Kinetics and Kinematics of Loading Response in Stroke Patients (A Review Article)

AHMED M.[1], AHMED S.[2]
Address For Correspondence: *MSc Physiotherapy, London, Therapies Dept. St. Georges Hospital, London (V),*

With the advancement in the field of assessment and treatment individualization, gait analysis is considered a routine matter in the health care settings during rehabilitation of walking. Gait analysis refers to the specialized assessment of walking which includes past medical history, objective examination of various characteristics of gait and the impact of disease on the pattern of walking. Kinematics and kinematics are the most useful parameters used to assess the disabilities associated with gait cycle. Infrared video cameras are normally used during gait analysis to define the three dimensional positions and movements of various lower limb joints. By analysing the kinetics and kinematics of the loading response; an important phase of the gait cycle, various deficits can be assessed in stroke patients including decreased musculoskeletal coordination, weakness and spasticity of the lower limb muscles. The detailed assessment of the kinetics and kinematics during loading response can enable the physiotherapists and rehabilitation specialists to design better treatment programs for the Neuro compromised stroke patients. This review article will address the kinetics and kinematics during loading response in stroke patients, but the technique can be used in a number of conditions.

Key Words: Loading response, kinetics and kinematics, gait analysis

Introduction

Stroke, described as cerebral vascular accident, is the most imperative cause of focal neurological deficits. It includes the impairments related to both sensory and motor tasks.[1] It is considered to be the third most common cause of death in the adult population in United Kingdom (UK).[2] According to the statistics of Stroke Association (2008), 150,000 persons suffer from stroke every year in the UK and around 250,000 live with the focal motor disabilities caused by this cerebral vascular accident.[3] Muscle activation nuisance and turmoil in the completion of the efficient ambulation are some of the major impairments resulted after stroke.[4] All of these impairments hamper in the execution of the everyday motor tasks.[5] It is estimated that rather being discharged after complete motor rehabilitation (37%), a large number of stroke patients with severe impairments involving upper and lower limbs are discharged to the institutionalized care (63%) every year.[2]

It is suggested that many of the stroke patients revert to unsupported walking after the first day of the onset of the initial symptom, depending upon the extent and the area of the brain involved.[6] But, almost 50% of these do not reach to their pre-injury level of activities even after 9 weeks of the onset.[7] Craik and Oatis[8] suggested that almost 70% of the stroke patients can regain their walking capacities after the cerebral vascular accident. During recovery period, stroke patients regain walking while maintaining the dynamic balance, but have hitches in loading the affected limb and it results in shambling over the affected side. It is suggested that perfection of this weight bearing evenness is the prime target of rehabilitation.[9-11]

The aim of this review article is to explore the effect of altered loading response; a phase of the gait cycle, in stroke patients of age between 35 to 60 years. In this review article, effect of altered loading response will be analyzed and functions of different lower limb joints will be discussed from the scope of available literature. Aberrations in the muscle activation patterns, kinematics of various lower limb joints and spatiotemporal gait characteristics will be considered while reviewing the effect of stroke in the walking abilities of the stroke patients.

Loading Response in Healthy Subjects

Gait, describes as walking, is a semi automated motor task controlled by the central nervous system.[12] Forward propulsion of the body from one position to the other depends upon the interrelated activities of various lower limb joints. Depending upon the relationship between the foot and the surface, gait is normally divided into two phases. The period of time when the foot is in contact with the surface is termed as stance, and the time when it is in space is named swing. The normal gait cycle consists of 60% of stance phase and 40% of swing phase.[13]

During the gait cycle, various lower limb joints perform three functional tasks of selective synergistic motions which serve to fulfil a functional goal. The first important task is the weight acceptance which includes initial contact and loading response. Loading response is the initial double stance phase and it begins with the initial foot contact and continues until the contralateral foot is lifting for swing.[6] Second task of gait cycle is to support the upper body on a single limb and it is comprised of mid stance and terminal stance. Limb advancement is the third functional component of gait cycle which includes pre-swing, initial swing, mid swing and terminal swing.[6] For reference purposes, the gait cycle can be started from any of its events as the starting point. However, initial contact is normally considered as the starting event of the gait cycle in healthy individuals.[14]

The interaction of the lower limb joints and generation of the muscle forces during gait cycle can be assessed by

performing gait analysis. The intricate process of gait analysis involves various biomechanical factors. It entails the understandings of the joint kinetics and kinematics. Kinematics deal with the evaluation of movement but not with the cause of these movement while kinetics deal with the forces which generate movement and muscle activity.[15]

Altered Loading Response in Stroke Patients

Among all the focal neurological impairments which result after stroke, altered loading response and abnormal postural control are the ones which influence most on the daily living activities of the stroke patients[4]. Altered joint activities are resulted due to the weakness and spasticity of different muscles on both the affected and unaffected lower limbs. *"Weakness of the flexor muscles, spasticity of the extensor muscles, and a synergistic extension motor pattern may be the main causes of gait disturbance"* (Pg. 580, 16).

