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Identification and Simulation as Tools for Measurement of Neuromuscular Properties

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ABSTRACT

Quantitative, objective methods for the evaluation of neuromuscular properties are required for the diagnosis of neuromuscular disorders and the evaluation of the effectiveness of treatment and rehabilitation. This paper describes how nonlinear identification and simulation methods may be used to evaluate joint properties quantitatively and distinguish the relative contributions of reflex and intrinsic mechanisms. Results from studies in normal and spinal cord injured subjects are presented to demonstrate the properties of the approach, its viability as a clinical research tool, and some of the issues that arise when comparing correlating its results with clinical evaluations.

Keywords: Ankle Stiffness - Spastic Hypertonia - Muscle tone - Spinal Cord Injury - Stretch Reflex – system Identification

INTRODUCTION

Clinical assessment of joint function typically involves limb manipulation, observations of functional capabilities and patient reports of limitations and pain. Information for other modalities such as X-ray, MRI, CT and ultrasound may also be used. These measures are very valuable for diagnosis and treatment provided that are utilized by skilled and experienced clinicians. However these measures are largely subjective and qualitative so there is a need for objective, quantitative measures of neuromuscular properties to supplement these traditional measurements. Consequently, batteries of quantitative tests have been developed to assess neurological function; quantitative gait assessments have been employed for surgical planning and outcome assessment. Static force-displacement relations have been used to assess joint integrity, the effects of remobilization and the outcome of joint replacement.

One difficulty with most previous quantitative methods is that they are oriented towards the measurement of a response rather than the underlying properties. This makes it difficult to compare the results from different test procedures and methods. In addition, they do not distinguish the contributions made by intrinsic and reflex mechanisms. Thus, as Figure 1 shows, two main mechanisms interact to determine the mechanical behavior of a joint: (1) intrinsic properties of the joint, connective tissues, and active muscles; (2) reflex changes in the level of activation of muscle. Distinguishing the relative contributions of these two mechanisms is important to understand the etiology of a disorder, select an appropriate treatment and evaluate effectiveness. This is particularly relevant when dealing with the abnormal muscle tone associated with spasticity and related clinical syndromes. Here the nature of the mechanical changes associated with spasticity is not yet know; nor is there agreement as to the underlying mechanisms. Some studies point to a hyperactive stretch reflex [1, 2] while others attribute it to changes in the passive stiffness of the joint [3] or to increased intrinsic muscle stiffness [4]. Treatment and rehabilitation programs will be very different depending upon which mechanism causes the abnormal behavior.

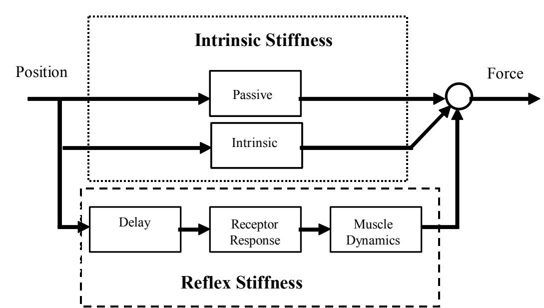

Figure 1: Intrinsic and reflex components of dynamic joint stiffness

A major reason for this continuing controversy is that it is difficult to distinguish the mechanical consequences of reflex activity from those due to the intrinsic properties of joint and muscle. We have developed a nonlinear system identification method that partitions the overall mechanical response into reflex and intrinsic components [5]. This paper will first review the basis of the method and then describe its use in exploring stretch reflex function at the ankle in normal and spinal-cord-injured subjects.

IDENTIFICATION METHOD

The method requires the application of position perturbations which (i) have an average velocity low enough to avoid attenuating reflex responses overly, (ii) containe power over a wide enough bandwidth to identify the dynamics.

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Abstract

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The method assumes that the mechanical response may be partitioned as shown in Figure 1. Intrinsic mechanisms contribute to the torque via an instantaneous response that is approximately linear for a given operating point. Reflex mechanisms are assumed to contribute through a pathway comprising a differentiator, a neural-conduction delay, a static nonlinearly, and linear dynamics. The two components add linearly.

