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Effect of Plantarflexor Spasticity and Ankle Joint Range of Motion on Sit to Stand Movement in Stroke Patients

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Abstract

Objective: The study was done to find out the relationship between plantarflexor spasticity, ankle joint range of motion with sit to stand kinematics and total time duration to complete sit to stand movement in stroke patients.

Methods: The study was of correlation design, with 35 stroke subjects (32 male, 3 female) carried out at different hospitals in New Delhi. Subjects were assessed for plantarflexor spasticity, plantarflexion and dorsiflexion range of motion of ankle joint and sit to stand movement. Sit to stand movement was videotaped and sit to stand kinematics and total time taken to complete the movement was analyzed through motion pro motion analysis software. Correlation between plantarflexor spasticity , ankle joint range of motion with sit to stand kinematics and total time duration to complete sit to stand movement determined using Karl Pearson's correlation coefficient.

Results: The results showed a strong positive correlation between plantarflexor spasticity and total time duration of sit to stand movement (r=0.81) and plantarflexion range of motion and total time duration of sit to stand movement (r=0.85). There was a strong negative correlation between dorsiflexion range of motion and total time duration of sit to stand movement (r=-0.80) Plantarflexion range of motion also has a moderate negative correlation with sit to stand kinematics in phase III of knee (r=-0.57) and phase III of hip (r=-0.33) respectively. Dorsiflexion range of motion also has a moderate positive correlation with sit to stand kinematics in phase III of knee (r=0.47) and phase III of hip (r=0.43) respectively.

Conclusion: The ankle impairments like plantarflexor spasticity and reduced ankle joint range of motion can affect sit to stand movement, so such impairments should be addressed during various therapeutic interventions.

Key Words: Plantarflexor spasticity; Ankle joint range of motion; Sit to stand movement; Kinematics.

Introduction

Stroke is a frequent cause of problem in body function, resulting in limitation on activity and participation.[1] Inability to stand up is common early on following stroke and predisposes the individual to further decreases in muscle strength and physical fitness, and to adaptive soft tissue changes, particularly in soleus muscle, associated with disuse and physical inactivity.[2] The ability to effectively sit to stand is a vital prerequisite for upright mobility.[3] It is biomechanically demanding, requiring more lower extremity joint torque and range of motion than walking and stair climbing.[4] Although executed in a small space and brief time, sit to stand is a movement of the whole body, involving a complete change in the interrelations of parts.

Central nervous system pathology may result in spasticity or a velocity dependent increase in stretch reflexes which contributes significantly to calf muscle hypertonia or stiffness.[5] Spastic hypertonia at the ankle

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joint is a major source of disabilities after stroke. Both reflex and nonreflex changes in ankles with spastic hypertonia can substantially affect the functional performance of stroke patients. Moreover, changes in ankle passive biomechanic properties could contribute to the internal ankle joint torque in functional movement, depending on the severity of spasticity.[6] Subjects with stroke have been found to have only half of dorsiflexion range of motion compared with healthy controls. Limited motion at the ankle may contribute to functional limitations, which are likely caused by the interaction of several complex factors such as spasticity, immobility, and structural adaptations.[6]

However, the ankle impairments of stroke patients on sit to stand performance are still undermined. Moreover, there is a lack of in-depth investigation on ankle impairments that affect sit to stand performance. It is also necessary to establish the dynamics of functional activities, such as rising to a standing position, as carried out by healthy individuals, in order to analyze and correct abnormality in individuals who have impairments.[7] According to Nuzik *et al*, during sit to stand the hip flexed during the first 40 % of the cycle and extended during the last 60 % of the cycle. The knee extended throughout the pattern of motion. The ankle moved toward dorsiflexion in the first 45 % of the movement cycle. The remainder of the motion was characterized by movement toward plantar flexion.[8] Electromyographic analyses of muscle activation during this task shows the following, presented in order of activation:

1. Tibialis anterior muscle: for preparatory placement of foot backward and tibial stabilization.
2. Simultaneous onset of activity in gluteus maximus, biceps femoris (hip extensors) and rectus femoris, vastus lateralis and medialis muscles (knee extensors); activity of extensors peaks at time thighs are lifted off seat.
3. Varying degrees of roles of gastrocnemius and soleus muscles for postural control.[2]

Hence, sit to stand needs exclusive attention in terms of evaluation and characterization. Thus, the purpose of this study was to study the extent of relationship between ankle impairments, including plantarflexor spasticity and decreased ankle joint range of motion in patients with stroke during sit to stand movement.

