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MECHANICAL ABNORMALITIES OF THE SPASTIC ANKLE IN CHRONIC STROKE SUBJECTS

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Abstract—**We examined the intrinsic and reflex contributions to ankle stiffness in people with chronic stroke and healthy subjects using the parallel system identification technique. Modulation of intrinsic and reflex stiffness was characterized by applying pseudorandom binary sequence (PRBS) perturbations to the ankle at different initial ankle joint over the entire range of motion (ROM). The experiments were performed for both paretic (stroke) and contralateral (control) side. Healthy (normal) subjects were used a secondary control.**

Reflex stiffness gain significantly increased in stroke than in control side at most positions. Intrinsic stiffness gain also increased significantly at dorsiflexing positions. These changes were position dependent. Thus, the abnormalities in intrinsic stiffness gain increased continuously from middle plantarflexion to full dorsiflexion while the major increase in reflex stiffness happened at the middle ROM. No significant changes were found in other intrinsic and reflex stiffness parameters. As compared to the normal ankle, the reflex stiffness gain of the control side was significantly larger, indicating that the control side is not normal.

These findings demonstrate that both intrinsic and reflex stiffness contribute significantly to the mechanical abnormality associated with spastic ankle in hemiplegic stroke subjects. The results also suggest that the contralateral limb may not be used as a suitable control.

Keywords—**spasticity, stiffness, reflex, intrinsic, ankle, stroke, identification**

I. INTRODUCTION

Spasticity, a common effect after stroke injury, is a motor disorder associated with several clinical symptoms including hypertonia [1-3]. Quantitatively, objective evaluation of spasticity remains difficult due to the lack of accurate tools for separating torque generated by reflex mechanisms from torque generated by passive and intrinsic mechanisms [4]. Recently, we used a parallel cascade system identification technique to quantify spasticity in terms of joint dynamic stiffness.

Joint dynamic stiffness is the dynamic relation between joint position and the resulting torque which can be categorized into intrinsic and reflex components. Intrinsic stiffness arises from the mechanical properties of the joint, passive tissue and active muscle fibers. Reflex stiffness arises from the changes in muscle activation due to stretch [4, 5].

We have already used this identification model to study spinal cord injured (SCI) subjects [4, 6]. The results indicated that both intrinsic and reflex stiffness contributes to ankle spasticity [4]. However, similar studies in stroke subjects did not find significant difference in the joint dynamic stiffness components [7]. It could be due to the fact that in their study the ankle of most subjects were examined from plantarflexion (PF) to neutral position (NP) whereas abnormalities in reflex and particularly intrinsic stiffness are usually more evident between NP and full dorsiflexion (DF) [7]. We therefore designed and performed this study (1) to quantify the ankle stiffness of the hemiparetic stroke subjects over the entire ROM, and (2) to study the contralateral ankle as well to find if it can be used as a suitable control.

II. EXPERIMENTAL PROTOCOL

Twenty chronic, hemiparetic stroke subjects with different degrees of spasticity and eleven healthy subjects were studied. Subjects were seated and secured in an adjustable, experimental chair with the ankle strapped to the footrest. The thigh and trunk were strapped to the chair (Figure 1). The seat and footrest were adjusted to align the ankle axis of the rotation with axis of the force sensor and the motor shaft.

The joint stretching device operated as a position control servo driving ankle position to follow a command input. An oscilloscope mounted in front of the subject displayed a command signal and provided feedback of low-pass filtered ankle torque.

Joint position, velocity and torque were recorded by a potentiometer, tachometer & torque transducer, respectively. Electromyograms (EMGs) from gastrocnemius and tibialis anterior were recorded using bipolar surface electrodes.

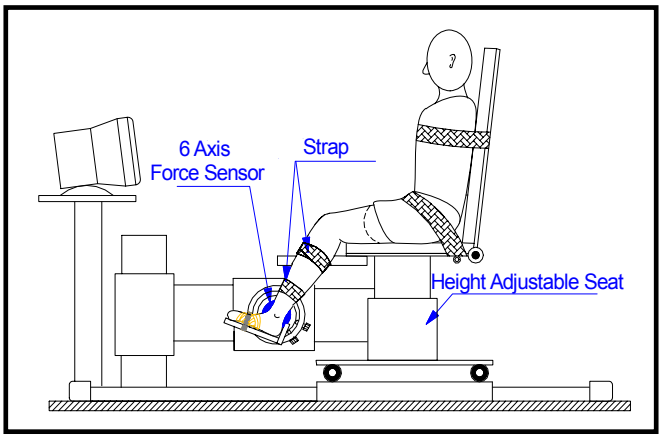

Figure 1: Experimental setup

A series of pseudorandom binary sequences with the amplitude of 0.03 rad and a switching-rate of 150ms were used to perturb the ankle at different positions from 50° PF to 20° DF at 10° intervals. A 90° angle of the elbow and ankle joint was considered to be the neutral position (NP) and defined as zero.

