Journal of Engineering and Applied Sciences 12 (Special Issue 5): 6904-6910, 2017
ISSN: 1816-949X

Postural Control and Gait Performance among Individual with Diabetic Peripheral Neuropathy and Stroke Survivors: A Review Paper

[1]Amirah Mustapa, [2]Nadia Mohd Mustafah, [1]Maria Justine and [1]Haidzir Manaf
[1]School of Physiotherapy, Faculty of Health Sciences, Universiti Teknologi MARA,
Puncak Alam Campus, 42300 Selangor, Malaysia
[2]Department of Rehabilitation Medicine, Faculty of Medicine, Universiti Teknologi MARA,
Sg Buloh Campus, 47000 Selangor, Malaysia

Abstract: Numbers of diabetic patients with Diabetic Peripheral Neuropathy (DPN) and stroke are increasing worldwide. Diabetes Mellitus can give rise to macrovascular and microvascular complications and among which is stroke and peripheral neuropathy, respectively. Literatures have revealed that DPN and stroke populations exhibited significant deterioration of postural control and gait performance and reported worsening in the presence of cognitive impairment. Walking with balance and gait dysfunction among DPN with additional stroke event may create destabilizing effects and fall. To date, there is no comprehensive study of postural control and gait performance among DPN with additional stroke has been published. Therefore, the aim of this study is to explore further on the postural control and gait performance following dual disability of DPN and stroke.

Key words: Diabetes mellitus, diabetic peripheral neuropathy, stroke, postural control, gait performance, worldwide

INTRODUCTION

Diabetes Mellitus (DM) and its debilitating microvascular complication is explained by the presence of signs and symptoms of peripheral nerve dysfunction in the diabetic patients after non-diabetes causes have been excluded (Tanenberg, 2009). Interactions of both metabolic and vascular factors by diabetes pathological with the contribution of degenerative process in aging cause Diabetic Peripheral Neuropathy (DPN) (Tesfaye and Selvarajah, 2012). Hyperglycemia stimulates the synthesis of an endogenous protein kinase C activator which may promotes peripheral nerves's ischemia through high vascular permeability and thickening of the basement membrane and then results DPN. Furthermore, advanced glycation end products (leading substances of aging process) may cause impairment in vascular function primarily deficit of the capillaries blood flow with subsequent putting off nitric oxide (oxidative stress) (Bucala *et al*., 1991) that have been implicated in impaired vasodilation in the diabetic patients (Zochodne, 2007). Afterward, the alterations of capillaries abilities to relax under chronic hyperglycemic conditions leads to the vasoconstriction of the blood vessels and a declination of nerve blood flow, ultimately lead to ischemic damage to neurons and structural damage of nerves pathway (Tomlinson and Gardiner, 2008).

On the other hand, DM is also a known modifiable risk factor for stroke (Megherbi *et al*., 2003; Lahano, 2015). World Health Organization (WHO) defined stroke as a rapidly developing clinical signs of focal (or global) disturbance of cerebral function, over than 24 h or leading to death with no apparent cause of nonvascular origin has two types; ischemic and hemorrhagic stroke (Sacco *et al*., 2013). The incidence of ischemic stroke among diabetic patients occurs due to occlusion of small arteries that lead to the small infarct in the brain (Asfandiyarova *et al*., 2006). While there is no association of hemorrhagic stroke risk and diabetes but the co-occurrence of diabetes with hypertension has been determined as the contribution towards cerebral hemorrhage (Hyvarinen *et al*., 2009). Similarly with DPN, hyperglycemia specifically affects vascular structure and function through activation of protein kinase C, diversion of glucose into the aldose reductase pathway and formation advanced glycosylation end products. Then, these processes have been proposed in causing macrovascular complication by increasing the progression of atherosclerosis in stroke (Idris *et al*., 2006).

