Author: M. Halaki, N. ODwyer, I. Cathers and S. Heritier
Title: Systematic nonlinear relations between joint mechanics and the neural reflex response with changes in stretch amplitude at the wrist
Journal: Journal of Biomechanics **ISSN**: 0021-9290
Year: 2012
Volume: 45
Issue: 16
Pages: 2755-2762
Abstract: The effects on both joint mechanics and the neural stretch reflex of changes in amplitude of stretch ranging from within the region of short range stiffness up to 3% of resting muscle length were quantified. The level of activation of the primary agonist was held constant but there was a small but systematic increase in activation of the other flexor and extensor muscles as stretch amplitude increased. The patterns of change with stretch amplitude in both the joint mechanics and stretch reflex properties were non-linear but systematic and were well described by power relationships that accounted for high proportions of variance. With an increase in stretch amplitude, joint stiffness, viscosity, damping ratio and natural frequency decreased. In parallel with these mechanical changes, the neural reflex coherence increased and the phase advance and gain decreased. Multiple regression analyses partitioned the variation in the joint mechanics that was attributable to the various factors and it was found that the variation of the mechanics was almost entirely attributable to the variation of the neural reflex gain, which accounted for 93% of the changes in the stiffness, viscosity and natural frequency and 82% of the changes in the damping ratio. It is concluded that joint mechanics are significantly affected by changes in the neural reflex gain.

Author Address: nodwyer@csu.edu.au

Systematic nonlinear relations between joint mechanics and the neural reflex response with changes in stretch amplitude at the wrist

Mark Halaki [a*], Nicholas O'Dwyer [a,b], Ian Cathers [a,c] and Stephane Heritier [d]

[a] Discipline of Exercise and Sport Science, Faculty of Health Sciences, The University of Sydney C42, NSW 2006, Australia

[b] School of Human Movement Studies, Charles Sturt University, Bathurst, NSW 2795, Australia

[c] Discipline of Biomedical Sciences, Sydney Medical School, The University of Sydney C42, NSW 2006, Australia

[d] NHMRC Clinical Trials Centre, The University of Sydney C42, NSW 2006, Australia
Present address: The George Institute for International Health, Sydney, NSW, Australia

*** Corresponding author:** Mark Halaki, Discipline of Exercise and Sport Science, The University of Sydney C42, NSW 2006, Australia. Email: mark.halaki@sydney.edu.au

Key words: stretch reflex; short range stiffness; joint mechanics; viscosity; damping

Word count (introduction-discussion): 3834

ABSTRACT

The effects on both joint mechanics and the neural stretch reflex of changes in amplitude of stretch ranging from within the region of short range stiffness up to 3% of resting muscle length were quantified. The level of activation of the primary agonist was held constant but there was a small but systematic increase in activation of the other flexor and extensor muscles as stretch amplitude increased. The patterns of change with stretch amplitude in both the joint mechanics and stretch reflex properties were non-linear but systematic and were well described by power relationships that accounted for high proportions of variance. With an increase in stretch amplitude, joint stiffness, viscosity, damping ratio and natural frequency decreased. In parallel with these mechanical changes, the neural reflex coherence increased and the phase advance and gain decreased. Multiple regression analyses partitioned the variation in the joint mechanics that was attributable to the various factors and it was found that the variation of the mechanics was almost entirely attributable to the variation of the neural reflex gain, which accounted for 93% of the changes in the stiffness, viscosity and natural frequency and 82% of the changes in the damping ratio. It is concluded that joint mechanics are significantly affected by changes in the neural reflex gain.

INTRODUCTION

The high degree of adaptability of the stretch reflex may render it a useful mechanism for 'tuning' joint mechanical characteristics (Neilson, 1993; Rack, 1981). However, it has been difficult to establish the relative importance of the stretch reflex in determining joint mechanical characteristics because of other factors such as the intrinsic muscle stiffness and muscle contraction and co-contraction levels.

