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Neurophysiologic and Rehabilitation Insights From the Split-Belt and Other Locomotor Adaptation Paradigms

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Locomotion is incredibly flexible. Humans are able to stay upright and navigate long distances in the face of ever-changing environments and varied task demands, such as walking while carrying a heavy object or in thick mud. The focus of this review is a behavior that is critical for this flexibility: motor adaptation. *Adaptation* is defined here as the process of adjusting a movement to new demands through trial-and-error practice. A key feature of adaptation is that more practice without the new demand is required to return the movement to its original state. Thus, motor adaptation is a short-term motor learning process. Several studies have been undertaken to determine how humans adapt walking to novel circumstances. Many of these studies have examined locomotor adaptation using a split-belt treadmill. The results of these studies of people who were healthy and people with neurologic damage suggest that the cerebellum is required for normal adaptation of walking and that the role of cerebral structures may be less critical. They also suggest that intersegmental and interlimb coordination is critical but readily adaptable to accommodate changes in the environment. Locomotor adaptation also can be used to determine the walking potential of people with specific neurologic deficits. For instance, split-belt and limb-weighting locomotor adaptation studies show that adults with chronic stroke are capable of improving weight-bearing and spatiotemporal symmetry, at least temporarily. Our challenge as rehabilitation specialists is to intervene in ways that maximize this capacity.

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Humans must precisely coordinate the activities of many muscles to control everyday movements such as walking. This is done in the face of a constantly changing environment—we hold full coffee cups, walk on icy surfaces, and wear high heels (sometimes all at the same time!). Locomotor commands, therefore, must be constantly updated or recalibrated in order to correctly predict what is required in each situation. Despite this complex problem, humans walk with relative ease—our locomotor patterns are smooth and accurate and can even be graceful. What are the specific neural mechanisms that produce this flexibility? What happens when there is damage to the nervous system or the motor apparatus? Can this flexibility be utilized to promote recovery of movement after injury?

The focus of this review is a behavior that is critical for motor flexibility: motor adaptation. First, motor adaptation will be defined and compared with more-permanent motor skill learning. Second, central nervous system (CNS) mechanisms of mammalian locomotion will be reviewed briefly, as an introduction to mechanisms of locomotor adaptation in humans and animals. Next, recent human locomotor adaptation studies will be evaluated in terms of the response of specific gait parameters to adaptive stimuli and how this response informs mechanisms of locomotor control. Finally, clinical implications to walking rehabilitation will be drawn for a variety of neurologic conditions. The split-belt treadmill locomotor adaptation paradigm will be highlighted throughout the article because studies using this approach have revealed interesting and novel concepts about the relevance of motor adaptation to walking rehabilitation.

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Motor Adaptation and Its Relationship to Motor Skill Learning

Motor learning forms a foundation for rehabilitation interventions used to treat patients with neurological disease or injury. It is important to distinguish motor adaptation from motor learning. *Motor adaptation* is defined here, using the terminology of Martin et al,[1] as the process of modifying or adjusting an already well-learned movement or motor skill that occurs over a period of trial-and-error practice when exposing the movement to a novel, perturbing context or environment. Initially, the CNS does not correctly predict the new demands, and, as a result, significant movement errors occur. After minutes to hours of practice, the movements become more and more accurate as the CNS makes the necessary adjustments to the feedforward motor plan. Once the adaptation is complete, if the new demand is removed, movements once again are erroneous, this time in the opposite manner, because the CNS adjustments remain. These initial oppositely directed errors are termed *negative aftereffects*. The presence of negative aftereffects demonstrates that the adaptation has been stored by the CNS.[2,3] Thus, hallmarks of motor adaptation are that it is acquired relatively quickly, occurs only with motor practice, and requires active de-adaptation.[1,4] That is, further practice without the new demand is required to return the movement to its original state (Figure).

Using this definition, therefore, motor adaptation can be said to be one specific component of true motor skill learning. *Motor learning* has been defined as a set of processes associated with practice or experience leading to relatively permanent changes in skilled behavior.[5] Thus, to fully learn (ie, retain permanently) a novel motor skill requires much longer time periods and is influenced not only by adaptive mechanisms but also by offline learning, consolidation, and long-term storage.[6,7] Learning a novel motor skill also is affected by other non–motor processes such as attention, decision-making factors, and reward mechanisms.[8–10] Thus, there are many mechanisms for learning a new movement, but the one of interest here is tied to the motor adaptation process.