Methods

Thirty five subjects with stroke were recruited from Jaipur Golden Hospital, Bhagwan Mahavir Hospital and Maharaja Agarsen Hospital, New Delhi. The study was of correlation design and of single session lasting for 45 minutes. The set inclusion criteria for the study were subjects who had first episode of stroke, age between 40-80 years, time since stroke more than 6 months, able to rise from a chair independently, the subject should have a minimum score of 8 out of 16 in composite spasticity score and follow verbal commands. Subjects were excluded if they had any brainstem, cerebellar, or subcortical lesion, ankle joint pain, any other musculoskeletal or neurological disorder and had any perceptual deficit. A written informed consent was taken from subjects after the verbal explanation of the procedure and purpose of the study. The study was approved by research and ethical committee of ISIC Institute of Rehabilitation Sciences, New Delhi.

Subjects were assessed for plantarflexor spasticity on composite spasticity scale and passive ankle plantarflexion, dorsiflexion range of motion measured using universal goniometer. Composite spasticity scale is a 0-16 point ordinal scale, developed by Chan, which has been shown to be reliable and valid in people with stroke. It has 3 components: tendon jerk (ankle), resistance to full range passive joint displacement (e.g., ankle dorsiflexion) and clonus (ankle).[9] For measuring available range of motion at ankle subjects were seated on an elevated plinth. The

hip and knee were maintained at a constant 90 degree of flexion during the measurements. The subjects were instructed to relax while the ankle was passively moved in the plantarflexion and dorsiflexion direction to the end of the available range of motion with the subtalar joint in a neutral position. Fulcrum of goniometer was centered over the lateral aspect of lateral malleolus. Proximal arm was aligned with lateral mid line of fibula using head of fibula and for reference distal arm to be aligned parallel of lateral aspect of 5th metatarsal. Two trials were performed for each movement and the average value of the two recorded readings was considered for analysis.[10]

For studying the kinematics of sit to stand subjects were dressed in lycra shorts and skin markers were placed on the affected lower limb on lateral aspect of head of 5th metatarsal, lateral malleolus, lateral epicondyle of femur, greater trochanter and top of mid-iliac crest.[8] Subjects were made to sit on a bench without back and arm rest with subject's greater trochanter at the leading edge of the seat with bare foot and arms folded in across the chest.[11] The height of seat was adjusted according to the length of lower leg so that ankle, knee and hip joint of each patient was at 90 degrees in starting postion.[1] The subject's feet were kept shoulder width apart.[12] Subjects were instructed to look straight ahead and stand up at a comfortable speed when given the verbal command 'stand up' and were asked to perform three times sit to stand task in his own pace after execution of a single practice trial.[13] Camcorder was placed on a tripod stand at a distance of 4 meter at right angle to the plane of movement to capture the task of sit to stand in sagittal plane on hemiplegic side.[5,8] One trial for each subject was selected for analysis. Criteria for trial selection was 1) ability to view all the data points on each frame, 2) subjective appearance of the movement as smooth or natural, 3) feet flat in the beginning of the movement and 4) a clearly defined completion of the motion.[8] Video obtained was transferred to computer. Motion pro motion analysis software was used to calculate angles at ankle, knee and hip and total duration taken to complete task sit to stand movement. A two dimensional link segment model was used in kinematic analysis. Ankle joint excursion during the movement was measured by a line joining fifth metatarsal and lateral malleolus and line joining lateral malleolus and lateral femoral epicondyle, knee joint excursion was measured by line joining lateral malleolus and lateral femoral epicondyle and line joining lateral femoral epicondyle and line joining lateral femoral epicondyle and greater trochanter and hip joint excursion was measured by line joining the lateral femoral epicondyle to greater trochanter and greater trochanter to mid iliac crest.[16] Entire sit to stand was divided in to three phases for analysis based on different phases of sit to stand described by Schenkman *et al*.[7]

