Kinematic and Electromyographic Analysis of Knee Motion During Rising from A Chair in Stroke Patients

Nevein M.M. Gharib*, Manal M Ismail and Sahar M. Adel*****
*Department of Neuromuscular Disorders and its Surgery, Faculty of Physical Therapy, Cairo University.
**Department of Orthopedic Physical Therapy, Faculty of Physical Therapy, Cairo University.
***Department of Physical Therapy for Basic Science. Faculty of Physical Therapy, Cairo University.

ABSTRACT

Purpose of the study: *To evaluate knee joint motion including kinematic and electromyographic (EMG) activities of vastus lateralis (VL) and biceps femoris (BF) muscles during rising from a chair in stroke patients (both affected and non-affected sides) and normal subjects.* ***Methods:*** *Forty stroke patients and ten normal age matched subjects participated in this study. The patients were assigned into two equal groups according to the degree of spasticity of the affected lower limbs (mildly spastic GIa and moderately spastic GIb). All subjects were assessed for range of motion (ROM) of the knee joint and EMG activity of VL and BF during rising from a chair.* ***Results:*** *There was a statistically significant differences among the three groups regarding the EMG activities of VL (the highest value in GIb) and BF (the lowest value in GIb) in both the affected and nonaffected sides. Additionally, there was a statistically significant differences of the knee joint ROM among the affected sides and normal subjects (except at phase II) with the highest value in GIb (phases I and IV) and GII (phases II and III) and among the nonaffected sides and normal subjects (except phase I) with the highest value in GIb (phases I and II) and GII (phases III and IV).* ***Conclusion:*** *Stroke patients suffer from asymmetrical pattern of ROM and EMG activities as compared to normal sujects. Therefore, assessment of knee joint motions during rising from a chair should be considered in rehabilitation of stroke patients.*
Key words: *Stroke – Rising from a chair – Knee – Kinematic analysis – EMG.*

INTRODUCTION

Rising from a chair is one of the most mechanically demanding functional tasks routinely undertaken during daily activities[9,32]. This movement is at least equally important to normal human function as walking because rising from a sitting position is commonly a prerequisite to the initiation of ambulation[39] and is considered a major determining factor for independence among elderly people and people with disabilities[16]. Impairments in this normal biomechanical movement may put a person at risk of falling[23,24].

Sit-to-stand (STS) movement was divided into four phases[36]: (1) Phase I (the flexion momentum phase), it begins with initiation of the movement and ends just before lifting the buttocks from the seat of the chair (lift-off). During this phase, the head, arms and trunk rotate forward around the pelvis and hips (toward flexion) therefore, displacing the body's centre of gravity (COG) forward generating upper-body momentum. (2) Phase II (the momentum-transfer phase), it begins with the initiation of vertical momentum as the buttocks are lifted off the chair. During this phase, the knee and hip extensor muscles show peak activity; as soon as; the lower extremities are loaded after seat-off. The centre of mass (COM) traveled anteriorly and upward reaching its maximal anterior point shortly after maximum dorsiflexion. There is concentric activity of the quadriceps muscle at the knee with eccentric activity of the biceps femoris (BF) muscle at the knee and gluteus maximus muscle at the hip. The peak muscle activity occurs during this phase, despite its short duration. Weakness of the quadriceps muscle and erector muscles of the spine, in particular, contributes to difficulties during this crucial phase[24]. This phase ends with maximum ankle dorsiflexion. 3) Phase III (the extension phase), it follows momentum-transfer phase and ends when the hips are in extension. This phase is characterized by vertical movement of the body to full stance. During phase III, the knee-extension and head-flexion motions are also coming to an end and the maximum COG vertical displacement is obtained as the hip, knee and ankle extensor muscles propel the body mass vertically[26]. 4) Phase IV (the stabilization phase), this phase begins just after the hip-extension velocity reached 0°/sec and continued until all motion associated with stabilization occurs. The separation between phase III and phase IV is not easily defined because the subjects in this phase normally experience some anterior-posterior (A-P) and lateral sway. Near the end of STS task, there is a decelerating force that causes brake for the translation of the body mass forward. This force is developed by the extensors of the lower limbs to achieve stability of the body in standing position[4].

STS movement is affected in stroke patients as a result of difficulty generating timing and sufficient force in the lower limb extensor muscles to propel the body vertically[33] balance impairment, disturbed muscle tone, lack of selective control and learned nonuse syndrome[23]. To overcome the difficulty and successfully execute the task, stroke patients modify their movement strategies by modifications in the displacement of the COM and by an asymmetrical weight-bearing pattern[34]. They make excessive trunk flexion to decrease the force exerted by the extensor muscles (specially, knee extensors), leaning toward the non affected side as they can develop more force to overcome the weakness in the affected limb[4] The patients also perform this movement slowly to overcome the impaired balance and to decrease the demand on the lower limb muscles[24].

Rising from a chair produces greater knee torques than gait or stair climbing[19,24] and requires large movement particularly of the hip and knee[15] that places more demands on the knee joint during this task[13]. Therefore, assessment of STS movement including lower limb joints especially, the knee joint (including kinematic and electromyographic assessment), should be included in routine functional assessment and in fall prevention programs. The purpose of this study was to conduct a kinematic and electromyographic analysis of knee joint motion during rising from a chair in both stroke patients (including the affected and non-affected sides) and normal healthy subjects.

