DERLEME REVIEW

DOI: 10.5336/healthsci.2019-64991

Integrated Evaluation of Upper Extremity Dysfunction After Stroke: An Overview for Clinical Practice

İnme Sonrası Üst Ekstremite Fonksiyon Bozukluğunun Bütüncül Değerlendirmesi: Klinik Uygulama İçin Genel Bakış

Esma Nur KOLBAŞI[a],
Burcu ERSÖZ HÜSEYİNSİNOĞLU[b]

[a]Department of Physiotherapy and Rehabilitation,
İstanbul Medeniyet University
Faculty of Health Sciences,
[b]Department of Neurological Physiotherapy Rehabilitation,
İstanbul University-Cerrahpaşa
Faculty of Health Sciences,
İstanbul, TURKEY

Received: 22 Jan 2019
Received in revised form: 19 Mar 2019
Accepted: 28 Mar 2019
Available online: 29 Mar 2019

Correspondence:
Burcu ERSÖZ HÜSEYİNSİNOĞLU
İstanbul University-Cerrahpaşa
Faculty of Health Sciences,
Department of Neurological Physiotherapy
Rehabilitation, İstanbul,
TURKEY/TÜRKİYE
burcu.ersoz@istanbul.edu.tr

ABSTRACT Stroke is a major cause of disability worldwide. Upper extremity (UE) impairments such as abnormal muscle tone or abnormal recruitment are extensively seen following the incident. The changes in the muscle tone, strength and recruitment pattern may lead to altered biomechanical properties in the UE. Thus, the functions and kinematic features of UE movements may also change. As a result of these changes, abnormal motor performance of UE may be obversed in patients with stroke. Therefore, the knowledge about healthy and abnormal biomechanical properties in addition to the knowledge on differentiated kinematic characteristics after stroke are extremely important in clinical evaluation and decision-making. In this article, we reviewed the current evidences of literature about the changes in biomechanical, kinematic and functional properties in the UE and related assessment methods after stroke. It was our primary purpose to underline key points during clinical assessment to guide researchers and clinicians. In conclusion, it was seen that none of the outcome measures in the literature were able to assess the entire aspects of the impairment because of their variable focal points and qualitative or quantitative structure. Therefore, it is suggested by the authors to include more than one outcome measurement with various aspects in order to evaluate the every components of UE impairment. Through this way, more accurate planning for rehabilitation approaches may be achievable.

Keywords: Stroke; kinematics; upper extremity; assessment

ÖZET İnme, dünya çapında önemli bir özürlülük nedenidir. İnmeyi takiben, anormal kas tonusu veya harekete katılım sırasında ortaya çıkan yetersizlikler gibi üst ekstremite (ÜE) etkilenimleri ile sıklıkla karşılaşılmaktadır. Kas tonusu, kas kuvveti ve harekete dahil olma paternlerindeki değişiklikler, ÜE'de biyomekanik değişikliklere yol açabilir. Bu duruma bağlı olarak, ÜE fonksiyonları ve hareketlerinin kinematik özellikleri de etkilenebilir. Bahsi geçen değişimlerin sonucunda, inmeli hastaların ÜE'lerinde anormal motor performans açığa çıkabilir. Bu nedenle, inme sonrası ortaya çıkan farklılaşmış kinematik özelliklere ek olarak sağlıklı ve anormal biyomekanik yapılar hakkında da bilgi sahibi olmak klinik değerlendirme ve klinik karar vermede oldukça önemlidir. Bu makalede, ÜE' nin biyomekanik, kinematik ve fonksiyonel özelliklerinin değişimi ve bu değişimlere ilişkin değerlendirme yöntemleri ile ilgili literatürde yer alan güncel bulguları derledik. Bu derleme ile birincil amacımız, inme sonrası klinik değerlendirmenin anahtar noktalarını belirleyerek araştırmacılara ve klinisyenlere yol göstermekti. Sonuç olarak, literatürdeki değerlendirme ölçeklerinin değişken odak noktalarına ve niteliksel ya da niceliksel yapılara sahip olduğu saptandı. Ölçeklerden hiçbirinin, bu yapıları nedeniyle, ÜE etkileniminin tüm yönlerini değerlendirmede yeterli olmadığı tespit edildi. Dolayısıyla, klinik muayenede ÜE etkileniminin tüm bileşenlerini değerlendirmek amacıyla, çok yönlü birden fazla ölçeğe yer verilmesi yazarlar tarafından önerilmektedir. Bu sayede, fizyoterapi ve rehabilitasyon yaklaşımları için daha doğru bir planlama yapılarak, etkili bir tedavi sunulabilir.

Anahtar Kelimeler: İnme; kinematik; üst ekstremite; değerlendirme

Impaired upper extremity (UE) function is one of the common sequelae after stroke.[1-4] Loss of UE function appears in 80% of the patients in the acute stage and in 50% in the chronic stage after stroke.[5,6] The impairments of UE include paresis, abnormal muscle tone, loss of fractioned movement and changes in somatosensorial system.[7] As a result of these impairments, alterations in UE biomechanics and kinematic proporties occur. Therefore, objective assessment of UE with the current outcome measurements after stroke becomes even harder. In addition, due to the quantitative nature of outcome scales, clinicians mainly focus on the quantity rather than the qualitative characteristics of movements' occured in UE.[8] As a result of this incomplete assessments, the treatment and/or the gains from the rehabilitation will eventually be affected. There is now a consensus on the inadequency of such scales and need for the kinematic analyses.[8,9] However, it is a well known fact that kinematic/quantitative assessment of UE is expensive, time consuming, experience-dependent and hard to establish in every clinic. Therefore, our purpose with this review is to provide a deep insight to the clinicians on the assessment of upper limb impairments by keeping in mind the importance of biomechanical and functional notice. So that they might have a better accuracy when evaluating the UE of patients in clinical settings after stroke.

