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Differences of Passive Ankle Stiffness in Post-Stroke Hemiplegia by Varying Angular Velocity between the Paretic and Non-Paretic Side

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Abstract

Background/Objectives: The purpose of this study was to investigate the effect of passive stiffness on joint function by analyzing the in the passive stiffness of the PS and NPS ankle joint at various angular velocity.

Method/Statistical Analysis: The differences of the stiffness of ankle according to different angular velocity in both sides were compared and analyzed in 11 hemiplegic patients. Analyses on the difference of stiffness of ankle joint at each angular velocity between the P and NP side were performed using a paired t-test. The ANOVA with repeated measure was performed to analyze the differences of the stiffness. Statistical significance was defined as $P< .05$.

Findings: In the comparison on the passive stiffness of ankle joint, the paretic side was turned out to be higher in the passive stiffness than the non-paretic side at all angular velocity (20, 40, 60 deg/sec, $P< .05$). In addition, the analysis on the differences of the stiffness according to the changes in angular velocity showed that the stiffness was found to be the largest at 60 deg/sec and the smallest at 20 deg/sec in both paretic and non-paretic side indicating that the stiffness increases in the higher velocity ($P< .05$).These results indicate that the range of movement of paralyzed muscles caused by stroke is limited due to the increased passive ankle stiffness and that the velocity of movement is related to the joint function.

Improvements/Applications: This study can be exploited as a basis to evaluate the passive ankle stiffness of paretic muscles of stroke patients affecting joint function and as a data for rehabilitation program.

Keywords: *Stroke, Hemiplegia, Muscle, Passive Stiffness, Angular Velocity, Rehabilitation.*

Introduction

Spasticity shows velocity-dependent characteristics in addition to abnormal increase of muscle tension. Since spasticity causes some serious problems, such as footdrop in plantarflexion of the ankle joint, which hinders basic functional behavior such as walking, it is an important part of rehabilitation [1]. The spasticity and hypertonia of muscles due to stroke are known to be induced by a wide variety of causes, the major causative factors, nevertheless, are yet to be clearly understood. Changes in muscle tension are caused by changes in the reflex and non-reflex factors, leading to an increase in the passive stiffness of joint and muscles[2]. Changes in muscle fascicle length, thickness, and pennation angle, in addition to the neurological factors, are known to cause the changes in passive ankle stiffness [3]. The passive stiffness is also induced by an increase in plantarflexion moment with changes in muscle viscoelasticity properties. As such, movement disorder such as paralysis after brain injury occur with changes in muscle architecture and in the passive characteristics of muscles as well, hence leading to serious influence on daily life in a negative manner. Previous studies have

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shown that the passive spasticity of the paretic muscle is 43% higher than that of the non-paretic muscle [4]. Hence, understanding the stiffness that increases in the ankle joint after stroke and analyzing the changes in stiffness in respect to the velocity of motion can be an important part of the evaluation of function. In particular, since the ankle joint is an important joint that is directly related to natural ability of human, walking, it is a clinically crucial joint for returning to daily living activity after stroke and it is a body segment that is deeply affected by paralysis and spasticity compared to other joints [5]. In addition, the typical walking pattern of stroke patients is caused by plantarflexion of the ankle joint, and the recovery is slower than other joints [6].It has been studied by many researchers since it is important to understand the factors listed above for functional recovery of the ankle joint [7].

Joints move the skeleton by generating torque, a muscle-generated force of rotating characteristics. Force is used as a moment in biomechanical analysis, the moment, which changes as the angle changes while the joint is moving, can be described as a function of stiffness, i.e., the stiffness is the moment divided by the angle (Nm/deg) [8]. The spasticity of ankle joint and an increase of muscle tension is a major causative factor and have been intensively studied thus far. It has been suggested that the degree of non-reflective factors may influence more than reflective factors and some studies have suggested that the degree of spasticity can lead to a change in the passive characteristics of the ankle joint, limiting the amount of torque generated internally[9]. The spasticity of paralyzed muscle is closely related to the velocity of joint motion since the spasticity shows a velocity-dependent feature due to excessive exaggeration of stretch reflexes. Hence, it is essential to understand the velocity of joint angle and the change pattern of spasticity since the velocity of joint motion in functional movement such as walking affects the rigid muscles[10,11].

In this study, the passive stiffness of the ankle joint after stroke was analyzed by measuring the difference of the joint angle and the angular velocity of the paretic and non-paretic side using the torque-angle relationship curve and slopes which indicate the relation between the torque generated from the ankle joint and the angle at a specific point in time. In addition, we aimed to analyze the velocity of motion and the trend of passive stiffness increase by means of measuring the motion of different velocities in different conditions to analyze the characteristics of the velocity-dependent rigid muscles.

Method

1. **Participants:** The participants voluntarily agreed to participate and signed on the consent form for the experiment after hearing the explanation of the contents and purpose of the study, the experimental procedure, human rights protection of the subject, and the safety of the study. The participants were selected for patients who were able to walk independently at a chronic stage 6 months after onset and had ankle stiffness graded as less than 2 on Modified Ashworth Scale (MAS). The physical characteristics and medical history of the subjects are shown in Table 1.

