QUANTITATIVE ASSESSMENT OF SPASTICITY USING COMPUTERIZED GAIT ANALYSIS

BİLGİSAYARLI YÜRÜME ANALİZİ İLE SPASTİSİTENİN KANTİTATİF OLARAK DEĞERLENDİRİLMESİ

Gunes Yavuzer*

Spasticity is one of the several components of the upper motor neuron syndrome, characterized by muscle overactivity, reduced inhibition of flexor reflexes, weakness, and loss of dexterity and fine motor function. The muscle overactivity results in limb stiffness and muscle spasm, to which there is both a neurogenic and a biomechanical component.

Spasticity can be very disabling, impairing motor performance and activities of daily living, such as walking, the proper use of arms and hands, and, in addition, it may provoke painful spasms and contractures. In this way it has a great impact on the patients' quality of life. The measurement of the spasticity is important for determining the goals of intervention as well as the outcomes of rehabilitation.

When spasticity produces a clinical disability by interfering with posture, motor capacity, nursing or daily living activities, medical treatment is recommended. The treatment of spasticity should be addressed by a multidisciplinary team, covering the full spectrum of neuroscience. Good management depends on an understanding of their role and application in relation to the needs of individual patients. It is mainly indicated when the muscle overactivity is diffusely distributed and should be implemented early, to prevent permanent musculoskeletal deformities or contractures. The aim of the treatment is to prevent provocative factors, treat muscle overactivity and prevent complications by decreasing spinal reflex excitability via reducing the release of excitatory neurotransmitters, or by potentiating the activity of inhibitory inputs. A variety of antispasticity interventions have been in use to improve function. They include traditional physiotherapy (with a range of motion-stretching exercises and splints or orthotics), oral medications, neurolytic blocking agents (such as phenol or botulinum toxin A), orthopedic surgery (in particular tendon-lengthening procedures), intrathecal baclofen pumps and selective dorsal rhizotomy. Evaluation of the efficacy of these drugs is determined by the therapeutic objectives which may be biomechanical, or functional.

Spasticity can be measured using clinical scales or biomechanical and neurophysiological techniques. In daily practice, clinical scales such as Ashworth and Tardieu are used, however, more objective, quantitative and robust measures are required for research purposes. The Wartenberg Pendulum Test has been devised for measuring spasticity at the knee, but has been shown to be unsuitable for measuring more severe spasticity. Powered systems and stress induced EMG responses have been used in research studies, but are rarely considered suitable for routine clinical use.

Quantitative gait analysis may have an important role to play in assessing spasticity since there are clear associations between lower limb spasticity and gait problems. Quantitative and reliable measures will serve

Yazışma Adresi / Correspondence Address:
Dr Gunes Yavuzer, Ankara University Faculty of Medicine Department of Physical Medicine and Rehabilitation Ankara Turkey, Tel: 90 312 5956022
e-mail: gyavuzer@medicine.ankara.edu.tr

* *Ankara University Faculty of Medicine Department of Physical Medicine and Rehabilitation Ankara Turkey*

both clinicians to monitor the progress of patients and to assess the treatment outcome and researchers to investigate the pathophysiology and new interventions. In a previous study, we investigated the clinical potential and reliability of kinematic gait data in quantitative measurement of spasticity at lower extremity after stroke (unpublished data). Our findings revealed that time-distance and kinematic gait data can be used as reliable outcome measures in spastic hemiparetic patients after stroke. Time since injury, lesion characteristics, impaired proprioception, level of motor recovery, spasticity or motor functioning have no effect on gait variability of stroke patients. We reported that clinicians should only be cautious when interpreting the quantitative gait data of stroke patients, as women and slow walkers may show different repeatability after stroke.