DPN affects distal and eventually proximal peripheral sensory and motor nerves (Dixit and Maiya, 2014). Sensory neuropathy is prominent in DPN with an exhibition of numbness and pricking sensation in a stocking and glove pattern that starts from the feet

Corresponding Author: Haidzir Manaf, School of Physiotherapy, Faculty of Health Sciences, Universiti Teknologi MARA, Puncak Alam Campus, 42300 Selangor, Malaysia

(Gupta and Gupta, 2014). While motor neuropathy cause distal muscle weakness (Gupta and Gupta, 2014). DPN also affects the autonomic nervous system (Vinik, 2004) which can cause exercise intolerance, orthostatic hypotension and sudden death (Vinik *et al*., 2013). Stroke will cause hemiparesis, hemisensory loss, visual impairment, cognitive deterioration, hemineglect (Perennou, 2006) and incoordination of the upper and lower limbs (Fujimoto *et al*., 2014). On top of that spasticity in post-stroke cause muscle tightness that may reduce volitional movements, limits mobility and Quality of Life (QoL) (Martin *et al*., 2014). Defects of central integration of afferent inputs (somatosensory, visual, vestibular) with combination of sensory, motor and/or autonomic impairments in DPN and stroke (Ng and Fong, 2014) are the chief contributor to postural instability (Bardawil *et al*., 2013) and high gait variability which increase the likelihood of fall (Dingwell and Cavanagh, 2001).

Patients with DPN are 15 times more likely to experience fall compared to the healthy persons (Dingwell and Cavanagh, 2001). While, 23-73% of community-dwelling stroke patients experience fall within three months to 1 year of post-stroke. Therefore, it is likely patients who had dual disability of DPN and stroke will have higher rate of fall. Thus, our motivation of this review is to explore further on the postural control and gait performance among DPN and stroke as it is important in fall prevention. To our knowledge, this is the first review to address the body systems and mechanisms involved in the stability of postural control and gait performance following dual disability of DPN and stroke.

MATERIALS AND METHODS

Postural control: Postural control is defined as the control of body's position in space for balance. While balance refers to the ability of the body to maintain the Centre of Gravity (COG) within stability limits that are determined by the Base of Support (BOS).

Normal postural control: In the normal adult, postural control is obtained from sensory feedbacks of the body which are the somatosensory, vestibular and visual system. The somatosensory system is a multiple sensors that will sense all the body parts's position (Winter, 1995). Somatosensory will respond by proprioceptive sensation through the early stretch of muscles, muscle length and tension of the lower limb and tactile sensation mainly sensations of touch, pressure and vibration of feet. The somatosensory inputs of muscles will continuously send messages to the Central Nervous System (CNS) about the position of the body part for balance correction (Oie *et al*., 2001). Vestibular system senses linear and angular head acceleration. Otolith organs in vestibular provide information about the head orientation in opposing the acceleration of gravity which then activate anti-gravity leg muscles in static standing state or/and gaze control of head stabilization in dynamic standing (Lackner and Zio, 2000). Visual also plays a role in planning the direction of mobility besides avoiding any obstacles throughout the way. Thus, visual information about the environment must be certain to stabilize the head in space (Kiemel *et al*., 2011). Therefore, the process of maintaining balance must rely on both good sensory and muscular function.

Besides, postural control in opposing postural sway is build up by a few basic systems. Postural sway is defined as the response of the postural muscle activity in standing with then, stimulates a continuous to-and-fro movement of the body against the point of gravity (Uccioli *et al*., 1995). One of the system is muscles tone that plays role in maintaining the joints in a significant position mainly in the extensor of postural muscle. Postural fixation maintains the position of the joints against an internal force (e.g., body weight) through antagonistic muscles co-contraction at the joints. During upright standing, compensatory torques must be activated to compete with the destabilizing torque from gravity. Then, spontaneous sway is produced by the continuous body deviations disputed by corrective torques. Interruption of other body segments during movement of one part of the body showed the instability of postural control (Hasan, 2005). Normal adult also can rock stably backward and forward by lifting the toe or the heel to keep the upright balance without stepping in opposing external disturbance (Fard *et al*., 2013).

