www.ThePharmaJournal.com

The Pharma Innovation

ISSN: 2277- 7695
TPI 2014; 3(8): 36-41

www.thepharmajournal.com
Received: 03-09-2014
Accepted: 16-09-2014

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Post-Stroke Hemiplegic Gait: A Review

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Abstract
Stroke is one of the leading causes of mortality and morbidity worldwide. After a stroke, the ability to control balance in the sitting and standing positions is a fundamental skill of motor behavior for achieving autonomy in everyday activities. Initial walking function is impaired in two-thirds of the stroke population and this impairment is the greatest contributor to functional disability after stroke. Post-Stroke Hemiplegic gait is typical pattern of walking. The gait is a spastic gait affecting one leg; the ipsilateral arm may be held in a decorticate posture with hand near chest. It occurs after a contralateral hemispheric stroke and other corticospinal lesion. It possesses a complex anatomical picture to understand. This paper aims at reviewing post-stroke hemiplegic gait in detail and to look at the anatomical involvement, etiology, kinematics of hemiplegic gait and also to review available treatment modalities in this modern era.

Keywords: Stroke; Hemiplegic gait; Spastic gait; Disability

1. Introduction
Stroke is the most commonest clinical manifestation of diseases of the cerebral blood vessels [1]. It is a syndrome characterized by the acute onset of a neurologic deficit that persists for at least 24 hours, which reflects focal involvement of the central nervous system, and is due to a disturbance of the cerebral circulation [2]. Acute stroke is characterized by the rapid appearance (generally over minutes) of a focal deficit of brain function, the result is most commonly a hemiplegia with or without signs of focal higher cerebral dysfunction (such as aphasia), hemisensory loss, brain-stem deficit or visual field defect [1].
Stroke is one of the leading causes of mortality and morbidity worldwide. Approximately 20 million people suffer from stroke each year and of these 5 million do not survive. Developing countries account for 85% of global deaths from stroke. Stroke is also a leading cause of functional impairments, with around 20% of survivors requiring institutional care after 3 months and on an average 15% - 30% being permanently disabled. According to the American Stroke Association (ASA), approximately 700,000 individuals are diagnosed with a stroke each year, of these individuals, who survive, up to 90% of them report one or more disabilities [3]. The incidence rises steeply with senesce, and in lots developing countries, the incidence is rising owing to the adoption of less healthy lifestyles [1].
Depending on its location, stroke can cause many perm disorders, suchlike paralysis on one side of the body and loss of speech. The clinical manifestations of stroke are highly variable because of the complex anatomy of the brain and its vasculature [4]. The disease kills 15%–35% of its victims and causes serious disability in more adults who survive than any other medical disease. [5] Most strokes are ischemic, but approximately 15% of strokes are caused by subarachnoid or intracerebral hemorrhage [6].
After a stroke, the ability to control balance in the sitting and standing positions is a fundamental skill of motor deportment for reaching autonomy in everyday activities. The postural performance of patients soon following a stroke has been found to be closely correlated with long-term functional improvement [7]. Initial walking function is impaired in two-thirds of the stroke population and this impairment is the greatest contributor to post stroke functional disability [8].
In epidemiologic studies, gait disorders are systematically identified as a greatest risk factor for falls and injury [4].

2. The Normal Human Gait
Walking is highly controlled, coordinated and repetitive series of limb movements whose

function is to set ahead the body from place to place with minimal expenditure of energy, it is also defined as a translatory progression of the body, as a whole acquired by coordinated, rotatory movements of body segments [9, 10].

Walking is a product of three interrelated nervous system functions: locomotion, balance, and adaptation. Locomotion comprises the stereotyped patterns of muscle activation (synergies) of limbs and trunk that produce repetitive stepping. The nervous system must be able both, to initiate and arrest stepping and to alter stepping patterns for turns, different speeds, and different support surfaces. Balance or equilibrium synergies include a number of postural responses that enable an individual to arise and remain erect during standing and locomotion. Standing is an active process (static postural response) in which the sway of the body is kept within the limits of the base of support provided by the feet. Anticipatory postural responses are changes in postural muscle groups that precede voluntary movements made to offset disturbances in balance that would result from the voluntary movement. For example, paraspinal and leg muscle activation precedes voluntary movements of the arm in a standing person to protect the person's upright stability. Anticipatory postural responses are feed-forward responses. They anticipate the perturbation of balance caused by the voluntary action. Reactive postural responses are feedback responses. They protect against unexpected external perturbations and are triggered by sensory cues indicating that the body is beyond the limits of stability [11].