METHODS

Subjects

Forty stroke male patients (Group I) and ten normal age matched male subjects (group II) participated in this study. Patients were selected from the outpatient's clinic of the Faculty of Physical Therapy, Cairo University. The patients were assigned into two equal groups, according to the degree of spasticity of the affected lower limb in which GIa had mild spasticity and GIb had moderate spasticity. The general characteristics of the patients are represented in table (1).

Table (1): General characteristics of the patients.

Variable	GIa (n=20)	GIb (n=20)
Age (year) Range (mean±SD)	46 - 64 (53.9±6.67)	45 - 64 (53.3±5.02)
Duration of illness (month) Range (mean±SD)	6 -13 (8.65±2.41)	6 - 13 (8.45±2.18)
Type of stroke Infarction Hemorrhage	 15 5	 13 7
Affected side Right Left	 11 9	 8 12

All patients were medically stable and had the ability to stand up from sitting position and sit down independently (without any assistance) for three times. Patients were excluded if they had: 1) Other neurological or orthopedic diseases that my affect STS movement (such as, recurrent stroke, parkinsonism, severe osteoarthritis or rheumatoid arthritis), 2) Balance disorders due to other diseases rather than stroke (e.g., inner ear, vestibular or cerebellar dysfunctions), 3) Blindness, deafness, mental and cognitive affection (4) Marked superficial and/or deep sensory loss.

Instrumentations

I- Qualysis Motion Capture System was used to conduct the kinematic analysis of the knee joint motion during the four phases of STS movement for all subjects. The system consisted of six ProReflex infrared high speed cameras to perform multi camera measurements and have a capture capability of 120 frames/sec. The basic principle of the system was to expose reflective markers to infrared light and to detect the light reflected by the markers. The 2-dimentional (2-D) image of the markers was processed and the 2-D data from the six cameras were combined for calculating the 3-D positions of the markers.

The software program used were Q trac and Q tools (provided by Qualisys Company). Ten skin markers (five in each side) were stuck to the skin over specific bony landmarks by double face adhesive plaster[20]. The location of cameras and their spatial orientation remain unchanged during the study. The markers were placed bilaterally as following; two on the acromion processes, two on greater trochanters, two on the lateral aspect of the knee joint line (the width of the lateral aspect of the knee when the patella is excluded; is divided into two equal parts and the marker is applied in the middle), two on the lateral malleoli, two over head of the fifth metatarsal bone. All markers were placed on all subjects by the same examiner for placement consistency[31].

II- Dynamic (Kinesiological) EMG unit (Biopac, TEL100-C, MP system) was used to evaluate the EMG activities (root mean square [RMS]) of quadriceps [vastus lateralis (VL)] and lateral part of hamstrings [biceps femoris (BF)] of the affected and non-affected sides in the stroke patients (GI) and from the right side in the normal subjects (GII). This EMG unit consists of: A) Four channels EMG apparatus, B) Disposable surface EMG electrodes: to record the EMG activity from the specified muscles. Each channel collects the data by three electrodes (active, reference and ground) and C) Data processing computer unit.

The EMG data collection was synchronized with Qualisys Motion Capture System so that when the Qualisys Motion Capture System works, the EMG apparatus works at the same time. This procedure allowed correlating the angle of the knee to the muscular activities of the two muscles.

Assessment procedure

- The camera system was calibrated, before any 3D capturing to enable the cameras to pick up the positions of the markers in the trajectory field. The position of cameras and their spatial orientation remain unchanged during the study. Any relocation of the cameras required re-calibration[18].
- Skin preparation including shaving of hair and cleaning of skin with cotton and alcohol was conducted.

EMG disposable electrodes were attached and secured in their locations after skin preparation by adhesive plaster. The electrodes pairs were placed with an inter-electrode distance of 20 mm (center to center), aligned with the direction of the muscle fibers and secured with adhesive tape. The ground electrode for each muscle was placed over the dorsal aspect of the hand (one on each hand). Recording of EMG data was collected from VL and BF.

The location of the electrodes for the selected muscles was determined by using marker pen, tape measurement and protractor. The electrodes were placed on the selected muscles as follows:

- Vastus lateralis (VL): 16 cm superior and six to eight cm lateral to the superior border of the patella and orientated 15° to the vertical[10].
- Biceps femoris (BF): At the level of mid thigh on a line drawn between the ischial tuberosity and the distal tendon of these muscles[25].

Proper electrode placement was verified by viewing on-line raw EMG on an oscilloscope during the corresponding motion of each muscle[37].

The skin markers were stuck to the skin over specific bony landmarks.

After calibration, the subjects were prepared by placing the markers at their proper positions over the specific bony land marks.

Each subject was seated on an arm restless and backless chair with adjustable height with the two feet were at the same level and fully supported on the ground, the knee angle was 105° (from full extension)[9,12].

The patient was asked to stand up with his self paced (natural, comfortable) speed without using his upper limb by folding them in front of his chest[21]. This procedure was repeated three to five times till the patient was familiar with the procedure then the measurements were recorded three times (with one minute rest in between to avoid fatigue) and the average was taken after analysis of the data. Patients were instructed to stand up and not to sit until they were asked to do so.