Postural control changes in DPN: Postural control instability in DPN occurs due to reduce accurate feedback of the proprioception sense along with the deterioration of somatosensory (Fahmy *et al*., 2014), vestibular systems and visual (Corriveau *et al*., 2000). Interruption of the afferent and efferent neuron function through the termination of the tibial, sural and deep peroneal nerves in the mechanoreceptors of the capsule and ligaments at the ankle joint (Chitra and Shetty, 2015) lead to diminish functions of the proprioceptive and tactile sensations in maintaining postural stability (Bardawil *et al*., 2013). Visual dysfunction in DM occurs when peripheral vision is occluded with high blood glucose level in the blood vessels of the retina (Maurer *et al*., 2005). Palma suggested DPN resultant in the impairment of proprioceptive and tactile sensation which result static

balance alteration in the eyes open while static balance alteration in the eyes close due to visual dysfunction (Maurer *et al*., 2005). DM affect vestibular function as the vestibular system is sensitive to high blood glucose and insulin level which cause DPN group exhibited impaired ability to detect short whole body anterior translation with large sway area (Hamada and Debrky, 2014). DPN patients also have difficulty in detecting minor postural disturbances and resistance towards gravity (Hyvarinen *et al*., 2009).

Besides that, postural sway in DPN occurs following the inability of the postural and lower limb muscles to provide an adequate activity level of muscles and joint. Muscle strength reduction is associated with relatively high glucose level and potentially less glucose uptake and hyperglycemia in muscles which can contribute to lower capability in resisting posturals way (Toosizadeh *et al*., 2015). Anderson reported 17 and 14% less strength in ankle flexor and extensor muscles of DPN patients. Additionally, DPN deteriorates the muscle sensory action through the defect in reflexive responses of lower limb muscles as it compromises the function of spindle muscle in the aspect of velocity, position and force sensation to maintain static standing (Toosizadeh *et al*., 2015). Normal adult maintains their postural from sway by an anterior-posterior x-y planes which is known as ankle strategy, resembles an inverted pendulum: the fulcrum is the ankle and the head is the opposite end of the pendulum. However, DPN group exhibited larger trace surface compared to the normal that indicated to reduce the ability of the ankle strategy to resist postural sway against gravity (Uccioli *et al*., 1995).

Postural control changes in stroke: Postural control instability in stroke occurs due to somatosensory impairments (Park *et al*., 2013), visual (Ng and Fong, 2014) and vestibular systems defect (Haral *et al*., 2014). Interruption of the somatosensory system through sensory impairments especially proprioception and tactile sensations are frequently reported by the stroke patients due to lesion on the cortical side of the brain that gives innervation for sensory (Peurala *et al*., 2007). Delay in motor activity onset, abnormal timing and sequences of motor activity further deteriorate postural control deficit among stroke survivors (Garland *et al*., 2009).

Postural sway in stroke occurs when stroke patients with damage of the central nervous system attempt to control the posture in the standing position and to maintain the center of mass (Cho *et al*., 2014). Indeed, the spastic motor in stroke also causes difficulty of the body part to adjust in maintaining postural control (Oliveira *et al*., 2008). Chronic stroke patients also has impaired capability to endure external perturbations mainly on the paretic side because of weakened short-latency of leg muscle reactions at the paretic side in the motions feedback of the support surface. Therefore, stroke patients will avoid large passive body mass displacements on paretic leg and rely excessively on the non-paretic leg muscles to maintain the stability of postural control (Geurts *et al*., 2005). In short, sufficient range of motion, muscular strength and proprioceptive sense of the lower limb joints is required in postural control stability, yet these functions are compromised in the stroke patients (Park *et al*., 2013).

RESULTS AND DISCUSSION

Gait performances: Gait performances refer to the human locomotion in walking with forward propulsion of the body by relying on the various lower extremities movement with the association of foot and surface (Kemu, 2008). Walking is also a motion control design with a repetitious sequence of limb movement while simultaneously maintains stance stability and forward motion.

Normal gait performance: Gait is a structural and complex process involving motor control system of the brain and spinal cord. Sensory, vestibular and vision is also Important to main a normal gait pattern (Petrofsky *et al*., 2005). Walking requires bilateral, symmetrical and coordinated movement of all limbs (Manaf *et al*., 2012). Initiation of gait is voluntarily command by the brain but the spinal centers control the activation of muscles after the leg movement (Dietz, 1997). One gait cycle encompasses 60% of the stance phase and 40% of swing phase (Kharb *et al*., 2011).