I. AN OVERVIEW ABOUT THE EVALUATION OF UPPER LIMB IMPAIRMENTS AFTER STROKE

The clinical assessment of UE includes observational assessment, manual assessment, kinematic assessment, tests and questionnairres.

MANUAL ASSESSMENT

Manual assessment consists of palpation, active and passive ROM assessment, evaluation of muscle tone and sensory assessment. These assessment methods are planned to evaluate common UE problems (paresis, loss of fractional movement, abnormal muscle tone and/or changes in somatosensorial system) after stroke.[7]

OBSERVATIONAL ASSESSMENT

At the beginning of the treatment, the functions that the patient can or cannot perform should be recorded and an observational movement analysis should be performed as described in the earlier paragraphs. The attention must especially be paid to postural control, effects on muscles contributing biomechanical changes in shoulder complex, sensory loss and ability to reach and grasp during the assessment. The impairments that may cause dysfunction need to be identified in this frame and a treatment plan should be established.

KINEMATIC ASSESSMENT

Kinematic analysis is a process in which the kinematic properties used to describe movement are measured. Kinematic studies may provide accurate and objective information about motor strategies associated with target-related tasks and may monitor the implementation of therapeutic techniques for UE.[10] Kinematic analysis has increasingly been used in clinical trials to show post-stroke motor recovery or to investigate the effects of various therapeutic interventions since functional and clinical assessments alone are inadequate to evaluate motor strategies used during movements.[11,12]

Many kinematic studies have been conducted in the laboratory environment in the last 15 years with the aim of measuring the UE movements used during daily activities in healthy individuals. Kinematic analysis are especially important in the measurement of the spatiotemporal parameters of the movements.

OUTCOME MEASURES

Outcome measures are highly important in the clinical assessment of the upper extremity. Some of the tests require different equipments (stopwatch, dynamometer, goniometer, etc.) or different objects to reach, to grasp, to handle or to carry. The majority of tests are performed in sitting or supine position.[13] Santisteban et al. reported that there were 48 different outcome measures in the assessment of UE after stroke and Fugl-Meyer Test (FMT) (36%) was the most common measurement.[14] This is a stroke-specific, performance-based impairment index which is designed to assess the movement, coordination and reflex actions of the shoulder, elbow, forearm, wrist and hand.[15] The FMA-UE has excellent inter-rater reliability and good concurrent validity when compared with similar tests of arm motor function.[16]

Furthermore, it is reported that outcome measures were combined in the most of the studies. For instance; FMT was mostly combined with Motor Activity Log (MAL), Action Research Arm Test (ARAT) and WMFT whereas its combination with Modified Ashworth Scale and Nine Hole Peg Test was infrequent. From these scales, only MAL evaluates the amount and quality of affected arm use in real life situations. It is a self-rating assessment which is scored between 0 and 5. A higher score indicates better performance. It is a valid and responsive assessment tool in clinical practice.[17,18] ARAT, WMFT and Nine Hole Peg Test are all performance related measurements and have high reliability and validity amoung stroke patients.[7] Modified Ashworth Scale measures impairment level of muscle tone. The elbow flexors are most easily and commonly assessed in the UE by Modified Ashworth Scale. Although it has excellent intrarater reliability for elbow, its inter rater reliability is poor.[19]

II. UPPER LIMB IMPAIRMENTS AFTER STROKE: KEY POINTS DURING CLINICAL ASSESSMENT

A. PARESIS

Paresis might be defined as decreased ability to volitionally activate motor units after central nervous system injuries such as stroke.[7,20] However, paresis mostly occurs in the form of a combination of different impairments, which leads to a syndrome. These impairments involved in paretic syndrome are weakness, spasticity, loss of fractionated movement and higher-order planning deficit.[20] Due to the impairments referred above, individuals with paresis have difficulty to execute the simpliest daily life functions which are highly related to quality of life.[21] A person with paretic syndrome is not able to efficiently perform movements as simple as reaching, grasping, etc. compared to their peers with intact central nervous system. For instance, when a stroke patient tries to reach to an object, it might be clearly seen that they are compensating the movement and excessively including the other parts of body such as trunk due to the increased spasticity and loss of fractioned movement. In some cases, they even help and support their affected side with the sound limb. But, if we look at the current outcome measures, we see that they are mostly assessing the accomplishment rather than the movement itself.[8] So, it is important for a clinician to discriminate the achievement and performance in case of paretic syndrome. Besides, the assessment of the muscle tone with scales such as Modified Ashworth Scale or Modified Tardeu Scale needs to be involved in the examination.

B. ABNORMAL MUSCLE SYNERGY

The generally accepted definition of muscle synergy is simply “a stable pattern of spatiotemporal movement between the muscles involved in the performance of an action”.[22] On the other hand, a pathological pattern of muscle co-activation after impairments in the motor systems is defined as abnormal muscle synergy.[23] Two complementary mechanisms may explain the synergies occured after stroke:

The disruption of healthy synergism by lesion and development of new synergy by cortical re-organization

The unmasking and up-regulation of alternative descending paths.[22]

The synergy patterns occurred in the upper extremity after stroke are flexor and extensor synergies. Usually, flexor synergy is observed and in this pattern, shoulder is placed in retraction, external rotation and 90° abduction, the elbow is in flexion and the forearm is placed in full supination. On the other hand, shoulder protraction, internal rotation and adduction, full elbow extension and full forearm pronation are observed in the extensor synergy.[24]

In studies investigating the changes in upper limb synergies in chronic stroke patients, it is reported that elbow-associated synergies were the same in healthy participants and in stroke patients regardless of impairment level.[25,26] However, shoulder-associated synergies were different in stroke patients. Stroke patients showed the activation of anterior deltoid muscle together with posterior and medial deltoid in abduction/extension synergy as opposed to healthy individuals and deltoid muscle activation was increased in stroke patients as level of impairment was increased. It was also found that pectoralis major was the mainly activated muscle in the adduction/flexion synergy. Anterior deltoid activation was limited in subjects with stroke and pectoralis major activation was increased as the level of impairment was increased.