Spasticity may involve the upper and lower extremities. Arm swing does have effects on gait and is altered with spasticity. In the lower extremity, the common gait patterns with spasticity are flexed hip, adducted thighs (scissoring gait), stiff knee, flexed knee, equinovarus foot, valgus foot and striatal toe. Distinction between fixed and dynamic deformity is mandatory for the clinical decision making in spastic gait. During clinical examination foot may correct to neutral if it is splintable however while walking clonus or severe muscle imbalance may lead to dynamic deformity.

Equinovarus with or without toe clawing is one of the most common deformity in patients with spastic gait. Initial contact occurs with the forefoot and weight acceptance along with the lateral aspect of the foot. There might be a skin breakdown along the lateral border of the foot together with an antalgic gait pattern in chronic cases. Equinovarus posturing limits single support dorsiflexion, prevents forward progression of the tibia, and leads to a hyperextension thrust of the knee and restrained forward translation of the body's center of mass. This abnormality compromises stability by decreasing the base of support and causing limb instability may prevent functional ambulation. Swing phase foot clearance is also compromised. Muscles that may contribute to this disorder include tibialis posterior, tibialis anterior, long toe flexors, medial and lateral gastrocnemius, soleus, extensor hallucis longus, and peroneus longus. A diagnostic posterior tibial nerve block may help to differentiate the causative muscles. Conservative management also includes administration of neurolytic and chemodenervation agents. Most common orthopaedic intervention is the tendon lengthening however one should keep in mind that this approach only decreases force output with no change in neural output.

Valgus foot results from inappropriate contraction of peroneal and triceps surae muscles. Stance phase stability is compromised by an abnormal base of support. This condition also may lead to genu valgum stresses and resultant pathology to medial knee joint structures.

Hitchhiker toe or striatal toe (persistent great toe hyperextension) is a common condition compromising gait in patients with spasticity. Selection of the footwear is a big problem. The usual offender is the extensor hallucis longus muscle. This pathology often is managed successfully with motor point block of the extensor hallucis longus muscle. Diagnostic blocks often are helpful if co-contraction by the flexor hallucis longus is suggested.

Another frequent complication of the lower extremity spasticity is the stiff knee. It is defined as the reduction of the knee flexion under 60 degrees during swing. Knee flexion during swing is a passive movement secondary to hip flexion so that if hip flexion reduces, knee flexion reduces too during swing. Overactivity by iliopsoas, gluteus maximus, quadriceps (inappropriate firing of the rectus femoris prevents knee flexion during swing), and hamstrings (as hip extensors) is other possible contributors to this condition. Functional deficits include increased effective limb length, with circumduction, contralateral vaulting, or hiking of the pelvis required for foot clearance. Energy consumption is increased, typically in patients with little functional reserve. Dynamic EMG analysis may be required to identify the contributing muscle(s) from the extensive list presented above.

Crouched gait is marked by excessive knee flexion throughout swing and stance phases. Limb advancement is attenuated as part of this crouch pattern shortening step length. Both hamstring and quadriceps muscles may be involved. Dynamic EMG may demonstrate prolonged activity of the medial hamstrings. Knee flexion contracture is common. Excessive hip flexion may interfere with multiple activities of daily living (ADL) and also may contribute to knee flexion deformity. Again, excessive hip flexion may interfere with limb advancement in swing, as well as decreased advancement of the center of mass over the flexed support limb. Initial treatment of both excessive knee and hip flexion is controversial. Therapeutic interven-

tions include motor point blocks, chemoneurolysis, tendon lengthening, tendon transfers, and muscle release.

Thigh adduction (scissoring gait) may interfere with hygiene, daily activities, as well as with walking. Limb advancement is curtailed by severe hip adduction. Balance may be impaired as a result of a narrowing of the base of support. Muscles that potentially contribute to gait scissoring include the adductor longus and brevis, adductor magnus, and gracilis. Less frequently, the iliopsoas and pectineus are contributors. Diagnostic obturator nerve block helps establish whether contracture is present. Before permanent nerve blocks one should remember that patient may need hip adductors in swing phase to compensate weak hip flexors and interventions to adductor muscles may compromise gait.