Why is adaptation important, given that it is a short-term learning process? First, locomotor adaptation allows for flexibility of walking patterns. Quickly learning and storing a new modification of the walking pattern is important to help walking remain relatively “automatic,” allowing attention to be focused on potentially more-important information. In many circumstances, the adapted pattern is needed only temporarily, so it is advantageous to be able to unlearn it fairly rapidly (ie, in minutes rather than days).

Second, adaptation allows for the study of long-term learning in a more controlled and timely way in the laboratory, providing a means to determine factors that drive adaptation and how damage to different brain regions affects this process. Assessment of motor adaptive capabilities may be useful for ascertaining whether people with certain types of brain damage have the capacity to generate a more normal motor pattern.[11,12]

Third, repeated adaptations may result in learning a more permanent motor pattern. For example, if subjects adapt and de-adapt certain movement patterns repeatedly over days or weeks, they can develop a new learned calibration for the context that initially drove adaptation.[1] That is, they no longer have to *adapt* from one behavior to the other but instead have 2 *learned* behaviors that they can switch between, without practice, immediately upon introduction of the different context. This concept is illustrated in the Figure. This method of learning may be most ideal for situations in which a person can already make a movement that approximates the new movement to be learned. Although the transition from adaptation to learning is not fully understood yet, it is thought to be an important process for motor learning and rehabilitation.

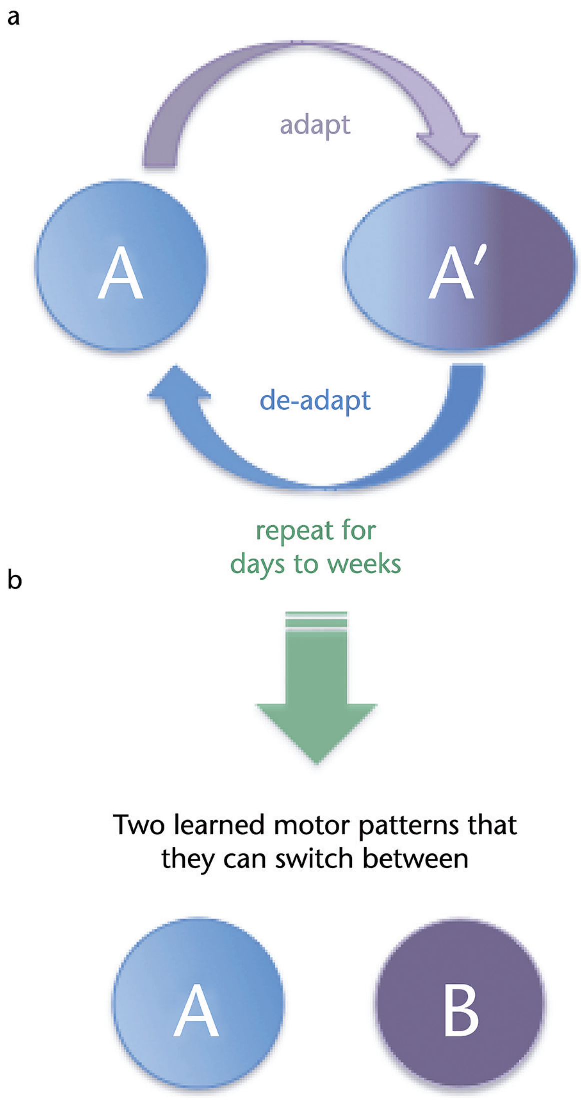

Figure.
(a) The process of motor adaptation. The typical walking pattern (A) is adapted through practice to accommodate a change in task demands (eg, walking on a split-belt treadmill with one leg moving twice as fast as the other), and this adaptation results in a modified pattern (A′). After the new demands are removed (eg, the belt speeds are returned to normal), the adapted pattern continues, and practice under the original task demands is required in order to return to the typical walking pattern (A). (b) The transition from short-term adaptation to longer-term learning. After days to weeks of practicing both the original pattern (A) and the adapted pattern (A′), people may be able to produce 2 patterns (A and B) that they can switch between, given the appropriate task demands.

CNS Regions Involved in Locomotion

Walking is a relatively unique motor behavior because, unlike other voluntary movements, spinal central pattern generators (CPGs) are thought to produce much of the basic locomotor synergy, at least in lower mammals. Central pattern generators have been demonstrated in cats[13–17] and other simpler vertebrates and, by definition, control movement without supraspinal influences or afferent feedback. When sensory information is available, spinal structures also provide some degree of flexibility to the basic locomotor pattern, even when separated from the brain.[18,19] Yet, in the intact animal, supraspinal structures, including the brain stem, cerebellum, and even the motor cortex, are thought to play a substantial role in locomotion.