The dynamics of these two pathways are identified separately using the parallel-cascade, nonlinear identification technique illustrated in Figure 2.

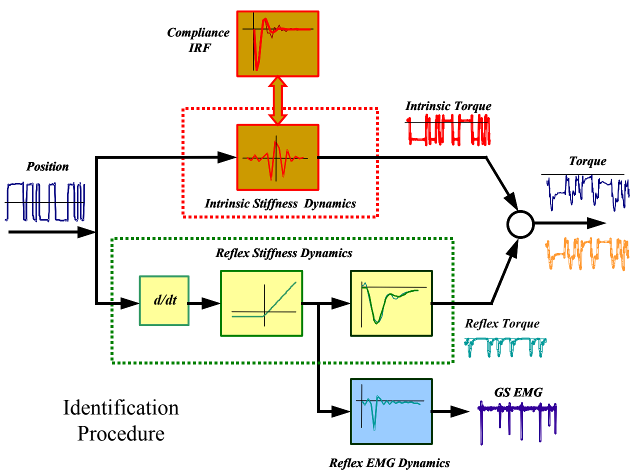

Figure 2: Nonlinear, parallel-cascade identification procedure to separate intrinsic and reflex contributions to joint stiffness.

The method proceeds as follows: (1) Intrinsic dynamics are estimated as a linear dynamic impulse response function (IRF) relating position and torque. The IRF length is always kept shorter than the conduction delay to eliminate any contribution from reflex mechanisms. The dynamic inverse of the intrinsic stiffness dynamics is computed to give the intrinsic compliance which is more convenient to interpret; (2) Reflex dynamics are estimated from the intrinsic pathway residuals using a Hammerstein identification procedure to determine the static nonlinear and linear dynamic elements. (3) Parametric models are fitted to the IRF functions. The dynamic compliance was fitted to the impulse response function (IRF) of

$$TQP_{IRF}(s)=\frac{P(s)}{TQ_I(s)}=\frac{1}{I s^2+Bs+K}$$

The reflex stiffness IRF was fitted to the IRF of:

$$VTQ_{IRF}(s)=\frac{TQ_R(s)}{V(s)}=\frac{G_R\,\omega_n^2\,p}{(s^2+2\xi\,\omega_n s+\omega_n^2)(s+p)}e^{-s\tau}$$

NORMAL SUBJECTS

Typical Results

We first used the method to examine ankle mechanics in normal subjects [6]. We found the system identification procedure yielded good estimates for all components of the model. Figure 3 shows the IRFs and parametric fits estimated from a typical trial with a normal subject.

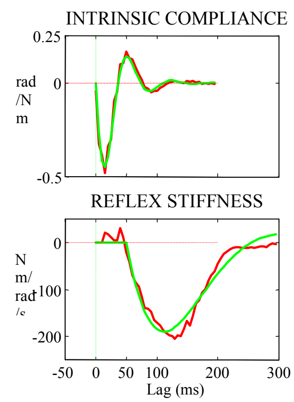

Figure 3: Intrinsic compliance and reflex IRFs estimates from a typical control subject. Parametric fits are superimposed.

These IRFs were used to predict the torques in each pathway to validate the model (Figure 4). For this trial, the overall model accounted for 95% of the torque variance. Intrinsic dynamics accounted for the majority (64%) but the reflex contribution was significant (30%). The shape and frequency content of the intrinsic and reflex torques were very different but they were comparable in magnitude. Frequency analysis demonstrated that reflex contributions were mainly in the 5-10Hz band.