Flexion momentum phase, starts with start of movement of trunk flexion and finishes just before the lift of buttocks from chair seat, momentum transfer, phase in which the buttocks leave contact with the seat and ankles attain maximum dorsiflexion and extension phase comprising of extension of all joints of body followed and continued till the cessation of movement marked by absence of any movement at the pelvis.[14]

Data Analysis

Data was analysed using the SPSS (version 17.0) for Windows. Mean ± standard deviation of composite spasticity score, ankle joint range of motion, total time, ankle, knee and hip joint excursion during sit to stand was calculated.

Karl Pearson's correlation coefficient was calculated to find out correlation between plantarflexor spasticity and ankle joint excursion, plantarflexor spasticity and time taken to complete the movement, ankle dorsiflexion range and time taken , ankle dorsiflexion range and ankle, knee and hip excursion. The significance level was set at $p \leq 0.05$.

Table 1: Mean and standard deviation value of duration and kinematics of sit to stand

Serial no.	Variables	Mean ±s.d.(degrees)
1.	Ankle angle in phase I(A)1	106.74±6.90
2.	Ankle angle in phase II(A2)	100.91±7.08
3.	Ankle angle in phase III(A3)	115.40±6.98
4.	Knee angle in phase I (K1)	92.26±9.97
5.	Knee angle in phase I (K2)	99.83±10.69
6.	Knee angle in phase III (K3)	162.86±8.22
7.	Hip angle in phase I(H1)	122.14±18.18
8.	Hip angle in phase II(H2)	118.63±18.40
9.	Hip angle in phase III(H3)	166.97±8.65

Results

The mean ± s.d. of age of the sample was 57.91± 7.67 years, mean ± s.d. of composite spasticity scale score was 10.40±1.66, mean ± s.d., of plantarflexion range of motion was 40.14±3.86^{o}, mean ± s.d. of dorsiflexion range of motion was 8.06±2.80^{0} and m ± s.d. of total time duration of sit to stand 3.53±0.95 seconds. The mean±s.d. of ankle angle, knee angle and hip angle in different phases of sit to sand is tabulated in Table 1.

A strong positive correlation was found between plantarflexor spasticity and total time duration of sit to stand (r=0.81) which was significant. (Table 2, Fig 1) A weak negative correlation was found, between plantarflexor spasticity and ankle excursion in phase I (r=-0.25) and in phase II (r=-0.25) and no correlation was found, in phase III (r=0.05) which were non-significant. (Table 3) A strong positive correlation was found, between plantarflexion range of motion and total time duration of sit to stand (r=0.85)

Table 2: Correlation between plantarflexor spasticity and total time duration of sit to stand, plantarflexion range of motion and total time duration of sit to stand and between dorsiflexion range of motion and total time duration of sit to stand.

Variable	Time (r)
Composite spasticity scale score	0.812**
Plantarflexion	0.853**
Dorsiflexion	-0.808**

**significant at 0.01

Fig 1: Correlation between composite spasticity score with total time duration of sit to stand

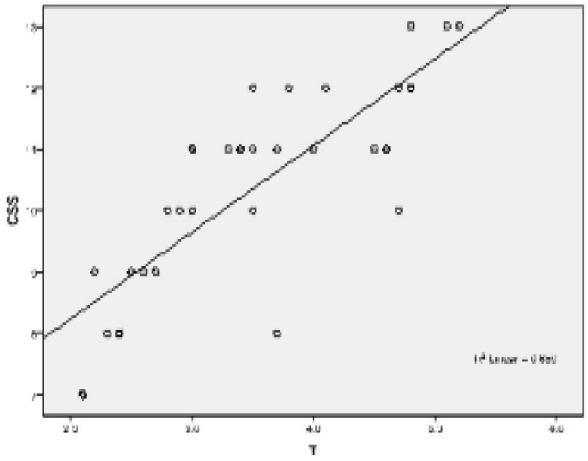


Table 3: Correlation between plantarflexor spasticity and ankle sit to stand kinematics