3. The Normal Gait Cycle

The normal gait cycle may be defined as the period between successive points at which the heel of the same foot strikes the ground. The stance phase, during which the foot is in contact with the ground, occupies 60 to 65 percent of the cycle. The swing phase begins when the right toes leave the ground. The fact is that, for 20 to 25 percent of the walking cycle, both feet are in contact with the ground (double limb support). In later life, when the steps shorten and the cadence (the rhythm and number of steps per minute) decreases, the proportion of double limb support increases. Surface electromyograms show an alternating pattern of movement in the legs, predominating in the extensors during the stance phase and in the flexors throughout the swing phase [12].

3.1 Phases of Gait Cycle

There are two phases in gait cycle

A. Stance phase: It has following five sub phases

1. Heel contact: 'Initial contact'
2. Foot-flat: 'Loading response', initial contact of forefoot w. ground
3. Midstance: greater trochanter in alignment with the vertical bisector of foot
4. Heel-off: 'Terminal stance'
5. Toe-off: 'Pre-swing'

B. Swing phase: it is composed of three sub phases

1. Acceleration: 'Initial swing'
2. Midswing: Swinging limb overtakes the limb in stance
3. Deceleration: 'Terminal swing'

4. Anatomy of Gait and Balance

Normal gait and posture depend on the normal function of various structures along the entire neuroaxis. This includes the cortical, subcortical, and spinal cord regions as well as the final common pathway of motor neurons, the neuromuscular junctions and the muscles they innervate. The function of spinal, subcortical, and cortical structures involved in gait and posture are discussed below [11, 4].

4.1 Spinal Cord

Locomotor synergies are present in the spinal cord. Spinally transected vertebrates, including humans, can produce coordinated stepping movements without any input from supraspinal neural structures. The spinal networks that produce patterned muscle activation, termed central pattern generators, are distributed throughout the spinal cord. The rhythmical muscle activation that is a part of coordinated stepping can be generated from even a few isolated segments of the hemi transected spinal cord. The central pattern generator has been deduced to consist of excitatory and inhibitory interneurons using glycine, glutamate, and acetylcholine as neurotransmitters. Interneurons and collaterals connect the ipsilateral and contralateral central pattern generators to produce reciprocal movements of the limbs.

Coordinated, sequential activation of the limbs and axial muscles involved in stepping is produced by the rhythmical excitation and inhibition of the motoneurons of various muscles. Input to central pattern generators includes local sensory input and descending input from the brain stem via the reticulospinal pathways. Spinal locomotion can be modified by sensory input and level of electrical or chemical stimulation.

However, the locomotor pattern is stereotyped and does not anticipate environmental constraints on locomotion or the needs of the animal. Nor does the spinal cord has the balance synergies that are essential for successful locomotion. The important point is that the actual programming of the muscle activation required for gait is present at the spinal level. Higher centers initiate gait and adapt it to individual needs. For straight forward walking, the higher centers need not specify the individual muscle activations necessary to produce locomotion but instead activate the central pattern generators. This arrangement does not prevent supraspinal control of individual muscles during locomotion when exact foot placement is requisite [4, 11].

4.2 Brain Stem

The brain stem has three important functions in balance and locomotion: (1) setting the activity of the spinal central pattern generators to determine the initiation and speed of locomotion; (2) setting postural tone; and (3) modulating the force generated by the muscles activated by the central pattern generators.

Control of the spinal cord central pattern generators is exerted by the subthalamic and midbrain locomotor regions and relayed to the spinal cord by the reticulospinal tracts. The subthalamic locomotor region is defined physiologically as an area in the posterior lateral hypothalamus from which locomotion can be evoked by stimulation in the decerebrate animal. The subthalamic locomotor region is not in the subthalamic nucleus but is medial and dorsal to it. This region is not associated with any particular cell group, and stimulation of it may excite axons passing through the area.

The midbrain locomotor region is also defined physiologically but appears to overlap at least partially the pedunculopontine nucleus. This fact is important because the pedunculopontine nucleus receives a major input from the basal ganglia and from the sensorimotor and limbic cortex. This area may represent

one way in which the cortex and basal ganglia affect the initiation of locomotion. Progressive increases in electrical stimulation of the midbrain locomotor region of decerebrate monkeys induce stepping first in the contralateral limb, then in both limbs, and finally in a gallop. The midbrain locomotor region can also be stimulated by injections of gamma-aminobutyric acid (GABA) antagonists that are thought to disinhibit the midbrain locomotor region from tonic GABAergic input from the basal ganglia. Stimulation of the midbrain locomotor region in the neurologically intact cat induces locomotion that appears to be initiated to avoid a painful or frightening stimulus.

