III STEP Series

Neuroplasticity After Spinal Cord Injury and Training: An Emerging Paradigm Shift in Rehabilitation and Walking Recovery

Physical rehabilitation after spinal cord injury has been based on the premise that the nervous system is hard-wired and irreparable. Upon this assumption, clinicians have compensated for irremediable sensorimotor deficits using braces, assistive devices, and wheelchairs to achieve upright and seated mobility. Evidence from basic science, however, demonstrates that the central nervous system after injury is malleable and can learn, and this evidence has challenged our current assumptions. The evidence is especially compelling concerning locomotion. The purpose of this perspective article is to summarize the evidence supporting an impending paradigm shift from compensation for deficits to rehabilitation as an agent for walking recovery. A physiologically based approach for the rehabilitation of walking has developed, translating evidence for activity-dependent neuroplasticity after spinal cord injury and the neurobiological control of walking. Advanced by partnerships among neuroscientists, clinicians, and researchers, critical rehabilitation concepts are emerging for activity-based therapy to improve walking recovery, with promising clinical findings. [Behrman AL, Bowden MG, Nair PM. Neuroplasticity after spinal cord injury and training: an emerging paradigm shift in rehabilitation and walking recovery. *Phys Ther.* 2006;86:1406–1425.]

Key Words: *Locomotion, Recovery of function, Spinal cord injuries.*

Andrea L Behrman, Mark G Bowden, Preeti M Nair

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

The purpose of this perspective is to summarize the evidence supporting an emerging paradigm shift[1] for the rehabilitation of walking after incomplete spinal cord injury (SCI) from compensation for deficits to activity-dependent neural adaptation and training. If new basic science findings are efficiently and effectively integrated into clinical practice, it will no longer be possible for scientists and clinicians to work independently. Rather, this integration will require a partnership between clinicians and scientists and a shift in mind-set from compensatory training to activity-based therapies as the foundation for rehabilitation. This shift could create a temporary period of discomfort as clinicians move from traditional practice to the challenge of creative translation of science into clinical practice.

A series of hypothesis-based studies are proposed to examine important issues defined by clinicians in partnership with neuroscientists, exercise and muscle physiologists, engineers, biomechanists, physicians, and consumers. Furthermore, the theoretical analysis supporting a recovery model of locomotor function after SCI based on activity-dependent plasticity and neurobiology may be applicable to other biological systems such as respiration and upper-extremity function.

Current Model of SCI and Rehabilitation: Compensatory Clinical Model

Since the 1928 work of Santiago Ramón y Cajal, famed neuroscientist, the prevailing assumption has been that the central nervous system (CNS) is hard-wired, nonmalleable, and incapable of repairing itself.[2] This perspective has provided the foundation that has buttressed and guided decision making for physical rehabilitation after SCI. Clinicians have selected compensation as a rehabilitation strategy for nonremediable deficits of strength (force-generating capacity), voluntary motor control, sensation, and balance. This approach enables, rather than remediates, disablement. The patient learns to compensate, using other abilities to complete a task, or to modify the task or the environment to accomplish the goal.[3] Clinical decision making has been guided by expected outcomes based on the degree of motor and sensory loss from total to partial.[4]

Advances in neuroscience are unlocking the mysteries of recovery after SCI. Although all of the pieces of the puzzle are not in place, physical therapists should shift their paradigm.

A review of texts published in 2000 and 2001 for instruction of adult neurorehabilitation in physical therapist education programs[5–7] supports compensation as a predominant foundation for physical rehabilitation practice for people with SCI. Typical goals of SCI rehabilitation are to strengthen available muscles under voluntary control; to support and compensate for paresis or paralysis using braces and assistive devices; to teach new movement strategies to accomplish activities of daily living, including dressing, transfers, and bed mobility; to teach new strategies for upright mobility that incorporate braces and assistive devices; and to teach wheelchair mobility skills.[5–7] Clinical decision making has stemmed from the associated expected functional outcomes according to the level and severity of complete SCI.

Basis for New Model for SCI and Rehabilitation: Activity-Dependent Plasticity and Recovery

Over the past 30 years, neuroscientists have sought to determine the role of the spinal cord in controlling movement in general and locomotion in particular. Some neuroscientists have investigated the capacity of the CNS, in particular the spinal cord, to learn, to respond, and to control walking in animals with complete spinal cord lesions. Seminal work in this area can be reviewed in numerous primary and review articles.[8–10] Lovely et al[11] demonstrated that cats with complete

AL Behrman, PT, PhD, is Associate Professor, Department of Physical Therapy, College of Public Health and Health Professions, University of Florida, PO Box 100154, UFHSC, Gainesville, FL 32610-0154 (USA), and Research Scientist, Brain Rehabilitation Research Center, Malcom Randall VA Medical Center, Gainesville, Fla. Address all correspondence to Dr Behrman at: abehrman@phhp.ufl.edu.

MG Bowden, PT, MS, is Research Physical Therapist, Brain Rehabilitation Research Center, Malcom Randall VA Medical Center.

PM Nair, BPhT, is a doctoral candidate, Rehabilitation Science Doctoral Program, University of Florida.

Dr Behrman provided concept/idea/project design. All authors provided writing.

This article is based on a presentation at the III STEP Symposium on Translating Evidence Into Practice: Linking Movement Science and Intervention; July 15–21, 2005; Salt Lake City, Utah.

This article was received July 8, 2005, and was accepted June 5, 2006.

DOI: 10.2522/ptj.20050212

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

spinal transections respond to intense walking training. When cats were provided with truncal support, manually assisted loading, and stepping kinematics over a treadmill, they generated a hind-limb stepping response even in the absence of supraspinal input. Additionally, the cats increased their cadence and step length appropriately when treadmill speed was increased. One explanation for this response is the spinal cord's capacity to respond to afferent input associated with the increased treadmill speed. Whether that input is proprioception, muscle length, cutaneous feedback, or load, it indicates a change in context and signals a change in motor output to meet the demand. An intact feedback loop between afferent and efferent nerves with an upper motor neuron (UMN) lesion (above the lumbosacral area) provides a means for input to the neural axis and for generation of a motor response. This phenomenon of appropriately responding to sensory input supports the view of the intrinsic capacity of the neural network at the level of the spinal cord to integrate incoming information, interpret it, and respond with a motor output. Similarly, Hodgson et al[12] observed that when cats with complete midthoracic spinal transections were trained either to stand or to hind-limb step on a treadmill, each group learned its respective task. Neither group, however, could perform the nontrained, alternate task: stepping or standing. This finding has lent support to the concept of "task specificity" when retraining after SCI.

The activity-dependent plasticity of the spinal cord, once thought to be unresponsive and incapable of recovery, serves as one prong of scientific evidence challenging the assumptions of current clinical practice. A second is the scope of research examining the role of specific afferent input to the neurobiological control of walking.[13,14] Two examples of this evidence and its implications for retraining walking after SCI are emphasized here. One example deals with the effect of hip position, and the second example deals with the effect of load.

Sherrington[15] was the first author to propose that proprioceptors responding to hip extension are important for initiating swing. Grillner and Rossignol[16] found that preventing the hip from extending in chronic spinal cats inhibited the generation of the flexor burst and thus the onset of the swing phase. The most direct evidence for this conclusion, however, came from vibrating the hip flexor muscle (iliopsoas) during stance. This vibration led to an earlier onset of swing in walking decerebrate cats.[17] Vibration likely stimulated the primary and secondary endings of muscle spindles in hip flexor muscles, simulating the stretch, which occurs when the hip is extended during stance. Similarly, in humans, involuntary and alternating stepping-like movements were observed in an individual after incomplete SCI when the hip was extended in the supine position.[18] The findings of research examining infant stepping also support the role of hip extension position for the initiation of swing. From the recorded hip motion and electromyographic (EMG) data, scientists concluded that the preferred hip position was extension in late stance, which stretches the hip flexors and triggers forward swing of the limb.[19] These data suggest that the hip position is important in initiating the transition from stance to swing.

Another important sensory input regulating the stance-to-swing transition is the extensor load relayed by the Golgi tendon organs (Ib) in the ankle extensor muscles.[13,14] During locomotor activity, electrical stimulation of the group Ib afferents from the ankle extensor inhibits the generation of flexor bursts, which prolongs the duration of extensor activity. Duysens and Pearson[20] observed that gradually increasing the load applied to the Achilles tendon resulted in increases in both amplitude and duration of the rhythmic EMG bursts of the ankle extensors. In humans, researchers found that unloading the ankle extensors by a portable device in the stance phase of walking reduced soleus muscle EMG activity; this reduction was maintained even when transmission in Ia afferents was blocked by local anesthesia. This finding pointed to group Ib or group II afferents contributing to the extensor EMG activity in the stance phase.[21] Harkema et al[22] observed that the amplitude of extensor muscle activation in the legs was directly related to the level of body weight loading on the legs during the manually assisted stepping of subjects with and without SCI on a treadmill. Furthermore, limb peak load was more closely associated with modulation of the extensor EMG amplitude than muscle stretch or velocity of stretch. Dietz et al[23] also found that physiological locomotor-like leg movements alone (100% body unloading) generated by the application of a driven gait orthosis on a treadmill were not sufficient to generate leg muscle activation in either subjects with complete paraplegia or tetraplegia or subjects without injury. In this study, leg movements in combination with loading of the legs led to appropriate leg muscle activation.