The raw EMG data were sampled at 500 Hz, amplified and band-pass filtered (30 to 500 Hz). According to the manufacturer's technical specifications, the common mode rejection ratio was 110 dB min. at 60 Hz, amplifier noise was 0.1 µV RMS and input impedance was equal two MΩ. The signals were analogue to digital converted and relayed to a computer-based data acquisition and analysis system for later analysis. EMG apparatus was set at a gain of 10,000 and acquisition time from five to eight sec. for each trial (according to the time taken to stand up from sitting position).

RESULTS

A- Kinematic analysis of knee joint motion:

As shown in table (2), there was a statistically significant difference in the mean values of the knee joint ROM among the affected side of GIa, GIb and the right side of GII during phases I, III and IV (P=0.0001 for all), However, there was a non-significant difference among them during phase II (P=0.26). Moreover, there was a statistically significant difference among the non-affected side of GIa, GIb and the right side of GII during phases II, III and IV (P=0.0001 for all). However, there was a non-significant difference among them during phase I (P=0.34) (Table 2, Fig. 1). Post Hoc LSD pair comparisons test between each two of the three groups as regarding to the mean values of the knee joint ROM revealed a significant difference between GIa and GIb concerning the affected side in all 4 phases, and a significant difference between GIa and GII was found concerning phase III and IV and phase II of the non affected side. A significant difference was found between GIb and GII concerning all phases for affected side except phase two and phase I of the non affected side.

Table (2): The mean values of knee joint ROM (degrees) and EMG activity of VL and BF muscles of both the affected and non-affected sides in GIa, GIb and the right side in GII during the four phases of STS movement.

Variable			GIa	GIb	GII (Rt. Side)	F-value	P-value
Knee joint ROM (degree)	Phase I	Affected	5.45±0.51	6.6±0.5	4.9±1.37	20.59	0.0001
		Non-affected	4.7±0.73	5.1±0.64		1.077	0.34*
	Phase II	Affected	37.45±3.21	38.35±2.3	36.5±3.5	1.37	0.26*
		Non-affected	39.3±2.57	40.95±2.39		8.998	0.0001
	Phase III	Affected	55.75±3.17	49.6±2.45	62.5±4.52	55.058	0.0001
		Non-affected	58.35±3.21	57.15±3.16		8.013	0.001
	Phase IV	Affected	4.95±4.4	11.1±4.9	1±0.81	21.815	0.0001
		Non-affected	0.5±0.51	0.15±0.36		8.446	0.001

*Non-significant

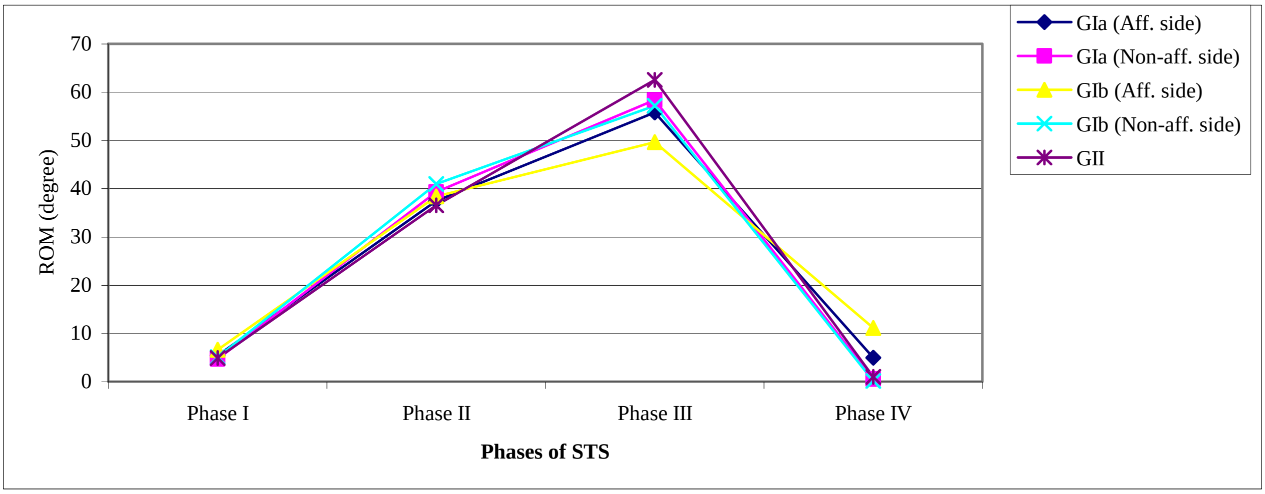

Fig. (1): The mean values of the knee joint ROM of both affected and non-affected sides in GIa, GIb and the right side in GII during the four phases of STS movement.

B- Electromyographic analysis of VL muscle activity:

There was a statistically significant difference in the mean values of the amplitude of VL activity (RMS) of both the affected and non-affected sides among GIa, GIb and the right side in GII during the four phases of STS movement (P=0.0001 for all) (Table 3, Fig. 2).

The Post Hoc LSD pair comparisons between each two of the three groups as regarding to the EMG activity of VL muscle revealed a significant difference between GIa and GIb, between GIa and GII and between GIb and GII in all phases for both affected and non-affected side.

Table (3): The mean values of the amplitude of the EMG activity (RMS) of VL muscle of both affected and non-affected sides in GIa, GIb and the right side in GII during the four phases of STS movement.