Output for the locomotor regions descends by way of the medullary reticulospinal tract, which arises from the ventral medial reticularis gigantocellularis and Magnocellularis nuclei. This tract traverses the ventral medial spinal cord, and lesions of the tract prevent locomotion and impair balance.

The role of the brain stem in postural control is evident by the postural responses that can be elicited in decerebrate animals. Vestibular, neck, and righting reflexes are present in decerebrate animals that have an intact brain stem. Stimulation in the dorsal tegmental fields of the pons causes an intact cat to cease walking and reduces postural tone, so that the cat sits and then lies down. Conversely, more inferior stimulation in the ventral tegmental fields of the pons increases postural tone and induces locomotion, so that a supine cat rises and then begins to walk. These stimulation sites indicate that postural and locomotor responses may be mediated, at least partially, by the same brain stem structures.

Brain stem systems do not simply turn on the spinal central pattern generators, as is done experimentally by electrical stimulation of the locomotor regions.

Recordings from reticulospinal neurons, that activate the central pattern generators, as well as from vestibulospinal and rubrospinal neurons demonstrate that activity in these pathways is phase-locked to the step cycle. This phasic activity is largely abolished if the cerebellum is ablated. The phasic activity in these descending tracts does not determine the frequency of stepping but enhances its force and rhythmicity. The tectospinal tract influences the posture of the head and neck and its orientation to visual targets. This tract arises from the superior colliculus, a structure that receives a major input from the substantia nigra pars reticulata, hence providing another connection between the basal ganglia and the postural and locomotor systems [4, 11].

4.3 Cerebellum

The cerebellum is not the origin of postural or locomotor responses because total ablation of the cerebellum does not abolish these responses. The cerebellum, however, refines the force and timing of locomotor and postural responses. Thus, the execution of these responses in an animal with cerebellar ablation is uncoordinated and dysmetric. The cerebellum receives information about each step cycle through the spinocerebellar pathways. The cerebellum partially exerts its influence on locomotion by affecting the activity of the reticulospinal, vestibulospinal, and rubrospinal pathways, which are physically active during stepping and contribute to coordinated rhythmic stepping. The cerebellar projections to the thalamus and thence to the frontal cortex may also affect gait and balance, although this possibility has not been explored [4, 11].

4.4 Basal Ganglia

The basal ganglia may have direct input to the brain stem nuclei controlling posture and balance via the pedunculopontine nucleus (midbrain locomotor region) and the superior colliculus (tectospinal tract). The majority of the basal ganglia output is directed back to the frontal cortex, and this influence is presumably responsible for the hallmarks of disease of the basal ganglia, hypokinesia and hyperkinesia. The execution of locomotor and postural responses, like other movements, may also be hypokinetic (parkinsonian) or hyperkinetic (choreic, dystonic) [4, 11].

4.5 Cortex

The cortex is completely unnecessary for routine walking in the cat, as evidenced by the performance of decorticate cats. However, the cat with frontal cortex or medullary pyramid lesions cannot perform stepping that requires precise placement of the feet, for example, walking on the rungs of a horizontal ladder. This indicates that "skilled walking" requires input from the motor cortex. The motor cortex has neurons that fire in phase with stepping and, therefore, like the vestibulospinal, reticulospinal and rubrospinal systems, it probably facilitates stepping. Stimulation of the medullary pyramid can reset the step cycle. This indicates that the corticospinal tract can affect the central pattern generators and may have a role in initiating and modifying the locomotor pattern produced by the central pattern generators. The sensorimotor cortex also has connections to the midbrain locomotor region and may affect the central pattern generators through this link. Stimulation of the motor cortex can induce discrete movements of a limb, preceded by the appropriate anticipatory postural responses, indicating that the cortex has a role in anticipatory postural responses. Lesions of the frontal cortex may also affect anticipatory reflexes.

Other anatomical areas are undoubtedly important in balance and locomotion. The areas reviewed here are responsible for the proper mechanical execution of balance and locomotion in a relatively stereotyped fashion [4, 11].