Hip extension position and load are 2 examples of sensory input specific to the task of walking that contribute to the inherent mechanisms in the neural axis generating stepping.[24] These sensory signals are interpreted by a network of spinal interneurons, often referred to as a "central pattern generator" (CPG), which combine with descending supraspinal input in order to control walking.[25] The pattern of locomotion is attributed to the CPG, which promotes the rhythmic oscillations of the extremities. Thus, it is intuitive to develop rehabilitative strategies that emphasize the provision of hip extension and load, as well as other sensory elements contributing to the control of walking. A

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

complete ensemble of sensory information relative to walking (ie, speed, interlimb and intralimb coordination, and kinematics) provided during training would likely enhance the neural output generating walking. Greater clarity of the sensory experience of walking may be necessary for people with more severe injuries and locomotor deficits.

Evidence from this literature can be translated into therapeutic guidelines and incorporated into interventions to promote the recovery of walking.[26–28] The term "locomotor training" (LT) has arisen to describe a physiologically based approach to retraining walking after neurologic injury that capitalizes on the intrinsic mechanisms of the spinal cord to generate stepping in response to specific afferent input associated with the task of walking.[26–31] Guidelines for LT, for instance, include maximizing loading of the lower limbs instead of the upper extremities during training. Although body-weight support systems provide this opportunity, facilitating more upright standing and adjusting the height of assistive devices (if used) overground also may promote greater load bearing by the lower limbs relative to the arms. Instructions to ensure that the leg hits the ground before a forearm crutch emphasizes load bearing through the legs. Synchronizing hip extension and limb unloading with simultaneous loading of the other lower limb to promote swing initiation and activation of contralateral limb extensors also is an important guideline. Equally critical is promoting the initiation of stepping from a stride position to allow weight transfer from an extended and loaded limb forward to the unloaded limb. Certainly other training guidelines may be translated from basic and clinical evidence.[25,26,28,32–34]

Translation of Animal Basic Science to Human Clinical Science

Findings from basic scientists provide a foundation for recovery after SCI based on an understanding of activity-dependent plasticity and of the neurobiological control of walking. The nervous system is responsive to input and can learn even after injury. The training experience afforded to the nervous system is critical and specific to the sensory experience associated with the goal: standing or walking.

The basis for a paradigm shift in clinical rehabilitation of people with SCI has been recorded in scientific publications as early as 1991[35] by the neuroscience community proposing "a physiological basis for development of rehabilitative strategies for spinally injured patients"[13,14] and continues through publications in 2004 describing plasticity after SCI and locomotor activity after SCI in humans.[10,36] Similarly and in parallel, publications by clinical scientists with doctoral training in neuroscience or motor control (or collaborating with neuroscientists) offer concepts that will form the basis for a new direction in locomotor recovery and rehabilitation after neurologic injury.[27,28,37–39] These "emerging rehabilitation concepts"[28,35,40,41] include recovery based on intense practice of the specific task, locomotion; providing appropriate sensory input (loading and unloading, trunk posture, hip extension, limb kinematics) associated with the locomotor task to tap the intrinsic neural networks generating stepping activity; permissiveness of the training environment (treadmill speed, body-weight support [BWS]) to enhance practice of the locomotor task; integration of postural control as a corequisite for locomotion; and minimizing compensation (load bearing through the legs versus load bearing through the arms, hip hiking for swing).[42]

The dialogue among neuroscientists, clinical scientists, and clinicians will allow them to inform one another of the critical questions unanswered in the translation from basic science to human application. Clinicians also may identify important questions from their experiences that require preliminary work in animal models prior to testing in humans. For instance, baclofen is a relatively common drug used to reduce spasticity by altering reflex activity. The potential interactive effect of baclofen on walking recovery, and in particular its effect on the activation of stepping, is not known. Neuroscientists can readily examine issues of severity of injury, dose, timing, and training interactions on walking recovery while using animal models of SCI. Basic science findings may direct clinical practice or clinical research relative to pharmacological interactions with training and enhance sensitivity of measures.[43]

A Proposed Shift in Clinical Decision Making Based on the Recovery Model

Evaluation

Evaluation from a compensatory model is primarily accomplished using the American Spinal Injury Association (ASIA) classification system of impairments, including both manual muscle testing and sensory testing.[44] From this evaluation, injury severity is classified as ASIA A, B, C, D, or E, and the neurological level of the lesion is established. This system is used to classify residual function, to group and compare patients in impairment categories, and to predict functional outcomes, including walking ability after SCI.[4,45–50] The evaluation of isolated, voluntary motor control during a manual muscle test has been identified, in conjunction with lesion level, as primary predictors for ambulation when in the acute stage after SCI.[50,51] Achievement of quadriceps femoris muscle strength greater than 3/5 within 3 months of SCI , in particular, has been strongly associated with ambulatory potential.[49] Interestingly, preservation or recovery of pinprick sensation after

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

acute SCI (within 72 hours) is highly predictive of recovery of walking function at time of discharge from rehabilitation.[48] These indicators, furthermore, are used to assess the more immediate potential for ambulation in people with chronic SCI. This evaluation model is set in a hierarchical model for the neural control of movement noting a top-down system for the control of walking[52] and the ability to perform isolated voluntary movements while lying supine. The ASIA classification system and lower-extremity motor scores (manual muscle test scores) are excellent predictors associated with walking recovery when applied acutely after SCI. However, evidence indicates that people with chronic, incomplete motor SCI may recover or improve walking function after training without associated improvements in ASIA lower-extremity motor scores.[26,27,31,53,54] Such evidence, as well as the basic science literature in animal models of SCI,[10,35] suggests an alternative mechanism for improved walking ability and warrants speculation as to how this neurally driven capacity may be assessed and trained.

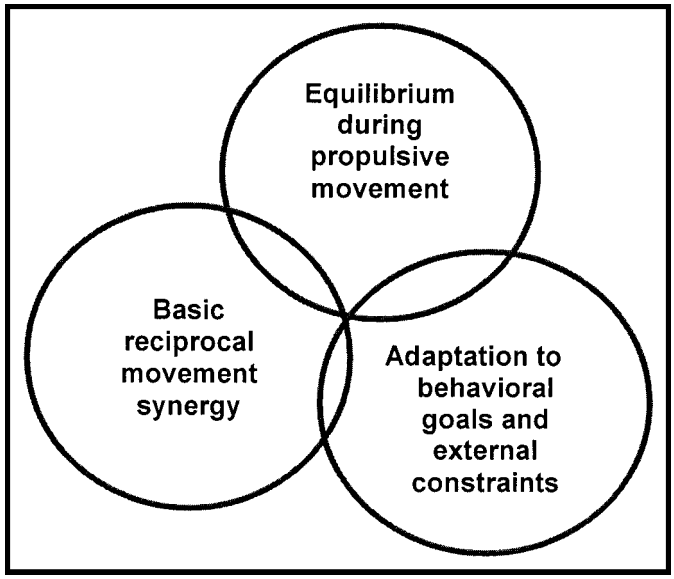

Figure.
Functional walking control requirements by the nervous system.

Within the recovery model, an evaluation is proposed[55] to examine the capacity to generate walking behavior within a conducive environment and within the context of a nonhierarchical model for the control of walking,[25] capitalizing on sensory input to generate a motor output for walking in combination with supraspinal drive. Use of a BWS system and treadmill may provide a permissive evaluation environment in that they afford the conditions for exhibiting such behavior. Walking overground for people with compromised nervous system function after SCI requires significant neurophysiological and biomechanical demand to support body weight, to balance, and to generate the necessary forces for walking. As a result, the individual walks with an altered pattern overground, compensating for various motor and sensory deficits and using braces and assistive devices.[42] Although walking capacity ultimately must be applied overground, the BWS and treadmill environment with manual assistance may provide an alternative means to discern the potential of the nervous system and a viable training environment. The Appendix provides a detailed comparison of the compensatory and recovery-based approaches to evaluation and treatment.

As illustrated in the Figure, walking entails 3 neural control mechanisms: a reciprocal stepping pattern (for propulsion), balance (upright and dynamic equilibrium), and adaptability (the ability of the individual to respond to the demands of the environment and to meet his or her own behavioral goals).[28,56] The evaluation of walking, therefore, should assess each of these elements of control. This evaluation approach would afford clinicians the means to classify people after SCI according to a discriminative examination of motor control deficits and abilities specific to the task of walking.[55] Each neural control mechanism can be expanded, identifying the specific subcomponents necessary to accomplish the task. For instance, the extensive work modeling the tasks required for the control of balance and the control of adaptability may provide the basis for an evaluation of these 2 subtasks.[57–59] Evaluating and categorizing locomotor abilities in such an environment ultimately may contribute to a clinical, decision-making algorithm for the rehabilitation of walking.[33,55] Such an approach also would afford researchers and clinicians a means to assess the differential effect of various modalities and training protocols on these 3 essential elements of walking: stepping, balance, and adaptability. Thus, the model serves not only as a framework for evaluation, but also as a framework for developing outcome measures and treatment planning.