Previous studies of the effects of increasing stretch amplitude on joint mechanics have reported decreases in joint stiffness (Halaki et al., 2006; Kearney and Hunter, 1982; Loram et al., 2007) and damping coefficient (Halaki et al., 2006; Kearney and Hunter, 1982; Milner and Cloutier, 1998). Muscles exhibit initial short range stiffness where the tension increases as the muscle is stretched up to ~1% of resting length. This is followed by a sudden yield in resistance with the tension then levelling off (Rack and Westbury, 1974). Sinkjaer et al (1988) reported that the reflex increased the ankle stiffness by 40-100% of the intrinsic stiffness and that stiffness did not change for stretches between 1° and 7°. This finding is at odds with other studies (Kearney and Hunter, 1982; Loram et al., 2007) which found that ankle stiffness decreased over the same range. Sinkjaer et al (1988) did not measure the neural reflex response but others have reported that both gain and phase of the neural reflex decrease with increasing stretch amplitude (Cathers et al., 1999).

The above mentioned studies have investigated relations between stretch amplitude and joint mechanics without reporting on the neural stretch reflex response, or on relations between amplitude and the neural reflex response without reporting the joint mechanics. The purpose of the present study was to better understand the relations between stretch

amplitude, joint mechanics, and concomitantly, the neural reflex response. Varying the stretch amplitude provides a way of manipulating the neural reflex while controlling for contraction level, hence elucidating the contribution of the reflex to the mechanical properties of the limb (stiffness, viscosity, inertia, damping, natural frequency).

METHODS

The methods used in the current study have been previously reported in detail (Halaki et al., 2006).

Subjects

Ten subjects (5 male; 5 female) aged 26-50 years with no history of neurological or musculoskeletal disease gave their written informed consent prior to testing. The procedures were approved by the relevant Human Ethics Committee.

Equipment

Subjects were seated with their shoulder abducted ~60°, elbow flexed ~30°, forearm in the neutral position and palm facing medially. The forearm was firmly strapped and hand firmly clamped in a 'U' bracket attached to a manipulandum with their wrist above its axis of rotation. The manipulandum was attached to a DC servomotor (Baldor, ASR-Servotron-SD55-15-AI) driven in position control (Baldor, UM4-150-2-045). The angular position (Sakae, KSM-2201) and torque (Transducer Techniques, TRT-500) were measured.

Pairs of 10 mm diameter Ag/AgCl surface electrodes placed ~25 mm apart measured electromyographic (EMG) signals over flexor carpi radialis (FCR) and carpi radialis

(ECR) as per Basmajian and Blumenstein (1980). An earth electrode was placed over the olecranon of the ulna.

To monitor the contraction levels in other muscles around the wrist, extensive sampling of muscle activity was carried out in one subject using intramuscular electrodes (221-24-730, Chalgren Enterprises Inc.) from the following prime flexors (Backdahl and Carlsoo, 1961) and extensors (McFarland et al., 1962): FCR, flexor carpi ulnaris, flexor digitorum superficialis, flexor digitorum profundus, ECR longus and brevis, extensor carpi ulnaris and extensor digitorum.

EMG signals were amplified (gain = 1000) and band-pass (10-500 Hz) filtered (Biopac Systems, EMG100B). All signals were sampled at 2000 Hz (16-bit A/D converter, Biopac Systems, MP100A) using AcqKnowledge (Version 3.5.3) software.

Procedures

Flexion and extension maximum voluntary isometric contractions (MVICs) were collected with the wrist in neutral position. Subjects were then instructed to maintain an EMG target contraction level of 15%MVIC in FCR using biofeedback but to otherwise not intervene in response to the imposed perturbations. This contraction level was chosen to avoid fatigue while still providing a reasonable signal-to-noise ratio in the EMG signals.

Displacement signals

Sixty sinusoidal signals of 20 seconds duration at 10 frequencies (3, 4, 5, 6, 7, 8, 9, 10, 11, 12 Hz) with 6 root mean square (RMS) amplitudes (0.4, 0.5, 0.6, 2, 4, 6° RMS) were employed in a randomised order. Calculations using

anatomical (Horii et al., 1993; Lieber et al., 1990) and physiological (Campbell and Lakie, 1998; Hill, 1968; Wiegner, 1987) data indicated that 0.4-0.6ºRMS displacements were within the short range (<1% of resting length) stiffness region (Rack and Westbury, 1974) for FCR, while 2-6ºRMS displacements were beyond that range.