Variable	Ankle angle in phase I (r)	Ankle angle in phase II (r)	Ankle angle in phase II (r)
Composite spasticity score	-0.25	-0.25	0.05

Fig 2: Correlation between plantarflexion range of motion with total time duration of sit to stand

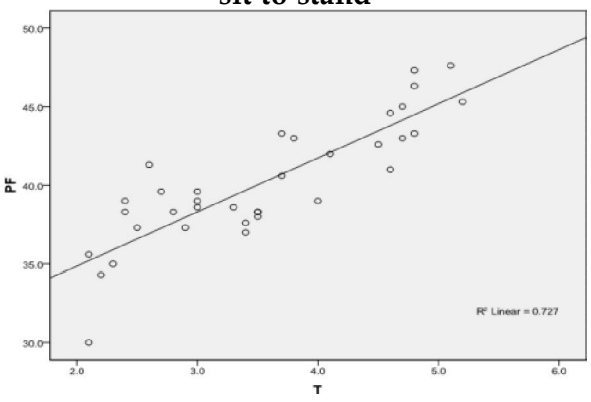
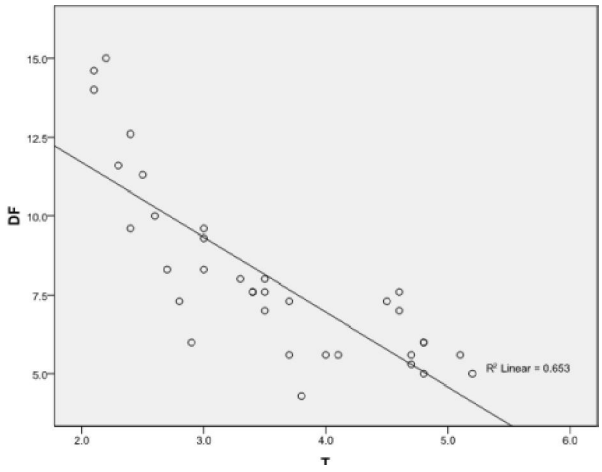


Fig 3: Correlation between dorsiflexion range of motion with total time duration of sit to stand



which was significant. (Table 1, Fig 2) A weak negative correlation was found between plantarflexion range of motion and ankle excursion in phase I (r=-0.29), in phase II (r=-0.27) and in phase III (r=-0.10) which were non-significant. No correlation was found, between plantarflexion range of motion and knee excursion in phase I (r=-0.09) and phase II (r=0.01) which was non-significant but moderate negative correlation was found with phase III (r=-0.57) which was significant. No correlation was found, between plantarflexion range and hip excursion in phase I (r=0.03) and in phase II (r=0.02) which were non-significant, but a moderate negative in phase III (r=-0.33) which was significant. (Table 4)

A strong negative correlation was found, between dorsiflexion range of motion and time taken to complete sit to stand (r=-0.80) which was significant. (Table 2, Fig 3) A weak positive correlation was found, between dorsiflexion range of motion and with ankle excursionin phase I (r=0.21), phase II (r=0.24) and phase III (r=0.15) which were non-significant. A very weak negative correlation was found, between dorsiflexion and knee kinematics excursion phase I (r=-0.13) and phase II (r=-0.17) which were non-significant but moderate positive correlation was found, between dorsiflexion and with knee excursion in phase III (r=0.47) which was significant. A very weak positive correlation was found, between and hip joint excursion in phase I (r=0.17), phase II (r=0.18) which were non-significant but moderate positive correlation was found, in phase III (r=0.43) which was significant. (Table 4)