Variable			GIa	GIb	GII (Rt. Side)	F-value	P-value
RMS of VL (volt)	Phase I	Affected	0.454±0.024	0.547±0.031	0.428±0.034	77.474	0.0001
		Non-affected	0.515±0.032	0.732±0.025		442.977	0.0001
	Phase II	Affected	1.567±0.037	1.9±0.087	1.441±0.154	109.653	0.0001
		Non-affected	1.757±0.029	2.153±0.098		209.899	0.0001
	Phase III	Affected	1.385±0.041	1.695±0.07	1.245±0.153	115.062	0.0001
		Non-affected	1.514±0.043	1.977±0.053		323.136	0.0001
	Phase IV	Affected	0.869±0.04	1.008±0.032	0.765±0.028	183.193	0.0001
		Non-affected	0.906±0.036	1.188±0.033		647.210	0.0001

RMS: Root Mean Square VL: Vastus Lateralis

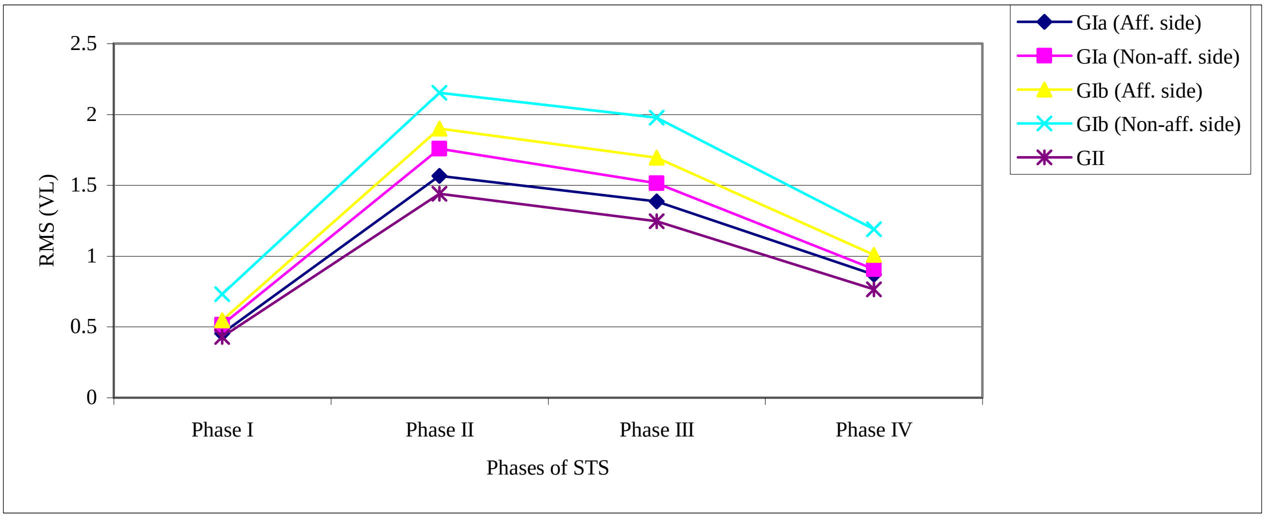

Fig. (2): The mean values of the amplitude of the EMG activity (RMS) of VL muscle of both affected and non-affected sides in GIa, GIb and the right side in GII during the four phases of STS movement.

C- Electromyographic analysis of BF muscle activity:

As regarding to the affected side, there was a statistically significant difference in the mean values of BF activity (RMS) among GIa, GIb and the right side in GII during phases I and II (P=0.0001 for both), III and IV (P=0.002 for both) (Table 4, Fig. 3). With respect to the non-affected side, there was a statistically significant difference among the three groups during all phases (P=0.0001 for all). Post Hoc LSD pair comparisons between each two of the three groups as regarding to the EMG activity of BF muscle revealed a significant difference between GIa and GIb, and between GIb and GII in all phase for both the affected and non affected side with a P-value ranged from 0.0001 to 0.04. However the LSD revealed a significant difference between GIa and GII in phase I for both the affected and non affected side and phase III and IV for the affected side only.

Table (4): The mean values of the amplitude of the EMG activity (RMS) of BF muscle of both affected and non-affected sides in GIa, GIb and the right side in GII during the four phases of STS movement.

Variable			GIa	GIb	GII (Rt. Side)	F-value	P-value
RMS of BF (volt)	Phase I	Affected	0.324±0.04	0.264±0.029	0.373±0.03	36.297	0.0001
		Non-affected	0.409±0.035	0.475±0.036		34.275	0.0001
	Phase II	Affected	0.549±0.044	0.477±0.047	0.579±0.034	22.953	0.0001
		Non-affected	0.609±0.029	0.699±0.053		36.992	0.0001
	Phase III	Affected	0.524±0.04	0.475±0.063	0.542±0.047	7.202	0.002
		Non-affected	0.598±0.028	0.695±0.036		70.385	0.0001
	Phase IV	Affected	0.356±0.063	0.308±0.072	0.396±0.026	7.156	0.002
		Non-affected	0.424±0.039	0.487±0.032		29.159	0.0001