Walking is not an automatic process. A certain level of attention is required during walking. Cognitive and attention have an important role in the maintenance of balance and postural control as the brain gives the command to the limb muscles to stabilize the body (Paul *et al*., 2009). The bottleneck theory proposes the creation of bottleneck for information process which two tasks are processed by the same neural processor or networks. The second task process will be delayed until the processor is free from processing the first task (Woollacott and Cook, 2002). Dual-task performance in the walking involves carrying out a primary task (i.e., walking) and a secondary task at the same time (Seligmann *et al*., 2008). According to this theory, the performance of dual tasking during walking might result reduce gait speed or delay performance of the second cognitive task (Seligmann *et al*., 2008).

Gait performances changes in DPN: DPN patients usually have a conservative gait pattern that occurs with high double support time, slow speed and shorter steps as an attempt to keep stability in walking. DPN exhibits this pattern because of the reduction of sensory feedbacks from the lower extremities and weakness of ankle plantar flexor and dorsiflexor muscles (Richardson, 2002). Muscle weakness of the ankle mainly Tibialis Anterior (TA) is one of the factors implicated to the balance alteration in gait among diabetics as innervation of peroneal nerve is the first nerve that showed deterioration of electrophysiological changes in DPN patients (Giacomozzi *et al*., 2002). This finding is in agreement with the study by Sawacha indicated that there is a significant deterioration in TA function in the gait of neuropathic patients which contribute alteration in normal midswing phase. Secondly, ankle plantar flexors muscles weakness in DPN lead to shorter steps and lower cadence and consequently, slower walking speed (Camargo *et al*., 2015).

Moreover, the improper input of vestibular and visual also cause CNS to lose coordination in gait and may cause decreased gait speed and wider step length (Petrofsky *et al*., 2005). In fact, diminish sensory feedback with a further contribution of vision impairments, muscle weakness and lack of neuromuscular control of distal joints in neuropathic patients resulting in increase gait instability (Menz *et al*., 2004). Additionally, DM is associated with dysregulation of glycemic variability that might contribute to brain atrophy and cognitive impairments (Cui *et al*., 2011). Possible reason for the findings observed in this study is that the reduce somatosensory information from the limbs in the diabetic patients results in compensation of attentional capacity to maintain gait performance, thus leaving less prioritize for other simultaneous cognitive tasks (Wrobel and Najafi, 2010).

Gait performance changes in stroke: Lack of dorsiflexion ankle during early stance phase which is initial contact will cause foot slapping on the floor (Park *et al*., 2013). Plus, spasticity of calf muscles such as gatrocnemius and soleus cause patient to walk at the lateral part of the foot and prevent forward body weight shifting that lead to lack of push off that is needed to initiate swing phase (Manaf *et al*., 2012). Besides, weakness of hip abductor and dorsiflexor muscle cause circumduction gait and toes dragging in the mid-swing phase of stroke patient. Furthermore, spatiotemporal characteristics of loading response in gait are changed mainly on the paretic side (Femery *et al*., 2001). Correspondingly, stroke has longer step lengths on the affected leg (Hsu *et al*., 2003). Dorsiflexors weakness and plantar flexors spasticity during loading response are noted to be a factor of the step length alteration (Marigold *et al*., 2004). Reduce velocity, cadence and stride length are a compensation of poor motor control because of balance deficiencies and difficulty in body movement over an unstable limb (Schroeder *et al*., 1995).

Attention is compulsory as a vision for postural control in stroke (Bensoussan *et al*., 2006). Deficits after damage to the intralaminar nuclei explain attentional demands in cognitive or postural tasks performance in hemiplegic people. Stroke survivors showed deterioration of gait activity in dual tasking task with similar effects in the dual-motor and dual-cognitive task. This could be explained by the choice of strategy taken by the participants for stability maintenance (Manaf *et al*., 2014).