It is suggested that in stroke patients, almost 40% of the required muscle work is performed by the muscles of the affected side during walking.[17] Hip flexors and extensors and ankle planter flexors symbolize the chief contributors of this required muscle work. It is suggested that in stroke patients, these lower limb muscles of the affected side lose their capabilities to generate the normal levels of muscular forces.[18] However, Olney et al[19] suggested that muscles of the affected side work with less potential due to the effect of paresis. Perry[6] attributed this loss of muscle strength as the major factor that hinders both the progression and weight bearing stability of stroke patients.

Paralytic involvement of one side of the body can affect spatiotemporal characteristics of walking and loading response in many ways. An excessive planter flexion and inversion, and stiffed knee gait are some of the manifestations after stroke. Mid swing is characterized by the dominant flexor pattern and terminal swing leads to the dominant extensor pattern consisting of knee extension and ankle planter flexion. *"The terminal swing and loading response phases of the tibialis anterior are lost as the ankle control of the flexor pattern shifts to the extensor pattern and soleus begins its action prematurely" (Pg. 312, 6).*

It is suggested that muscle weakness at the ankle joint causes reduced planter flexion during loading response in stroke patients.[17] Richards et al[20] also observed a reduction in the power of the medial gastrocnemius and limited ankle planter flexion during walking in stroke patients. It is also observed that weakness in the muscles of the paretic side alters dorsi flexion at the ankle joint during the initial contact. In response, tibia does not role forward over the calcanium to complete the transfer of body weight from the contralateral limb.[21] Also, previous literature reveals that planter flexors of the paretic side does not facilitate toe off during the stance phase.[21,22] Spasticity of these muscles results in the hyper active stretch reflex which functions during swing phase as excessive co-activation pattern and hiders with the tibialis anterior work to dorsiflex the ankle joint during forward propulsion of the foot.[21] Lamontagne[22] observed the weakness in the muscles of the non paretic limb in stroke patients, which he described as the effect of excessive co-activation of the antagonists, and not merely the result of paresis. This shows that stroke does only effects the loading response on the paretic side, but functioning of the muscles on the non paretic limb is also compensated.

Effect of Altered Joint Kinematics on Spatiotemporal Parameters

Olney and Richards[23] classified hemiplegic gait abnormalities according to the variations in the joint kinematics on the affected side into three categories.

- Hemiplegic gait characterized by reduced hip joint amplitude in saggital plane due to reduced hip flexion at the initial contact and decreased hip extension at toe off.
- Reduced knee joint amplitude due to an increased knee flexion at loading response, and
- Increased planter flexion at heel strike or initial contact.

A decreased hip joint extension at the end of loading response is also observed by Kim and Eng.[24] This decrease in the hip extension during loading response can be the result of insufficient active hip extensors. However, Hsu et al[25] explained that this reduced hip extension on the affected side during and after the loading response can be involved in producing the decreasing step length and reduced walking speed on the contralateral side. Muscles of the contralateral side continuously perform various compensatory mechanisms during loading response to assist the paretic muscles of the affected side by co-contraction mechanisms. These compensatory actions of the contralateral muscles may be due to the passive stretch of the spastic muscles or may be due to co-contraction more than the contraction produced by the agonists. However, these altered joint kinetics and kinematic; in turn, lead to the more energy demanding circumstances during walking in stroke patients.

Asymmetry in the Step Length of Stroke Patients

Previously, spatiotemporal parameters have been commonly used to clinically assess the impact of stroke on walking.[24] It is suggested that various spatiotemporal characteristics of gait cycle are altered during loading response in stroke patients, such as step length asymmetry, walking speed and contact time of the stance foot.[26] Previous literature suggests that stroke patients have longer paretic step lengths.[27,25] Chitralakshmi[28] described that step length is increased in stroke patients on the paretic side, and this is not related to the overall walking speed. However, Kim et al[24] reported that increase in the step lengths of the stroke patients is not only present in the paretic limb, but can also be found in the non paretic limb. Therefore, Kim et al[24] hypothesized that the altered loading response in stroke patients is not involved in the step length asymmetry. However, this asymmetry in the step length might result due to the compensatory strategies of the antagonists on the non paretic side.

