage.

Research indicates that interventions in which physical therapists are expert have tremendous potential for achieving improvement in functional outcomes in indi-
uals with SCI. I contend it is in the best interest of our patients and our profession to investigate fully how we might best use these skills, in conjunction with treadmill training and FES and along with newer approaches such as BWS training, to develop evidence-based interventions. I believe we are doubly bound to use caution in this arena because of the great and emotion-laden appeal of programs that are aimed at restoring locomotor function. Although there are many hurdles yet to overcome, the potential rewards, in terms of quality of life, are incalculable. So, let us move onward into the fray with a cautious but open mind.

References