As previously noted, the compensation model targets isolated muscle strength as critical for ambulation. From a recovery and task-specific view, however, the locomotor requirement is not simply for isolated, voluntary muscle strength. For instance, neuromuscular control of an extensor moment is required for a synergistic, reciprocal stepping pattern throughout the gait cycle and during the transfer of body weight. Furthermore, control of the extensor moment provides a stable base of support to the head, arms, and trunk. Although quadriceps femoris muscle strength is an important contributor to this capacity, the control of this extensor force during the upright and propulsive task specific to walking is critical for successful ambulation.[60]

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

Treatment

In the compensatory model, the goal of upright mobility is achieved by modifying the task and environment. These conventional strategies "enable" rehabilitation.[3,4] In the recovery model, decisions regarding the use of equipment, assistive devices, or braces are considered within the context of providing a training experience consistent with the "emerging rehabilitation concepts" to maximize the intrinsic mechanisms within the CNS to generate stepping. The introduction and use of assistive devices, braces, wheeled mobility, the overall training environments, and the progression process will differ for these 2 perspectives. Comparisons of the 2 decision-making processes are identified in the Appendix.[5–7,26]

An example of divergent paradigms is the initial assessment of the locomotor limitation and choice of interventions. In the compensatory model, weakness and loss of voluntary motor control are considered problems for which a walker or parallel bars can provide compensation. Weight bearing through the arms on an assistive device and a forward flexed trunk may restrict hip extension, loading of the lower limbs, production of ground reaction forces associated with propulsion, and activation of flexion during transition from stance to swing. Visintin and Barbeau[42] investigated the consequences of weight bearing on the upper extremities compared with load bearing through the legs, both with 40% BWS provided. Upper-extremity weight bearing resulted in decreased EMG activity in the lower limbs and more asymmetry in the limb kinematics. Thus, a compensatory strategy emerged when individuals used an assistive device for weight-bearing support. In contrast, overhead BWS resulted in a more symmetrical pattern of EMG activity and gait. Thus, a recovery-based approach incorporating this evidence would suggest a benefit to using overhead BWS without a handrail or upper-extremity support while training on the treadmill and diminished use of the upper extremity during training overall. In this case, erect posture and diminishing load bearing on the arms become critical components of the training. The BWS provides a permissive environment to elicit walking capacity.

Decision making for use of a walker or overhead BWS illustrates the process of translation from animal to human research to clinical practice. Other hypotheses may be generated around this single domain, use of BWS assistance, in developing a physiologically based LT program. Certainly, guidelines for BWS will continue to be refined as evidence for varied training protocols are identified for specific walking deficits.[33,34]

In examining the introduction of a walker relative to balance control, compensation and recovery models again result in 2 different approaches. When clinicians are training balance specific to the task of walking, use of a walker is not appropriate, because its presence modifies the task. Maintaining balance while using a walker becomes "task specific" to the presence of a walker. As a patient so honestly explained to us when asked if he could be evaluated while walking without his walker, "I was trained to walk with 6 legs, not with 2." If we anticipate training the corequisite task of balance,[61,62] we must train it during the task of walking with 2 legs and not with "6 legs." Balance is a corequisite of the task of walking and a significant requirement for successful walking.[61,62] Retraining balance may more effectively be trained within the specific task of walking without upper-extremity support.

Comparable elements such as the use of an ankle-foot orthosis (AFO) during training or for walking overground may be hypothesized and studied. Use of an AFO may be recommended after SCI due to weakness, an unstable and uncontrolled position of the foot during swing or stance, or lack of foot clearance during swing, or for safety. Clinicians suggest that the AFO solves the problem of ankle control by controlling the degrees of freedom at the hip and knee, thereby allowing the patient to gain more proximal control for walking. The brace is used to enable mobility. Several researchers purposefully do not train with an AFO when using a physiological, activity-dependent therapy for retraining walking.[26,27,37] The AFO, if used during training, may alter the limb mechanics, ground reaction forces, and afferent input, thus inhibiting the responsiveness to sensory input to generate stepping. Whether the patient ultimately uses an AFO for walking in the home and community is a matter of benefit, safety, and clinical judgment. The implications of training with or without an AFO provide a hypothesis-driven study critical to determining an effective training protocol.

State-of-the-Translation to Clinical Science and Practice

The discovery of the spinal cord's capacity for activity-dependent plasticity and afferent-based generation of locomotion after SCI in animal models led to a translation of these findings to humans after SCI. Application to the human condition formed the basis for a bridge in communication across the neuroscience and clinical rehabilitation professional communities. Hugues Barbeau, a physical therapist and neuroscientist, was one of the early scientists to translate the animal model findings for SCI and LT to humans.[63] He explored the use of BWS placed over a treadmill within the context of providing the sensory experience of walking in order to generate walking after SCI and stroke. The BWS and treadmill were tools used to provide an environment "permissive" to practice the task of walking intensely and to afford the specific, sensory experience associated with the task. Although a primary training environment to

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

develop the capacity to step and the corequisite posture and equilibrium is critical, this capacity and skill must transfer and be practiced in the overground environment as well.[26,64] Other researchers[65,66] and the medical equipment industry responded with the development of commercially available BWS systems. Current clinical enthusiasm for the BWS system may be premature without evidence for practice guidelines addressing clinical decision making, precautions, and safety. Translation to the human condition after SCI has been investigated in both clinical and research settings over the past 15 years. Here we review 3 studies relative to the training effects of LT for people with acute SCI and 11 studies relative to people with chronic SCI (>5 months after SCI); these studies may provide some clinical guidance. Cross-sectional studies also have targeted an understanding of the parameters of training and their immediate effect.[22,32,42]

Six criteria for evaluating how physical therapy treatment approaches should be critiqued for scientific merit have been proposed: (1) theories underlying the treatment approach are supported by valid anatomical and physiological evidence, (2) the approach is designed for a specific type of patient population, (3) potential side effects are presented, (4) studies from peer-reviewed journals are provided that support the treatment's efficacy, (5) studies include well-designed, randomized, controlled clinical trials or single-subject experimental studies, and (6) proponents of the treatment approach are open and willing to discuss its limitations.[67] We applied these criteria to examine LT as a new therapy being translated into clinical practice.

In the example of LT, the available evidence indicates that the first criterion for theoretical support validated by biological evidence has been well met through the work of Edgerton et al,[35] Barbeau and Rossignol,[68] and other researchers[12,69] in animal models and basic science. With regard to the second criterion, there is a substantial body of literature based on translation of the basic science findings for clinical application after SCI and stroke, and evidence is emerging for other neurological disorders. The third criterion for safety of the intervention has been addressed by the lack of significant adverse events when compared with conventional gait training. A recent SCI trial of LT in acute rehabilitation demonstrated safety and feasibility for delivery of this intervention in early rehabilitation after SCI.[29] Other studies[27,70,71] support safe delivery to people with chronic SCI. Safe delivery is important relative to the timing of delivery, the chronicity of injury, and the severity of injury (complete or incomplete). Complications such as bone loss, sensory deficits, range-of-motion and flexibility limitations, heterotopic ossification, habitual compensatory behaviors, autonomic dysreflexia, orthostatic hypotension, and others may complicate the ability to provide LT safely and effectively, but the current literature has not addressed these points directly. In order for LT to be transitioned to the clinic for appropriate populations, these critical safety issues will need to be examined.

To address the fourth criterion of efficacy, a synopsis of peer-reviewed and published studies is provided in Tables 1 and 2. These tables summarize: (1) LT and outcomes in people with SCI and (2) specific training parameters and their immediate effects in people with SCI, respectively. To examine the efficacy established via these studies, Sackett's levels of evidence were applied to each study, and the resulting levels are indicated in Table 1.[72] For clarity, the studies have been separated into those that examined LT in people with acute SCI and those that focused primarily on people with chronic SCI (greater than 5 months postinjury).

The best evidence (level I) for people with acute SCI comes from a recently completed RCT comparing LT and a control group that received an overground training program matched in intensity for people with ASIA B-, C-, and D-classified injuries within 8 weeks of their SCI. Sixth-month outcomes for both ASIA C and D with UMN injuries regardless of the therapy received indicated remarkable achievements of normal walking speed (1.1 m/s) after 45 to 60 sessions of therapy during acute rehabilitation.[29] Much of the additional, positive evidence for LT, though, has come from case reports and small-group studies of people with chronic SCI.[29] Although the results are promising in people with chronic SCI, the majority of evidence is either Sackett level IV or V, and efficacy cannot fully be determined without an appropriately designed randomized clinical trial (RCT).

Regarding the sixth and final criterion of being open and willing to discuss the limitations, it should be noted that the scientific community has led the effort to show restraint in the not-yet-justified translation to unstudied populations and clinical practice. The advent of technology has inspired many people in the clinical community to assume efficacy because of novelty of the equipment, and attempts to brand a therapeutic intervention by the equipment used should be met with caution. The term "body-weight–supported treadmill training" has become a lexicon for any therapy that uses these 2 pieces of equipment regardless of the therapeutic goal. The use of this language may mask the active ingredients of the therapy by overemphasizing the role of the equipment as opposed to emphasizing the goal of the therapy and the scientific underpinnings guiding clinical decision making for this physiologically based intervention. Thus, not all studies identified as "body-weight–supported tread-

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

mill training" adhere to the same principles of training, training variables, progression, or theoretical context.