In conclusion, the use of altered muscle synergies was associated with abnormal motor performance. Yet, there are limited number of questionnaires which include assessment of synergistic patterns in or along with the examination of UE performance. To the best of our knowledge, only the Fugl-Meyer Test- Upper Extremity explicitly assesses the synergies whereas Arm Motor Ability Test and Wolf Motor Function Test consider the involvement of synergies in the UE performance.[27-29] In another scale called "Motor Assessment Scale", the examiner does not allow the patient to use synergistic patterns in the activities and evaluates the performance of functional tasks.[30] In addition to these outcome scales, electro-myography (EMG) analyses and kinematic measurements are also used for the investigation of abnormal synergies in stroke survivors. Despite the high reliability of these methods, they are not available in every clinical setting. Therefore, clinicians must discriminate the use of abnormal synergies from the actual performance by choosing the right measurement scales and observing the action performed.

C. LOSS OF SOMATOSENSATION

What we know from the prevalence studies investigating the somatosensor deficits after stroke is that around half of the stroke patients are suffering from the loss of somatosensation causing to discomfort and functional impairment.[31-33] Besides, it is a very well-known fact that an intact somatosensation is required for motor control and motor recovery.[32,34,35] However, somatosensation is usually neglected in the clinical assessment by the clinicians. Furthermore there are limited numbers of outcome measures which evaluate somatosensorial function of stroke patients. Revised Nottingham Sensation Assessment and Fugl-Meyer Assessment Sensory Subscale are two easy scoring outcome measures that allow detailed sensory evaluation. The only disadvantage of the scales is the length of the completion time but it will be useful to reveal the sensory loss of the stroke patients.

III. BIOMECHANICAL AND KINEMATIC ALTERATIONS OF THE UPPER LIMB AFTER STROKE: KEY POINTS DURING CLINICAL ASSESSMENT

TRUNK

Unlike extremities, the muscles in the trunk are affected multidirectionally and bilaterally after stroke. Electromyography studies searching anticipatory postural adjustments of axial-lateral and posterior-anterior trunk mucles during UE and lower extremity flexion reported severe impairments in the activity of trunk muscles. These impairments are characterized as reduced activity of lateral muscles, reduced synchronized activation of pertinent muscles and delayed onset.[36] Dickstein, Shefi, Marcovitz and Villa have shown a reduction in the activity of latissimus dorsi, rectus abdominus and external oblique muscles of the impaired side.[37] This reduced activity has been especially between ipsilateral lateral trunk muscles, i.e. latissimus dorsi and external oblique muscles. In addition, reduced activity of bilateral erector spinae muscles compared to healthy subjects and significant difference between impaired and non-impaired side have been demonstrated.

In early isokinetic studies, it is reported that the strength of trunk extensor, flexor and rotator muscles in stroke patients were weaker compared to healthy subjects. Additionally, peak torks of flexor and extensor muscles were lower, but there was no difference in the performance of rotator muscles.[38,39] In the current literature, the opinion of less trunk muscle performance compared to healthy peers still stand, however, it is particularly investigated in relation with gait performance.[40-42] In a recent meta-analysis investigating the trunk and upper extremity kinematics during reaching to a target, it is stated that stroke patients showed a greater trunk displacement and trunk contribution during reaching.[43] The reason for the increased flexion (i.e.contribution) of trunk may be the compensation of reduced elbow extension during the reaching activity of impaired UE.[44] Similarly to the early findings, trunk rotation kinematics were similar to healthy subjects.[43]

De Baets, Van Deun, Monari and Jaspers studied scapular and trunk kinematics during shoulder flexion in stroke subjects and found that healthy subjects showed ipsilateral lateral flexion and contralateral axial rotation in the trunk at the beginning of the movement whereas contralateral lateral flexion and ipsilateral axial rotation was seen in the subjects with stroke.[45] In another study, Johansson, Grip, Levin and Häger investigated kinematic parameters of Finger-to-Nose Test (FNT) and reported that total movement time, pointing time, time to peak speed, scapular and trunk movements were increased whereas accuracy and peak speed were decreased in stroke subjects compared to control subjects.[46]

As described above, trunk muscles are impaired after stroke affecting the UE performance. Thus, the assessment of trunk muscles with either a questionnaire or different analysis methods should be included in the examination. There are several scales for the assessment of trunk performance in the literature such as Trunk Impairment Scale, the Trunk Control Test, the Postural Assessment Scale for Stroke, the Ottowa Sitting Scale.[47] It is worthy of note that The Trunk Impairment Scale by Fujiwara et al. is the most used, valid and reliable standardized scale in patients with stroke.[48]