It is suggested that the alterations in the walking speed does affect the mobility patterns of the lower limb joints in stroke patients.[29] Femery[26] suggested an increase in the spatial characteristics of the gait cycle in stroke patients. These asymmetries in the spatial characteristics are significantly prominent on the affected side. Furthermore, contact time and step duration are 16% and 18% higher in stroke patients than in the healthy subjects.[26] This increase in the step length is attributed to be the results of the spasticity of the planter flexors.[29] Weakness of dorsi flexors and spasticity of the planter flexors during loading response is also considered to be the cause of alterations in the step length.[30] Therefore, changes in the muscle forces and abnormal movements of the ankle joint leads to the landing of the fore foot all of a sudden, and thus the whole loading mechanism is disturbed.

Effect of Altered Loading Response at Knee Joint

Lucareli[15] suggested that loading response is the most central part of gait cycle. It lessens the impact of sudden weight being transferred from the contralateral limb, and it harmonises the displacement of the centre of mass within the base of support. Knee is the most important joint to study the impact of stroke on loading response as it shows the significant changes in moments and muscle work.[31] It is suggested that that the interdependence of the muscles of the paretic limb alters the overall walking speed.[17] This study concluded that the transfer of the body weight from the contralateral limb during loading response is affected by the anomalous functioning of the muscles around the knee joint. However, Lucareli[15] suggested that there is no significant difference in the angular positions of the knee joint during loading response. But, the time point for the peak knee flexion was significantly different between the study groups in the study done by Lucareli.[15] It may be due to the fact that subjects were walking with varying walking speeds. Also, knee joint flexion values at initial contact observed by Gamble[32] in healthy subjects were in correspondence with the values of Lucareli (15) in stroke patients. Furthermore, the amount of knee flexion was also reported to be increased in the stroke patients when they were asked to walk at their normal speeds.[31] Therefore, it can be concluded that the variations in the walking speeds of the stroke patients does have a role on the initial knee flexion values during loading response.

Previous literature revealed that in some studies, walking with normal speed resulted in decreased knee flexion at initial contact.[33-36] However, Olney[17] suggested a greater knee flexion during initial contact when subjects were walking at higher speeds. This shows a positive relationship between amount of knee flexion and the walking speeds of the stroke patients. Walking speed does influence on the knee joint mobility during loading response which is against the observation of Lucareli.[15] However Literature does not clarifies the role of walking speed in modifying knee joint mobility during early loading response. If walking speed does have a role, then walking with normal or slow speed will give different values of temporal gait characteristics in stroke patients.

It is suggested that the stroke patients walk slower and often have longer step lengths as compared to healthy subjects.[25] In the studies which focussed on the walking patterns of the stroke patients,[31,37,38,5] subjects were allowed to walk at their normal speeds which would have resulted in varied knee joint nobilities. Moreover, a positive relationship is also suggested to exist between the stride lengths and walking speeds of the stroke patients.[39] Therefore, it is not reasonable to compare the joint mobility and temporal gait characteristics of the stroke patients with the healthy subjects walking at their normal speed. Because, the normal walking speed of the healthy subjects is slightly higher than the normal walking speed of stroke patients.[5] For comparison, healthy subjects should be encouraged to walk at slower speeds to match the walking speeds of the stroke patients. The impact of the walking speeds on the knee joint mobility is also reported by Olney.[19] In words of Chen[40] *"comparison of gait differences between hemiparetic and non-disabled individuals walking at the same speeds may provide biomechanical insights that elaborate the mechanisms of locomotor impairment"* (Pg. 878).

The changes in the knee joint movements can be linked to various factors. Mulroy[41] reported that these changes may be due to quadriceps weakness which results in increased knee flexion or spastic quadriceps which result in premature knee extension. Planter flexion contractures and spasticity of the soleus muscle may also result in altered ankle movements during initial contact and loading response.[6] Finally inhibition of the heel rocker mechanism and reduced rolling of tibia over the calcanium, due to an absent heel strike, may be the cause of altered ankle joint kinematics during loading response.

Clinical Implications

Understanding of all these biomechanical abnormalities during loading response in the walking patterns of the stroke patients can provide a rationale for the physiotherapists to target their treatments to the appropriate areas. It is suggested that decreased strength of plantar flexors during toe off can be easily compensated by strengthening the quadriceps as it will provide the additional power for push off.[23,42] Nadeau[42] also noted a strong correlation between hip flexors and the overall gait speed. Therefore, a combination therapy for quadriceps and the plantar flexors of the paretic side including dynamic resistance training and gait specific exercises could be favourable.[43] Body weight supported treadmill training is also evidenced to be beneficial for improving the locomotor functions of hemiplegic subjects.[44] However, Chen[45] reported no change in the altered knee joint kinematics after treadmill training even after doing adjustment for the training parameters like walking speed and body weight support. Therefore, further research is needed in this area of clinical practise to evaluate and standardized the treatment

protocols in order to rehabilitate various gait abnormalities during and after the loading response.

