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Comparison of Neuromuscular Abnormalities between Upper and Lower Extremities in Hemiparetic Stroke

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Abstract—**We studied the neuromuscular mechanical properties of the elbow and ankle joints in chronic, hemiparetic stroke patients and healthy subjects. System identification techniques were used to characterize the mechanical abnormalities of these joints and to identify the contribution of intrinsic and reflex stiffness to these abnormalities. Modulation of intrinsic and reflex stiffness with the joint angle was studied by applying PRBS perturbations to the joint at different joint angles. The experiments were performed for both spastic (stroke) and contralateral (control) sides of stroke patients and one side of healthy (normal) subjects.**

We found reflex stiffness gain (G_R) was significantly larger in the stroke than the control side for both elbow and ankle joints. G_R was also strongly position dependent in both joints. However, the modulation of G_R with position was slightly different in two joints. G_R was also larger in the control than the normal joints but the differences were significant only for the ankle joint. Intrinsic stiffness gain (K) was also significantly larger in the stroke than the control joint at elbow extended positions and at ankle dorsiflexed positions. Modulation of K with the ankle angle was similar for stroke, control and normal groups. In contrast, the position dependency of the elbow was different. K was larger in the control than normal ankle whereas it was lower in the control than normal elbow. However, the differences were not significant for any joint.

The findings demonstrate that both reflex and intrinsic stiffness gain increase abnormally in both upper and lower extremities. However, the major contribution of intrinsic and reflex stiffness to the abnormalities is at the end of ROM and at the middle ROM, respectively. The results also demonstrate that the neuromuscular properties of the contralateral limb are not normal suggesting that it may not be used as a suitable control at least for the ankle study.

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

I. Introduction

Damage to descending pathways, as occurs in stroke, results in several forms of motor and/or sensory impairment [1, 2] and typically results in spasticity, which can directly and indirectly change the mechanical properties of the neuromuscular system [3]. Spasticity is a motor disorder associated with lesions at different levels of the nervous system and different clinical symptoms including hypertonia [1, 2]. Hypertonia is regarded as the defining feature of spasticity that can result in unstable posture and motor disabilities in stroke [2]. Despite intensive investigation, the nature and origin of the mechanical changes in muscle and related tissues associated with spastic hypertonia remain incompletely understood [3, 4]. To study the origin of spasticity, the overall joint dynamic behavior must be characterized in terms of its mechanical properties. The mechanical properties of stretch reflexes have been difficult to study in a clinical setting due to the longstanding lack of accurate and sensitive tools for separating torque generated by reflex mechanisms from torque generated by passive and intrinsic mechanisms [3, 4]. Grossly, joint dynamic stiffness is determined by (i) *intrinsic* mechanisms arising from active muscle fibers, limb inertia and connective tissue, and (ii) *reflex* mechanisms arising from the neural response to stretch. Recently, we have developed and used a novel system identification technique [5, 6] (the parallel-cascade technique) that enables us to characterize joint dynamic stiffness and to separate the relative contributions of its components.

In published studies of spinal cord injured persons using this novel technique, we reported that overall ankle dynamic stiffness was abnormally high. Both intrinsic and reflex mechanical responses were significantly increased, but the major mechanical abnormality arose from increased reflex stiffness [3, 4]. Moreover, we showed that these mechanical abnormalities were dependent on joint position [3, 4].

The objective of the present study was to examine whether similar abnormalities are associated with spasticity in different spastic populations and different extremities using the same system identification technique.

Accordingly, the objectives of the present study were:

1- to characterize the relative contributions of intrinsic and reflex stiffness to mechanical abnormalities associated with spastic hypertonia in chronic, hemparetic stroke subjects,

2- to examine position dependency of these mechanical abnormalities in the elbow and ankle joints,

3- to find whether the neuromuscular properties of the contralateral side is normal in this stroke population.

We thus used the system identification technique to study the modulation of intrinsic dynamic stiffness and reflex function of the elbow and ankle joint throughout their ROMs in chronic stroke patients and normal subjects.

II. Experimental Protocol

A. Subject Recruitment

Twenty chronic, hemiparetic stroke subjects with different degrees of spasticity and eleven healthy subjects were used for the ankle study. For the elbow study, we used 14 stroke subjects with the same inclusion criteria and 14 healthy subjects. The experiments were performed for both stroke and contralateral sides of patients and one side of the healthy subjects. The contralateral limb was used as control to reduce the effects of inter-subject variability and the healthy limb used as a secondary control.