SCAPULA

Trunk deviates toward the impaired side in the flaccid stage and therefore, scapula descend from its normal horizontal level. This is called as "scapular depression".[49] Since trapezius and serratus anterior muscles are flaccid, scapular downward rotation is also seen in addition to scapular depression.[50-52] Humeral head moves towards the inferior as a result of contributing factors such as decreased tone in the rotator cuff, upper trapezius and serratus anterior muscles, downward rotation posture of the scapula and the gravity. This may lead to glenohumeral joint subluxation.[49] However, it is important to underline the findings of a study by Price et al. which show that the scapular resting position (such as scapular downward rotation) may not responsible for the glenohumeral subluxation.[51]

Pectoralis major and minor, rhomboids, levator scapulae and latissimus dorsi muscles become hypertonic with the initiation of spastic stage and scapula is pulled towards downward rotation.[49,53] As the muscle groups affected by the spasticity become more dominant, muscular imbalance may change and this creates a posture called "spastic muscle pattern". Scapular depression and retraction occur due to dominant flexor tone of upper extremity.[54]

At the level of glenohumeral complex, motor impairments such as muscle weakness, increased muscle tone, pathological muscle synergies and altered temporal muscle activity may inhibit scapulohumeral control. This results in the adaptation of scapular position and movement according to the humeral position.[55-57]

The scapular changes that occur in normal or dynamic states are called "scapular dyskinesia".[58] Scapular dyskinesia is a general term to define loss of scapular control and movement.[53] The reduction in scapulohumeral control creates a difficulty during movements of extremities of the impaired side and the risk of hemiplegic shoulder pain may increase.[59,60] De Baets, Jaspers, Janssen and Van Deun reported that the shoulder pain in stroke patients originated from abnormal recruitment of infraspinatus, serratus anterior and inferior trapezius muscles during humeral movement.[55] This abnormal recruitment was also reported in subjects with shoulder impingement syndrome in the literature.[61,62] De Baets et al. found that stroke subjects showed lesser posterior scapular tilt during the elevation phase of 90° shoulder flexion and grater scapular lateral rotation during the lowering phase of 90° shoulder flexion than the healthy control group.[45] In the EMG analysis, earlier lower trapezius and late infraspinatus offset in addition to late onset and earlier offset in the serratus anterior muscle were found in stroke patients during the 45° shoulder flexion. Along with these results, it is also found that onset of upper trapezius, anterior deltoid and infraspinatus muscles were earlier than onset of lower trapezius and serratus anterior muscles. However, offset of lower trapezius and serratus anterior muscles were earlier than offset of the upper trapezius, anterior deltoid and infraspinatus muscles.

Similarly, Lixandrao et al. studied scapular kinematics in scapular and self-selected plans during arm elevation and lowering and during hair combing.[63] As a result, the authors found increased scapular internal rotation of impaired and non-impaired side extremities in stroke subjects during arm lowering in scapular plane and during arm elevation and lowering in self-selected plans in addition to increased scapular internal rotation seen in the stroke subjects during hair combing. Increased scapular anterior tilt of impaired side was observed in stroke subjects during elevation phase of hair combing, and during the arm elevation and lowering in the scapular and self-selected plans. Similar results were found in the non-impaired side and increased anterior tilt of scapula was observed during arm elevation and depression in the self-selected plans. There was no difference in scapular upward rotation among neither arms. Additionally, a current study showed that the acromial displacement during FNT was significantly different between mild and moderate stroke patients.[46]

Numerous assessment methods are used for the measurement of the degree of scapular dyskinesis, subjectively by visual evaluation and objectively by either a 3-dimensional electromagnetic device or 2-dimensional clinically applicable methods.[64-67] However, a recent review suggested that visual observation and inclinometric methods are also applicable since the 3-dimensional devices are rarely available in the clinics.[67] In addition to this recommendation, we would like to underline the fact that the most-known and applied visual assessment method is Scapular Dyskinesis Test which may be easily applied by clinicians.[65,68]

SHOULDER

As the flaccidity leaves its place to the spasticity after stroke, humeral internal rotation starts in response to increased activity of subscapularis and pectoralis muscles. Other internal rotator muscles of glenohumeral joint such as teres major and latissimus dorsi also contribute to the deformity. Adduction is another component of shoulder tightness in stroke patients and is a consequence of increased activity of teres major and latissimus dorsi muscles.[69] The most frequent problem in the shoulder assessment is the co-existence of flexion, internal rotation and adduction pattern due to hyperactivity in the pectoralis major muscle. One should be aware of that if the shoulder extension is accompanied by adduction and internal rotation, it may be the result of latissimus dorsi muscle contributing the deformity.[69]

It has also been reported that glenoid cavity was placed in a more downward rotation due to scapular depression and rotation that occured as a result of decreased muscle tone seen in flaccid stage.[49,70,71] Besides, they found that the downward rotation of glenoid cavity may harm to the "locking mechanism" normally provided by upper tilt of glenoid cavity and thus it may cause glenohumeral subluxation.

While these disorders and biomechanical changes should be evaluated together, it is easy to see that the structured scales remain in one dimension. For instance; the upper extremity subscale of Fugl-Meyer Assessment evaluates the severity of UE synergies whereas the WFMT merely examine the disorder in the shoulder. On the other hand, MESUPES, which is less mentioned in the literature, provides information particularly about the quality of the proximal movement in the upper extremity. However, the evaluation of all of these dimensions should be performed in one setting, i.e. together. In this way, it may become clearer which of the rehabilitation applications should be given priority.