RMS: Root Mean Square BF: Biceps Feromis

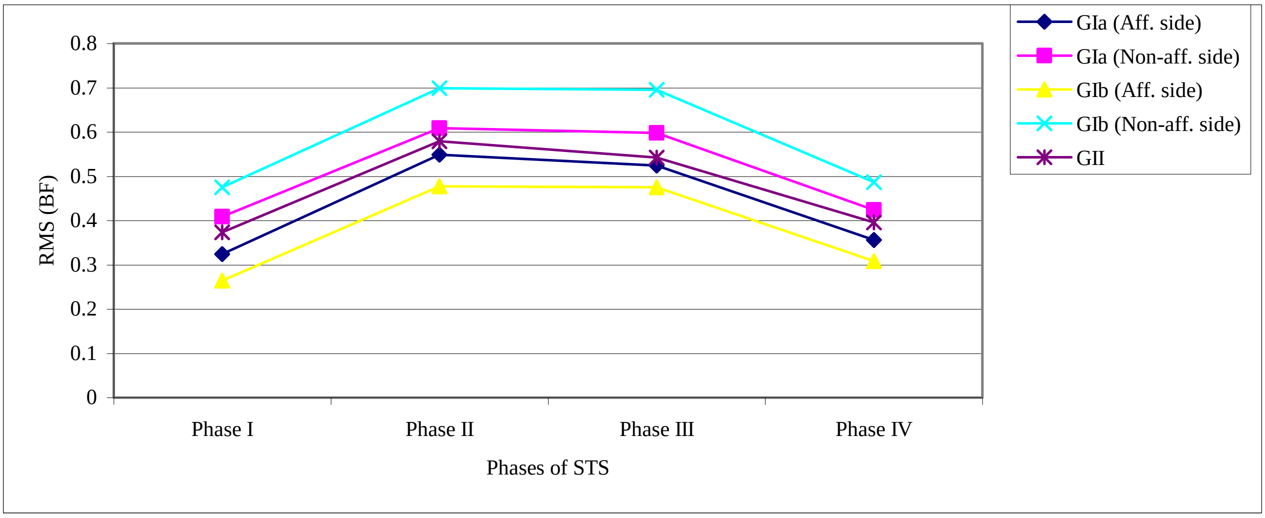

Fig. (3): The mean values of the amplitude of the EMG activity (RMS) of BF muscle of both affected and non-affected sides in GIa, GIb and the right side in GII during the four phases of STS movement.

DISCUSSION

Concerning VL muscle activity of the affected side during STS, the results showed a statistically significant difference in the mean values of the activity of this muscle during all phases, with the lowest value in GII and the highest one in GIb in all phases. The significant increase in GIb as compared to GIa and GII might be attributed to the following: there are many problems contributing to the dysfunction of the motor control in the affected side in hemiplegic patient such as: lack of awareness in the affected side, spasticity[28,29], loss of selective muscle control and release of primitive modes of muscle activation in the form of release of some abnormal reflexes[14]. These problems become exaggerated with the increase in the degree of spasticity (as in GIb) and may play a role in increasing the EMG activity of VL muscle in this group specially with exerting high effort[30].

Concerning VL muscle activity of the non-affected side during STS, the results revealed an increase in the activity of the non-affected side as compared to the affected side because the patients put more load on the non-affected side to compensate for weakness of the affected one by leaning toward the non-affected side[7,27]. As a result of leaning, the COG moves toward the non-affected side and may move out of BOS so, the patient in phase II starts to exert high force by the non-affected quadriceps muscle. This is in consistent with the opinion of Lee et al.,[24] who reported that as spasticity increases, the compensation by using the non-affected side increases. This might justify the increased activity of GIb more GIa and GII.

Regarding to ROM of the knee joint of affected side in GIa and GIb and the right side in GII: During phases I and II, ROM in GIa and GIb was higher than GII. This might be attributed to the tendency of body to lean forward and towards the non-affected side (compensation to avoid falling). This in turn pushes the affected knee towards extension in GIa and GIb more than in GII. The more the leaning is, the more the ROM of the affected knee joint. This might explain the increase in ROM in GIb than GIa. This explanation comes in agreement with the opinion of Vander Linden et al.,[38], Baer and Ashburn[2], and Cheng et al.,[8] who postulated that the hemiplegic subjects show marked asymmetry, with increased weight bearing through the non-affected leg during STS. Additionally, it might be reasonable to assume that marked asymmetry of weight distribution could be associated with marked asymmetrical movement pattern.

During phase III, ROM in GIa and GIb was smaller than GII. This might be explained as follows: the starting position of STS movement is 105° of knee flexion (for all subjects) and the knee joint ROM ends by reaching to full knee extension (ROM of the knee joint is limited because it is controlled by full knee extension)[12]. During phases I and II, the knee joint (in both GIa and GIb) moves a larger range than GII and subsequently the remained ROM of the knee will becomes smaller in phase III in GIa and GIb as compared to GII.

During phase IV, ROM of the knee joint was high in GIa and GIb as compared to GII (with a higher range in GIb). This might be attributed to two main reasons: i) During phase IV, as the body reaches the erect position, the patient tries to put more weight on the affected side to ensure stability[7]. As a result of this, the affected knee continues to extend more in both GIa and GIb as compared to GII with more extension in GIb. ii) The presence of genu recurvatum in some patients in both GIa and GIb (five in GIb and three in GIa).