For the purpose of this article, the aim of LT is to enhance or restore walking after neurologic injury or disease. Locomotor training is a physiologically based therapy affording intense practice and repetition of the task of walking in environments affording a locomotor-specific experience of walking, skill progression and acquisition, and transfer of this capacity to community ambulation. The theoretical underpinnings recognize a tripartite model of the neural control of walking and thus the opportunity to both activate stepping pattern generation from supraspinal, descending pathways and afferent, ascending pathways in the neural axis.[25] In the presence of a compromised and dysfunctional descending circuitry, training that capitalizes on the intrinsic mechanisms of the CNS to generate rhythmic movements via sensorimotor pathways and its capacity to learn is the basis for locomotor training. Available supraspinal drive is incorporated by engagement of the patient in critical tasks (ie, holding an upright posture, weight transfer) and goal identification and setting.[26]

Analysis using the suggested set of criteria illustrates that LT, despite its detailed investigation from animal models to RCTs, cannot be indiscriminately recommended for widespread application across all people with SCI for the recovery of walking. Case reports and case series have described little effect in people with lower motor neuron injuries. There are no reports of someone with ASIA A or B injuries being able to translate improved walking behavior to an overground environment, and the only RCT that has been published on the topic indicates that LT is equally effective in achieving overground gait speed in people with acute SCI, ASIA C and D.[29] Achievement of gait speed outcomes in both groups exceeded expectations for recovery; 92% walked as opposed to 58% historically.[29] Evidence from studies with smaller samples of subjects with SCI offer very compelling evidence; however, recovery of locomotion beyond that currently seen with compensation-based approaches may be actualized in human populations. Translation into clinical practice, according to the Megans and Harris criteria,[72] would be contingent upon several factors that are currently absent from the current state of evidence, including: (1) standardization of critical elements of training parameters, (2) identification of individuals for whom the therapy is most appropriate (beyond the ASIA classification),[73,74] and (3) evidence for benefits in people with chronic SCI in the form of a well-designed RCT. Other areas also warrant consideration, including timing of delivery postinjury, staffing patterns, cost-benefit, equipment options, and by-products of training for health.[26]

In order to develop the "best practice" for the recovery of walking, certain critical questions must be answered. To most effectively apply LT, clinicians need to know which patients will benefit and when postinjury that benefit will be maximized. The interventions that maximize recovery of walking, the interventions that should be paired with each other as a hybrid treatment, and the interventions that will augment the training by addressing other deficits such as strength and activation should be identified. Finally, questions about how best to deliver the intervention (including intensity, duration, frequency, safety, decision making, and progression) should be investigated. Such information would form a "Guidelines for the Recovery of Walking After SCI" comparable to the Paralyzed Veterans of America guidelines for functional outcomes after SCI.[4] The current literature begins to answer these questions to inform clinical practice (Tab. 1), but many questions remain unanswered. When reviewing the literature, each of these elements should be identified, as should an understanding of the critical components (active ingredients) of the therapy to which its success is attributed. Future studies are needed to address these questions and to continue to provide evidence for the parameters and progression for specific training protocols,[26,33] hybrid therapies,[75–77] and augmented therapies.

As shown in Table 1, the majority of work has focused on people with incomplete SCI. Although incomplete SCIs accounted for 55.3% of the total number of annual SCIs in 2004,[78] clinicians have not developed clinical decision-making guidelines for this population as they have for people with complete spinal injuries.[4] When motor and sensory function are evident, the capacity for recovery for walking, upper-extremity function, and bowel and bladder or other biological system functions is likely significant.[79] This population may benefit immensely from a model of recovery for locomotion[79] or for other system functions.

As described in Table 1, outcomes for people with incomplete SCI, ASIA C and D, UMN lesions vary following training with BWS and a treadmill. Current evidence indicates that people after SCI with sparing of some voluntary control of isolated leg movements (ASIA C and D) likely benefit more from locomotor training for improved walking ability[73,74] than people lacking voluntary control (ASIA A and B). Improvements in gait speed varied widely. Additionally, individuals achieved the ability to walk more independently (less assistive device use or removal of braces), increased endurance, transitioned from being a limited household ambulator to a full-time ambulator, and made changes in muscle activation patterns and coordination. Many outcomes are presented and vary from physiological measures such as strength and endurance to mechanistic measures such

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

as EMG quantification and H-reflex analysis, but walking speed was by far the most common outcome assessed and is reflected in a separate column in Table 1. Although gait speed correlates with functional ability in people with stroke,[80] and undoubtedly has great implications for measuring physical performance in people with SCI as well, results of changes in gait speed should be reviewed critically within the context of clinically meaningful change. In particular, percentage change scores for people whose gait speeds fall far below normal values (0.8–1.2 m/s)[81] may not reflect a functional gain or increase in walking capacity. At this time, the standard error of measurement has yet to be determined for people with incomplete SCIs and severe ambulation deficits, and minimal changes expressed in high percentages may fall below a minimal detectable change necessary to describe meaningful improvement.[82] Alternatively, walking speed may not be the most appropriate outcome measure for people who are highly impaired, and other measures of functional performance also should be used in addition to walking speed.

Variability in outcomes may reflect protocol differences, intensity and duration of training, and the heterogeneity within the population of people with incomplete SCIs. Heterogeneity is associated with direct consequences of the injury (ie, severity and location of injury, age at time of injury, time since injury, and presence of interactive medications) and personal factors (ie, premorbid history, personal motivation, and family support). Being able to better characterize people with SCI beyond the ASIA classification system and to thus categorize the population according to neurophysiological, lesion, and neural control measures[73,74,83] may assist in identifying who specifically benefits from an intervention and thus advance clinical decision making. The severity and specific constellation of deficits that contribute to gait disability (stepping, balance, and adaptability) may be important for evaluation and treatment planning. Identifying the mechanisms accounting for benefit are critical needs for future research and for developing "best practice" guidelines. The theoretical basis for LT is readily applied to people with intact lumbosacral sensorimotor neural circuits, as in people with UMN SCI lesions. Application of LT for recovery of walking for people with mixed injuries or lower motor neuron injuries should be studied specifically, although the theoretical basis for benefit is likely different than for UMN lesions. The majority of studies have been conducted following inpatient and outpatient rehabilitation in people from 1 month up to 18 years after SCI. Dobkin et al,[29] Wernig et al,[31] and Nymark et al,[84] however, tested LT during inpatient rehabilitation and extending into outpatient rehabilitation.

Inherent in these evaluative criteria is the examination of the state-of the-evidence for continuity of the critical training components. For example, in examining LT relative to these criteria, it is essential to identify the specific aspects of the training that define the intervention in comparison with other interventions or training protocols. Table 2 highlights some of these components and the variability that is seen in the literature describing training programs incorporating BWS and treadmill equipment. Consumers of this literature are encouraged to assess it examining the critical training components and the scientific basis provided for selection of the training components. It may be more important to identify the therapeutic goal (ie, recovery or restitution of walking, endurance training) and its guidelines, decision making, and progression first, and then to select the equipment and decide how it is used consistent with the therapeutic paradigm to achieve the goal. Although the BWS and treadmill are current tools to optimize delivery of this intervention, other equipment or devices may yet offer alternative modes for delivery.[85] In addition, application of physiologically based training guidelines are not limited to training on a treadmill, but have been extended to the overground environment.[26,27,31,53]

A review of published studies requires an identification of the specific training that is used. All training protocols using a BWS system or treadmill are not alike simply because of the common equipment, and comparisons among studies therefore are often difficult. Furthermore, differences exist among BWS systems that may be critical to training benefit.[86,87] Inquiry relative to the role of the BWS system itself is needed because its use has become more prevalent in clinical practice. Guidelines for progression during training also vary among the studies and may include simple directions to increase walking speed and decrease BWS, predesignated rules for altering walking speed and BWS, or more complex algorithms of decision making.[26] As Hidler[33] and Field-Fote et al[34] contended, there is little consistency among training protocols. More importantly, many questions remain to be answered in order to define the optimal training for people after SCI.

In summary, the basic premise guiding current clinical practice is that recovery is not expected after SCI. Consequently, clinical decision making for rehabilitation of patients after SCI is founded on a model of compensation. However, neuroscientists are providing new therapeutic intervention strategies based on the neurobiological control of walking and physiologically based activity-dependent plasticity. Essential elements for LT have been proposed from experimental evidence, and translation to the human condition continues to be examined. Comparable training strategies that remediate disability, in lieu of compensating for impairments,

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

may promote recovery of function for other biological systems after SCI.

Recommendations for Advancement of Paradigm Shift

The translation of scientific evidence into clinical practice is challenging,[88] and advancing a paradigm shift requires overcoming many obstacles. The shift means a change in expectation, realized by a change in recovery in people with SCI after injury and after training. Clinicians and neuroscientists partnered in this effort as one community provide multiple perspectives and insights into problem-solving recovery and rehabilitation after SCI. This partnership has led and will continue to lead to more meaningful scientific inquiry and the more rapid infusion of evidence into clinical practice.

In order to promote advancement of this translation, it is critically important to take advantage of current innovative approaches to research partnerships and to disseminate findings.[88] One current opportunity includes responding to a new request for calls (June 2005 and 2006) for applications from the Christopher Reeve Foundation to develop specialized centers to join the NeuroRecovery Network to "apply advances from basic science and applied research for intensive activity-based rehabilitation treatments" assessing outcomes and cost-benefit.[89] A second opportunity involves responding to a National Institutes of Health request for applications ("Research Partnerships for Improving Functional Outcomes, PAR-04-077")[90] encouraging basic, applied, and translational research directed toward improving the health of people with acute or chronic diseases who may benefit from rehabilitation. Additionally, we would suggest new directions, including: (1) updating current physical therapy curricula to include teaching the basis and evidence for a paradigm shift in recovery and rehabilitation after SCI and its implications for clinical practice, (2) refocusing the National Institute on Disability and Rehabilitation Research model SCI Centers as a network for implementation and assessment of new therapies partnering neuroscientists and doctorally trained therapists with each clinic, (3) developing a Paralyzed Veterans of America clinical practice guidelines specific to locomotor rehabilitation after SCI, (4) developing a report on the state of locomotor rehabilitation with researchers and clinicians through the National Institutes of Health to identify specific research needs for future requests for applications (with III STEP proceedings serving as an initial step), (5) incorporating dissemination strategies into grant funding mechanisms to train therapist teams at clinics and provide support for translation into practice, (6) establishing dissemination of research as a priority of the Foundation for Physical Therapy,[88] and (7) including consumers (eg, Working 2 Walk[91]) throughout each of these processes.