These experiments were conducted on both spastic and contralateral sides in stroke patients and one side of the healthy subjects, while subjects were relaxed. The contralateral ankle was used as control to reduce the effects of inter-subject variability. The healthy ankle was used as a secondary control. Flexion was considered negative by convention.

III. Analysis Methods

A. Parallel-cascade Identification Technique

A parallel-cascade identification technique was used to separate intrinsic and reflex components (Figure 2) [5, 6]. Intrinsic stiffness dynamics were estimated by determining the Impulse Response Function (IRF) between position and torque. Reflex stiffness dynamics used a differentiator, a static nonlinear element followed by the dynamic linear element. Reflex stiffness dynamics were estimated by determining the impulse response function between velocity as the input and the reflex-torque as the output, using Hammerstein identification methods [6].

B. Parametric Model

Intrinsic and reflex stiffness dynamics were estimated by fitting parametric models to the IRFs. The intrinsic compliance is the inverse dynamic of the intrinsic stiffness and is defined by a linear, second-order high pass system. The linear, dynamics of the reflex stiffness were well described by a third order system.

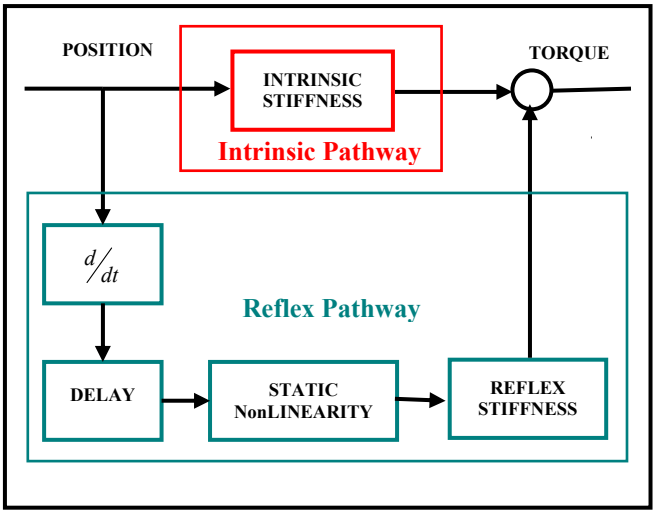

Figure 2: The parallel-cascade system identification model

IV. Results

A. Intrinsic Stiffness Gain versus. Ankle Position

Figure 3 shows the group average of intrinsic stiffness gain *(K)* as a function of ankle angle in both spastic and control sides of hemiparetic stroke patients and healthy subjects. There are three major findings:

(1) *K* was strongly position dependent; it first decreased sharply from full PF flexion to mid-PF, then increased slowly from mid-PF to mid-DF, and finally it increased sharply from mid-DF to full-DF. This position dependency was consistent in all groups.

(2) *K* was significantly larger in the stroke than in the control sides at dorsiflexing positions.

(3) There was no significant difference between the control and normal groups.

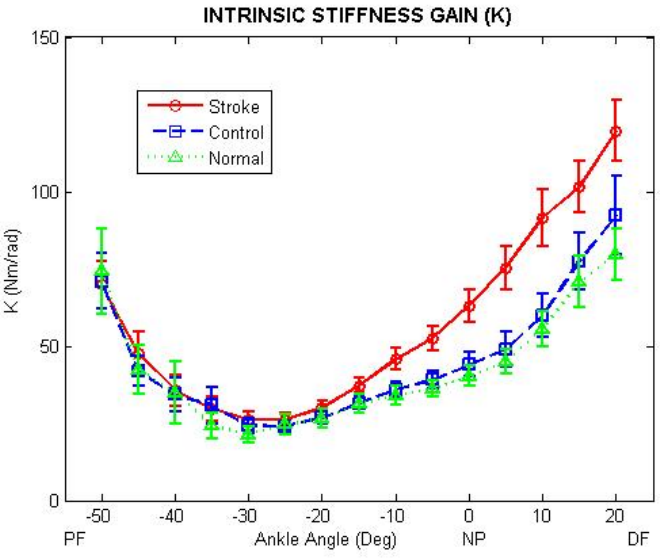

Figure 3: Intrinsic stiffness (*K*) as a function of ankle angle in stroke, control and normal ankles

B. Intrinsic Stiffness Viscosity versus Ankle Position

Figure 4 shows the group average of intrinsic stiffness viscosity *(B)* as a function of ankle angle in stroke, control and normal groups. The results indicate that:

(1) *Similar to K, B* was position dependent; it first decreased from full PF flexion to mid-PF and then increased slowly from mid-PF to full-DF.

(2) *B* was larger in the stroke than in the control at dorsiflexing positions but the differences were not significant.