B. Apparatus for Elbow Study

Subjects were seated on an adjustable, experimental chair with their forearm attached to the beam of a stiff, PID controlled motor by a custom fitted fiberglass cast. The seat was adjusted to provide shoulder abduction of 80° and align the elbow axis of the rotation with axis of the torque sensor and the motor shaft (Figure 1).

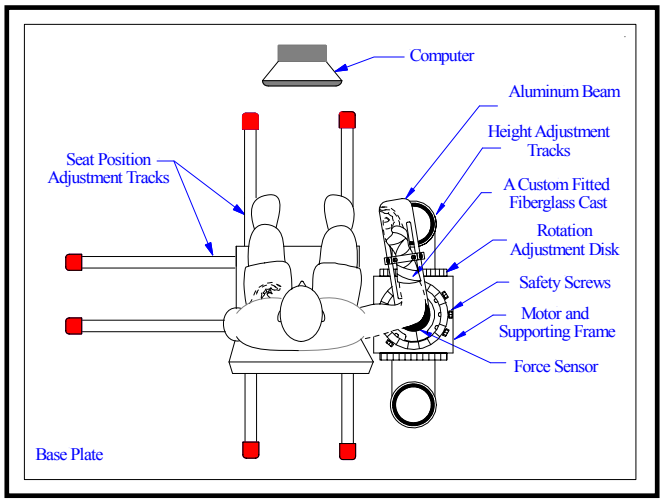

Figure 1: Experimental setup for upper extremity

C. Apparatus for Ankle Study

Subjects were seated and secured in an adjustable, experimental chair with the ankle strapped to the footrest and the thigh and trunk were strapped to the chair (Figure 2). The seat and footrest were adjusted to align the ankle axis of the rotation with axis of the force sensor and the motor shaft.

D. Recording

Joint position, velocity and torque were recorded by a potentiometer, tachometer & torque transducer, respectively. Electromyograms (EMGs) from biceps, brachoradialis, and triceps for the elbow, and from gastrocnemius and tibialis anterior for the ankle were recorded using bipolar surface electrodes (Delsys, Inc. Boston, MA).

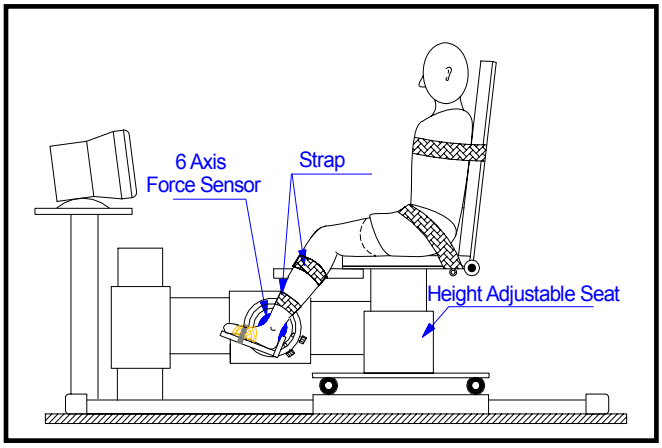

Figure 2: Experimental setup for lower extremity

E. PRBS perturbations

A series of pseudorandom binary sequences (PRBS) with the amplitude of 0.03 rad and a switching-rate of 150ms were used to perturb the elbow at different positions:

- from 45° flexion to 75° extension, at 15° intervals for the elbow joint
- from 50° PF to 20° DF at 5° intervals for the ankle joint

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

III. Analysis Methods

A. Parallel-cascade Identification Technique

Intrinsic and reflex contributions to the elbow stiffness dynamics were separated using a parallel-cascade identification technique (Figure 2) [5, 6].

Intrinsic stiffness dynamics were estimated by determining the Impulse Response Function (IRF, PTQ_{IRF}) between position and torque.

Reflex stiffness dynamics were modeled as a differentiator, in series with a delay, a static nonlinear element and then a dynamic linear element. Reflex stiffness dynamics were estimated by determining the impulse response function (VTQ_{IRF}), between velocity as the input and the reflex- torque as the output, using Hammerstein identification methods [5].