This paradigm shift requires that new generations of therapists enter the clinic with the perspective that people with SCI recover and that physical rehabilitation is an agent for recovery. Today's therapists will need to come face-to-face with possibilities for recovery that challenge their current practice and its assumptions, and they will need to learn a new perspective that will change how they think and how they practice. This is not an easy task, but partnerships between clinicians and neuroscientists (and other scientists) may more effectively garner a new era for rehabilitation and greater recovery after SCI. Advances in SCI medical care and physical rehabilitation that actually change how we practice and alter the course of outcomes following SCI have been few. When such advances have occurred, though, they have been meaningful. The advent of antibiotics, spine stabilization by emergency medical technicians at the scene of an accident, external stabilization devices, the modular and custom-fit wheelchair, and methylprednisolone are such advances that have improved the care and rehabilitation of people after SCI. We propose that intense activity-based therapies, such as LT based on the afferent experience of walking and understanding of the neurobiological control of walking, are the basis for an emerging paradigm shift advancing rehabilitation and recovery after SCI. Questions remain, however, and the opportunity to harness this newfound potential for recovery of locomotion and perhaps other biological functions after SCI is upon us. Behavioral therapies alone will not produce full recovery, but they may play an important role in enhancing recovery potential. As regenerative neuroscience advances,[92,93] physical therapy interventions will be paired, as complementary agents for recovery, with plasticity-enhancing neurophysiologic agents to optimize recovery from neurologic injury and disease.

Summary

Advances in neuroscience are unlocking the mysteries of recovery after SCI. Although all of the pieces of the puzzle are not in place, physical therapists should shift their paradigm of rehabilitation from compensation to recovery. Clinicians examining people after SCI for walking capacity may in the future add an evaluation of the neural control mechanisms of walking (eg, reciprocal stepping, balance, and adaptability) to current classification of voluntary movement by ASIA impairment paired with manual muscle testing. New BWS and treadmill systems may provide an alternative and permissive environment for training and allow clinicians to differentiate mechanisms of control. The partnership among clinicians, physical therapist researchers, neuroscientists, and consumers will allow patients to benefit from current applications of science during rehabilitation and provide feedback from clinicians to the neuroscientists modeling SCI and recovery.

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

Table 1.
Review of Intervention Studies Incorporating Body-Weight Support and a Treadmill for the Goal of Walking Recovery After Incomplete Spinal Cord Injury[a]

Article	n	Population	Time Since Injury	Walking Speed	Other Results	Sackett Level[b]
LT studies (incorporating BWS and TM)—acute						
Wernig et al,[31] 1995	45	ASIA C, D	2–20 wk	OG walking speed not reported	36/45 acute w/c bound; at end, 33 walked at least 200 m	IV, nonrandomized historical cohort
Nymark et al,[84] 1998	1 1 1 1	Frankel C—C2 Frankel D—T9 Frankel C—C5 Frankel C—T5	1–2 mo	OG walking speed not reported	Strength, endurance, and %BWS improved in 3/4 Improvements in gait speed and spatiotemporal parameters on TM	V, no control group
Dobkin et al,[29] 2006	30 25	ASIA C ASIA D	<8 wk	Posttraining speed=1.1±0.6 m/s	Posttraining walking speeds not significantly different for LT group and control group for those with UMN ASIA C or D injuries 92% of those with ASIA C or D injuries walked independently	I
LT studies (incorporating BWS and TM)—chronic						
Wernig and Muller,[53] 1992	8	iSCI, 5/8 had "complete functional paralysis"	5–20 mo postinjury	Speed improved an average of 0.12 m/s	Subjects who initially were nonambulatory walked 32–61 m (105–200 ft), average speed=0.15 m/s Average speed=0.22 m/s	V, no control group
Wernig et al,[31] 1995	44	ASIA C, D	6 mo–18 y	OG walking speed not reported	33/44 chronic w/c-bound; at end of therapy, 25 walked independently, 7 walked with help Other 11 had improved speed and endurance 6 capable of staircase walking before therapy compared with 34 afterward	III
Dietz et al,[c] 1995	7 5	Incomplete paraplegia (2 with LMN injuries) Complete paraplegia	49–323 d posttrauma	OG walking speed not reported	EMG modulated similarly to subjects without neurological impairment, except less "dynamic" Gastrocnemius muscle EMG amplitude lower in subjects with SCI, but increased with LT, and inappropriate TA decreased Clonidine reduced and epinephrine enhanced EMG No effects of training seen in subjects with low tone (cauda equina)	V, no control group
Behrman and Harkema,[27] 2000	1 1 1 1	ASIA A—T5 ASIA C—T5 ASIA D—C6 ASIA D—T9	Mean postinjury time=6 mo	1) 0–0.53 m/s 2) 0.09–0.33 m/s 3) 0.6–1.6 m/s	One subject achieved OG walking, 2 improved All improved stepping on TM In subject 3, distance increased from 12 to 68 m, Berg Balance Test scores=30–43	V, no control group
Protas et al,[d] 2001	1 1 1	ASIA D—T12 ASIA D—T10 ASIA C—T8	2–13 y	Speed increased from 0.12 to 0.32 m/s	Endurance: 20.3 m/5 min to 63.5 m Oxygen costs decreased from 1.96 to 1.33 mL/kgm Mild increases in MMT strength	V, no control group

(Continued)

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

Table 1.
Continued

Article	n	Population	Time Since Injury	Walking Speed	Other Results	Sackett Level[b]
LT studies (incorporating BWS and TM)—acute						
Trimble et al,[e] 1998	1	T9 sensory, T2/S1 motor	1 y	OG walking speed not reported	Maximum H/M ratio higher than in controls, unchanged by training LFD lower than in controls, but improved with training	NA, only case series are rated
Gardner et al,[f] 1998	1	iSCI (community ambulator)	7 mo	Self-selected speed increased from 1.22 to 1.36 mph	FVC and running speeds also increased Stride length increased Resting HR decreased Main improvements seen in running performance	NA, only case series are rated
Wirz et al,[g] 2001	16 2 14	ASIA A or B ASIA C ASIA D (includes LMN)	1 mo–1 y	OG walking speed not reported	LE extensor EMG increased in all subjects 3 y posttraining, EMG constant for subjects with iSCI, decreased for subjects with complete SCI	V, no control group
Behrman et al,[26] 2005	1	ASIA D	5 mo	Walking speed improved from 0.19 to 1.01 m/s	Walking activity/24 hr increased from 1,054±543 steps to 3,924±1,629 steps Improved from home ambulator using a rolling walker and right AFO to a full-time ambulator using a cane only for community mobility	NA, only case series are rated
Effing et al,[h] 2006	2 1	ASIA C ASIA D	>48 mo	Actual speed not reported; 2/3 had significant increases	1/3 showed increase in QoL 1/3 improved in ADL 1/3 improved OG walking ability	V, no control group
Hicks et al,[i] 2005	14	ASIA B, C	Mean=7.4 y postinjury	OG walking speed not reported	54% decrease in BWS on TM 180% increase in TM walking speed 335% increase in distance/session Increased satisfaction with life correlated with TM walking ability 6/14 improved OG walking	V, no control group

(Continued)

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

Table 1.
Continued

Article	n	Population	Time Since Injury	Walking Speed	Other Results	Sackett Level[b]
Hybrid (combine LT using BWS with FES)						
Field-Fote,[75] 2001	19	iSCI (ASIA C)	≥1 y	OG: 0.12–0.21 m/s TM: 0.23–0.49 m/s	TM walking distance from 93 to 243 m and the LE motor score improved in the FES leg from 8 to 11 and in the nonassisted leg from 15 to 18	V, no control group
Field-Fote and Tepavac,[76] 2002	14	iSCI	1–16 y	TM: 0.22–0.44 m/s OG: 0.10–0.18 m/s	Improved intralimb coordination	IV, subjects acted as their own control group
Postans et al,[77] 2004	14	iSCI (ASIA C, D)	1–6 mo	TM: ~0.16-m/s improvement	Increased distance (by~300 m), OG walking speed/endurance Decrease in %BWS (by ~19%)	V, no control group
Nymark et al,[84] 1998	1	Frankel C—T10	1–2 mo	OG walking speed not reported	Strength, endurance, and %BWS improved Improvements in gait speed and spatiotemporal parameters on TM	NA, only case series are rated

[a] ADL=activities of daily living, AFO=ankle-foot orthosis, ASIA=American Spinal Injury Association classification of injury, BWS=body-weight support, EMG=electromyographic activity, FES=functional electrical stimulation, FVC=forced vital capacity, H/M ratio=H-reflex to maximum M-wave ratio, HR=heart rate, iSCI=incomplete spinal cord injury, LE=lower extremity, LFD=low-frequency depression, LMN=lower motor neuron, LT=locomotor training, MMT=manual muscle test, NA=not applicable, OG=overground, QoL=quality of life, SCI=spinal cord injury, TA=tibialis anterior muscle, TM=treadmill, UMN=upper motor neuron, w/c=wheelchair.

[b] Definitions of Sackett levels[72]: level I—large randomized controlled trial with low false-positive or false-negative errors (high power); level II—small randomized controlled trial with high false-positive or false-negative errors (low power), level III—nonrandomized, concurrent cohort comparisons between contemporaneous subjects who did and did not receive the intervention; level IV—nonrandomized, historical cohort comparisons between current subjects who received the intervention and former subjects who did not receive the intervention; and level V—case series without controls.

[c] Dietz V, Colombo G, Jensen L, Baumgartner L. Locomotor capacity of spinal cord in paraplegic patients. *Ann Neurol.* 1995;37:574–582.

[d] Protas EJ, Holmes SA, Qureshy H, et al. Supported treadmill ambulation training after spinal cord injury: a pilot study. *Arch Phys Med Rehabil.* 2001;82:825–831.

[e] Trimble MH, Kukulka CG, Behrman AL. The effect of treadmill gait training on low-frequency depression of the soleus H-reflex: comparison of a spinal cord injured man to normal subjects. *Neurosci Lett.* 1998;246:186–188.