Signal analysis

Since the frequencies of interest were <15 Hz, the displacement and torque signals were low-pass filtered at 20 Hz (8^{th} order Butterworth, zero-lag). The EMG signals were first high-pass filtered (20 Hz, 8^{th} order Butterworth, zero-lag), rectified, low-pass filtered (20 Hz, 8^{th} order Butterworth, zero-lag) then normalised by the maximum of the processed EMG from three MVIC trials of each muscle, and henceforth referred to as IEMG. Signals were then re-sampled at 100 Hz (Matlab, The Mathworks Inc., Version-6).

The wrist mechanics and neural reflex properties were obtained as before (Cathers et al., 1999, 2004; Halaki et al., 2006) using a cross-correlation and spectrographic analysis (Bendat and Piersol, 1971) which identifies the best-fit linear relation between the torque (input) and position (output) signals for the mechanical response and between the position (input) and IEMG (output) signals for the neural reflex response. These relations were described by coherence square, gain and phase relations between the signals with a frequency resolution of 0.25 Hz and were calculated as follows.

For input signal $X = x_1, x_2, x_3, \ldots x_N]$ and output signal $Y = y_1, y_2, y_3, \ldots y_N]$ (where N is the number of sample point in each signal), let G_x and G_y be their respective frequency power spectra and let G_{xy} be the cross-power spectrum between X and Y. The coherence

square frequency response function between Y and X provides the ratio of the coherent output to the total output at each frequency and is calculated using Equation 1:

(1)

The gain frequency response function provides the amplitude ratio of Y/X (output/input) at each frequency and is derived from the coherent component of the cross-power spectrum and the spectrum of X as shown in Equation 2:

(2)

For the neural reflex gain the ratio is IEMG/position.

The phase frequency response function provides the relative phase between Y and X at each frequency and is calculated using of Equation 3 in the complex plane, which is the arctangent of the imaginary part and the real part of the cross-power spectrum G_{xy} at each frequency:

(3)

To determine the torque contribution of the wrist, torque due to the manipulandum alone was subtracted from total torque due to the manipulandum+wrist. This was possible because the motor was running in position control mode with identical position signals.

Modelling the mechanics

The mechanics of the wrist were modelled previously (Halaki et al., 2006) using the following second-order system:

$$\tau(t)=I\cdot d^2\phi(t)\cdot dt^{-2}+B\cdot d\phi(t)\cdot dt^{-1}+K\cdot\phi(t) \quad (4)$$

where:

τ = Torque (Nm)

ϕ = Displacement (rad)

K = Stiffness ($Nm\cdot rad^{-1}$)

B = Damping coefficient or viscosity ($Nm\cdot s\cdot rad^{-1}$)

I = Limb moment of inertia ($kg\cdot m^2$)

For each subject, values of *I*, *B* and *K* at each amplitude were chosen to provide the best fit modelled mechanical gain and phase to the measured mechanical gain and phase for the 10 frequencies at that amplitude by minimising the RMS of the difference between measured and modelled values (Halaki et al., 2006). The natural frequency (Hz) was then calculated as Wn= — – and the damping ratio as ——.

Relationships between amplitude, neural reflex and mechanics

The stretch amplitude may affect joint mechanics via one of two paths illustrated in Figure 5. In one path, stretch amplitude influences the neural reflex response (path 2), which in turn affects the mechanical properties of the limb (path 3). The other possible path is independent of the reflex whereby stretch amplitude may produce a direct mechanical effect on the limb. This path includes both passive and intrinsic muscle and joint properties. Since we did not measure the effect of stretch amplitude on joint

mechanics independent of reflex effects, both paths could not be separated experimentally. However, the relative contribution of each path was determined analytically by investigating the following relationships:

1) joint mechanics (i.e., stiffness, viscosity, damping ratio and natural frequency) obtained from the second order mechanical model (Equation 4) and amplitude previously reported (Halaki et al., 2006)
2) the neural reflex response (i.e., gain, phase and coherence square) obtained from the neural reflex response (Equations 1-3) and amplitude
3) joint mechanics and the neural reflex response.