CONCLUSION

Alterations in the kinetics and kinematics of the lower limb joints in stroke patients can be involved in changing the joint movements during walking. The decreased ankle dorsi flexion during initial contact is due to the paretic weakness of plantar flexors. To compensate this decreased dorsiflexion, knee joint shows increased flexion and flexion at the hip joint is also limited. Lower limb joints on the unaffected side show the similar movements, but these movements are not due to the paretic muscle weakness but mainly because of the enhanced co-contraction mechanisms.

All these mechanisms result in decreased walking speed and increased stride time. Step length is greatly decreased associated with enhanced contact time. Therefore, stroke patients have increased double support period and decreased single limb support during walking. Most of the body weight is supported on the unaffected side as weight bearing on the affected side results in limping of the limb. Manifestation and compensation mechanism of various lower limb muscles and joints result in various abnormal postures like foot drop, stiffed knee gait, stiffed hip gait, limping gait etc.

References

1. Macko RF, Gerald VS, Dobrovolny L, Sorkin JD, Goldberg AP, Silver KH. Treadmill training improves fitness reserve in chronic stroke patients. Arch Phys Medi Rehab 2000; 82 (7): 879-884.
2. Hunter SM, Crome P. Hand function and stroke. Revi Clini Geron 2002; 12: 68-81.
3. The Stroke Association (Homepage online). (Cited 2008); Available from: URL: http://www.stroke.org.uk/information/what_is_a_stroke/index.html
4. Fong KN, Chan CC, Au DK. Relationship of motor and cognitive abilities to functional performance in stroke rehabilitation. Brain Injer 2001; 15: 443–453.
5. Donders R, Mulder T, VanLimbeek J, Schoonderwaldt H. Long-term outcome after stroke: A Disability-Orientated Approach. Inte Jour Reha Rese 1996; 19: 189–200.
6. Perry J. Gait analysis: Normal and pathological function. New Jersey: Slack; 1992.
7. Smith MT, Baer GD. Achievement of simple mobility milestones after stroke. Arch Phys Med Rehab 1999; 80: 442–447.
8. Craik RL, Oatis CA. Gait analysis: theory and application. St. Louis: Mosby-Year Book; 1995.
9. Mumma CM. Perceived losses following stroke. Reha Nurs 1986; 11 (3): 19-24.
10. Bohannon RW, Horton MG, Wikholm JB. Importance of four variables of walking to patients with stroke. Int J Rehab Res 1991; 14: 246-250.
11. Forster A, Young J. Incidence and consequences of falls due to stroke: A systematic inquiry. Brit Med J 1995; 311: 83–86.
12. Hausdorff M, Yogev G, Springer S, Simon S, Giladi N. Walking is more like catching than tapping; Gait in the elderly as a complex cognitive task. Exp Brain Res 2005; 164: 541-548.
13. Kirtley C. Clinical gait analysis. Washington: Churchill Livingstone; 2006
14. Downey CA. Observational gait analysis handbook. Path kinesiology department, Physical therapy department, the professional staff association of Rancho Los Amigos Medical Centre; 1989.
15. Lucareli P, Greve J. Alteration of the loading response mechanism of the knee joint during hemiparetic gait following stroke analyzed by 3-D kinematic. Clinics 2006; 61 (4): 295-300.
16. Yelnik A, Albert T, Bonan I, Laffont I. A clinical guide to assess the role of lower limb extensor over activity in hemiplegic gait disorders. Stroke 1999; 30 (3): 580-585.
17. Olney SJ, Griffin MP, Monga TN, McBride ID. Work and power in gait of stroke patients. Arch Phys Med Rehab 1991; 72: 309-314.
18. Bourbonnais D, Vanden S. Weakness in patients with hemiparesis. Amer jour occup ther 1989; 43: 313-319.
19. Olney SJ, Griffin MP, McBride ID. Temporal, Kinematic, and Kinetic variables related to gait speed in subjects with hemiplegia: A regression approach. Phys Ther 1994; 74: 872-885.
20. Richards CL, Malouin F, Dumas F, Lamontagne A. Recovery of ankle and hip power during walking after stroke. Cana Jour Rehab 1998; 11: 271-273.
21. Winter DA. The biomechanics and motor control of human gait: Normal, Elderly and Pathological. Waterloo Ontario: University of Waterloo Press; 1991.
22. Lamontagne A, Malouin F, Richards CL, Dumas F. Mechanisms of disturbed motor control in ankle weakness during gait after stroke. Gait Post 2001; 15: 244-255.
23. Olney SJ, Richards C. Hemiparetic gait following stroke. Part 1: Characteristics. Gait Posture 1999; 4 (2): 136-148.
24. Kim CM, Eng JJ. Symmetry in vertical ground reaction force is accompanied by symmetry in temporal but not distance variables of gait in persons with stroke. Gait Posture 2003; 18: 23-28.
25. Hsu AL, Tang PF, Jan MH. Analysis of impairments influencing gait velocity and asymmetry of hemiplegic patients after mild to moderate stroke. Arch Phys Med Rehab 2003; 84: 1185-1193.
26. Femery V, Moretto P, Renaut H, Thévenon A, Lensel G. Measurement of plantar pressure distribution in hemiplegic children: Changes to adaptative gait patterns in accordance with deficiency. Clin biomech 2002; 17: 406-413.