The non significant difference in ROM between GIa and GII during phases I and II might be justified as follows: in case of mild spasticity there is a little impairment that will not prevent the patient from using his affected side. Additionally, during phase I, there is trunk flexion which is carried out with high speed to get the benefit from the horizontal momentum that will be converted to the vertical one during phase II[2]. Phase II is short in stroke patients (i.e., occurs with high speed). With high speed the abnormality may not appear clearly. This is consistent with the opinion of[17]. This opinion may justify the non-significant difference between GIa and GIb and between GIb and GII during phase II. On the other hand, during phase III and IV, there was a statistically significant difference between GIa and GII. This might be attributed to the increased knee ROM that occurs during phase III and the longer duration during phase IV that may cause appearance of the impairment. This opinion is supported by that of Dean and Shepherd[11] who reported that, the increased ROM and the increased duration may give a chance for the appearance of the impairment of movement control. While during phase IV, the significant difference might be justified as follows: during this phase, as the patient tried to put more weight on the affected side the knee extended more than that of GII. Additionally, presence of genu recurvatum in some patients caused the affected knee joint to move range higher than GII. This justification may explain the increase in the mean in GIb higher GIa.

Concerning ROM of the knee joint of the non-affected side in GIa and GIb and the right side in GII: The results of this study showed a small knee ROM during phase I, then the range increased during phases II and III and decreased again during phase IV. This can be explained as follows: At phase I, leaning by the trunk shifts COG toward the non-affected side and may move out of BOS so, the patient in phase II starts to exert high force by the non-affected quadriceps muscle causing more ROM in the non-affected. This might justify the non significant difference between GIa and GIb at phases II and III. The increase in the activity of VL at phases II and III explains the significant differences.

During phase III, the knee of the non-affected side extends more than the affected side but still less than GII. This may be due to the limited ROM that the knee should move. While, during phases I and II, the knee joint (in both GIa and GIb) moved a larger range than GII and subsequently the remained ROM of the knee will become smaller in phase III in GIa and GIb as compared to GII. At phase IV, as a result of fear from falling, the patients carries more weight on the non-affected limb and the knee reaches full extension during phase III to ensure stability. This might justify the higher value of ROM in GIa and GIb than GII. This also may explain the non significant difference between GIa and GII, GIb and GII and GIa and GIb.

Regarding the EMG activity of BF muscle of the affected side in GIa, GIb and the right side in GII: The results showed that, there was a significant decrease in the activity of BF in GIa and GIb as compared to GII. This might be attributed to the presence of weakness of this muscle[1,24] and the increased dependence on the non-affected side[7].

The non significant difference in the EMG activity of BF muscle between GIa and GII during phases II, III and IV might be attributed to the presence of mild degree of spasticity that will not hinder the patient from using his affected lower limb[7]. On the other hand, during phase I, there was a significant difference between GIa and GII that may be attributed to the leaning by the trunk forward and toward the non-affected side and in this case the activity of these muscles may be required to control this leaning[22]. The presence of significant difference between GIb and GII might be due to the inability of those patients to use their affected side properly as a result of the higher degree of spasticity[35].

The activity in the non-affected side increases more than in GII because as a result of stroke the patients depends on his non-affected side and less weight is carried by the affected side to guard against falling. As spasticity increases, the compensation by using the non-affected side increases that's why the activity of the non-affected side in GIb increases more than that in GIa. The presence of significant differences between the GIa and GIb and GII explains how these patients depend on their non-affected side. During phase II the non-significant difference between GIa and GII may be due to the less contribution from the non-affected side as a result from using of the affected side.

The asymmetrical movement may result from the abnormality in the activation pattern in hemiplegic patients as there is reduced rate of force generation with the paretic muscles. While rising from a chair is a dynamic task that requires that force to be generated quickly[6]. Also, this asymmetry in carrying weight may result from weakness of the affected side and loss of postural control[59]. The more the abnormality the more the asymmetry as the asymmetry in moderately spastic patients is higher than those with mild spasticity patients during rising because of weakness of the affected side and loss of postural control. The paretic leg carries about 24 - 37.9 % of body weight[5,8,9]. The non-paretic limb compensates the paretic limb. It shows significantly higher weight-bearing abilities compared with the paretic limb[3,13].

It can be conclude that stroke patients are suffering from asymmetrical movement pattern during rising from a chair. There is disturbance of EMG activity of both VL and BF. They also are suffering from asymmetrical movement of the knee joint which may result from the disturbed muscular activities and the compensations used. We can recommend that, to avoid complications that may occur in the lower limb, particularly the knee joint symmetrical pattern movement should be facilitated during activities of daily living. Also, program that encourage increase weight-bearing on the affected side sould be encouraged.

REFERENCES

1- Adams, R.W., Gandevia, S.C. and Skuse, N.F.: The distribution of muscle weakness in upper motoneuron lesions affecting the lower limb. Brain: 113: 1459-1476, 1990.

2- Baer, G.D. and Ashburn, A.M.: Trunk movements in older participants during sit-to-stand. Arch Phys Med Rehabil: 76: 844-849, 1995.

3- Bohannon, R.W. and Andrews, A.W.: Limb muscle strength is impaired bilaterally after stroke. J Phys Ther Sci, 7: 1-7, 1995.

4- Brunt, D., Greenberg, B., Wankadia, S., Trimble, M.A. and Shechtman: The effect of foot placement on sit to stand in healthy young subjects and patients with hemiplegia. Arch Phys Med Rehabil, 83: 924-929, 2002.