Postural control and gait performance in patients with DPN and stroke: The pattern of gait impairments demonstrated by DPN patients may further deteriorate gait difficulties in stroke patients. Plus, the ability to analyze and select the necessary sensory information to prevent falls also may be affected in stroke survivors following the interruption of brain function. Command of movements execution from the brain is commonly affected in the stroke. Thus, CNS from the brain to the spinal cord unable to give proper input in starting the gait with the maintenance of stepping rhythm while walking (Amato *et al*., 2012). Hence, abnormal interactions between the CNS instability might be the source of gait deficit in the stroke patients. Stroke survivors diagnosed with DPN might demonstrated postural control and gait performance deficits based on the following loss of function in the foot and ankle joints through the reduction of sensory feedbacks from the lower extremities and weakness of ankle muscles in post DPN (Hamada and Debrky, 2014; Fulk and Echternach, 2008) with combination of impairments in post-stroke survivors for instead hemiparesis, spasticity, muscle weakness and impaired sensory feedbacks especially proprioception and tactile sensation (Tyson *et al*., 2006; Amato *et al*., 2012). Also, cognitive and attention which are compulsory in balance maintainance may impaired and further deteriorated postural control and gait performance in this dual disability (Toosizadeh *et al*., 2015; Wrobel and Najafi, 2010). Apparently, co-existence of the deterioration body functions in the DPN patients and stroke survivors

Table 1: Balance modulation in DPN and stroke

Systems	DPN	Stroke
Sensory input	Proprioception and tactile (Hamada and Debrky, 2014)	Proprioception and tactile (Tyson *et al*., 2006)
	Vestibular deficit (Fulk and Echternach, 2008)	Vestibular deficit (Merwick and Werring, 2014)
	Vision deficit (Maurer *et al*., 2005)	Vision deficit (Merwick and Werring, 2014)
Motor output	Muscle weakness and delay reaction (Fulk and Echternach, 2008)	Flaccidity and spasticity (Cui *et al*., 2011)
Cognitive	Brain atrophy and cognitive impairment (Cui *et al*., 2011)	Cognitive impairment (Danovska and Peychinska, 2012)

(Table 1) may imply greater deterioration in postural control and gait performances among stroke patients diagnosed with DPN.

CONCLUSION

Overall, this review has made an endeavor to state two points. First, DPN and stroke patients have been demonstrated significant postural instability and gait imbalance and worsening with pathologic condition on the cognitive that contribute to falls incidence. Second, the recent studies also noted that fall is one of the most common complications of DPN and stroke result from deficit of sensory, motor and cognitive systems. Therefore, it is hypothesized that both DPN and stroke populations exhibit most similar impairments. The literature review warranted the need for investigation of the characteristics of postural control and gait pattern that is considered risky to fall in the chronic stroke survivors diagnosed with DPN.

ACKNOWLEDGEMENTS

Researchers thank the Ministry of Education, Malaysia for funding the research project through the RAGS (600-RMI/RAGS 68/2014) and the Research Management Institute (RMI), Universiti Teknologi MARA (UiTM) for the administrative support.

REFERENCES

Amato, P.D.P., A. Kyvelidou, D. Sternad, B. Najafi and R.M. Villalobos *et al*., 2012. Training dual-task walking in community-dwelling adults within 1 year of stroke: A protocol for a single-blind randomized controlled trial. BMC. Neurol., 12: 129-129.

Asfandiyarova, N., N. Kolcheva, I. Ryazantsev and V. Ryazantsev, 2006. Risk factors for stroke in type 2 diabetes mellitus. Diabetes Vasc. Dis. Res., 3: 57-60.

Bardawil, E.M.M., E.M.M.A. Hamid, E.N.A.E.H. Sawy, M.H.Z. Megallaa and W. S.A.E.G. El, 2013. Postural control and central motor pathway involvement in type 2 diabetes mellitus: Dynamic posturographic and electrophysiologic studies. Alexandria J. Med., 49: 299-307.

Bensoussan, L., S. Mesure, J.M. Viton and A. Delarque, 2006. Kinematic and kinetic asymmetries in hemiplegic patients'gait initiation patterns. J. Rehabil. Med., 38: 287-294.