[f] Gardner MB, Holden MK, Leikauskas JM, Richard RL. Partial body weight support with treadmill locomotion to improve gait after incomplete spinal cord injury: a single-subject experimental design. *Phys Ther.* 1998;78:361–374.

[g] Wirz M, Colombo G, Dietz V. Long-term effects of locomotor training in spinal humans. *J Neurol Neurosurg Psychiatry.* 2001;71:93–96.

[h] Effing TW, van Meeteren NL, van Asbeck FW, Prevo AJ. Body weight-supported treadmill training in chronic incomplete spinal cord injury: a pilot study evaluating functional health status and quality of life. *Spinal Cord.* 2006;44:287–296.

[i] Hicks AL, Adams MM, Martin Ginis K, et al. Long-term body-weight-supported treadmill training and subsequent follow-up in persons with chronic SCI: effects on functional walking ability and measures of subjective well-being. *Spinal Cord.* 2005;43:291–298.

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

Table 2.
Training Parameters for Intervention Studies Incorporating Body Weight Support and a Treadmill for the Goal of Walking Recovery After Incomplete Spinal Cord Injury[a]

Article	Initial BWS	Speed	Manual Assist	Arm Support	Overground Training	Orthotic Device Use	Intensity	Duration	Frequency
LT studies (incorporating BWS and TM)—acute									
Wernig et al,[31] 1995	40%	Self-selected speed	Legs only	Balance only	Added to conventional gait training as soon as possible	NA	30 min	2–22 wk	5×/wk
Nymark et al,[84] 1998 (4 subjects)	Up to 80%	0.15–0.6 m/s	Legs and trunk	Initially for balance, then removed	Added to conventional gait training	NA	60 min	12 wk	3×/wk
Dobkin et al,[29] 2006	High enough to achieve speed goals	At least 0.72 m/s, goal of 1.07 m/s	Legs and trunk	None	Same training goals as on TM	Removed during training	20–30 min with BWS and TM; 10–20 min overground	12 wk	5×/wk
LT studies (incorporating BWS and TM)—chronic									
Wernig and Muller,[53] 1992	40%	Self-selected speed	Legs only	Balance only	1 time/wk added to conventional gait training	NA	30 min	1.5–7 mo	5×/wk
Dietz et al,[b] 1995	Up to 80%	0.42 m/s	Legs only	NA	NA	NA	NA	12 wk	5×/wk
Wernig et al,[31] 1995	40%	Self-selected speed	Legs only	Balance only	Added to conventional gait training as soon as possible	NA	30 min	3–20 wk	5×/wk
Behrman and Harkema,[27] 2000	40%	0.75–1.25 m/s	Legs and trunk	None	Modified to match training principles	Removed during training	90 min	9 wk	5×/wk
Protas et al,[c] 2001	40%	0.04 m/s	Legs only	Balance only	NA	NA	60 min (20 min of stepping)	12 wk	5×/wk
Trimble et al,[d] 1998	NA	Matched overground fast walking	NA	NA	NA	NA	30 min	10 d 4 mo at gym	Every other day 3×/wk
Gardner et al,[e] 1998	32%	0.67–2.01 m/s	None	Balance only	NA	NA	20 min of TM stepping	6 wk	3×/wk
Wirz et al,[f] 2001	Up to 80%	0.42 m/s	Legs only	NA	NA	NA	15 min of stepping	27 wk (average)	5×/wk
Behrman et al,[26] 2005	40%	0.75–1.25 m/s	Legs and trunk	None	Modified to match training principles	Removed during training	90 min	9 wk	5×/wk

(Continued)

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

Table 2.
Continued

Article	Initial BWS	Speed	Manual Assist	Arm Support	Overground Training	Orthotic Device Use	Intensity	Duration	Frequency
LT studies (incorporating BWS and TM)—chronic									
Effing et al,[g] 2005	50%	0.03 m/s to self-selected speed	Legs only	Allowed initially	NA	NA	30 min	12 wk	5×/wk
Hicks et al,[h] 2005	60%+	0.17 m/s	NA	NA	NA	NA	Three 5- to 15-min walking bouts	Until 144 sessions were completed	3×/wk
Hybrid (combine LT using BWS with FES)									
Field-Fote,[75] 2001	30%	Fastest comfortable speed	FES to legs	NA	NA	NA	90 min	12 wk	3×/wk
Field-Fote and Tepavac,[76] 2002	NA	Fastest comfortable speed	FES to legs	Balance only	NA	NA	90 min	12 wk	3×/wk
Postans et al,[77] 2004	40%	Self-selected speed	Legs and FES	Balance only	NA	NA	25 min of walking	4 wk	5×/wk
Nymark et al,[84] 1998 (1 subject)	Up to 80%	0.15–0.6 m/s	Legs and trunk	Initially for balance, then removed	Added to conventional gait training	NA	60 min	12 wk	3×/wk

[a] BWS=body-weight support, FES=functional electrical stimulation, NA=not addressed in "Method" section of article, TM=treadmill.
[b] Dietz V, Colombo G, Jensen L, Baumgartner L. Locomotor capacity of spinal cord in paraplegic patients. *Ann Neurol.* 1995;37:574–582.
[c] Protas EJ, Holmes SA, Qureshy H, et al. Supported treadmill ambulation training after spinal cord injury: a pilot study. *Arch Phys Med Rehabil.* 2001;82:825–831.
[d] Trimble MH, Kukulka CG, Behrman AL. The effect of treadmill gait training on low-frequency depression of the soleus H-reflex: comparison of a spinal cord injured man to normal subjects. *Neurosci Lett.* 1998;246:186–188.
[e] Gardner MB, Holden MK, Leikauskas JM, Richard RL. Partial body weight support with treadmill locomotion to improve gait after incomplete spinal cord injury: a single-subject experimental design. *Phys Ther.* 1998;78:361–374.
[f] Wirz M, Colombo G, Dietz V. Long-term effects of locomotor training in spinal humans. *J Neurol Neurosurg Psychiatry.* 2001;71:93–96.
[g] Effing TW, van Meeteren NL, van Asbeck FW, Prevo AJ. Body weight-supported treadmill training in chronic incomplete spinal cord injury: a pilot study evaluating functional health status and quality of life. *Spinal Cord.* 2006;44:287–296.
[h] Hicks AL, Adams MM, Martin Ginis K, et al. Long-term body-weight-supported treadmill training and subsequent follow-up in persons with chronic SCI: effects on functional walking ability and measures of subjective well-being. *Spinal Cord.* 2005;43:291–298.

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

References

1 Kuhn TS. *The Structure of Scientific Revolutions*. Chicago, Ill: University of Chicago Press; 1970.

2 Ramón y Cajal S. *Degeneration and Regeneration of the Nervous System*. London, United Kingdom: Oxford University Press; 1928.

3 Guide to Physical Therapist Practice. 2nd ed. *Phys Ther.* 2001:81: 9–746.

4 *Outcomes Following Traumatic Spinal Cord Injury*. Washington, DC: Paralyzed Veterans of America; 1999. Consortium for Spinal Cord Medicine Clinical Practice Guidelines for Spinal Cord Injury.

5 Umphred DA. *Neurological Rehabilitation*. St Louis, Mo: Mosby Inc; 2001.

6 O'Sullivan SB, Schmitz TJ. *Physical Rehabilitation: Assessment and Treatment*. Philadelphia, Pa: FA Davis Co; 2000.

7 Somers M. *Spinal Cord Injury: Functional Rehabilitation*. London, United Kingdom: Prentice-Hall Inc; 2001.

8 Wolpaw JR, Tennissen AM. Activity-dependent spinal cord plasticity in health and disease. *Annu Rev Neurosci.* 2001;24:807–843.

9 Rossignol S, Chau C, Brustein E, et al. Locomotor capacities after complete and partial lesions of the spinal cord. *Acta Neurobiol Exp.* 1996;56:449–463.

10 Edgerton VR, Tillakaratne NJ, Bigbee AJ, et al. Plasticity of the spinal neural circuitry after injury. *Annu Rev Neurosci.* 2004;27:145–167.

11 Lovely RG, Gregor RJ, Roy RR, Edgerton VR. Effects of training on the recovery of full-weight-bearing stepping in the adult spinal cat. *Exp Neurol.* 1986;92:421–435.

12 Hodgson JA, Roy RR, de Leon R, et al. Can the mammalian lumbar spinal cord learn a motor task? *Med Sci Sports Exerc.* 1994; 26:1491–1497.

13 Van de Crommert HW, Mulder T, Duysens J. Neural control of locomotion: sensory control of the central pattern generator and its relation to treadmill training. *Gait Posture.* 1998;7:251–263.

14 Dietz V, Duysens J. Significance of load receptor input during locomotion: a review. *Gait Posture.* 2000;11:102–110.

15 Sherrington CS. Flexion reflex of the limb, crossed extension reflex, and reflex stepping and standing. *J Physiol.* 1910;40:28–121.

16 Grillner S, Rossignol S. On the initiation of the swing phase of locomotion in chronic spinal cats. *Brain Res.* 1978;146:269–277.

17 Hiebert GW, Whelan PJ, Prochazka A, Pearson KG. Contribution of hindlimb flexor muscle afferents to the timing of phase transitions in the cat step cycle. *J Neurophysiol.* 1996;75:1126–1137.

18 Calancie B, Needham-Shropshire B, Jacobs P, et al. Involuntary stepping after chronic spinal cord injury: evidence for a central rhythm generator for locomotion in man. *Brain.* 1994;117(pt 5):1143–1159.

19 Pang MY, Yang JF. The initiation of the swing phase in human infant stepping: importance of hip position and leg loading. *J Physiol.* 2000;528(pt 2):389–404.