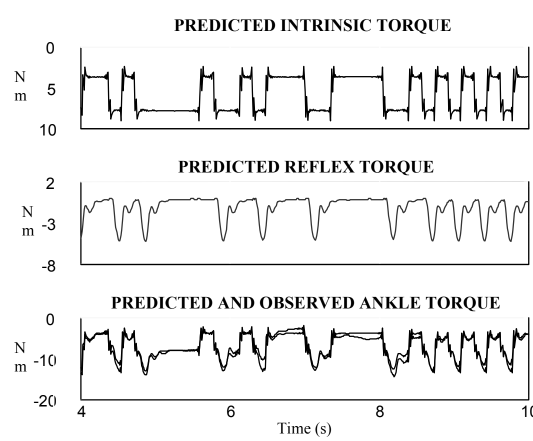

Figure 4: Intrinsic and reflex torques predicted for a typical control subject. The bottom row shows the total predicted torque superimposed on that observed experimentally.

Reliability & Repeatability

We examined the repeatability and reliability of the parameter estimates using a test-retest protocol. Experiments were carried out at the same operating points, on the same subjects on two different days [7]. Results from the same subject were very repeatable as Figure 5 demonstrates although inter-subject variability was high ($r < 0.3$).

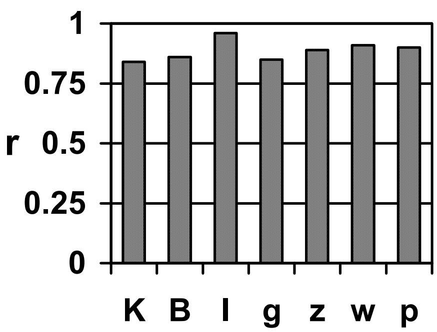

Figure 5:Test-retest reliability of intrinsic and reflex parameters estimated from normal subjects.

Operating Point Effects

Joint Position

Ankle stiffness depended strongly on joint position; both intrinsic and reflex stiffness increased progressively as the joint was moved towards dorsiflexion (Figure 6).

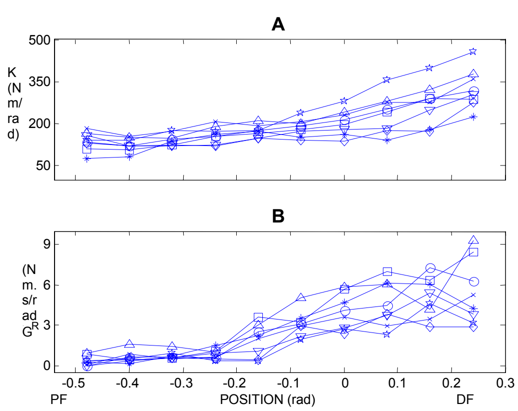

Figure 6: Variation of Intrinsic and Reflex gain with joint position in normal subjects. Each curve represents a different subject.

Voluntary Torque

Reflex stiffness gain behaved in an unexpected manner. It reached a maximum at low levels of voluntary activation and then decreased progressively as activation continued to increase. This was quite different from the behavior of the EMG response where the gain increased. This probably reflects a nonlinear response of the motoneuron pool to voluntary and reflex activation

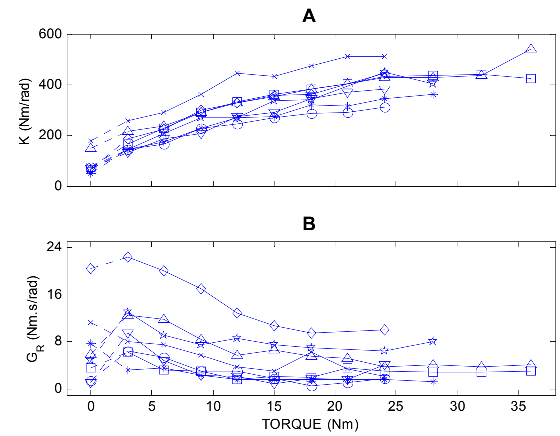

Figure 7: Variation of intrinsic and reflex gain with voluntary torque in normal subjects. Each curve represents a different subject.

Perturbation Properties

Reflex gain varied strongly with the amplitude and frequency content of the position perturbation used to interrogate the system. Both effects could be explained as the result of a decrease in reflex gain in proportion to mean square value of the perturbation velocity

Summary

The nonlinear identification method yields models that describe joint stiffness very well in normal subjects and successfully distinguishes intrinsic and reflex mechanisms. Intra-subject variability is low indicating that the method can be used to track changes with therapy. Inter-subject variability is large suggesting that sensitivity to pathological conditions may be low unless the stiffness changes are large. Model parameters depend strongly on the operating point defined by voluntary torque, joint position and stimulus parameters indicating that clinical studies must (a) match operating points and (b) examine operating point dependence of reflex parameters.