Table 1. Medical history and physical characteristics of each subject.

No.	Gender	Age (Years)	Diagnosis	Paretic side	MAS	Onset (months)	Height (cm)	Weight (kg)
1	F	52	Rt. Subarachnoid Hemorrhage	Lt	1+	33	157	53
2	M	57	Lt. Basal Ganglia Infarction	Rt	1	29	171	71
3	F	52	Lt. Cerebral Infarction	Rt	1+	10	152	52
4	M	59	Rt. Subarachnoid Hemorrhage	Lt	1	34	173	67
5	M	54	Lt. Thalamus Infarction	Rt	2	17	178	83
6	F	57	Rt. Cerebral Infarction	Lt	2	44	161	61
7	M	51	Rt. MCA Infarction	Lt	1+	45	169	64
8	M	53	Lt. Subarachnoid Hemorrhage	Rt	1	9	155	63
9	M	55	Lt. Pontine Infarction	Rt	2	23	165	67
10	F	53	Rt. Basal Ganglia Infarction	Lt	1+	8	165	65
11	F	58	Rt. Cerebral Hemorrhage	Lt	1+	28	153	60
Mean±SD		54.6±2.7				25.5±13.3	163.5±8.7	64.2±8.5

 M: Male, F: Female, Rt: Right, Lt: Left, MCA: Middle Cerebral Artery, MAS: Modified Ashworth Scale, NP: Non-paretic, P: Paretic.

2. **Experimental Procedures:** Torque was measured by dynamometer (cybex, lumex, USA) to calculate the passive stiffness of the ankle joint according to the velocity of movement. For the measurement, participants sat comfortably in the dynamometer chair, straightened the knees, set the ankle joint to anatomical 0 degree, and secured it with a strap to the footrest. The range of ankle joint motion was measured within the range from 10 degrees of dorsiflexion to 30 degrees of plantarflexion. The motion was repeatedly performed for 10 times for each velocity and the data were collected for 6 intermediate excluding the beginning and ending two times. A 5 minute break was set between the measurements to rule out the interference effect between the velocities and the velocity condition was scheduled in a random basis.

3. **Data Analysis:** The raw data were collected on a personal computer using Lab View 8.0 (National Instrument, USA). The dynamometer received the synchronization signal of torque and angle and converted each analog signal into digital one and stored them in a personal computer. The voltage value gained from the dynamometer was recalibrated, converted to actual Nm value and subjected for analysis. The remaining signals were derived from real data using Chart 5 for Window (AD Instrument, USA) program. Passive stiffness was analyzed by determining slopes ranging from 10 degrees of plantarflexion to 10 degrees of dorsiflexion of the torque-angle curve during the operation at each velocity [8].

Result and Discussion

As shown in Table 2, the passive stiffness of the ankle joint, which was determined by measuring the slope between 10 degrees of plantarflexion and 10 degrees of dorsiflexion, was measured at an angular velocity of 20 deg/sec as 2.61±1.25 of non-paretic side and 4.46±2.11 paretic side, indicating that the stiffness of paretic side was larger than that of non-paretic side with statistical significance. The passive stiffness at angular velocity of 40 deg/sec was measured as 3.40±1.57 in non-paretic side and 6.93±2.63 in paretic side indicating consistent patterns as in the 20 deg/sec as paretic side showed higher value with statistical significance. In case of60 deg/sec, it was measured as 3.94±1.88 in non-paretic side and 8.69±3.85 in paretic side also resulting in the consistent pattern as the paretic side showed higher value with statistical significance. The passive stiffness of paretic side turned out to be higher in all angular velocity tested and all the differences were statistically significant ($P< .05$). The degree of stiffness increase according to the velocity tended to increase as the angular velocity increases.

Table 2. Passive ankle stiffness of post stroke hemiplegia in different angular velocity

Side / Velocity	Nom-Paretic side	Paretic side
20 deg/sec	2.61±1.25	4.46±2.11*
40 deg/sec	3.40±1.57	6.93±2.63*
60 deg/sec	3.91±1.88	8.69±3.85*

The result above was consistent with the results of previous studies that the stiffness increased during passive movement of the joints [10-12]. This means that neurological changes after cerebral infraction and changes in the material and biomechanical properties of the muscles and tendons cause negative consequences on joint movement, and velocity-dependent properties greatly influence on adaptation to external environmental changes and on controlling the proper velocity of joint movement. Biomechanical changes in the ankle joint correlate with decreased joint range and increased ankle resistance and stiffness together with spasticity after stroke[15].

Figure 1 shows the difference in ankle joint stiffness between the paretic side and non-paretic side at ankle joint angular velocity of 60 deg/sec. As shown in the figure, the torque increases as the ankle joint flexes to the dorsal side and as the slope increases. The slope is higher in paretic side than in non-paretic side, so the hardness is higher indicating the stiffness is higher in paretic side.