20 Duysens J, Pearson KG. Inhibition of flexor burst generation by loading ankle extensor muscles in walking cats. *Brain Res.* 1980;187: 321–332.

21 Sinkjaer T, Andersen JB, Ladouceur M, et al. Major role for sensory feedback in soleus EMG activity in the stance phase of walking in man. *J Physiol.* 2000;523:817–827.

22 Harkema SJ, Hurley SL, Patel UK, et al. Human lumbosacral spinal cord interprets loading during stepping. *J Neurophysiol.* 1997;77: 797–811.

23 Dietz V, Muller R, Colombo G. Locomotor activity in spinal man: significance of afferent input from joint and load receptors. *Brain.* 2002;125:2626–2634.

24 Duysens J, Van de Crommert HW, Smits-Engelsman BC, Van der Helm FC. A walking robot called human: lessons to be learned from neural control of locomotion. *J Biomech.* 2002;35:447–453.

25 Zehr EP. Neural control of rhythmic human movement: the common core hypothesis. *Exerc Sport Sci Rev.* 2005;33:54–60.

26 Behrman AL, Lawless-Dixon AR, Davis SB, et al. Locomotor training progression and outcomes after incomplete spinal cord injury. *Phys Ther.* 2005;85:1356–1371.

27 Behrman AL, Harkema SJ. Locomotor training after human spinal cord injury: a series of case studies. *Phys Ther.* 2000;80:688–700.

28 Barbeau H. Locomotor training in neurorehabilitation: emerging rehabilitation concepts. *Neurorehabil Neural Repair.* 2003;17:3–11.

29 Dobkin BH, Apple D, Barbeau H, et al. Weight-supported treadmill vs over-ground training for walking after acute incomplete SCI. *Neurology.* 2006;66:484–493.

30 Barbeau H, Visintin M. Optimal outcomes obtained with body-weight support combined with treadmill training in stroke subjects. *Arch Phys Med Rehabil.* 2003;84:1458–1465.

31 Wernig A, Muller S, Nanassy A, Cagol E. Laufband therapy based on "rules of spinal locomotion" is effective in spinal cord injured persons [published erratum appears in *Eur J Neurosci.* 1995;7:1429]. *Eur J Neurosci.* 1995;7:823–829.

32 Beres-Jones JA, Harkema SJ. The human spinal cord interprets velocity-dependent afferent input during stepping. *Brain.* 2004;127: 2232–2246.

33 Hidler JM. What is next for locomotor-based studies? *J Rehabil Res Dev.* 2005;42:10–16.

34 Field-Fote EC, Lindley SD, Sherman AL. Locomotor training approaches for individuals with spinal cord injury: a preliminary report of walking-related outcomes. *J Neurol Phys Ther.* 2005;29:127–137.

35 Edgerton VR, Roy RR, Hodgson JA, et al. A physiological basis for the development of rehabilitative strategies for spinally injured patients. *J Am Paraplegia Soc.* 1991;14:150–157.

36 Dietz V, Harkema SJ. Locomotor activity in spinal cord-injured persons. *J Appl Physiol.* 2004;96:1954–1960.

37 Dobkin BH, Apple D, Barbeau H, et al. Methods for a randomized trial of weight-supported treadmill training versus conventional training for walking during inpatient rehabilitation after incomplete traumatic spinal cord injury. *Neurorehabil Neural Repair.* 2003;17:153–167.

38 Basso D, Behrman AL, Harkema SJ. Recovery of walking after central nervous system insult: basic research in the controlled locomotion as a foundation for developing rehabilitation strategies. *Neurology Report.* 2000;24:47–54.

39 Field-Fote EC. Spinal cord control of movement: implications for locomotor rehabilitation following spinal cord injury. *Phys Ther.* 2000; 80:477–484.

40 Edgerton VR, Leon RD, Harkema SJ, et al. Retraining the injured spinal cord. *J Physiol.* 2001;533:15–22.

41 Barbeau H, Nadeau S, Garneau C. Physical determinants, emerging concepts, and training approaches in gait of individuals with spinal cord injury. *J Neurotrauma.* 2006;23:571–585.

42 Visintin M, Barbeau H. The effects of parallel bars, body weight support and speed on the modulation of the locomotor pattern of spastic paretic gait: a preliminary communication. *Paraplegia.* 1994;32: 540–553.

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

43 Wang DC, Bose P, Parmer R, Thompson FJ. Chronic intrathecal baclofen treatment and withdrawal, I: changes in ankle torque and hind limb posture in normal rats. *J Neurotrauma.* 2002;19:875–886.

44 Maynard FM Jr, Bracken MB, Creasey G, et al; American Spinal Injury Association. International Standards for Neurological and Functional Classification of Spinal Cord Injury. *Spinal Cord.* 1997;35: 266–274.

45 Burns SP, Golding DG, Rolle WA Jr, et al. Recovery of ambulation in motor-incomplete tetraplegia. *Arch Phys Med Rehabil.* 1997;78: 1169–1172.

46 Waters RL, Yakura JS, Adkins RH, Sie I. Recovery following complete paraplegia. *Arch Phys Med Rehabil.* 1992;73:784–789.

47 Waters RL, Adkins RH, Yakura JS, Sie I. Motor and sensory recovery following complete tetraplegia. *Arch Phys Med Rehabil.* 1993;74: 242–247.

48 Crozier KS, Graziani V, Ditunno JF Jr, Herbison GJ. Spinal cord injury: prognosis for ambulation based on sensory examination in patients who are initially motor complete. *Arch Phys Med Rehabil.* 1991;72:119–121.

49 Crozier KS, Cheng LL, Graziani V, et al. Spinal cord injury: prognosis for ambulation based on quadriceps recovery. *Paraplegia.* 1992; 30:762–767.

50 Burns AS, Ditunno JF Jr. Establishing prognosis and maximizing functional outcomes after spinal cord injury: a review of current and future directions in rehabilitation management. *Spine.* 2001;26: S137–S145.

51 Waters RL, Adkins R, Yakura J, Vigil D. Prediction of ambulatory performance based on motor scores derived from standards of the American Spinal Injury Association. *Arch Phys Med Rehabil.* 1994;75: 756–760.

52 Craik RL. Recovery processes: maximizing function. In: Lister MJ, ed. *Contemporary Management of Motor Problems: Proceedings of the II STEP Conference.* Alexandria, Va: Foundation for Physical Therapy; 1991: 165–174.

53 Wernig A, Muller S. Laufband locomotion with body weight support improved walking in persons with severe spinal cord injuries. *Paraplegia.* 1992;30:229–238.

54 Behrman AL, Bowden MG, Herget B, et al. Locomotor training and recruitment of developmental neuroplasticity: insights from a pediatric SCI case study. Presented as an abstract at: 35th Annual Meeting of the Society for Neuroscience; November 12–16, 2005; Washington, DC.

55 Ladouceur M, Pepin A, Norman KE, Barbeau H. Recovery of walking after spinal cord injury. *Adv Neurol.* 1997;72:249–255.

56 Forssberg H. Spinal locomotor functions and descending control. In: Sjolund B, Bjorklund RA, eds. *Brainstem Control of Spinal Mechanisms.* Amsterdam, the Netherlands: Elsevier Biomedical; 1982:253–271.

57 Patla AE, Shumway-Cook A. Dimensions of mobility: defining the complexity and difficulty associated with community mobility. *Journal of Aging and Physical Activity.* 1999;7:7–19.

58 Shumway-Cook A, Patla AE, Stewart A, et al. Environmental demands associated with community mobility in older adults with and without mobility disabilities. *Phys Ther.* 2002;82:670–681.

59 Shumway-Cook A, Patla A, Stewart A, et al. Environmental components of mobility disability in community-living older persons. *J Am Geriatr Soc.* 2003;51:393–398.

60 Huxham FE, Goldie PA, Patla AE. Theoretical considerations in balance assessment. *Aust J Physiother.* 2001;47:89–100.

61 Horak FB, Macpherson JM. Postural orientation and equilibrium. In: Rowell LB, Shepherd JT, eds. *Handbook of Physiology and Exercise: Regulation and Integration of Multiple Systems.* Bethesda, Md: American Physiological Society; 1996:255–292.

62 Patla AE, Prentice SD, Robinson C, Neufeld J. Visual control of locomotion: strategies for changing direction and for going over obstacles. *J Exp Psychol Hum Percept Perform.* 1991;17:603–634.

63 Barbeau H, Wainberg M, Finch L. Description and application of a system for locomotor rehabilitation. *Med Biol Eng Comput.* 1987;25: 341–344.

64 Winstein CJ. Balance retraining: does it transfer? In: *Balance: Proceedings of the American Physical Therapy Association Forum, Nashville, Tennessee, June 13–15, 1989.* 1990:95–103.

65 Harburn KL, Hill KM, Kramer JF, et al. An overhead harness and trolly system for balance and ambulation assessment and training. *Arch Phys Med Rehabil.* 1993;74:220–223.

66 Norman KE, Pepin A, Ladouceur M, Barbeau H. A treadmill apparatus and harness support for evaluation and rehabilitation of gait. *Arch Phys Med Rehabil.* 1995;76:772–778.

67 Harris SR. How should treatments be critiqued for scientific merit? *Phys Ther.* 1996;76:175–181.

68 Barbeau H, Rossignol S. Enhancement of locomotor recovery following spinal cord injury. *Curr Opin Neurol.* 1994;7:517–524.

69 de Leon RD, Hodgson JA, Roy RR, Edgerton VR. Locomotor capacity attributable to step training versus spontaneous recovery after spinalization in adult cats. *J Neurophysiol.* 1998;79:1329–1340.