SPASTIC SCI SUBJECTS

Methods

To explore the clinical utility of the method we examined a homogenous group of spinal cord injured subjects (SCIs) having (i) clinically evident spasticity, associated with traumatic spinal cord injury with incomplete motor function loss, (ii) enough retained voluntary control to generate contractions in the ankle muscle, and (iii) long-term spasticity of at least two years duration

SCI subjects were evaluated clinically prior to each experiment using: (i) the modified 5-point Ashworth scale

[8, 9] to assess muscle tone, and (ii) the Fugel-Meyer scale [10] to assess function.

Identification Results

The identification procedure worked well with data from the SCI patients. The nonparametric IRFs fitted the data well and were in turn well described by the parametric models. The most dramatic difference between the results from the normal subjects was that the reflex gain was much higher in the SCI subjects. This is illustrated in Figure 8 that shows reflex stiffness IRFs for typical SCI and control subjects estimated at comparable operating points. The reflex stiffness IRF was much larger in the SCI than the control. This increased reflex gain is reflected in the reflex torques that were much larger in the SCI than the control subject (lower panel).

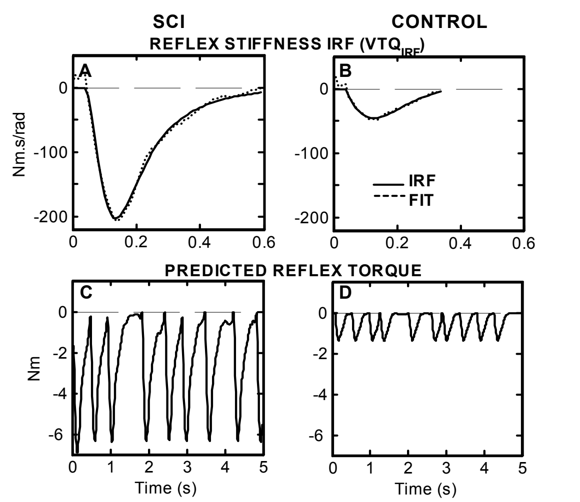

Figure 8: Reflex stiffness IRFS estimates from SCI and control subjects at comparable operating points. Bottom row: reflex torques predicted in response to the same input.

<u>Position Dependence</u>

SCI responses also displayed a reflected the strong dependence on operating point. The dependence on position was qualitatively similar to that of normal subjects – reflex and intrinsic grain increased progressively as the ankle was dorsiflexed as shown in Figure 9 and 10 below. However, reflex gain was consistently much greater in SCI than in normal subjects at all positions. Moreover, strong reflex responses were observed at plantarflexed positions where reflexes were never observed in control subject.

Intrinsic parameters also displayed the same type of position dependence as normal subjects although intrinsic stiffness and viscosity became greater than normal, as the ankle was dorsiflexed.

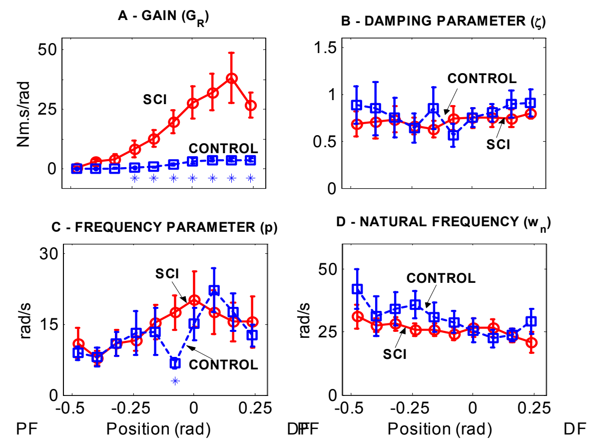

Figure 9: Variation of reflex parameters with joint position in SCI and Control Subjects. Group means and standard errors.