Discussion

The results showed that there was a strong positive significant correlation between plantarflexor spasticity and total time duration of sit to stand, interpreted as more the plantarflexor spasticity more time it will take to perform sit to stand movement. Richards *et al* who noted that time taken to perform sit to stand in hemiplegic subjects was more as compared to healthy subjects.[15] Unlike total time duration, result showed that there is weak non-significant correlation between plantarflexor spasticity and ankle excursion in all phases of sit to stand, interpreted as there is no effect of plantarflexor spasticity on ankle excursion in sit to stand as most of the ankle angle in phase I and phase II are dorsiflexion than plantar flexion and which can be explained by the results of study done by Sahrmann and Norton *et al* who demonstrated that impairment of movement following stroke is not primarily due to reflexes

Table 4: Correlation between ankle joint range of motion and sit to stand kinematics

	A1 (r)	A2 (r)	A3(r)	K1 (r)	K2(r)	K3(r)	H1(r)	H2 (r)	H3(r)
PF	-0.291	-0.276	-0.105	-0.099	0.010	-0.574**	0.037	0.021	-0.336*
DF	0.218	0.249	0.153	-0.130	-0.171	0.477**	0.171	0.184	0.436**

*significant at 0.05,**significant at 0.01,

PF- Plantarflexion,DF- Dorsiflexion,A1- Ankle angle in phase I,A2- Ankle angle in phase II,A3- Ankle angle in phase III,K1- Knee angle in phase I,K2- Knee angle in phase II,K3- Knee angle in phase III,H1- Hip angle in phase I,H2- Hip angle in phase II,H3- Hip angle in phase III

in the spastic antagonistic muscles but to abnormalities of agonist contraction.[16]

There was a strong positive significant correlation between plantar flexion range of motion and total time duration of sit to stand on the contrary there is a strong negative significant correlation between dorsiflexion range of motion and total time duration of sit to stand which suggests that with more the time subject takes for sit to stand lesser is the dorsiflexion range of motion and more is the plantar flexion range of motion. It may be explained by the above result that more the plantarflexor spasticity more time it will take to complete the sit to stand movement as with more plantarflexor spasticity more restricted dorsiflexion range of motion. Lomaglio *et al* in a study reported that ankle dorsiflexion and knee extension moments on the paretic side as well as the degree of weight-bearing asymmetry significantly correlate to a prolonged duration of self-paced sit to stand.[4] And it can also be explained as of hemiparetic muscle weakness, the subjects with stroke may not be able to generate sufficient forces for propelling the body forward and upward. The subjects with stroke may also have problems with coordination of centre of mass horizontal and vertical momentum during rising to walk.[2]

There was a weak non-significant correlation between plantar flexion range of motion and ankle kinematics of sit to stand. Similarly there is a weak non-significant correlation between dorsiflexion range of motion and ankle kinematics of sit to stand suggested that there is no effect of ankle joint range of motion on ankle kinematics of sit to stand. This is in accordance with study done by Kluding *et al* who reported that an increase in ankle range of motion did not improve ankle joint kinematics.[5]

The result showed that there was no correlation between plantarflexion range of motion and knee kinematics in phase I and phase II and has moderate significant negative correlation in phase III of sit to stand. Similarly there is very weak non-significant positive correlation between dorsiflexion range of motion and knee kinematics in phase I and phase II and has moderate significant positive correlation in phase III of sit to stand. Significant result occurred between ankle joint range of motion (plantarflexion and dorsiflexion) and knee kinematics in phase III of sit to stand as phase duration of phase III is more (mean=1.92 s) among three phases of sit to stand. According to Margaret K.Y. Mak *et al*[17], during the kinematics of sit to stand movement, the initial period (initiation phase) involves dorsiflexion of the ankle joint (as center of pressure of the foot ground force moved posterior towards the heel) and flexion at the hip joint which usually increases from 80 degree to 120 degree, at the instant of seat off (forward acceleration phase). The knee joint angle remained constant during the forward acceleration phase, and reached its peak torque very close to the instant of seat off and then started to extend at seat-off until full extension at the end of the rising phase. Afterward, the extension torques at the hip and the knee joints started to descend, while the ankle torque reversed direction as the centre of pressure of the foot-ground forces moved anteriorly to the ankle joint at the end of the movement. From the above discussion of kinematics it can be seen that during phase I and II there is little involvement of knee and ankle together but in phase III (rising phase) we see that there is involvement of knee and ankle together that is when a knee go for extension the ankle torque reverse the direction (that is towards plantar flexion) as the centre of pressure of the foot ground force moved anterioly to the ankle joint at the end of the movement.[17]