The relationship between amplitude and joint mechanics (path 1 in Figure 5) has been reported to be a power relationship (Halaki et al., 2006; Loram et al., 2007). It is not known what functional forms are appropriate to describe the relationships in paths (2) and (3) though it has been suggested that nonlinearities are involved (Kearney and Hunter, 1982; Sinkjaer et al., 1988).

Statistical methods

Repeated measures analyses of variance (ANOVA) with two factors (amplitude and frequency) were performed to assess the effects of stretch amplitude and frequency on the reflex response. Individual subject profile plots and average group plots were systematically used to explore the functional form of the relationships between variables (Diggle et al., 1995). In all cases where there was not a linear relation between the original variables, logarithmic transformations applied to the response and/or predictor produced linear relations. Since values of coherence square tended to saturate close to 1

as amplitude increased (see Figure 2), log(1-reflex coherence square) was used. Plots of log(1-reflex coherence square) versus log(amplitude) revealed linear relationships between the variables for all subjects. Similarly, plots of log(neural reflex gain) versus log(amplitude) showed linear relationships for all subjects. The relationships between phase and stretch amplitude were linear without logarithmic transformation.

It was found that the linear relationships for each subject had a similar slope but a different intercept. Therefore, a linear regression model with the subject factor set as random was used, i.e. using a linear model with a random intercept for each subject but with the same slope. Such a model also accounted for the changes in a variable across trials for each subject. This procedure is essentially similar to a one-way repeated measures ANOVA, where the variability due to individual subjects is taken into account.

The patterns of change with amplitude of the parameters of coherence square, gain and phase were found to be similar across all frequencies (see Figure 2). Consonant with this, the patterns of change with amplitude of log(1-reflex coherence square) versus log(amplitude), log(neural reflex gain) versus log(amplitude) and phase versus amplitude also were found to be similar across all frequencies, with high r^2 values (0.88 ± 0.20). The linear regression models showed similar linear functions at all frequencies for each parameter but with a different slope at each frequency. Therefore, in order to provide a single representative measure of the changes in these parameters with amplitude, their values were averaged across frequency at each amplitude for each subject. These averages were then used in the regression analyses presented in the Results.

RESULTS

Raw data

A sample of the raw data obtained from one subject for the 0.4°RMS and 6°RMS stretches at 5 Hz is presented in Figure 1. Reflex responses can be seen during the smaller stretches but a higher level reflex entrainment can be seen during the larger amplitude stretches by clear bursts of EMG. The neural reflex phase advance, indicated by the start of the EMG bursts before the stretch, can be seen in the closer alignment of the reflex response with the velocity traces than the displacement traces. In fact the reflex response was slightly in advance of the velocity traces, reflecting a phase advance >90° at 5 Hz (see Figure 2). ECR activity level was minimal with little entrainment to stretch.

The average neural reflex coherence square, gain and phase for all amplitudes and frequencies are plotted in Figure 2. The coherence, gain and phase changed across frequency ($F[9,81] \geq 58.72$, $p<0.001$) and amplitude ($F[5,45] \geq 54.85$, $p<0.001$). The coherence and gain increased with frequency, whereas the phase advance was roughly constant between 3-7 Hz and then decreased for higher frequencies. Patterns of change of the neural reflex parameters with amplitude will be described in detail in the following sections.

Contraction levels and wrist torque

Subjects kept the level of activation of FCR very close to the 15%MVIC target with a mean ± standard deviation of 14.8±0.4%MVIC. The ECR activation level was low but

increased with stretch amplitude (see Figure 1) from 2.5 to 5.5%MVIC ($F[5,45]=18.10$, $p<0.01$). This increase in co-contraction, combined with small but systematic changes in activation levels of the other flexor and extensor muscles for which no activation feedback or instructions were given, most likely accounts for a decrease observed in net flexor torque from 1.5 to 1.0Nm ($F[5,45]=19.68$, $p<0.001$) as stretch amplitude increased. The intramuscular EMG recordings in a single subject showed that these other flexor and extensor muscles behaved similarly to the surface ECR recordings for the group. The activation levels were low and increased by a small amount from the smallest (mean=36±9μV) to the largest (mean=61±14μV) stretch amplitude at which, by contrast, the average level of activity recorded intramuscularly in FCR was 129±1μV.