The brain stem houses several locomotor regions (eg, mesencephalic locomotor region, subthalamic locomotor region), which when stimulated electrically or chemically, generate a variety of locomotor patterns in cats.[20] Brain-stem reticular and vestibular nuclei have projections to the spinal cord and assist with maintaining postural tone and facilitating the reciprocal pattern of flexor and extensor muscle activations required during walking.[21,22] The cerebellum also plays a role in locomotion, although its connections to spinal locomotor neurons are less direct.[23] Lesioning a portion or the entire cerebellum can result in profound locomotor impairments, ranging from the inability to control upright postural supporting reactions and maintaining dynamic balance during walking[24,25] to disrupted precision of limb placement and poor regulation of agonist-antagonist muscle pairs during stance.[26–28] In general, medial cerebellar regions are more involved in balance, muscle tone, and modulating the reciprocal patterns of leg muscle activation,[21,26] and the lateral regions are more involved in precision limb placement, particularly when visual guidance is required or when adjusting locomotor patterns in more-novel or more-complex environments.[25,29–30] The motor cortex also contributes to locomotor control, especially when adaptability and precision are required.[31–33] For example, cats with discrete lesions to the corticospinal and rubrospinal tracts but intact vestibulospinal and reticulospinal pathways have very few, if any, long-term gross deficits during simple treadmill walking.[34,35] However, if these same animals are made to place their legs more precisely during walking (eg, walking on rungs of a horizontal ladder) or to walk in a more challenging environment (eg, stepping over obstacles), clear deficits are easily apparent.[36–39]

Importantly, the role of supraspinal structures such as the cerebellum and motor cortex is presumed to be even more substantial in humans because compared with quadrupeds, bipeds have a smaller base of support, a higher center of mass, and only half the number of contact points with the ground, all of which makes human walking a much more complicated skill. Indeed, cerebellar or cerebral brain injury typically results in devastating locomotor impairments in humans.[40–46]

CNS Regions Involved in Locomotor Adaptation

From the previous section, it is clear that the spinal cord, brain stem, cerebellum, and cortex are all involved in human locomotor control to varying degrees. Yet, this does not mean that all are also involved in *adaptation* of locomotion. Converging evidence from the fields of neuroimaging, electrophysiology, and computational modeling and from behavioral studies in humans with lesions now strongly suggests the cerebellum is a critical structure for predictive feedforward adaptations of a wide variety of arm and eye movements.[2,47–52] It stands to reason, therefore, that the cerebellum also is involved in walking adaptation, although this has not been studied nearly as thoroughly.

One tool that has been used recently to test locomotor adaptation in humans and animals is the split-belt treadmill. With a split-belt treadmill, the speeds of the right and left legs can be controlled independently. When the legs are forced to walk at 2 different speeds, both rapid and longer-duration (adaptive) changes to the gait pattern take place in adult humans who are healthy.[53,54] In decerebrate cats, the firing rates of complex spikes in cerebellar Purkinje cells increase dramatically during the initial period of split-belt treadmill walking, suggesting that the cerebellum has at least some involvement in this form of adaptation.[55] In addition, nitric oxide deprivation, which is thought to prevent cerebellar long-term depression (a principal mechanism proposed for the cerebellar plasticity required for learning), abolishes the adaptive walking behavior altogether in these cats.[56] The few studies that have examined true motor adaptations of walking in humans with cerebellar damage seem to be in agreement with the physiological studies in cats. Individuals with cerebellar damage show significant impairments of acquisition and storage of novel walking adaptations.[11,57,58] Together, these data strongly suggest that the cerebellum is a necessary component for this type of locomotor adaptation.