5. Post-Stroke Gait

Stroke is associated with a number of impairments, of which, walking impairment is most commonly reported. [13] Initially after stroke, 50% of victims will have no walking activity [14].

Common digression of post-stroke gait comprises of decreased speed. (Bohannon RW, 1987. Brandstater ME, *et al* 1983) [15, 16] decreased stride length (Nakamura R, *et al* 1988) [17] increased step width (Bohannon RW, 1987) [15] decreased cadence (Bohannon RW, 1987. Nakamura R, *et al* 1988) [15, 17] also greater than before time spent in double limb support (Goldie PA, 2001) *et al* [18] as compared with healthy adults. These problems lead to prolonged swing phase on the affected side and prolonged stance phase on the healthy side. [19] In addition of being asymmetric and slow, post-stroke gait is also inefficient with respect to energy spends. Individuals with stroke walk lesser distances with higher oxygen consumption, indicating they walk with higher oxygen demand. [20] Also the incidence of falls is higher in community-dwelling stroke individuals than in the general healthy elderly population. [21] The majority of falls occur during walking [22] which suggests that dynamic balance control during gait is an important issue.

6. Kinematics of Hemiplegic Gait

The joint kinematics of hemiplegic patients exhibit differences from normal individuals in both, the stance and swing phases of gait.

Reduced ranges of motion (ROM) of joints were observed on the involved lower extremity [23].

During the stance phase, the sagittal plane ROM at the hip appears to have a greater variety of atypical joint motions in groups of hemiparetic patients than swing phase. During the stance phase, it has been observed that these patients may exhibit hip motions that range from not being significantly different from normal; [24] they have reduced hip flexion at initial contact or more hip flexion than normal at initial contact [25, 26, 27].

It has also been reported that the hip may actually remain flexed at toe-off [28].

Knee joints also exhibits atypical patterns. Some participants have been found to exhibit increased knee flexion during the stance phase as compared to normal individuals, [24, 29, 30] especially at initial contact. Other have been reported to exhibit reduced knee flexion during early stance followed by knee hyperextension in late stance and delayed movement into knee flexion in preparation for swing. [24, 27] The third group of participants exhibited excessive knee hyperextension throughout most of the stance phase [24, 30, 31].

The ankle joint during the stance phase shows following deformities. Initial contact on the paralysed side is typically made with the foot flat, [32, 33] and ankle in a plantar-flexed position, [27, 34] which results in initial toe contact or a moderate decrease in toe elevation. [33] After initial contact, the ankle has been reported to perform irregular movements into dorsiflexion during stance phase, [24] reduced dorsiflexion in midstance and push off, [26] and/or increased plantarflexion in stance [24]. Reduced ankle plantarflexion at toe-off on the paretic lower extremity has also been found [26, 28].

The swing phase patterns of hip, knee, and ankle motions on the hemiplegic side have been characterized by limited or reduced hip flexion [24, 28] and hip tilted upward, lack of or decreased knee flexion,[26, 28, 30, 35] and reduced dorsiflexion or continuous ankle plantarflexion [24, 26, 28, 30, 35]. The increased leg length produced by the limited hip and knee flexion and reduced ankle dorsiflexion results in reduced floor clearance by the foot during swing, which produces dragging of the toes or circumduction of the leg [24, 26, 28, 30, 33, 35].

Due to this reason, the hemiplegic gait has been characterized by a stiff knee during swing [26, 33, 35].

It has been advocated that the upward tilt of the hip is also compensation to ensure toe clearance as the involved lower extremity is swinging forward [33, 35].

The limited range of knee flexion during swing is not limited to the involved lower extremity only, but also seen in the uninvolved lower extremity in cases of severe impairment [23].

It has been little reported on the movements of the upper limbs of the hemiplegic patient over the walking cycle; it has been reported that there are alterations in the upper extremity kinematics also. It has been observed that movements of the arms are less, [33] with the shoulders remaining relatively fixed in extension and the elbow remaining flexed [24]. A very little is known to the trunk movements during the walking cycle. It has been reported that the trunk is flexed forward during stance, which moves the center of gravity forward when there is recurvatum of the knee [36]. Forward trunk leans at push-off of the involved leg is a compensation for weak musculature on the paretic leg and that this forward trunk lean was not evident during push-off of the uninvolved leg [37]. It has also been reported that there is lateral shift of the trunk over the uninvolved stance to assist in weight shifting during swing of the affected leg [36].