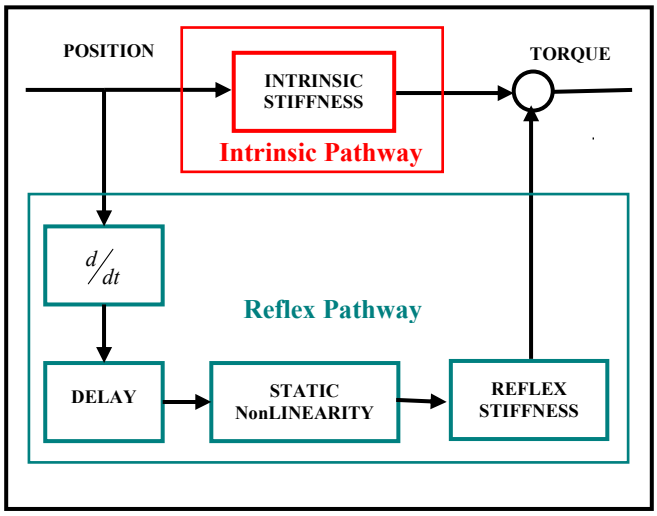

Figure 3: The parallel-cascade system identification model

B. Parametric Model

Non-linear least squares methods were used to fit parametric models to the IRFs as follows:

(1) The intrinsic compliance dynamics were computed from the intrinsic stiffness IRF and well defined by a linear, second-order high pass system.

(2) The linear, dynamics of the reflex stiffness were well described by a third order system.

IV. RESULTS

A. Reflex stiffness Gain versus Elbow and Ankle Position

Figure 4 shows the group average of reflex stiffness gain (G_R) as a function of elbow (top panel) and ankle (bottom panel) angle in both spastic and control sides of hemiparetic stroke patients and healthy subjects. There are three major findings:

(1) G_R was significantly larger at most joint positions in the stroke than control and normal side for both elbow and ankle joints. In the elbow (Fig. 4A), the differences of G_R between the stroke and control joint increased as the elbow was extended. In the ankle (Fig. 4B), the major differences were observed in the mid-ROM.

(2) G_R was strongly position-dependent in both joints. However the modulation of G_R with position was slightly different in two joints; it increased continuously as the elbow was moved from full flexion to full extension (Fig. 4A), whereas it first increased from mid-PF to mid-DF and then declined as the ankle moved toward full DF (Fig. 4B).

(3) G_R was also larger in the control than the normal joints but the differences were significant only for the ankle joint.

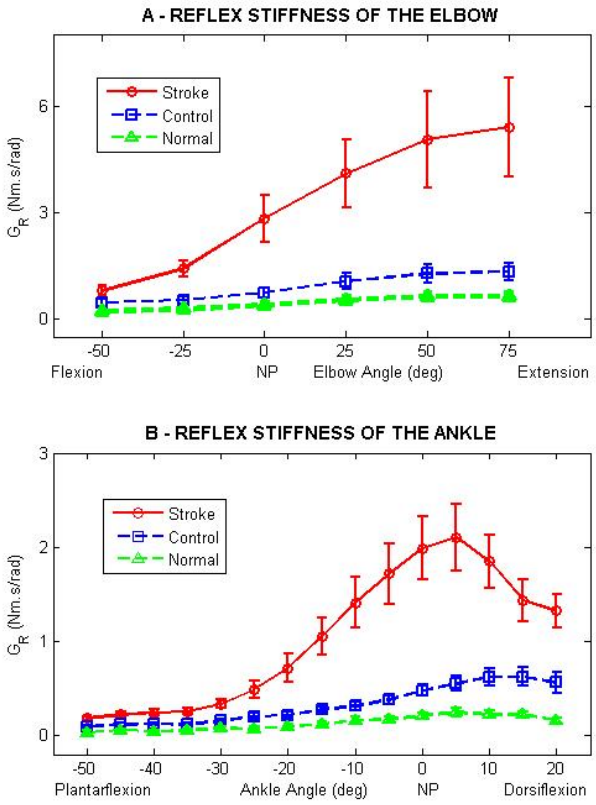

Figure 4: Reflex stiffness gain (G_R) as a function of the elbow (top panel) and ankle (bottom) angle for stroke, control and normal groups. Group Mean ±1 Standard Error