Bucala, R., K.J. Tracey and A. Cerami, 1991. Advanced glycosylation products quench nitric oxide and mediate defective endothelium-dependent vasodilatation in experimental diabetes. J. Clin. Invest., 87: 432-438.

Camargo, M.R., J.A. Barela, A.J. Nozabieli, A.M. Mantovani and A.R. Martinelli *et al*., 2015. Balance and ankle muscle strength predict spatiotemporal gait parameters in individuals with diabetic peripheral neuropathy. Diabetes Metab. Syndrome Clin. Res. Rev., 9: 79-84.

Chitra, J. and S.S. Shetty, 2015. Screening of proprioception of ankle joint in patients with diabetic neuropathy an observational study. Int. J. Therapies Rehabilitation Res., 4: 104-107.

Cho, K., K. Lee, B. Lee, H. Lee and W. Lee, 2014. Relationship between postural sway and dynamic balance in stroke patients. J. Phys. Ther. Sci., 26: 1989-1992.

Corriveau, H., F. Prince, R. Hebert, M. Raiche and D. Tessier *et al*., 2000. Evaluation of postural stability in elderly with diabetic neuropathy. Diabetes Care, 23: 1187-1191.

Cui, R., H. Iso, K. Yamagishi, I. Saito and Y. Kokubo *et al*., 2011. Diabetes mellitus and risk of stroke and its subtypes among Japanese. Stroke, 42: 2611-2614.

Danovska, M. and D. Peychinska, 2012. Post-stroke cognitive impairment-phenomenology and prognostic factors. J. IMAB. Ann. Proceeding Sci. Pap., 18: 290-297.

Dietz, V., 1997. Neurophysiology of gait disorders: Present and future applications. Electroencephalography Clin. Neurophysiol., 103: 333-355.

Dingwell, J.B. and P.R. Cavanagh, 2001. Increased variability of continuous overground walking in neuropathic patients is only indirectly related to sensory loss. Gait Posture, 14: 1-10.

Dixit, S. and A. Maiya, 2014. Diabetic peripheral neuropathy and its evaluation in a clinical scenario: A review. J. Postgraduate Med., 60: 33-40.

Fahmy, I.M., G.M. Ramzy, N.A. Salem, G.M. Ahmed and A.A. Mohammed, 2014. Balance disturbance in patients with diabetic sensory polyneuropathy. Egypt. J. Neurol. Psychiatry Neurosurg., Vol. 51,

Fard, B.M., A. Bagherib and A.S. Khoskbijarib, 2013. Receding horizon based control of disturbed upright balance with consideration of foot tilting. Int. J. Eng., 26: 1243-1254.

Femery, V., P. Moretto, H. Renaut and A. Thevenon, 2001. Spasticity and dynamic plantar pressure distribution measurements in hemiplegic spastic children. Ann. Readapt Med. Phys., 44: 26-34.

Fujimoto, H., M. Mihara, N. Hattori, M. Hatakenaka and T. Kawano *et al*., 2014. Cortical changes underlying balance recovery in patients with hemiplegic stroke. Neuroimage, 85: 547-554.

Fulk, G.D. and J.L. Echternach, 2008. Test-retest reliability and minimal detectable change of gait speed in individuals undergoing rehabilitation after stroke. J. Neurologic Phys. Therapy, 32: 8-13.

Garland, S.J., V.L. Gray and S. Knorr, 2009. Muscle activation patterns and postural control following stroke. Motor Control, 13: 387-411.

Geurts, A.C., D.M. Haart, V.I.J. Nes and J. Duysens, 2005. A review of standing balance recovery from stroke. Gait Posture, 22: 267-281.

Giacomozzi, C., A. Caselli, V. Macellari, L. Giurato and L. Lardieri *et al*., 2002. Walking strategy in diabetic patients with peripheral neuropathy. Diabetes Care, 25: 1451-1457.

Gupta, A. and Y. Gupta, 2014. Recent advances in endocrinology. Diabetic Neuropathy, 64: 714-718.

Hamada, S.M. and H.M.E. Debrky, 2014. Monitoring of motor function affection and postural sway in patients with type 2 diabetes mellitus. Egypt. J. Ear, Nose, Throat Allied Sci., 15: 241-245.