Path (1): Stretch amplitude and joint mechanics

The main findings previously reported (Halaki et al., 2006) were that stiffness, viscosity, damping ratio and natural frequency decreased nonlinearly with increasing displacement amplitude. The average (± standard deviation) inertia as calculated from the model was 0.0024±0.0013 $kg \cdot m^{-2}$ which was virtually identical to the value of 0.0024±0.0011 $kg \cdot m^{-2}$ that calculated by Winter (1990) using anthropometric data. The relationship between stretch amplitude and joint mechanics (path 1 in Figure 5) was described by power functions between the mechanical parameters (MP) and displacement amplitude (*A*) measured in (°RMS) as follows:

$$MP = \beta 1 \; A \; \alpha_1 \qquad (5)$$

where β_1= subject-specific constant and α_1= MP specific constant (Table 1). High values of r^2 (0.86-0.95) were obtained for all regression models. The relationships between the changes of stiffness, viscosity and inertia as amplitude changed were

variable across subjects (between stiffness and viscosity: $0.10 \leq r \leq 0.94$, between stiffness and inertia: $-0.47 \leq r \leq 0.44$, between viscosity and inertia: $-0.98 \leq r \leq 0.20$).

Path (2): Stretch amplitude and neural reflex gain

The mean across frequencies for all subjects of neural reflex coherence square, gain and phase for each amplitude are plotted in Figure 3. It can be seen that a power relation applied for both gain and coherence square.

Reflex gain (RG) measured in (%MVIC·degree^{-1}) was well described by the following function:

$$RG = \quad (6)$$

The fitted values were $\alpha_{2RG} = -0.76$ (0.02) with a subject-specific constant β mean of 5.6 (range: 4.8-8.6).

Reflex coherence square (RC) was equally well represented by the following function:

$$RC = \quad - \quad (7)$$

Again the fitted values were $\alpha_{2RC} = -0.61$ (0.03) with a subject-specific constant β mean of 0.06 (range: 0.05-0.09).

Finally, there was no clear evidence of a power relation between neural reflex phase (RP) measured in degrees and amplitude. A graphical analysis shows that the initial plot is somewhat linear (Figure 3c), making the use of a log-transformation doubtful. A possible function was:

$$RP = \quad + \quad (8)$$

The fitted values were α_{2RP}= -4.13 (0.44) with a subject-specific constant β_{2RP} mean of 95 (range: 82-113). Note that all models fitted the data well as represented by r^2 = 0.75-0.98.

The coherence square, gain and phase of the neural reflex response were correlated with each other with a coefficient (r) of -0.65 between coherence square and gain, of 0.42 between gain and phase and of -0.58 between coherence square and phase.

Path (3): Neural reflex gain and joint mechanics

To relate the changes observed in the mechanics to the neural reflex response, similar regression analyses were performed between the neural reflex parameters and the mechanical parameters and power relationships were again found to apply. Because of the multicollinearities between neural reflex gain, coherence square and phase, all three variables could not be accounted for in the multiple regressions simultaneously. Using neural reflex gain in a stepwise multiple regression accounted for most of the variance; therefore, only the relationships between the mechanical parameters and neural reflex gain are presented (Figure 4). The relationship was described by the following equation:

$$MP = \quad \quad (9)$$

where β = subject-specific constant and α = MP-specific constant (Table 1).

Comparison of path (1) versus paths (2) and (3) combined

The contribution of the stretch reflex to the changes in joint mechanics with changes in stretch amplitude can be determined by combining the mathematical relationships for

paths (2) and (3) in Figure 5. By substituting (Equation 6) into (Equation 9), we obtain:

$$MP = \quad ^{\alpha}$$

Simplifying the above equation we get:

$$MP = \qquad (10)$$

where β = is a subject-specific constant.