B. Intrinsic Stiffness Gain versus Elbow and Ankle Position

Figure 5 shows the group average of intrinsic stiffness gain (K) as a function of elbow (top panel) and ankle (bottom panel) angle in both spastic and control sides of hemiparetic stroke patients and healthy subjects. There are three major findings:

(1) K was significantly larger in the stroke than the control and normal joint at elbow extended positions (Fig. 5A)

(2) Modulation of K with the ankle angle was similar for stroke, control and normal groups; it increased slowly from mid-PF to mid-DF but increased sharply at the extremes of ROM (Fig. 5B). In contrast, the position-dependency of the elbow was different. K of the elbow joint decreased sharply from full- to mid-flexion and then increased continuously with the elbow extension in the stroke side, whereas it remained invariant in the control side (Fig. 5A).

(3) K was larger in the control than normal ankle but surprisingly it was lower in the control than in the normal elbow. However, the differences were not significant for any joint.

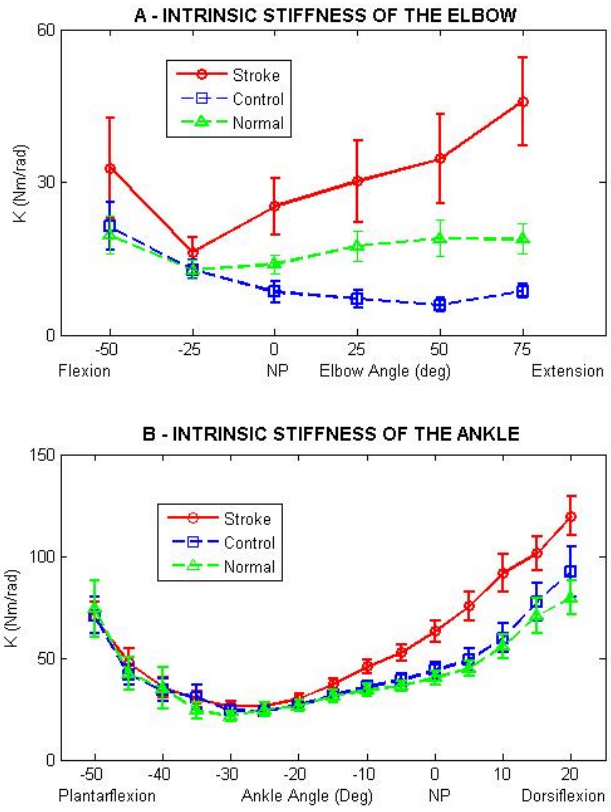

Figure 5*:* Intrinsic stiffness gain (K) as a function of the elbow (top panel) and ankle (bottom) angle for stroke, control and normal groups. Group Mean ±1 Standard Error

V. Discussion and Conclusions

The mechanical behavior of the spastic joint in both upper and lower extremities was quantified over their entire ROM in the chronic stroke subjects using a novel system identification technique

The parallel cascade identification technique has been successful in characterizing elbow and ankle dynamic stiffness and separating their intrinsic and reflex components, simultaneously.

Similar to SCI subjects [3, 4], both intrinsic and reflex stiffness were significantly larger in the stroke than in the control limb indicating that the nature and origins of mechanical abnormalities associated with spasticity are the same in SCI and stroke subjects although the underlying mechanisms of these abnormalities might be different [7, 8].

Intrinsic and reflex stiffness were abnormally high in both spastic ankle and elbow indicating that the nature of mechanical changes in upper and lower extremities caused by stroke is the same. However, intrinsic and reflex contributions to these changes were position dependent; the reflex contributions were maximal at the mid-ROM but intrinsic major contributions were at the end of ROM where spastic muscles were stretched.

These mechanical abnormalities might arise from augmented reflex stiffness that lead progressively to shortening of fiber length in spastic muscle [9] and to changes in the neutral position of the spastic joint. Persistent positional deformity and the accumulation of connective tissues in atrophic muscles secondary to the lesions might increase the passive stiffness [1, 10].

Intrinsic and reflex stiffness of the control joint were different from those of the normal joint for both ankle and elbow. However, the differences were significant only for the ankle joint indicating that the contralateral ankle may not used as an appropriate control [11] for the study of neuromuscular properties in stroke subjects.

These findings help better understanding of the origins of mechanical abnormalities associated with spasticity – fundamental issues about spasticity.

Acknowledgement

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