7. Management

Gait recovery is a major objective in the rehabilitation of patients of stroke [38]. The primary goals of rehabilitation of people with stroke include being managed to perform daily activities and to walk independently [39] and in the same manner, rehabilitation programs for post stroke individuals mainly focus on gait training, at least for sub-acute patients [40]. The approaches used in gait rehabilitation after stroke includes neurophysiological and motor learning techniques, robotic devices, FES, and BCIs [41].

7.1 Neurophysiological techniques

In this technique the physiotherapist supports the patient's movement patterns, acting as an active participant and decision maker, so the patient acts as relatively passive recipient [42]. The most commonly used Neurophysiological techniques in gait rehabilitation are summarized in the following:

Bobath Method is the most widely accepted treatment. This method consists on trying to inhibit increased muscle tone (spasticity) by passive mobilization associated with tactile and proprioceptive stimuli. Accordingly, during exercise, pathologic synergies or reflex activities are not stimulated. This approach begins from the trunk and the scapular and pelvic waists and then it progresses to more distal segments [42, 43].

The Brunnstrom method is also well known but practiced less commonly. It is different to the Bobath strategy and focus on raising pathologic synergies in order to achieve a normal movement pattern and promotes return of voluntary movement through reflex facilitation and sensory stimulation [43].

The Rood technique uses peripheral input (sensory stimulation) to facilitate movement and postural responses in the same automatic way as they normally occur [44].

The Johnson method assumes that damaged reflex mechanisms responsible for spasticity are the leading cause of posture and movement impairment. These pathological reflexes are controlled through positioning and splinting [44].

7.2 Motor learning techniques

It is Just opposite to the neurophysiological techniques, in it there is active participation of patients. Thus, patient collaboration is a prerequisite and neuropsychological evaluation is required. This theoretical framework is constituted of practice of context-specific motor tasks and related feedbacks. These exercises promote the learning motor strategies and in the same manner support recovery. Task-specific and context-specific training are two legitimate principles in motor learning framework, suggesting that training should target the goals that are relevant for the needs of the patients.

The motor learning approach has been applied by different authors to develop specific methodologies, common methods are the Perfetti method, Carr and Shepherd motor relearning method, Conductive education or Peto method, The Affolter method, Sensory integration or Ayres method [41].

7.3 Robotic devices

It is believed that the orthodox gait training does not restore a normal gait pattern in the majority of stroke patients [45]

Robotic devices are nowadays widely consented among many clinicians and researchers [46].

These devices provide safe, intensive and task-oriented rehabilitation with a minimal physical assistance required to walk, thus reduces health care costs. It also provides kinematic and kinetic data in order to control intensity of practice, assess changes and measure motor impairments with improve sensitivity and reliability in comparison to standard clinical scales [41].

Robotic systems for gait recovery have been projected as simple electromechanical assistances for walking, such as the treadmill with body weight support (BWS) , as end-effectors, such as the Gait Trainer (Reha-Technologies, Germany, GT), or as electromechanical exoskeletons, suchlike the Lokomat [41].

7.4 Functional Electrical Stimulation

FES has been used in rehabilitation of chronic hemiplegia since the 1960s [41].

FES consists delivering an electric current through electrodes to the muscles. The current elicits action potentials in the peripheral nerves of axonal branches and thus generates muscle contractions [47]. This technique is found effective to improved gait performance in hemiplegic subjects and thought to give better results if used along with BWS [41].

7.5 Brain Computer Interfaces

BCIs establish a direct link between a brain and a computer without any use of peripheral nerves or muscles thereby enabling communication and control without any motor output by the user [41]. The users' brain activity extracts specific features from brain signals that reflect the intent of subjects and transform them into action [48].

8. Conclusion

Gait deformity after stroke is a common entity, since stroke is a leading cause of morbidity and mortality worldwide, post stroke hemiplegic gait is also a matter of great concern. Ability to control balance and walking is considered a basic task in post stroke rehabilitation but modern techniques only aims generate adaptive reversibility in functions and structures of the undamaged brain. However, there are restrictions in the abilities of hemiplegic patients with marred mobility to actively participate in such training programs, and it is not easy to activate the brain and revive the neuroplasticity through sensory and kinesthetic stimulation [49, 50]. So it is a need of hour to reconsider about the possible drugs and regimens for the improvement of post stroke hemiplegic gait in better survival and improve ADL, alternative traditional medicines like Unani medicine knowledge may be used to better results as suggested by recent studies [51].

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