Haral, P.P., S. Yardi and A. Karajgi, 2014. Effect of sensorimotor integration on balance and gait in chronic stroke patients. Indian J. Physiother. Occup. Therapy, 8: 64-70.

Hasan, Z., 2005. The human motor control system's response to mechanical perturbation: Should it, can it and does it ensure stability?. J. Motor Behav., 37: 484-493.

Hsu, A.L., P.F. Tang and M.H. Jan, 2003. Analysis of impairments influencing gait velocity and asymmetry of hemiplegic patients after mild to moderate stroke. Arch. Phys. Med. Rehabil., 84: 1185-1193.

Hyvarinen, M., J. Tuomilehto, M. Mahonen, C.D. Stehouwer and K. Pyorala *et al*., 2009. Hyperglycemia and incidence of ischemic and hemorrhagic stroke-comparison between fasting and 2-hour glucose criteria. Stroke, 40: 1633-1637.

Idris, I., G.A. Thomson and J.C. Sharma, 2006. Diabetes mellitus and stroke. Int. J. Clin. Pract., 60: 48-56.

Kemu, M.A., 2008. Kinetics and kinematics of loading response in stroke patients a review article. Ann. King Edward Med. Univ., Vol. 14,

Kharb, A., V. Saini, Y.K. Jain and S. Dhiman, 2011. A review of gait cycle and its parameters. Int. J. Comput. Eng. Manage., 13: 78-83.

Kiemel, T., Y. Zhang and J.J. Jeka, 2011. Identification of neural feedback for upright stance in humans: Stabilization rather than sway minimization. J. Neurosci., 31: 15144-15153.

Lackner, J.R. and D.P. Zio, 2000. Human orientation and movement control in weightless and artificial gravity environments. Exp. Brain Res., 130: 2-26.

Lahano, A.K., M.A. Chandio and M.I. Bhatti, 2015. Frequency of common modifiable risk factors of stroke. Gomal J. Med. Sci., Vol.12,

Manaf, H., M. Justine, H.G. Ting and A.L. Latiff, 2014. Comparison of gait parameters across three attentional loading conditions during timed up and go test in stroke survivors. Top. Stroke Rehabil., 21: 128-136.

Manaf, H., M. Justine, M. Omar, M.K.A. Isa and Z. Salleh, 2012. Turning ability in stroke survivors: A review of literature. Int. Sch. Res. Network, 2012: 8-8.

Marigold, D.S., J.J. Eng, C.D. Tokuno and C.A. Donnelly, 2004. Contribution of muscle strength and integration of afferent input to postural instability in persons with stroke. Neurorehabilitation Neural Repair, 18: 222-229.

Martin, C.L., J.W. Albers and P.R. Busui, 2014. Neuropathy and related findings in the diabetes control and complications trial epidemiology of diabetes interventions and complications study. Diabetes Care, 37: 31-38.

Maurer, M.S., J. Burcham and H. Cheng, 2005. Diabetes mellitus is associated with an increased risk of falls in elderly residents of a long-term care facility. J. Gerontology Ser. A. Biol. Sci. Med. Sci., 60: 1157-1162.

Megherbi, S.E., C. Milan, D. Minier, G. Couvreur and G.V. Osseby *et al*., 2003. Association between diabetes and stroke subtype on survival and functional outcome 3 months after stroke. Stroke, 34: 688-694.

Menz, H.B., S.R. Lord S.R. George and R.C. Fitzpatrick, 2004. Walking stability and sensorimotor function in older people with diabetic peripheral neuropathy. Arch. Phys. Med. Rehabil., 85: 245-252.

Merwick, A. and D. Werring, 2014. Posterior circulation ischaemic stroke. BMJ., Vol. 348,

Ng, S.S. and S.S. Fong, 2014. Visual perception correlates with balance and motor performance in patients with stroke. Indian J. Physiother. Occup. Therapy, 8: 54-58.

Oie, K.S., T. Kiemel and J.J. Jeka, 2001. Human multisensory fusion of vision and touch: Detecting non-linearity with small changes in the sensory environment. Neurosci. Lett., 315: 113-116.