ELBOW, FOREARM AND WRIST

Post-stroke spasticity results an alteration in the movement patterns such as decreased muscle extensibility and decreased cortical involvement required to achieve upper extremity functions. The patterns, which are called "compensatory movement strategies", consist of flexor synergy (shoulder flexion, shoulder abduction, elbow flexion, forearm supination) and extension synergy (shoulder extensor, shoulder adduction, elbow extension, forearm pronation).[72] Brachialis is the strongest flexor in the elbow and elbow flexion is accompanied by the pronation of forearm in resting. However, it is necessary to observe which position the forearm is located to find out which muscle is more affected by the tone increase in the elbow.

The posture of the forearm (supination, neutral or pronation) should be observed to determine muscles contributing the flexion deformity in the elbow. The depression of the humeral head following the elbow flexion is associated with hyperactive biceps brachii muscle and in this case, forearm is positioned in supination by prominent biceps tone. Full flexion and extension movements may be used for determination of pronator tightness.

The pronation that occurs when the elbow is in full flexion shows the hyperactivity of the pronator quadratus muscle whereas the pronation that occurs when the elbow is in full extension indicates the overactivity of the pronator teres muscle.[69]

The flexor muscles of the wrist (flexor carpi radialis and ulnaris muscles) contributes the wrist flexion deformity after stroke. Radial or ulnar deviation seen in the elbow is associated with flexor carpi radialis or ulnaris, respectively. The postures of the hand and fingers occur in a similar manner. Middle or distal phalanx of the fingers (in some cases both of the phalanxes) are placed in a flexion posture by either flexor digitorum superficialis or flexor digitorum profundus muscles, respectively.[69]

In studies analyzing the upper extremity function focusing on the elbow and forearm region after stroke, more kinematic data related to these regions are presented. To the best of our knowledge, most studied function in the literature is reaching activity. Studies have shown that trunk flexion and shoulder abduction are more performed to compensate the reduced elbow extension during the extension of the impaired limb to reach a target.[73] Whereas greater shoulder abduction and wrist extension are involved during the bringing the cup grasped with impaired hand to the mouth, lesser shoulder flexion and forearm pronation are occured.[72]

In a study by Lee et al. it was stated that there was no significant difference in total movement time and reaction time between control and mild stroke subjects during the door handling task, however total movement units (pronation+supination) and time of the supination phase were increased and peak velocity in the supination was decreased in stroke subjects.[73] When the authors compared the control subjects and moderate stroke subjects, they found a significant difference in reaction time, peak velocity (pronation+supination), total movement time and total movement units (pronation+supination). In addition to these findings, authors reported that there was significant difference in reaction time, total movement time and total movement units between mild stroke subjects and moderate stroke subjects.

CONCLUSION

The altered UE biomechanics and kinematics associated with the impairments after stroke may cause several difficulties during the assessment procedure. Besides, considering the result-oriented structures of the measurements, it is hard for a clinician to assess UE qualitatively and examine the true performance. This leads to lack of understanding about the situation of UE for the clinicians. However, in this paper, the key points of assessments related to these alterations were highlighted and numerous evaluation techniques were mentioned. In the light of current literature, it was seen that there is no outcome measure that assess the whole aspects of the impairment. We suggest to include more than one outcome measurements which is related to the activity limitation to be able to examine all the above-mentioned impairments and alterations at once. In this way, a more accurate planning on the rehabilitation interventions may be possible.

Source of Finance

During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.

Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

***Idea/Concept:** Esma Nur Kobaşı, Burcu Ersöz Hüseyinoğlu; **Design:** Esma Nur Kolbaşı, Burcu Ersöz Hüseyinoğlu; **Control/Supervision:** Burcu Ersöz Hüseyinoğlu; **Analysis and/or Interpretation:** Esma Nur Kobaşı, Burcu Ersöz Hüseyinoğlu; **Literature Review:** Esma Nur Kobaşı; **Writing the Article:** Esma Nur Kobaşı, Burcu Ersöz Hüseyinoğlu; **Critical Review:** Esma Nur Kobaşı, Burcu Ersöz Hüseyinoğlu.*