The result showed that there is no correlation between plantarflexion range of motion and hip kinematics in phase I and phase II and has moderate significant negative correlation in phase III of sit to stand. Similarly, there is very weak non-significant correlation between dorsiflexion range of motion and hip kinematics in phase I and phase II and has moderate significant positive correlation in phase III of sit to stand. This difference in significance level occur may be because the phase duration of phase III is

more (mean=1.92) when compared to phase I (mean=0.91) and II (mean=0.71) respectively. And it was explained by Kluding *et al* who showed that the kinematics in proximal joints compensate for the deficits in the distal limb and proximal kinematics may have affected the amount of dorsiflexion measured during sit to stand and gait.[5]

For the purpose of analyzing sit to stand movement, we followed study done by Schenkman *et al*.[15] as sit to stand movement was divided into four phases (flexion momentum, momentum transfer and extension phase) in the present study. A fourth phase (stabilization phase) has not been analyzed because it is difficult to ascertain as there is no easy method of reliably identifying the transition between the postural movements resulting from rising and normal postural sway. A two-dimensional analysis was considered adequate since it has been demonstrated that sit to stand is primarily a sagittal plane activity in healthy subjects.[18] The subjects were instructed to keep their arms folded in across the chest in order to prevent the use of the upper extremities while executing sit to stand as in accordance with Janssen *et al* and prevent asymmetrical weight bearing, and the position was also adopted to avoid obstructing the camera's view of the markers.[18] Etnyre *et al* have reported that arms could contribute asymmetrical forces more readily, as with the arms free and hands on arm rests conditions.[19] In the present study, performance of sit to stand transfer has been standardized for starting position (hip, knee and ankle angle at 90 degrees position) and speed of movement to acquire a more or less universal movement pattern.[20] Subjects were asked to rise from the bench during sit to stand movement at their normal pace in accordance with Chou *et al* who reported that from sit to stand, the maximum oscillation in the anteroposterior direction was significantly greater at fast speeds than natural speeds, suggesting that the faster movement had greater instability during sit to stand transfer.[21]

To establish the reliability of the motion analysis tool, 2 raters, tested the repeatability of ankle kinematics and total time duration measure during sit to stand, on a group of 18 normal subjects. Inter-rater intra-class correlation coefficient (ICC) was found to be 0.99 for ankle kinematics and 0.99 for total time duration of sit to stand movement whereas, intra-rater intra-class correlation coefficient (ICC) was found to be 0.98 for ankle kinematics and 0.99 for total time duration of sits to stand movement. This indicates high consistency on repeated analysis.[22,23]

The results of the study support a rehabilitation program which focuses on ankle plantarflexion, dorsiflexion range of motion and spasticity to improve sit to stand preferably with a control intervention in stroke patients. It has also been reported that those with prolonged sit to stand movement duration experience more falls therefore proper intervention strategies to sit to stand training may be developed and implemented.

Future studies can focus on understanding the other impairments affecting sit to stand movement. The missing components identified during the task can be trained and effect of continual practice of these components can be analyzed. The limitations of the study were small number of sample, all the other variables affecting the sit to stand such as stage of recovery ,strength of lower limb musculature was not controlled.

Conclusion

Plantarflexor spasticity and ankle joint range of motion is significantly correlated with total time duration of sit to stand movement and ankle joint range of motion also has a significant correlation with sit to stand kinematics in phase III of knee and hip. The ankle impairments like plantarflexor spasticity and reduced ankle joint range of motion can affect sit to stand movement, so such impairments should be addressed during various therapeutic interventions.

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