Because decerebrate cats maintain the ability to walk on a split-belt treadmill,[59] it appears that cerebral structures are not essential for walking adaptations of this kind, at least in the cat. Recently, this question was addressed in a study of humans with chronic cerebral stroke and hemiparesis.[12] These individuals were found to adapt similarly to healthy controls, suggesting that unilateral cerebral damage does not affect the ability to acquire a novel locomotor adaptation, despite the presence of significant paresis and somatosensory loss. In contrast, a number of children who have had a hemispherectomy (surgical hemidecortication as treatment for intractable seizures, removing all cortical gray matter unilaterally and sometimes portions of the basal ganglia or thalamus but sparing underlying white matter) show partial disruption of the split-belt treadmill adaptation.[60] Interestingly, the adaptive deficits in this group appear to be specific to the temporal domain; adaptation of spatial walking parameters (eg, step length) is not impaired in children with hemispherectomy. Further studies are needed to determine exactly why adults with cerebral stroke show better adaptive capabilities than children with hemispherectomy, but it might relate to the relatively larger lesion size in the case of hemispherectomy.

Control of Specific Walking Parameters: Insights From Adaptation Studies

As previously discussed, the control of walking in humans is complex, involving both spinal and supraspinal structures. Furthermore, it must be flexible and adaptable to accommodate an ever-changing environment. Results from a variety of locomotor adaptation studies provide insight into this complex, yet flexible, control. In this section, we discuss what has been learned about the control of walking from human locomotor adaptation studies. Recall that according to the definition provided earlier, motor adaptation is characterized by an altered movement pattern that is generated within seconds to minutes following exposure to a novel perturbing context and results in negative aftereffects when the perturbation is removed. Many treadmill training studies have successfully produced walking pattern changes in patients with neurological deficits.[61,62] However, to induce these gait pattern changes (typically using treadmill speed changes or body-weight–support systems) requires training (ie, repeated exposure to the new condition occurring over many—often 10 to 12—sessions) and cannot be induced within minutes. Nor do these training sessions produce negative aftereffects. Thus, based on the criteria provided here for motor adaptation,[1] these studies provide information more in line with the concepts of motor learning than motor adaptation. Therefore, discussion of conventional treadmill training paradigms is not included here.

Humans are remarkably adept at maintaining appropriate spatial orientation and navigating a straight path during walking. Insight into how this is accomplished has been gained from a series of studies that examined locomotor adaptation during stepping on a circular treadmill. In these studies, participants adapted to stepping on a rotating circular disk for a brief period of time and then either walked, stepped, or hopped overground while blindfolded.[63–66] People who are healthy consistently walk or hop overground in a curved trajectory following circular treadmill stepping and are unaware of the curvature.[63,64] This negative aftereffect is called a "podokinetic" after-rotation (PKAR), and it is thought to be due to a recalibration of the proprioceptive relationship between the trunk and stance limb that occurs when stepping on the circular treadmill. Interestingly, the PKAR effect is observed when either forward or backward walking or hopping is undertaken following circular treadmill stepping[65,66] and appears to be influenced by vestibular input, despite the fact that individuals do not perceive moving in a curved trajectory.[67,68] Thus, information from studies of PKAR indicate that vestibular input and information about trunk rotation relative to the feet is integral to the control of locomotor trajectory and spatial orientation,[64–68] regardless of the direction (forward or backward) or form (walking or hopping) of locomotion. Notably, if participants are not blindfolded, there is no PKAR. This is evidence of the strong reliance on visual guidance humans have during walking and demonstrates our ability to override other conflicting sensory information when vision is available.

Strict coordination within a limb and between limbs is a requirement for normal human walking. A variety of locomotor adaptation studies have provided insight into the flexibility and control of intralimb and interlimb coordination during walking.[11,12,53,60,69–72] One group of studies has examined locomotor adaptation through weighting of a leg or applying resistance (through a motorized robotic device) to a leg during walking.[69–71] Robot-applied, velocity-dependent resistance against hip and knee movements of one leg during the swing phase of walking results in decreased hip and knee flexion when the resistance is initially applied. As people walk with the resistance, they gradually adapt by increasing flexor muscle activity, and hip and knee flexion values return to normal. When the resistance is removed, they continue to produce the adapted pattern of increased flexor activity, which then results in increased hip and knee flexion for approximately 20 steps after removal of the resistance (the negative aftereffect).[71]

Similar adaptations of hip and knee flexion are observed when a weight is applied to one leg during walking.[69] Application of a weight to one leg also results in adaptation of interlimb coordination. For example, the initial weighting causes a decrease in single-limb support time and step length on the weighted side and an increase in single-limb support time and step length on the unweighted side. Over a period of minutes walking with the weight, people gradually adapt such that single-limb support time and step length values return to their original values bilaterally. They continue to produce the adapted pattern when the weight is removed, resulting in increased single-limb support time and step length on the previously weighted side and vice versa on the previously unweighted side for a short period of walking after weight removal[69,70] (negative aftereffect). These studies illustrate that human locomotor intralimb and interlimb coordination is quite plastic and adaptable. Furthermore, both intralimb and interlimb coordination is adapted when a unilateral perturbation is applied during walking. This bilateral response to a unilateral perturbation supports the suggestion that there is a strong neural coupling between the legs during walking.[73–75]