REFERENCES

1. Nakayama H, Jørgensen HS, Raaschou HO, Olsen TS. Compensation in recovery of upper extremity function after stroke: the Copenhagen Stroke Study. Arch Phys Med Rehabil. 1994;75 (8):852-7. [Crossref]
2. Parker VM, Wade DT, Langton Hewer R. Loss of arm function after stroke: measurement, frequency, and recovery. Int Rehabil Med. 1986;8(2):69-73. [Crossref] [PubMed]
3. Wade DT. Measurement in neurological rehabilitation. Curr Opin Neurol Neurosurg. 1992;5(5): 682-6.
4. Persson HC, Parziali M, Danielsson A, Sunnerhagen KS. Outcome and upper extremity function within 72 hours after first occasion of stroke in an unselected population at a stroke unit. A part of the SALGOT study. BMC Neurol. 2012;12:162. [Crossref] [PubMed] [PMC]
5. Lawrence ES, Coshall C, Dundas R, Stewart J, Rudd AG, Howard R, et al. Estimates of the prevalence of acute stroke impairments and disability in a multiethnic population. Stroke. 2001;32(6): 1279-84. [Crossref] [PubMed]
6. Cramer SC, Nelles G, Benson RR, Kaplan JD, Parker RA, Kwong KK, et al. A functional MRI study of subjects recovered from hemiparetic stroke. Stroke. 1997;28(12):2518-27. [Crossref] [PubMed]
7. Lang CE, Bland MD, Bailey RR, Schaefer SY, Birkenmeier RL. Assessment of upper extremity impairment, function, and activity after stroke: foundations for clinical decision making. J Hand Ther. 2013;26(2):104-14. [Crossref] [PubMed] [PMC]
8. Aprile I, Rabuffetti M, Padua L, Di Sipio E, Simbolotti C, Ferrarin M. Kinematic analysis of the upper limb motor strategies in stroke patients as a tool towards advanced neurorehabilitation strategies: a preliminary study. Biomed Res Int. 2014;2014:636123. [Crossref] [PubMed] [PMC]
9. van Dokkum L, Hauret I, Mottet D, Froger J, Metrot J, Laffont I. The contribution of kinematics in the assessment of upper limb motor recovery early after stroke. Neurorehabil Neural Repair. 2014;28(1):4-12. [Crossref] [PubMed]
10. de los Reyes-Guzmán A, Dimbwadyo-Terrer I, Trincado-Alonso F, Monasterio-Huelin F, Torricelli D, Gil-Agudo A. Quantitative assessment based on kinematic measures of functional impairments during upper extremity movements: a review. Clin Biomech (Bristol, Avon). 2014;29(7):719-27. [Crossref] [PubMed]
11. Alt Murphy M, Willén C, Sunnerhagen KS. Movement kinematics during a drinking task are associated with the activity capacity level after stroke. Neurorehabil Neural Repair. 2012;26(9):1106-15. [Crossref] [PubMed]
12. Cacho EW, de Oliveira R, Ortolan RL, Varoto R, Cliquet A Jr. Upper limb assessment in tetraplegia: clinical, functional and kinematic correlations. Int J Rehabil Res. 2011;34(1):65-72. [Crossref] [PubMed]
13. Johansson GM, Grip H, Levin MF, Häger CK. The added value of kinematic evaluation of the timed finger-to-nose test in persons post-stroke. J Neuroeng Rehabil. 2017;14(1):11. [Crossref] [PubMed] [PMC]
14. Santisteban L, Térémetz M, Bleton JP, Baron JC, Maier MA, Lindberg PG. Upper limb outcome measures used in stroke rehabilitation studies: a systematic literature review. PLoS One. 2016;11(5):e0154792. [Crossref] [PubMed] [PMC]
15. See J, Dodakian L, Chou C, Chan V, McKenzie A, Reinkensmeer DJ, et al. A standardized approach to the Fugl-Meyer assessment and its implications for clinical trials. Neurorehabil Neural Repair. 2013;27(8):732-41. [Crossref] [PubMed]
16. Woodbury ML, Velozo CA, Richards LG, Duncan PW, Studenski S, Lai SM. Dimensionality and construct validity of the Fugl-Meyer Assessment of the upper extremity. Arch Phys Med Rehabil. 2007;88(6):715-23. [Crossref] [PubMed]
17. van der Lee JH, Beckerman H, Knol DL, de Vet HC, Bouter LM. Clinimetric properties of the motor activity log for the assessment of arm use in hemiparetic patients. Stroke. 2004;35(6):1410-4. [Crossref] [PubMed]
18. Ersöz Hüseyinsinoğlu B, Razak Özdinçler A, Erkan Oğul Ö, Krespi Y. [Reliability and validity of Turkish version of motor activity log-28]. Turk J Neurol. 2011;17(2):83-9.
19. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther. 1987;67(2):206-7. [Crossref] [PubMed]
20. Sathian K, Buxbaum LJ, Cohen LG, Krakauer JW, Lang CE, Cobetta M, et al. Neurological principles and rehabilitation of action disorders: common clinical deficits. Neurorehabil Neural Repair. 2011;25(5 Suppl):21S-32S. [Crossref] [PubMed] [PMC]
21. Hatem SM, Saussez G, Della Faille M, Prist V, Zhang X, Dispa D, et al. Rehabilitation of motor function after stroke: a multiple systematic review focused on techniques to stimulate upper extremity recovery. Front Hum Neurosci. 2016;10:442. [Crossref] [PubMed] [PMC]
22. McMorland AJ, Runnalls KD, Byblow WD. A neuroanatomical framework for upper limb synergies after stroke. Front Hum Neurosci. 2015;9:82. [Crossref] [PubMed] [PMC]
23. Brunnström S. Movement Therapy in Hemiplegia: A Neurophysiological Approach. 1st ed. New York: Harper & Row; 1970. p.192.
24. Otman S. Hemipleji Rehabilitasyonunda Nörofizyolojik Yaklaşımlar. 2. Baskı. Ankara: HÜ FTR YO Yayınları; 2001. p.17-20.

25. Roh J, Rymer WZ, Beer RF. Evidence for altered upper extremity muscle synergies in chronic stroke survivors with mild and moderate impairment. Front Hum Neurosci. 2015;9:6. [Crossref] [PubMed] [PMC]

26. Roh J, Rymer WZ, Perreault EJ, Yoo SB, Beer RF. Alterations in upper limb muscle synergy structure in chronic stroke survivors. J Neurophysiol. 2013;109(3):768-81. [Crossref] [PubMed] [PMC]

27. Fugl-Meyer AR, Jääskö L, Leyman I, Olsson S, Steglind S. The post-stroke hemiplegic patient. 1. a method for evaluation of physical performance. Scand J Rehabil Med. 1975;7(1):13-31.