The effect of stretch amplitude on the joint mechanics via neural reflex gain (paths 2 and 3 in Figure 5) can now be compared to the total effect of amplitude on the joint mechanics via reflex, intrinsic and passive factors (path 1 in Figure 5). If amplitude does act on the mechanical parameters independently of the neural reflex gain, there should be a difference between α and the product of the two parameters α , α . As displayed in Table 1, α_1 and $\alpha_{2RG} \cdot \alpha_{3RG}$ are almost identical for all endpoints. Therefore, there is a close agreement between the two paths suggesting that almost all of the observed variation of the joint mechanics due to amplitude can be explained via the neural reflex gain. Amplitude does not appear to act independently on the joint mechanics in the presence of stretch reflex.

To confirm the validity of using the average across frequencies to represent the changes of the neural reflex parameters, the products of $\alpha_{2RG} \cdot \alpha_{3RG}$ at each frequency are presented in Table 2. These products matched closely with the product derived from the average across frequency (Table 1), except at the lowest frequencies where the neural reflex coherence was lower (Figure 2) and hence the estimates of gain were less reliable. Both sets of $\alpha_{2RG} \cdot \alpha_{3RG}$ products matched closely with α_1. Therefore, averaging

across frequencies provided a valid but simplified measure of the changes of the neural reflex parameters with amplitude.

Finally, a check was made of the relation between the neural reflex gain and stretch velocity. Because stretch velocity is correlated with stretch amplitude - at a given frequency of sinusoidal displacement, velocity increases as amplitude increases - it was necessary to ensure that the relation observed between reflex gain and stretch amplitude was not simply a by-product of changes in stretch velocity. Figure 6 shows the reflex gain for a typical subject plotted against RMS velocity for the 60 sinusoidal signals - six amplitudes of stretch at 10 frequencies. It can be seen that at each amplitude the neural reflex gain increased with the increase in RMS velocity associated with increasing frequency. However, it is also clear that a different relationship existed between reflex gain and velocity for each amplitude. If the neural reflex response were due only to velocity dependency, then the gain should have increased with increasing stretch amplitude, whereas in fact it decreased. Therefore, it can be concluded that there was a primary relationship between reflex gain and stretch amplitude that was not simply a by-product of changes in stretch velocity.

DISCUSSION

This study has quantified the systematic effects on both joint mechanics and the stretch reflex of changes in stretch amplitude ranging from within the region of short range stiffness (<1%) up to 3% of resting muscle length. The patterns of change in both the joint mechanics and neural reflex properties were nonlinear and well described by power relationships with subject-specific constants.

The neural reflex responses changed markedly over the range of stretch amplitudes studied. The increase in the neural reflex coherence square (~15%) and decrease in phase advance (~20%) with increasing amplitude were small compared to the decrease in gain (~90%). The increase in neural reflex coherence with increased amplitude has been reported previously and was attributed to the fact that a larger input can be expected to produce greater modulation of neural activity in reflex pathways compared to smaller stretches (Cathers et al., 1999). The novelty is that we have given a quantitative expression for the variations of the coherence with amplitude. Similarly, the decrease in the neural reflex gain with amplitude is consistent with previous findings (Cathers et al., 1999; Neilson and McCaughey, 1981). A power function related amplitude and both the neural reflex gain and coherence square. Although the phase advance decreased with amplitude, it was not a clear power relationship. This is consistent with previous findings where both a phase independent of amplitude and a negative association have been reported. The finding of Neilson and McCaughey (1981) that phase was independent of stretch amplitude could be due to different instructions to the subjects i.e. to keep their arm as still as possible, which required co-contraction, whereas in the present study subjects were to maintain a flexion force with little or no co-contraction. Such differences in the subject's intention may vary the amplitude and latency of the neural reflex response (Colebatch et al., 1979; Crago et al., 1976). The subject instructions in the present study were similar to those given by Cathers et al. (1999) who observed similar decreases in phase advance with increasing amplitude.