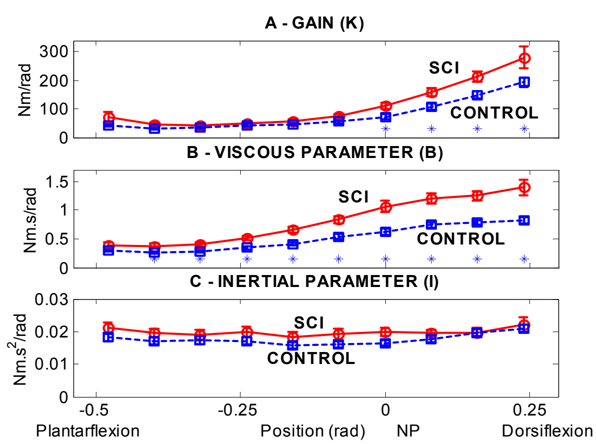

Figure 10: Variation of intrinsic stiffness parameters with joint position in SCI and control subjects. Group means and standard errors

Intrinsic and Reflex Contributions to Torque

The parameters of the stiffness IRFs provide a convenient means of summarizing the overall behavior and comparing groups. However, intrinsic stiffness is position dependent while reflex stiffness is velocity dependent and nonlinear; this makes it difficult to predict how parameter changes will translate into torque changes. Consequently, it is useful to simulate the models response to the experimental input and predict the intrinsic and reflex torques Once this has been done the variances of the different components provides a convenient means of assessing the relative contributions of the two mechanisms as shown in Figure 11.

The top row demonstrates conclusively that ankle mechanics in SCI subjects are very different than normal. The total torque variance is greater in SCIs than normals

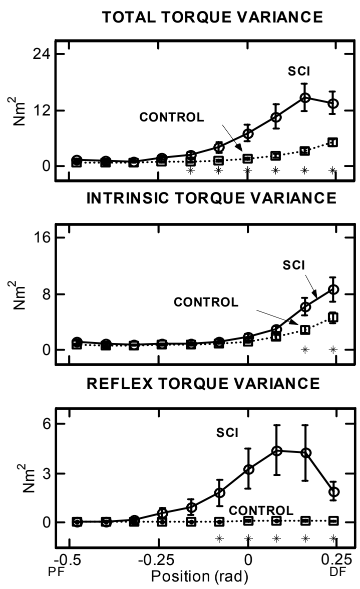

Figure 11: Total, intrinsic and reflex torque variances as functions of joint postion. Group means and standard errors for SCI and control subjects

for all positions > -0.15 rad. Furthermore, an examination of the variance of the intrinsic torque (second panel) and reflex torques (third panel) shows that reflex torques were responsible for this increases at most positions except for the most dorsiflexed.

It is also useful to consider the relative contribution made by reflex torques to the total torque. Figure 12 compares the relative contributions in SCI and normals. The relative contributions of reflex mechanisms were much larger in SCIs, reaching 40% in some cases, than in normals where it never exceeded 10%.

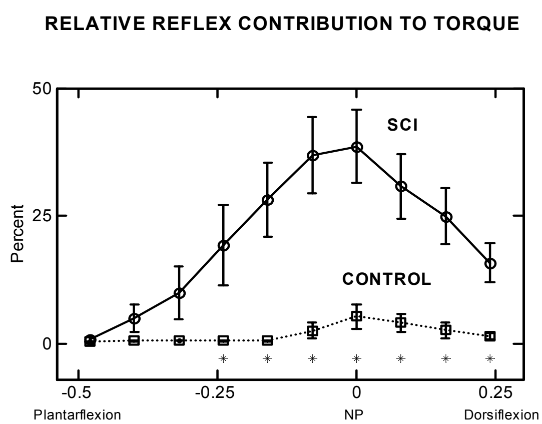

Figure 12: Relative contributions of reflex torque to total ankle torque as functions of joint position. Groups means and SE for SCI and control subjects

Correlation with Clinical Measurements

One surprising result was that there was no significant correlation between our objective measures of joint mechanics and clinical assessments. Figure 13 shows a scatter plot of reflex and intrinsic gain plotted against the Ashworth and Fugl-Meyer scores for all SCI subjects. Thus, while reflex gain was clearly greater than normal its variation did not correlate with clinical assessments. This may well be because the clinical assessments involve moving the limb through a wide range of operating points and so may not correlate with behavior at any one operating point. Rather they may reflect some type of meta-information describing the variation with operating point. This suggests that a more comprehensive nonlinear model would be useful.