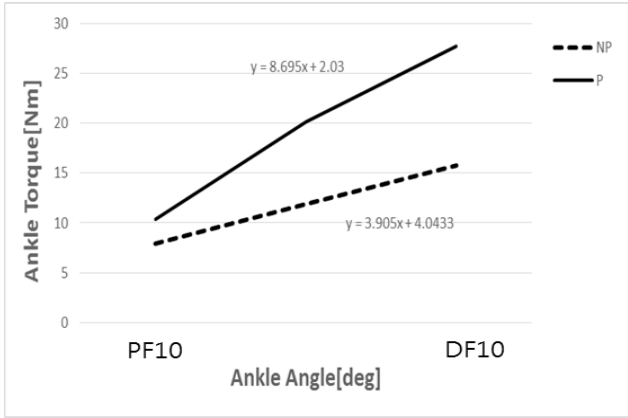

Figure 1. Torque-angle relationship slope of paretic and non-paretic side in 60 deg/sec ankle movement. NP: non-paretic, P: paretic

The torque-angle curve in the whole range of joint motion given in Figure 2 shows the overall pattern. The paretic side increases slowly in the plantarflexion compared to the non-paretic side, and the torque increases rapidly toward the dorsiflexion. This means the intrinsic muscle ability to stretch is deteriorated as resistance due to spasticity and contracture largely increased when the plantarflexion muscle becomes longer. These results mean that the resisting torque of the ankle joint generated during dorsiflexion is relatively greater than the resisting torque during plantarflexion, thus, the heel strike of the stance phase and the toe off, which is the beginning of the swing phase, are prevented during walk [16]. The stiffness increase with the elevating velocity of the joint movements indicates that the resistance of rigid muscle increases according to the joint movement [17]. Although the increase in exaggeration of stretch reflex, which is intrinsic characteristics of spasticity, cannot be directly observed with the velocity suggested in this study, the increasing passive stiffness will also affect active movement and muscle traits and will interfere with various movements given that voluntary movements are not smooth due to the nature of stroke [18].

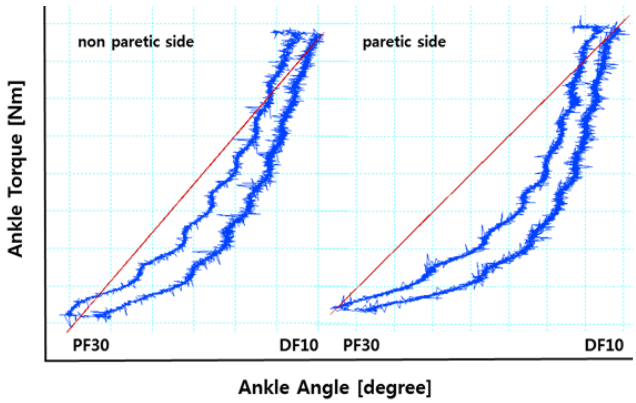

Figure 2. The torque-angle curve in the full range of ankle joint

Changes in the material properties of muscles are secondary to nerve damage. The stiffness of the paralyzed muscles is associated with changes in the reflex mechanisms and passive mechanical properties of the muscles at rest, resulting in contractures and reduced joint range. Many current clinical tests cannot clearly distinguish the contributions between the two, but the abnormal increase in torque occurs equally. Furthermore, as stiffness increases, the range of motion decreases and the motor function of the limbs decreases [6].

Passive stiffness may be related with increased amount of collagen in muscle, however the evident for long term accumulation in connective tissue has not been elucidated [19]. After stroke, the paralyzed muscles are immobilized for a long while, thus reducing the longitudinal load on the muscles. This reduction is reportedly known to cause muscle atrophy, shorten sarcomere and accumulate fat in connective tissue. A long-term study with immobilization of rats has revealed an increase in collagen fibers in the endo/epimysium of connective tissue, and a change in the ratio of muscle fibers and collagen related to gene expression has been observed[20]. When the muscle loses its intrinsic stretch ability in the process of the force generated by the muscles acting on the skeleton via the muscle, the magnitude of the force is proportional to eh size of the cross sectional area that is the thickness of the muscle, and is determined by various factors such as the pennation angle and muscle fiber length. Debilitation of muscles results in a decrease in thickness and the array angle and this phenomenon frequently occurs in paralyzed muscles after stroke. Eventually, all changes in biomechanical properties from muscle fibers to joint levels can cause functional impairment; it is hence thought to be important to analyze the complex correlations among the variables and the contribution of each factor to the malfunction in the studies of paralyzed muscles due to stroke.

Conclusion

As a result, the paretic side showed resistance in joint toward movement than the non-paretic side. The difference in stiffness appeared to be generally larger in higher velocity of movement than in lower velocity. Spasticity, a representative symptom of paralyzed muscles, directly affects joint movement, and the resistance is increased by shortened muscles due to changes in its material properties, thus increasing joint stiffness. In addition, the changes of the degree of change in stiffness dependent according to the velocity of movement means that it is difficult to adapt to various changes between the inside the body and external environment. Hence, it will be important to resolve these problems through various interventional method.

Ethical Clearance: Not required

Source of Funding: Self

Conflict of Interest: Nil

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