27. Dettmann MA, Linder MT, Sepic SB. Relationships among walking performance, postural stability, and functional assessments of the hemiplegic patient. Amer J Phy Med 1987; 66: 77-90.
28. Chitralakshmi KB, Mark GB, Richard RN, Steven AK. Relationship Between Step Length Asymmetry and Walking Performance in Subjects With Chronic Hemiparesis. Arch Phys Med Rehab 2007; 88: 43-9.
29. Intiso D, Santili V, Grasso MG, Rossi R, Caruso I. Rehabilitation of walking with electromyographic feedback in foot drop after stroke. Stroke 1994; 25: 1189-1192.
30. Marigold DS, Janice JE, Inglis JT. Modulation of ankle muscle postural reflexes in stroke: influence of weight-bearing load. Clin Neur 2004; 115: 2789-2797.
31. Burdett RG, Borello-France D, Blatchly C, Poptter C. Gait comparison of subjects with hemiplegia walking unbraced, with ankle-foot orthosis and with Air-Stirrup brace. Phys Ther 1988; 68: 1197-1203.
32. Gamble JG, Rose J. Marcha Humana. Baltimore: Premier; 1998.
33. Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiplegic patients. Brain 1979; 102: 405-430.
34. Knutsson E. Gait control in hemiparesis. Scand J Reh Med 1981; 13: 101-108.
35. Lehmann JF, Condon SM, Price R, DeLateur BJ. Gait abnormalities in hemiplegia: Their correction by ankle-foot orthosis. Arch Phys Med Rehab 1987; 68: 763-671.
36. Cozean CD, Pease WS, Hubbel HS. Biofeedback and functional electric stimulation in stroke rehabilitation. Arch Phys Med Reha 1988; 69: 401-405.
37. Chung SG, Rey EV, Bai Z, Roth EJ, Zhang LQ. Biomechanical changes impassive properties of hemiplegic ankle with spastic hypertonia. Arch Phys Med Reha 2004; 85: 1-6.
38. Dietz V, Berger W. Normal and impaired regulation of muscle stiffness in gait: A new hypothesis about muscle hypertonia. Expe Neural 1983; 79: 680-7.
39. De-Quervain IA, Simon SR, Leurgans S, Pease WS, McAllister D. Gait pattern in the early recovery period after stroke. Jour Bone Joint Surg 1996; 78: 1506-1514.
40. Chen G, Patten C. Joint moment work during the stance-to-swing transition in hemiparetic subjects. Journal of Biomechanics 2007; 41: 877–883.
41. Mulroy S, Gronley J, Weiss W, Newsam C, Perry J. Use of cluster analysis for gait pattern classification of patients in the elderly and late recovery phases following stroke. Gait Posture 2003; 18: 114-125.
42. Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantar flexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. Clinical Biomechanics 1999b; 14: 125–135.
43. Patten C, Dozono JM, Jonkers I. Gait speed improves significantly following dynamic, high-intensity resistance training in person post-stroke. Stroke 2007; 38: 465.
44. Sullivan KJ, Knowlton BJ, Dobkin BH. Step training with body weight support: Effect of treadmill speed and practice paradigms on post stroke locomotor recovery. Arch Phys Medi Reha 2002; 83: 683–691.
45. Chen G, Patten C, Kothari DH, Zajac FE. Gait deviations associated with post-stroke hemiparesis: Improvement during treadmill walking using weight support, speed, support stiffness, and handrail hold. Gait & Posture 2005a; 22: 57–62.