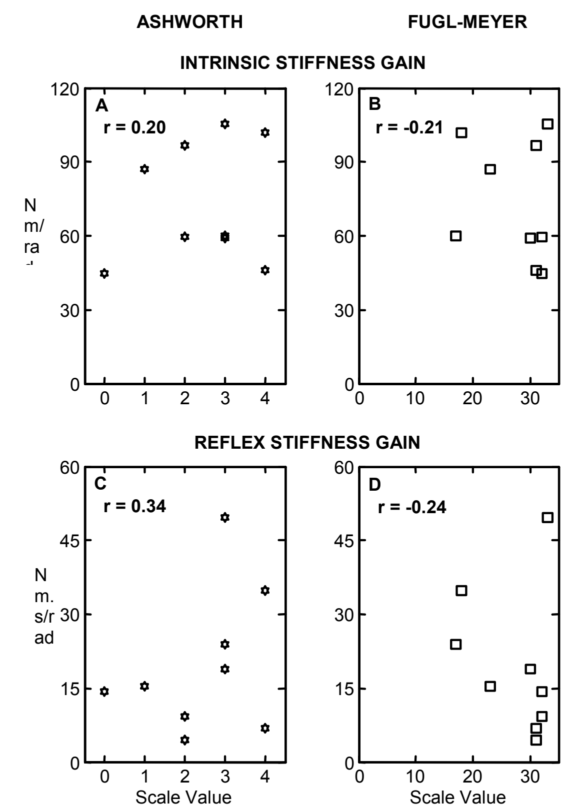

Figure 13: Correlation between Reflex and intrnisc stiffness and clinical evaluations of joint properties.

Assessment of Therapeutic Effects of FES

We have also used the method to track the changes in joint mechanics associated with long term use FES-assisted walking [11]. We examined four subjects before and after using FES-assisted walking for 16-18 months. A fifth SCI subject, who did not use FES, was also tested twice, 17 months apart for control purposes. Intrinsic stiffness gain and viscosity and reflex stiffness gain, dropped substantially in the FES subjects the SCI control subject.

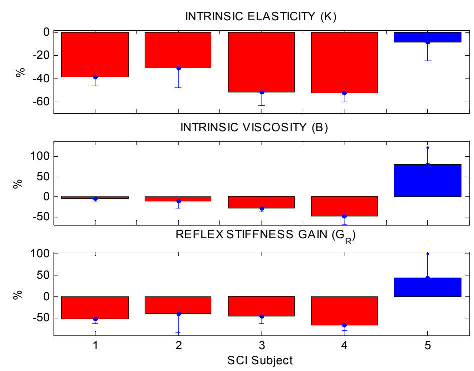

Figure 14:Percentage change in intrinsic and reflex parameters between two examinations separated by 16-18 months. Subjects 1-4 used FES, subject 5 did not.

CONCLUSIONS

System identification provides the means to obtain objective, quantitative measures of the mechanical properties of joints. Furthermore, it makes it possible to separate the intrinsic and reflex contributions in a single, non-invasive experiment. Studies in normal subjects indicate that intra-subject reliability is good while inter-subject variability is high. Moreover, the parameters are strongly dependent on the operating point making it essential to match operating points to compare results.

Experiments with spastic SCI subjects demonstrate that the method can be applied successfully in this population. Changes in reflex parameters were large enough to be statistically significance despite the large inter-subject variability. The feasibility of using the method to assess treatment over long periods of time was demonstrated in a group of SCI patients using FES assisted walking.

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