28. Kopp B, Kunkel A, Flor H, Platz T, Rose U, Mauritz KH, et al. The Arm Motor Ability Test: reliability, , and sensitivity to change of an instrument for assessing disabilities in activities of daily living. Arch Phys Med Rehabil. 1997;78(6):615-20. [Crossref]

29. Wolf SL, Thompson PA, Morris DM, Rose DK, Winstein CJ, Taub E, et al. The EXCITE trial: attributes of the Wolf Motor Function Test in patients with subacute stroke. Neurorehabil Neural Repair. 2005;19(3):194-205. [Crossref] [PubMed]

30. Malouin F, Pichard L, Bonneau C, Durand A, Corriveau D. Evaluating motor recovery early after stroke: comparison of the Fugl-Meyer assessment and the Motor Assessment Scale. Arch Phys Med Rehabil. 1994;75(11):1206-12. [Crossref]

31. Carey LM. Somatosensory loss after stroke. Crit Rev Phys Rehabil Med. 1995;7(1):51-91. [Crossref]

32. Kessner SS, Bingel U, Thomalla G. Somatosensory deficits after stroke: a scoping review. Top Stroke Rehabil. 2016;23(2):136-46. [Crossref] [PubMed]

33. Tyson SF, Crow JL, Connell L, Winward C, Hillier S. Sensory Impairments of the lower limb after stroke: a pooled analysis of individual patient data. Top Stroke Rehabil. 2013;20(5):441-9. [Crossref] [PubMed]

34. Patel AT, Duncan PW, Lai SM, Studenski S. The relation between impairments and functional outcomes poststroke. Arch Phys Med Rehabil. 2000;81(10):1357-63. [Crossref] [PubMed]

35. Vidoni ED, Acerra NE, Dao E, Meehan SK, Boyd LA. Role of the primary somatosensory cortex in motor learning: an rTMS study. Neurobiol Learn Mem. 2010;93(4):532-9. [Crossref] [PubMed]

36. Karthikbabu S, Chakrapani M, Ganeshan S, Rakshith KC, Nafeez S, Prem V. A review on assessment and treatment of the trunk in stroke:A need or luxury. Neural Regen Res. 2012;7(25):1974-7.

37. Dickstein R, Shefi S, Marcovitz E, Villa Y. Electromyographic activity of voluntarily activated trunk flexor and extensor muscles in post-stroke hemiparetic subjects. Clin Neurophysiol. 2004;115(4): 790-6. [Crossref] [PubMed]

38. Tanaka S, Hachisuka K, Ogata H. Trunk rotatory muscle performance in post-stroke hemiplegic patients. Am J Phys Med Rehabil. 1997;76(5):366-9. [Crossref] [PubMed]

39. Tanaka S, Hachisuka K, Ogata H. Muscle strength of trunk flexion-extension in post-stroke hemiplegic patients. Am J Phys Med Rehabil. 1998;77(4):288-90. [Crossref] [PubMed]

40. Quintino LF, Franco J, Gusmao AFM, Silva PFS, Faria CDCM. Trunk flexor and extensor muscle performance in chronic stroke patients: a case-control study. Braz J Phys Ther. 2018;22(3):231-7. [Crossref] [PubMed] [PMC]

41. Van Criekinge T, Saeys W, Hallemans A, Velghe S, Viskens PJ, Vereeck L, et al. Trunk biomechanics during hemiplegic gait after stroke: a systematic review. Gait Posture. 2017;54:133-43. [Crossref] [PubMed]

42. Aguiar LT, Camargo LBA, Estarlino LD, Teixeira-Salmela LF, Faria CDCM. Strength of the lower limb and trunk muscles is associated with gait speed in individuals with sub-acute stroke: a cross-sectional study. Braz J Phys Ther. 2018;22(6):459-66. [Crossref] [PubMed] [PMC]

43. Collins KC, Kennedy NC, Clark A, Pomeroy VM. Kinematic components of the reach-to-target movement after stroke for focused rehabilitation interventions: systematic review and meta-analysis. Front Neurol. 2018;9:472. [Crossref] [PubMed] [PMC]

44. Roby-Brami A, Feydy A, Combeaud M, Biryukova EV, Bussel B, Levin MF. Motor compensation and recovery for reaching in stroke patients. Acta Neurol Scand. 2003;107(5):369-81. [Crossref] [PubMed]

45. De Baets L, Van Deun S, Monari D, Jaspers E. Three-dimensional kinematics of the scapula and trunk, and associated scapular muscle timing in individuals with stroke. Hum Mov Sci. 2016;48:82-90. [Crossref] [PubMed]

46. Johansson GM, Grip H, Levin MF, Häger CK. The added value of kinematic evaluation of the timed finger-to-nose test in persons post-stroke. J Neuroeng Rehabil. 2017;14(1):11. [Crossref] [PubMed] [PMC]

47. Sorrentino G, Sale P, Solaro C, Rabini A, Cerri CG, Ferriero G. Clinical measurement tools to assess trunk performance after stroke: a systematic review. Eur J Phys Rehabil Med. 2018;54(5):772-84. [Crossref] [PubMed]

48. Fujiwara T, Liu M, Tsuji T, Sonoda S, Mizuno K, Akaboshi K, et al. Development of a new measure to assess trunk impairment after stroke (trunk impairment scale): its psychometric properties. Am J Phys Med Rehabil. 2004;83(9):681-8. [Crossref] [PubMed]

49. Caillet R. The Shoulder in Hemiplegia. 3rd ed. Philadelphia (PA): FA Davis Co; 1980. p. 130.

50. Paci M, Nannetti L, Rinaldi LA. Glenohumeral subluxation in hemiplegia: an overview. J Rehabil Res Dev. 2005;42(4):557-68. [Crossref] [PubMed]

51. Price CI, Rodgers H, Franklin P, Curless RH, Johnson GR. Glenohumeral subluxation, scapula resting position, and scapula rotation after stroke: a noninvasive evaluation. Arch Phys Med Rehabil. 2001;82(7):955-60. [Crossref] [PubMed]