5- Cameron, D.M., Bohannon, R.W., Garrett, G.E., Owen, S.V. and Cameron, D.A.: Physical impairments related to kinetic energy during sit-to-stand and curb-climbing following stroke. Clinical Biomechanics, 18: 332-340, 2003.

6- Caning, C.G., Ada, L. and O'Dwyer, N.: Slowness to develop force contributes to weakness after stoke. Arch Phys Med Rehabil, 80: 66-70, 1999.

7- Carr, J.H. and Shepherd, R.B.: Impairments and adaptations. In: Stroke rehabilitation: guidelines for exercise and training to optimize motor skill (Carr J H, Shepherd RB, edrs). London: Butterworth Heinemann, 209-232, 2003.

8- Cheng, P.T., Liaw, M.Y. and Wong, M.K.: The sit-to-stand movement in stroke patients and its correlation with falling. Arch Phys Med Rehabil, 79: 1043-1046, 1998.

9- Chou, S., Wong, M., Leong, C., Hong, W., Tang, F. and Lin, T.: Postural control during Sit to Stand and gait in stroke patients. Am J Phys Med Rehab, 82: 42-46, 2003.

10- Cowan, S.M., Hodges, P.W., Bennell, K.L. and Crossley, K.M.: Altered vastii recruitment when people with patellofemoral pain syndrome complete a postural task. Arch Phys Med Rehabil: 83: 989-995, 2002.
11- Dean, C.M. and Shepherd, R.B.: Task-related training improves performance of seated reaching tasks after stroke. Stroke: 28: 722-728, 1997.
12- Doorenbosch, C.A.M., Harlaar, J., Roebroeck, M.E. and Lankhorst, G.J.: Two strategies of transferring from sit-to-stand; the activation of monoarticular and biarticular muscles. J Biomech, 27: 1299-1307, 1994.
13- Eng, J.J. and Chu, K.S.: Reliability and comparison of weight-bearing ability during standing tasks for individuals with chronic stroke. Arch Phys Med Rehabil, 83: 1138-1144, 2002.
14- Fish, D.J.: Characteristic gait patterns in neuromuscular pathologies. J Prosthet Orthot, 9: 163-167, 1997.
15- Fleckenstein, S.J., Kirby, R.L. and Macleod, D.A.: Effects of limited knee flexion range on peak hip moments of force while transferring from sitting to standing. Biomech, 21: 915-918, 1988.
16- Galumbeck, M.H., Buschbacher, R.M. and Wilder, R.P.: The sit and stand chair. A revolutionary advance in adaptive seating systems. Long Term Eff Med Implants, 14: 535-543, 2004.
17- Hesse, S., Werner, C., Paul, T., Bardeleben, A. and Chaler, J.: Influence of walking speed on lower limb muscle activity and energy consumption during treadmill walking of hemiparetic patients. Arch Phys Ther Rehabil, 82: 1547-1550, 2001.
18- Hsiang, S.M., Brgmus, G.E., Martin, S.E. and Bezvfrkhny, L.B.: Video based lifting technique coding system. Ergonomics, 41: 239-256, 1998.
19- Hughes, M.A., Myers, B.S. and Schenkman, M.L.: The role of strength in rising from a chair in the functionally impaired elderly. J Biomech, 29: 1509-1513, 1996.
20- Ikeda, E.R., Schenkman, M.L., Riley, P.O. and Hodge, W.A.: Influence of age on dynamics of rising from a chair. Phys Ther, 71: 473-481, 1991.
21- Jeng, S.F., Schankman, N., Riley, P.O. and Lin, S.J.: Reliability of a clinical kinematic assessment of the sit-to-stand movement. Phys Ther, 73: 511-520, 1990.
22- Khemlani, M.M., Carr, J.H. and Crosbie, W.J.: Muscle synergies and joint linkages in sit-to-stand under two initial foot positions. Clin Biomech, 14: 236-246, 1999.
23- Kim, C.M. and Eng, J.J.: The relationship of lower-extremity muscle torque to locomotor performance in people with stroke. Phys Ther, 83: 49-57, 2003.
24- Lee, M.Y., Wong, M.K., Tang, F.T., Cheng, P.T. and Lin, P.S.: Comparison of balance responses and motor patterns during sit-to-stand task with functional mobility in stroke patients. , 76: 401-410, 1997.
25- Lyons, K., Perry, J., Gronley, J.K., Barnes, L. and Antonelli, D.: Timing and relative intensity of hip extensor and abductor muscle action during level and stair ambulation: An EMG study. Phys Ther, 63: 1597-1605, 1983.
26- Millington, P.J., Myklebust, B.M. and Sambes, G.M.: Biomechanical analysis of sit-to-stand motion in elderly persons. Arch Phys Med Rehabil, 73: 609-617, 1992.
27- Okamoto, K., Okamoto, T. and Ogawa, R.: Electromyographic case study of recovery of walking in an elderly man after cerebral infarction. J Phys Ther Sci, 10: 19-25, 1998.
28- Ounpuu, S.: Clinical gait analysis. In: Evaluation and management of gait disorders (Spivack BS, eds). New York: Marcel Dekker, 1995.
29- Perry, J.: Gait analysis: normal and pathological function. Thorofare, NJ: Slack, Inc., 185-220, 1992.
30- Perry, J.: Determination of muscle function in the spastic lower extremity. Clin Orthop Relat Res, 288: 10-26, 1993.
31- Rash, G.S., Belliapppa, P.P., Wachowiak, M.P., Somia, N.N. and Gupta, A.: Ademonstration of the validity of a 3-D video

