Hemiparetic Gait

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KEYWORDS

• Stroke rehabilitation • Gait • Hemiparesis

KEY POINTS

- The most common pattern of walking impairment poststroke is hemiparetic gait.
- Hemiparetic gait is characterized by asymmetry associated with an extensor synergy pattern of hip extension and adduction, knee extension, and ankle plantar flexion and inversion.
- There are characteristic changes in the spatiotemporal, kinematic and kinetic parameters, and dynamic electromyography (EMG) patterns in hemiparesis, which may be assessed most accurately in a motion studies laboratory.
- Increased energy cost of walking is due to the muscle work necessary to lift the body's center of mass (CM) against gravity during the paretic limb swing phase.
- An understanding of normal human gait is necessary to assess the complex interplay of motor, sensory, and proprioceptive loss; spasticity; and/or ataxia on hemiparetic gait.

INTRODUCTION

Seventy-five percent of patients who sustain a stroke have limitations in walking,[1] and the most common pattern of walking impairment poststroke is hemiparetic gait. This review provides a comprehensive overview of the characteristic features and analysis of poststroke hemiparetic gait. An understanding of normal human gait, including basic concepts of the neural control of gait and gait cycle terminology, is a prerequisite for analyzing and treating poststroke patients with hemiparetic gait dysfunction. This article also defines spatiotemporal and kinematic parameters of normal gait as well as key changes in these same parameters in hemiparesis. Although patients with

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poststroke hemiparesis may demonstrate classic changes in spatiotemporal, kinematic, and kinetic parameters, they also demonstrate greater gait variability than neurologically intact controls.[2] More advanced assessments of gait, including kinematic, kinetic, and dynamic electromyography (EMG) analyses, can also be done using quantitative gait analysis. The energy cost of normal walking and the increase in energy costs associated with maintaining walking velocity in poststroke hemiparesis are described. Lastly, the authors assert that good clinical care of patients for poststroke hemiparetic gait does not always require the sophisticated analysis provided by a gait laboratory but instead an astute clinician's understanding of the complex interplay of motor strength deficits, variable spasticity, sensory and proprioceptive loss, and/or ataxia on motor control and functional gait performance. Thus, a rubric for the clinician to describe hemiparetic gait in the clinical setting is presented.

NEURAL CONTROL OF NORMAL HUMAN GAIT

The neural control of human gait is dependent on both spinal and supraspinal mechanisms; thus, neurologically based gait impairment results from an interruption at either level. The concept of a locomotor center located at the level of the spinal cord was originally proposed in 1911 by T.G. Brown.[3,4] In his research, Brown noted that cats with transected spinal cords and cut dorsal roots were still capable of producing rhythmic alternating contractions in the ankle flexors and extensors. One century later, the term, *central pattern generator (CPG)*, is now commonly used to describe a spinal locomotor center. CPGs, a term first coined and described in invertebrates and fish, are innate neural networks that are capable of generating self-sustained, rhythmic patterned output, independent of sensory input.[5] Although only indirect evidence exists for human pattern generation, it is generally believed that CPGs, existing within the low thoracic and lumbar regions of the human spinal cord, generate rhythmic patterned outputs largely responsible for control of human locomotion. CPGs are capable of responding to multiple afferent inputs, which modulate human gait patterns, and include a complex interplay of supraspinal afferents originating from the brainstem reticular formation, basal ganglia, premotor and motor cortex, and cerebellum as well as sensory afferents from vestibular, visual, and proprioceptive systems. Information regarding limb positioning and kinesthesia is relayed from proprioceptors within tendons, muscles, ligaments, and joints via the medial leminiscus within the dorsal column of the spinal cord. The convergence of spinal reflex pathways and descending pathways on common spinal interneurons are known to be integrative.[6] In human and other primate gait patterns, however, it is believed that supraspinal input may play a greater role in the regulation of gait than in other species, due to the importance and prominence of the corticospinal tract.[7,8] Supraspinal afferents specifically play a prominent role in the volitional control of gait, such as occurs with change in walking speed, walking direction, or obstacle avoidance. In humans, damage within the central nervous system, secondary to a cerebrovascular accident, ultimately results in an alteration of the normal neural control of gait.

NORMAL HUMAN WALKING

Normal human walking is characterized by a repetitive sequence of limb motion whereby an ipsilateral lower limb alternately provides support for the contralateral lower limb as the body advances forward. Optimal human walking results in the forward translation of the body, using bipedal support, with maximal symmetry, stability, and energy efficiency. Although there is no specific standard for measurement of poststroke gait symmetry, Patterson and colleagues[9] recommend using a symmetry ratio