70 Wernig A, Nanassy A, Muller S. Laufband (treadmill) therapy in incomplete paraplegia and tetraplegia. *J Neurotrauma.* 1999;16: 7197–7126.

71 Field-Fote EC. Spinal cord stimulation facilitates functional walking in a chronic, incomplete spinal cord injured subject. *Spinal Cord.* 2002;40:428.

72 Megans A, Harris SR. Physical therapist management of lymphedema following treatment for breast cancer: a critical review of its effectiveness. *Phys Ther.* 1998;78:1302–1311.

73 Thomas SL, Gorassini MA. Increases in corticospinal tract function by treadmill training after incomplete spinal cord injury. *J Neurophysiol.* 2005;94:2844–2855.

74 Norton JA, Gorassini MA. Changes in cortically related intermuscular coherence accompanying improvements in locomotor skills in incomplete spinal cord injury. *J Neurophysiol.* 2006;95:2580–2589.

75 Field-Fote EC. Combined use of body weight support, functional electric stimulation, and treadmill training to improve walking ability in individuals with chronic incomplete spinal cord injury. *Arch Phys Med Rehabil.* 2001;82:818–824.

76 Field-Fote EC, Tepavac D. Improved intralimb coordination in people with incomplete spinal cord injury following training with body weight support and electrical stimulation. *Phys Ther.* 2002;82:707–715.

77 Postans NJ, Hasler JP, Granat MH, Maxwell DJ. Functional electric stimulation to augment partial weight-bearing supported treadmill training for patients with acute incomplete spinal cord injury: a pilot study. *Arch Phys Med Rehabil.* 2004;85:604–610.

78 *The 2004 Annual Statistical Report for the Model Spinal Cord Injury Care Systems.* Birmingham, Ala: National Spinal Cord Injury Statistical Center; 2004.

79 Raineteau O, Schwab ME. Plasticity of motor systems after incomplete spinal cord injury. *Nat Rev Neurosci.* 2001;2:263–273.

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

80 Perry J, Garrett M, Gronley JK, Mulroy SJ. Classification of walking handicap in the stroke population. *Stroke.* 1995;26:982–989.

81 Craik RL, Dutterer L. Spatial and temporal characteristics of foot fall patterns. In: Craik RL, Oatis CA, eds. *Gait Analysis: Theory and Application.* St Louis, Mo: Mosby-Year Book; 1995:143–158.

82 Stratford PW. Getting more from the literature: estimating the standard error of measurement from reliability studies. *Physiother Can.* 2004;56:27–30.

83 Curt A, Schwab ME, Dietz V. Providing the clinical basis for new interventional therapies: refined diagnosis and assessment of recovery after spinal cord injury. *Spinal Cord.* 2004;42:1–6.

84 Nymark J, Deforge D, Barbeau H, et al. Body weight support treadmill gait training in the subacute recovery phase of incomplete spinal cord injury. *J Neurol Rehabil.* 1998;12:119–138.

85 Hornby TG, Zemon DH, Campbell D. Robotic-assisted, body-weight-supported treadmill training in individuals following motor incomplete spinal cord injury. *Phys Ther.* 2005;85:52–66.

86 Gordon K, Ferris D, Roberton M, et al. The importance of using an appropriate body weight support system in locomotor training. *Society for Neuroscience Proceedings.* 2000;26:160.

87 Ratliff RA, Kent DM, Fuller SA, Ratliff RT. Physiological response comparison of upper and lower torso harnesses for body weight support during treadmill walking. *Med Sci Sports Exerc.* 1993;25:S38.

88 Jette AM. Editor's note: "Invention is hard, but dissemination is even harder." *Phys Ther.* 2005;85:390–391.

89 Christopher Reeve Foundation Web site. Available at: http://www.christopherreeve.org/. Accessed June 25, 2006.

90 Research Partnerships for Improving Functional Outcomes. Available at: http://grants.nih.gov/grants/guide/pa-files/PAR-04–077.html. Accessed June 25, 2006.

91 Working 2 Walk. Available at: http://www.working2walk.org/. Accessed June 25, 2006.

92 Anderson DK, Beattie M, Blesch A, et al. Recommended guidelines for studies of human subjects with spinal cord injury. *Spinal Cord.* 2005;43:453–458.

93 Reier PJ. Cellular transplantation strategies for spinal cord injury and translational neurobiology. *NeuroRx.* 2004;1:424–451.

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

Appendix.
Comparison of Compensation Model and Recovery Model for Rehabilitation of Walking After Incomplete Spinal Cord Injury[a]

	Compensation Model	Recovery Model
Assumption of capacity for repair and recovery after SCI	• Natural recovery: rate of recovery is greatest early after injury; after 6 months, expect fewer gains • After spinal injury, unable to repair, respond, or learn	• Capacity for recovery dependent on UMN lesion, activity-dependent experience, and understanding neurobiological control of walking • Unclear relative to LMN injuries
Who will benefit	• Prediction of walking recovery dependent on level of lesion, degree of voluntary motor control demonstrated by manual muscle test scores	• People with incomplete lesions demonstrate greater capacity for recovery • Unclear from ASIA scores or level of lesion who will benefit
Evaluation of potential for benefit or walking outcome	• Manual muscle test results and lesion level • Walking potential evaluated overground	• Walking capacity evaluated while in the constrained BWST environment, yet permissive for promoting upright posture and activating stepping
When will person benefit after SCI	• Rehabilitation services provided predominantly within first year of injury	• Acute: early evidence indicates improved ambulatory mobility; a recent RCT demonstrated a high degree of successful ambulation for LT intervention and control groups • Chronic: effect of locomotor training in people with chronic, motor incomplete SCI demonstrates benefit (Sackett levels IV–V)
Training environment	• Training is conducted overground • May use parallel bars for support or introduce assistive devices immediately	• Locomotor training occurs in 2 environments with guidelines from basic science incorporated into both environments: 1) BWS and treadmill with manual assistance as needed to provide appropriate sensory experience; retraining capacity primarily occurs in the BWST environment 2) overground, the ability to transfer skills acquired on the treadmill to overground is assessed, and instructions for community mobility/home practice are provided
Use of assistive devices	• Assistive devices are introduced early in gait training and compensate for UE and LE weakness and provide balance • Assistive device may alter gait pattern and gait kinematics (ie, forward flexed trunk) for walking • Assistive devices shift load-bearing capacity from the legs to the arms • Assistive devices may alter speed ability	• Assistive devices are introduced only in translation of skills to community ambulation • Assistive devices are not introduced immediately because primary training occurs in the BWST environment • The least-restrictive device or most-permissive device is selected • More than one device may be recommended; one device may be selected for limited and challenging practice, and another device may afford speed, a more upright posture, or better kinematics or safety within the home or community • The device may be adjusted to promote upright posture and limit UE load bearing • Alternative patterns for use of the device may be instructed to increase load bearing on the legs versus the arms
Use of braces	• Braces are selected to compensate for weakness, paralysis, or overactivity resulting in adverse limb positioning during stance or swing phases of walking • Braces (AFOs) are introduced early in the rehabilitation process • Braces, particularly at the ankle, eliminate toe drop, provide ankle stability, and eliminate the degrees of freedom at the ankle, allowing the individual the ability to relearn to walk by concentrating on hip and knee control	• Braces are not used while training in the BWST environment • Braces are not used while assessing the translation of skills from the treadmill to overground • Braces may be recommended if required for safety • A hinged AFO is recommended over a nonhinged posterior leaf brace • Braces may be used in the community; however, practice without is encouraged in a safe, home environment • Braces may alter the sensory experience critical to the recovery of walking

(Continued)

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024

Appendix.
Continued

	Compensation Model	Recovery Model
Speed of walking during training	• Walking training speed is limited by the capacity of the individual and the interactive effect of bracing and assistive device	• Walking training speed can be within normal walking limits • Manual assistance may be required at the trunk, pelvis, or legs to meet the kinematic demands at increased speeds • Walking speeds may be externally varied
Balance training for the task of walking	• Balance often is defined by the incorporation of an assistive device for support	• Balance is a corequisite of the task of walking • BWS assists in maintaining upright posture and development of balance of trunk over the base of support • Weight bearing through the arms is not used while training over the treadmill • Arm swing is encouraged as an important component of balance activity while walking on the treadmill and, if possible, overground • Assistive device height is adjusted and patterns of use are selected to limit UE weight bearing
Endurance training	• Endurance training may incorporate braces and assistive devices	• Endurance training begins on the treadmill with BWS and manual assistance to achieve 20 minutes of total stepping time as an intensity goal for training • Endurance training will persist in conjunction with changes in the requirements for BWS, speed, and manual assistance
Adaptability to the environment and behavioral demands of the individual	• Adaptability is trained using braces and assistive devices for negotiation of environmental obstacles • Demands of the home environment (eg, stairs, uneven terrain) are addressed	• Adaptability may be initiated on the treadmill after the capacity to step and balance (upright posture) have been adequately developed at a moderate to normal walking speed • Adaptations to stop/start, speed changes, and obstacles may be challenged on the treadmill • Transfer of adaptability may be practiced overground without assistive devices or with their introduction • Stair climbing may be introduced early as a mechanism requiring interlimb coordination

[a] AFO=ankle-foot orthosis, ASIA=American Spinal Injury Association classification of injury, BWS=body-weight support, BWST=body-weight–supported treadmill, LE=lower extremity, LMN=lower motor neuron, LT=locomotor training, RCT=randomized clinical trial, SCI=spinal cord injury, UE=upper extremity, UMN=upper motor neuron.

Downloaded from https://academic.oup.com/ptj/article/86/10/1406/2805283 by guest on 24 April 2024