Locomotor adaptations that occur when walking on a split-belt treadmill also provide insight into the control of intralimb and interlimb coordination during walking. When people walk on a split-belt treadmill, where each leg moves at a different speed, there are 2 types of changes that occur in the walking pattern. The first change in the walking pattern is an immediate reaction that is necessary to accommodate the differing belt speeds and results in the slower leg spending more time in stance and the faster leg spending less time in stance.[11,12,53,54] This reaction persists throughout split-belt walking and then immediately reverses when the belts are returned to normal treadmill conditions (ie, the belts tied at the same speed). Because there is no gradual adaptation of these parameters, nor are there any aftereffects, this is an example of a reactive, or feedback, type of adjustment.[11] The second change in the walking pattern that occurs during split-belt walking is adaptive and feedforward in nature. During split-belt walking, step length, double support time, and interlimb phasing are asymmetric initially, but people slowly adjust the coordination between their legs to reduce the initial asymmetry created by split belts. When the belts are returned to the same speed, they continue to produce this new adjusted pattern. In people who are healthy, these aftereffects result in step length, interlimb phasing, and double support asymmetries that are in the opposite direction from the asymmetries observed during early adaptation.[11,12,53] In contrast, there is essentially no change in intralimb joint timing during or after split-belt walking.[53] These results show that interlimb coordination (eg, step length) can be independently controlled and modified without necessarily altering many aspects of intralimb coordination (eg, stance time, intralimb joint timing), which suggests that the neural elements that control interlimb coordination are dissociable from those that control intralimb coordination during human bipedal walking.

This suggestion has been supported by the results of a study where individuals who were healthy walked on a split-belt treadmill in a *backward* configuration (with the belts moving backward) or in a *hybrid* configuration with the right belt moving forward while the left belt was moving backward.[72] The results from this study show that adaptation to forward and backward walking is independent in that there is no transfer between directions, nor any interference with one another. Thus, each leg can be adapted separately from its contralateral counterpart, and the effects from adaptation are stored individually for each leg. These findings support the suggestion that there are independently adaptable locomotor networks for each leg in humans. This type of specialized locomotor adaptability is functionally useful because it means the intact adult human locomotor system can learn new patterns without compromising other related patterns.[72]

In summary, studies of human locomotor adaptation in adults who are healthy illustrate that the control of walking is extremely flexible, allowing the system to readily adapt coordination between the limbs and trunk to accommodate changes in the environment. This flexibility is critical for the wide-ranging functional capacity of normal human locomotion.

Clinical Implications

The information gained from studying locomotor adaptation has a number of clinical implications. First, motor adaptation can help us begin to assess the motor learning capabilities of an individual. Both motor adaptation and motor learning require trial-and-error practice,[1,5] and motor adaptation may be an initial component in the process of motor learning.[4] Thus, studying motor adaptation allows us to begin to assess whether and to what extent the capacity for motor learning may be intact in an individual. This information is critical for appropriate planning in rehabilitation. For example, as discussed previously, chronic damage to the cerebellum appears to reduce adaptive walking capabilities in humans, but this does not seem to be the case for adults with cerebral damage due to stroke. Thus, it might be reasonable to expect some improvement in the walking pattern of an adult with cerebral stroke with an intervention that focuses on trial-and-error learning, yet this expectation may be less realistic for a person with chronic cerebellar damage or cerebellar degeneration.

Studying motor adaptation in people with neurologic damage also allows us to assess whether and to what extent the injured nervous system is capable of producing normal movement patterns.[4] For instance, the locomotor adaptation that occurs with split-belt walking results in asymmetric step length and double support time in humans who are healthy during the aftereffect period. However, in adults with unilateral stroke who show step length or double support asymmetry during unperturbed walking (as a consequence of their neurological injury), the aftereffects can result in improved symmetry.[12,76] This improvement occurs because the initial asymmetry is exaggerated during split-belt walking. The nervous system adjusts the interlimb coordination to correct for the exaggerated asymmetry, and when the belts are returned to the same speed, this corrected pattern persists and results in improved asymmetry compared with baseline. This result demonstrates that the compromised nervous system of an adult with stroke is still capable of producing a more-normal spatiotemporal walking pattern.