motion analysis method for measuring finger flexion and extension. J Biomech, 32: 1337-1341, 1999.
32- Riley, P.O., Schenkman, M.L., Mann, R.W. and Hodge, W.A.: Mechanics of a constrained chair-rise. J. Biomechanics, 24: 77-85, 1991.
33- Roy, G., Nadeau, S., Gravel, D., Malouin, F., McFadyen, B.J. and Piotte, F.: The effect of foot position and chair height on the asymmetry of vertical forces during sit-to-stand and stand-to-sit tasks in individuals with hemiparesis. Clin Biomech, 21: 585-593, 2006.
34- Roy, G., Nadeau, S., Gravel, D., Piotte, F., Maloiun, F. and McFradyen, B.: Side difference in the hip and knee joint moments during sit to stand and stand to sit tasks in individuals with hemiparesis. Clinical Biomech, 22: 795-804, 2007.
35- Sawner, K.A. and LaVigne, J.M.: Gait patterns in hemiplegia. In: Brunnstrom's movement therapy in hemiplegia. A neurophysiological approach. Philadelphia: JB Lippincott Company. 2nd ed., 1992.
36- Schenkman, M.L., Berger, R.A., Riley, P.O., Mann, R.W. and Hodge, W.A.: Whole-body movements during rising to standing from sitting. Phys Ther, 70: 638-651, 1990.
37- Shields, R.K. and Heiss, D.G.: An electromyographic comparison of abdominal muscle synergies during curl and double straight leg lowering exercises with control of the pelvic position. Spine, 22: 1873-1879, 1997.
38- Vander Linden, D.W., Brunt, D. and McCulloch, M.U.: Variant and invariant characteristics of the sit-to-stand task in healthy elderly adults. Arch Phys Med Rehabil, 75: 653-660, 1994.
39- Whitney, S.L., Wrisley, D.M. and Marchetti, G.F.: Clinical measurement of sit-to-stand performance in people with balance disorders: validity of data for the Five-Times-Sit-To-Stand Test. Phys Ther, 85: 1034-1045, 2005.

الملخص العربي

تحليل كينيماتيكي وكهروفسيولوجي لحركة الركبة وذلك أثناء القيام من الجلوس على كرسي في مرضى السكتة الدماغية

الهدف من هذا الدراسة هو تقييم حركة الركبة أثناء القيام من الجلوس على كرسي وذلك من خلال دراسة المدى الحركي لمفصل الركبة والنشاط العضلي الكهربي للعضلة المتسعة الجانبية والعضلة الفخذية ذات الرأسين في مرضى السكتة الدماغية (للناحيتين المصابة والغير مصابة) مقارنة بالأشخاص العاديين . وقد اختير لهذه الدراسة أربعون مريضاً من مرضى السكتة الدماغية حيث تم تقسيمهم إلى مجموعتين وذلك حسب درجة التوتر العضلي بعضلات الطرف السفلى المصاب (مجموعة ذات توتر عضلي بسيط وأخرى ذات توتر عضلي متوسط) ، ذلك بالإضافة إلى عشرة أشخاص عاديين لمقارنة نتائج المرضى بهم . وقد تم تقييم المدى الحركي لمفصل الركبة باستخدام جهاز تحليل الحركة ثلاثي الأبعاد كما تم قياس النشاط العضلي باستخدام جهاز رسم العضلات الكهربائي . أوضحت المعالجة الإحصائية للنتائج وجود اختلاف واضح ذو دلالة إحصائية بين المجموعات الثلاثة في النشاط العضلي للناحيتين المصابة والغير مصابة أثناء القيام من الجلوس. هذا بالإضافة إلى وجود اختلاف واضح ذو دلالة إحصائية في المدى الحركي لمفصل الركبة في الناحية المصابة لمرضى السكتة الدماغية مقارنة بالأشخاص العاديين (أثناء كل المراحل باستثناء المرحلة الثانية) والقيمة العظمى كانت في المجموعة ذات التوتر العضلي المتوسط (أثناء المرحلتين الأولى والرابعة) وفى الأشخاص العاديين (أثناء المرحلتين الثانية والثالثة) . وكان هناك اختلاف ذو دلالة إحصائية في المدى الحركي للركبة في الناحية الغير مصابة للمرضى مقارنة بالأشخاص العاديين (أثناء كل المراحل باستثناء المرحلة الأولى) والقيمة العظمى كانت في المجموعة ذات التوتر العضلي المتوسط (أثناء المرحلتين الأولى والثانية) وفى الأشخاص العاديين (أثناء المرحلتين الثالثة والرابعة). وقد استنتج من هذه الدراسة أن مرضى السكتة الدماغية يعانون من عدم تناظر بين الناحيتين المصابة والغير مصابة بالنسبة للنشاط العضلي والمدى الحركي للركبة مقارنة بالأشخاص العاديين وذلك أثناء القيام من الجلوس . وبناءا على هذا تقييم حركة الركبة أثناء القيام من وضع الجلوس يجب أن تؤخذ في الاعتبار أثناء تأهيل مرضى السكتة الدماغية .

الكلمات الدالة : السكتة الدماغية – حركة الركبة – تحليل كينيماتيكى - رسم العضلات الكهربي .