52. Karaahmet OZ, Eksioglu E, Gurcay E, Karslı PB, Tamkan U, Bal A, et al. Hemiplegic shoulder pain: associated factors and rehabilitation outcomes of hemiplegic patients with and without shoulder pain. Top Stroke Rehabil. 2014;21(3):237-45. [Crossref] [PubMed]

53. Dabholkar A, Mehta D, Yardi S, Dabholkar T. Assessment of scapular behavior in stroke patients. Int J Health Rehabil Sci. 2015;4(2):95-102. [Crossref]

54. Murie-Fernández M, Carmona Iragui M, Gnanakumar V, Meyer M, Foley N, Teasell R. [Painful hemiplegic shoulder in stroke patients: causes and management]. Neurologia. 2012;27(4):234-44. [Crossref] [PubMed]

55. De Baets L, Jaspers E, Janssens L, Van Deun S. Characteristics of neuromuscular control of the scapula after stroke: a first exploration. Front Hum Neurosci. 2014;8:933. [Crossref] [PubMed] [PMC]

56. Frontera WR, Grimby L, Larsson L. Firing rate of the lower motoneuron and contractile properties of its muscle fibers after upper motoneuron lesion in man. Muscle Nerve. 1997;20(8):938-47. [Crossref]

57. De Baets L, Jaspers E, Van Deun S. Scapulohumeral control after stroke: a preliminary study of the test-retest reliability and discriminative validity of a clinical scapular protocol (ClinScaP). NeuroRehabilitation. 2016;38(4):359-70. [Crossref] [PubMed]

58. Kibler WB, Ludewig PM, McClure PW, Michener LA, Bak K, Sciascia AD. Clinical implications of scapular dyskinesis in shoulder injury: the 2013 consensus statement from the 'Scapular Summit'. Br J Sports Med. 2013;47(14):877-85. [Crossref] [PubMed]

59. Rundquist PJ, Dumit M, Hartley J, Schultz K, Finley MA. Three-dimensional shoulder complex kinematics in individuals with upper extremity impairment from chronic stroke. Disabil Rehabil. 2012;34(5):402-7. [Crossref] [PubMed]

60. Niessen M, Janssen T, Meskers C, Koppe P, Konijnenbelt M, Veeger D. Kinematics of the contralateral and ipsilateral shoulder: a possible relationship with post-stroke shoulder pain. J Rehabil Med. 2008;40(6):482-6. [Crossref] [PubMed]

61. Moraes GF, Faria CD, Teixeira-Salmela LF. Scapular muscle recruitment patterns and isokinetic strength ratios of the shoulder rotator muscles in individuals with and without impingement syndrome. J Shoulder Elbow Surg. 2008;17(1 Suppl):48S-53S. [Crossref] [PubMed]

62. Worsley P, Warner M, Mottram S, Gadola S, Veeger HE, Hermens H, et al. Motor control retraining exercises for shoulder impingement: effects on function, muscle activation, and biomechanics in young adults. J Shoulder Elbow Surg. 2013;22(4):e11-9. [Crossref] [PubMed] [PMC]

63. Lixandrão MC, Camargo PR, Scarpa CE, Prado-Medeiros CL, Salvini TF. Bilateral changes in 3-D scapular kinematics in individuals with chronic stroke. Clin Biomech (Bristol, Avon). 2017;47:79-86. [Crossref] [PubMed]

64. Tate AR, McClure P, Kareha S, Irwin D, Barbe MF. A clinical method for identifying scapular dyskinesis, part 2: validity. J Athl Train. 2009;44(2):165-73. [Crossref] [PubMed] [PMC]

65. De Baets L, Jaspers E, Desloovere K, Van Deun S. A systematic review of 3D scapular kinematics and muscle activity during elevation in stroke subjects and controls. J Electromyogr Kinesiol. 2013;23(1):3-13. [Crossref] [PubMed]

66. Larsen CM, Juul-Kristensen B, Lund H, Søgaard K. Measurement properties of existing clinical assessment methods evaluating scapular positioning and function. A systematic review. Physiother Theory Pract. 2014;30(7):453-82. [Crossref] [PubMed]

67. McClure P, Tate AR, Kareha S, Irwin D, Zlupko E. A clinical method for identifying scapular dyskinesis, part 1: reliability. J Athl Train. 2009;44(2):160-4. [Crossref] [PubMed] [PMC]

68. Marciniak C. Poststroke hypertonicity: upper limb assessment and treatment. Top Stroke Rehabil. 2011;18(3):179-94. [Crossref] [PubMed]

69. Davies PM. Steps to Follow: A Guide to the Treatment of Adult Hmiplegia. 1st ed. Berlin: Springer-Verlag; 1985. p.300. [Crossref]

70. Ryerson S, Levit K. The shoulder in hemiplegia. In: Donatelli RA, ed. Physical Therapy of The Shoulder. 3rd ed. New York: Churchill Livingstone; 2004. p.205-27. [Crossref]

71. Liu W, McCombe Waller S, Kepple TM, Whitall J. Compensatory arm reaching strategies after stroke: induced position analysis. J Rehabil Res Dev. 2013;50(1):71-84. [Crossref] [PubMed] [PMC]

72. McCrea PH, Eng JJ, Hodgson AJ. Saturated muscle activation contributes to compensatory reaching strategies after stroke. J Neurophysiol. 2005;94(5):2999-3008. [Crossref] [PubMed] [PMC]

73. Lee JA, Kim EJ, Hwang PW, Park HR, Bae JH, Kim JN. Three-dimensional kinematic motion analysis of door handling task in people with mild and moderate stroke. Phys Ther Rehabil Sci. 2016;5(3):143-8. [Crossref]