Oliveira, C.B.D., I.R.T.D. Medeiros, N.A.F. Frota, M.E. Greters and A.B. Conforto, 2008. Balance control in hemiparetic stroke patients: Main tools for evaluation. J. Rehabil. Res. Dev., 45: 1215-1226.

Park, Y.H., Y.M. Kim and B.H. Lee, 2013. An ankle proprioceptive control program improves balance, gait ability of chronic stroke patients. J. Phys. Therapy Sci., 25: 1321-1324.

Paul, L., B.M. Ellis, G.P. Leese, M.A.K. Fadyen and M.B. Murray, 2009. The effect of a cognitive or motor task on gait parameters of diabetic patients, with and without neuropathy. Diabetic Med., 26: 234-239.

Perennou, D., 2006. Postural disorders and spatial neglect in stroke patients: A strong association. Restor. Neurol. Neurosci., 24: 319-334.

Petrofsky, J., M. Macnider, E. Navarro and S. Lee, 2005. Motor control and gait characteristics in people with type 1 and type 2 diabetes without sensory impairment in the foot. Basic Appl. Myol., 15: 75-86.

Peurala, S.H., P. Kononen, K. Pitkanen, J. Sivenius and I.M. Tarkka, 2007. Postural instability in patients with chronic stroke. Restor. Neurol. Neurosci., 25: 101-108.

Richardson, J.K., 2002. Factors associated with falls in older patients with diffuse polyneuropathy. J. Am. Geriatr. Soc., 50: 1767-1773.

Sacco, R.L., S.E. Kasner, J.P. Broderick, L.R. Caplan and A. Culebras *et al*., 2013. An updated definition of stroke for the 21st century a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke, 44: 2064-2089.

Schroeder, V.H.P., R.D. Coutts, P.D. Lyden and V.L. Nickel, 1995. Gait parameters following stroke: A practical assessment. J. Rehabil. Res. Dev., 32: 25-31.

Seligmann, Y.G., J.M. Hausdorff and N. Giladi, 2008. The role of executive function and attention in gait. Mov. Disord., 23: 329-342.

Tanenberg, R.J., 2009. Diabetic peripheral neuropathy: Painful or painless. Hos. Physician, 45: 1-8.

Tesfaye, S. and D. Selvarajah, 2012. Advances in the epidemiology, pathogenesis and management of diabetic peripheral neuropathy. Diabetes Metab. Res. Rev., 28: 8-14.

Tomlinson, D.R. and N.J. Gardiner, 2008. Glucose neurotoxicity. Nat. Rev. Neurosci., 9: 36-45.

Toosizadeh, N., J. Mohler, D.G. Armstrong, T.K. Talal and B. Najafi, 2015. The influence of diabetic peripheral neuropathy on local postural muscle and central sensory feedback balance control. PloS one, 10: 1-12.

Tyson, S.F., M. Hanley, J. Chillala, A. Selley and R.C. Tallis, 2006. Balance disability after stroke. Phys. Therapy, 86: 30-38.

Uccioli, L., P.G. Giacomini, G. Monticone, A. Magrini and L. Durola *et al*., 1995. Body sway in diabetic neuropathy. Diabetes Care, 18: 339-344.

Vinik, A.I., 2004. Advances in diabetes for the millennium: New treatments for diabetic neuropathies. Medscape Gen. Med., Vol. 6,

Vinik, A.I., M.L. Nevoret, C. Casellini and H. Parson, 2013. Diabetic neuropathy. Endocrinol. Metab. Clin. North Am., 42: 747-787.

Winter, D.A., 1995. Human balance and posture control during standing and walking. Gait Posture, 3: 193-214.

Woollacott, M. and S.A. Cook, 2002. Attention and the control of posture and gait: A review of an emerging area of research. Gait Posture, 16: 1-14.

Wrobel, J.S. and B. Najafi, 2010. Diabetic foot biomechanics and gait dysfunction. J. Diabetes Sci. Technol., 4: 833-845.

Zochodne, D.W., 2007. Diabetes mellitus and the peripheral nervous system: Manifestations and mechanisms. Muscle Nerve, 36: 144-166.