Regnaux and colleagues[77] weighted the nonparetic leg (2 kg for women, 4 kg for men) of adults with stroke during 20 minutes of treadmill walking. After removal of the load, weight bearing on the paretic leg was increased, leading to improved short-term weight-bearing symmetry during overground walking. This result demonstrates that despite nervous system damage, an adult with stroke is still capable of producing more-normal weight-bearing symmetry during walking. Together, these adaptation studies show that adults with stroke are capable, at least in the short-term, of walking with a more-normal pattern (in terms of spatiotemporal and weight-bearing symmetry). This is a critical finding, given that previous studies have shown that changes in coordination and symmetry during locomotor activities can be difficult to achieve in adults with stroke, even with training.[78–80]

Similarly, it has been shown that people with Parkinson disease (PD) retain the ability to adapt the locomotor trajectory in response to podokinetic stimulation.[81] Just as has been found in individuals who were neurologically intact, when people with PD step on a rotating treadmill for a period of time and then step overground while blindfolded, they consistently step with a trunk rotational velocity as would be observed when walking in a curved trajectory.[81] This adaptation study shows that adults with PD are capable, at least in the short-term, of producing trunk rotation required for turning during walking, which typically is impaired in people with PD. Future studies are needed to determine whether the rotating platform could be used in rehabilitation to remediate turning difficulties in people with PD.

Utilizing locomotor adaptation as an intervention has appeal because these paradigms result in short-term improvements in many of the most pervasive gait deficits observed in people with neurologic damage or disease.[12,70,76,77] For example, as discussed previously, a split-belt treadmill walking adaptation poststroke can lead to short-term improvements in step length and double support asymmetry.[12,76] Two critical questions must be addressed in order to better understand the direct utility of locomotor adaptation paradigms as rehabilitation interventions. First, do the effects observed during treadmill walking or rotating platform stepping transfer to overground walking? Second, can the short-term improvements, through repeated practice of the adapted pattern, produce long-term changes in the walking pattern of those with gait deficits? There is evidence to support the hypothesis that short-term improvements in the walking pattern of adults with stroke following locomotor adaptation are not just observed on the treadmill, but transfer to overground walking.[76,77] Whether the short-term changes observed can result in long-term improvements is an open question. Studies investigating long-term changes in spatiotemporal asymmetry following multiple sessions of split-belt treadmill training are under way in adults with cerebral damage due to stroke and children who have undergone a hemispherectomy.

An important concept when considering the use of a locomotor adaptation paradigm as a rehabilitation intervention for people with gait deficits is the direction the perturbation is applied during adaptation. The studies in stroke described above have all shown that adapting walking so that the movement deficits (asymmetry) are initially worsened is what leads the adaptation to result in aftereffects that improve symmetry. That is, the nervous system tries to correct the exaggerated asymmetry, and this correction results in aftereffects of improved symmetry.[4]

Selecting the correct perturbation direction is critical, although perhaps not intuitive. For example, an adult with stroke who has a baseline asymmetry of longer step length on the paretic side compared with the nonparetic side would need to train on the split-belt treadmill such that this asymmetry is initially exaggerated during the early adaptation period (that is, the paretic leg should be placed on the slower belt because the leg on the slower belt will initially produce a longer step length in response to the asymmetric belt speeds). Over the period of adaptation, the initially exaggerated asymmetry will be restored to near-baseline levels, therefore, the negative aftereffect will temporarily establish a new symmetric walking pattern. If the same person with stroke is trained with the paretic leg on the faster belt during the split-belt period, his or her asymmetry will be temporarily worsened following the adaptation.[12] Similarly, in a limb loading paradigm, the paretic leg of the adult with stroke must be weighted during adaptation in order to observe increased single-limb support time on the paretic leg after the weight is removed.[70] This is an interesting concept for rehabilitation: movement error enhancement may cue the nervous system to attempt to make a movement correction. This could be particularly important for people with chronic gait deviations, where the deviation may no longer be perceived by the nervous system as a movement error that requires correction.

Currently, there are few studies of locomotor adaptation in humans.

However, these studies have provided critical information about the walking capacity of many types of patients frequently seen in rehabilitation. Specifically, adaptation studies have revealed that many people with neurological impairments appear to retain the capacity to produce a more-normal walking pattern. Our challenge as rehabilitation specialists is to intervene in ways that maximize this capacity.

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