(3) *B* was slightly larger in the control than the normal ankle. There was no significant difference between stroke and normal ankles.

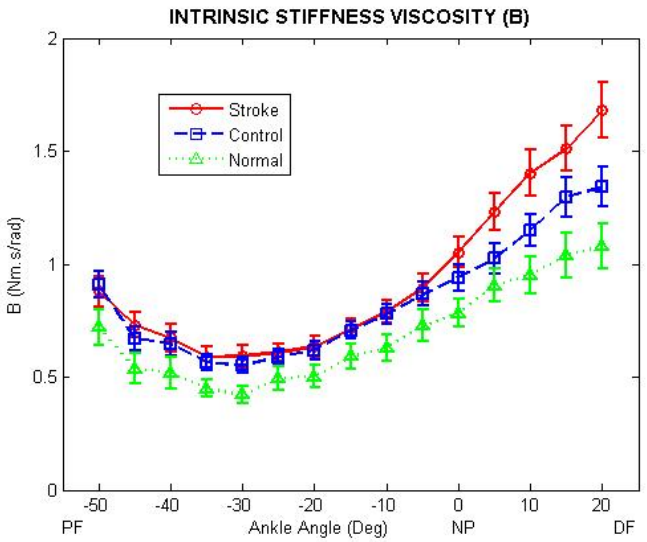

Figure 4: Intrinsic stiffness viscosity (*B*) as a function of ankle angle in stroke, control and normal ankles

C. Reflex Stiffness Gain versus Ankle Position

Figure 5 shows the group average of reflex stiffness gain (G_R) as a function of ankle angle in all groups. The results show that:

(1) G_R was significantly larger in the stroke than in the control and normal ankles.

(2) G_R was also significantly larger in the control than normal ankles.

(3) G_R was strongly position dependent; it first increased from mid-PF to mid-DF and then declined. The peak value of G_R was around NP in the stroke ankle whereas it was around full-DF in the control and normal ankle.

V. Discussion and Conclusions

Position dependency of the abnormalities in intrinsic and reflex stiffness was investigated in hemiparetic stroke subjects using the parallel cascade system identification technique.

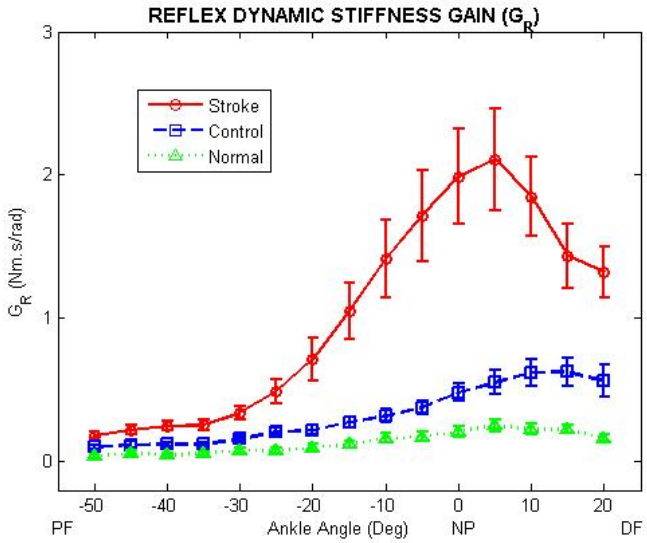

Figure 5: Reflex stiffness gain (G_R) as a function of ankle angle in stroke, control and normal ankles

Intrinsic and reflex stiffness gains were strongly position dependent and their modulation with ankle angle was similar in stroke, control and normal ankle. The position dependency of these parameters was also similar to SCI subjects [4, 8]. On the other hand, the decline in reflex stiffness gain at DF was not observed in a study done by Galiana et al. (2005) using a similar analysis technique because the end of the ROM of their stroke subjects were limited to NP [7].

We found that reflex stiffness gain increased significantly in the spastic ankle, consistent with Meinders et al. [9]. This is in contrast with Galina et al. who found a substantial changes only in a few stroke subjects [7]. This discrepancy could be due to the smaller number of subjects used in their study.

We show that the intrinsic stiffness gain increased significantly in the stroke subject at dorsiflexing positions. In contrast, as mentioned above Galiana et al. [7] did not find significant difference between stroke and normal ankles because the ROM of their subjects was limited to NP.

The findings demonstrate that both intrinsic and reflex stiffness contribute to abnormalities in ankle dynamic stiffness in hemiparetic stroke subjects.

Our results showed that reflex stiffness was significantly larger in the control than normal ankles. Furthermore, intrinsic stiffness gain and viscosity were slightly larger in the control than normal ankles. This demonstrates that the contralateral side of the hemiparetic stroke subjects is not normal, consistent with Thilmann et al. [10], suggesting that the contralateral limbs may not be a suitable control for study the neuromuscular mechanical abnormalities.

ACKNOWLEDGEMENT

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