It is well established that the stretch reflex significantly modifies the joint mechanics. The reflex has been reported to change the total stiffness from 200% (Allum and Mauritz, 1984) to 500% (Houk et al., 1970) of areflexive muscle and contribute ~20-

45% of total knee stiffness (Dhaher et al., 2005). Sinkjaer and Hayashi (1989) reported that when compared to the areflexive wrist, the stretch reflex at low contraction levels caused changes >100% in wrist stiffness, ~12% in inertia and viscosity, ~42% in the damping ratio and ~50% in the natural frequency. The stretch reflex has also been reported to change the form of the mechanical response from one dominated by viscosity to one dominated by elasticity (Lin and Rymer, 1993). Kearney et al. (1997) also reported that the reflex accounted for ~85% of the ankle torque variance at contraction levels of 10%MVIC.

The changes in the neural reflex response found in the current study were consistent with the changes in the joint mechanics. The patterns of change in both neural reflex and mechanical properties were nonlinear and well described by power relationships. The decrease in muscle spindle gain in response to increasing stretch amplitude also has been shown to be a power relation (Matthews and Stein, 1969; Poppele, 1973). This reinforces the role of the neural reflex gain in determining joint mechanics.

CONCLUSION

An increase in stretch amplitude produced a decrease in reflex gain and phase advance, joint stiffness and viscosity, natural frequency and damping ratio. Regression analyses showed that the changes in joint mechanics with changes in stretch amplitude could be accounted for by changes in reflex gain due to changes in stretch amplitude. All regression relations were well described by power functions incorporating a subject-specific constant term. It is concluded that joint mechanics are significantly affected by changes in reflex gain when the muscle contraction level of the primary agonist is held

constant. This knowledge may assist in understanding various movement disorders and the role of the stretch reflex in altered joint mechanics.

ACKNOWLEDGEMENTS

This work was funded by National Health and Medical Research Council of Australia.

AUTHOR CONTRIBUTIONS

Experiments were conducted at the Reflex Laboratory, The University of Sydney. Authors MH, NOD and IC contributed to the conception and design of the experiment, collection, analysis and interpretation of data, drafting the article or revising it critically for important intellectual content. Author SH contributed to the statistical analysis, interpretation of data and revising the article critically for important intellectual content.

CONFLICT OF INTEREST

There are no conflicts of interest for any of the authors.

REFERENCES

Allum, J.H., Mauritz, K.H., 1984. Compensation for intrinsic muscle stiffness by short-latency reflexes in human triceps surae muscles. Journal of Neurophysiology 52, 797-818.

Backdahl, M., Carlsoo, S., 1961. Distribution of activity in muscles acting on the wrist. (An electromyographic study). Acta Morphologica Neerlando-Scandinavica 4, 136-144.

Basmajian, J.V., Blumenstein, R., 1980. Electrode placement in emg biofeedback. Williams & Wilkins, Baltimore.

Bendat, J.S., Piersol, A.G., 1971. Random data: Analysis and measurement procedures. Wiley-Interscience, New York.

Campbell, K.S., Lakie, M., 1998. A cross-bridge mechanism can explain the thixotropic short-range elastic componenet of relaxed frog skeletal muscle. Journal of Physiology 510, 941-962.

Cathers, I., O'Dwyer, N., Neilson, P., 1999. Dependence of stretch reflexes on amplitude and bandwidth of stretch in human wrist muscle. Experimental Brain Research 129, 278–287.

Cathers, I., O'Dwyer, N., Neilson, P., 2004. Variation of magnitude and timing of wrist flexor stretch reflex across the full range of voluntary activation. Experimental Brain Research 157, 324-335.

Colebatch, J.G., Gandevia, S.C., McCloskey, D.I., Potter, E.K., 1979. Subject instruction and long latency reflex response to muscle stretch. journal of physiology 292, 527-534.

Crago, P.E., Houk, J.C., Hasan, Z., 1976. Regulatory actions of human stretch reflex. Journal of Neurophysiology 39, 925-935.

Dhaher, Y.Y., Tsoumanis, A.D., Houle, T.T., Rymer, W.Z., 2005. Neuromuscular reflexes contribute to knee stiffness during valgus loading. Journal of Neurophysiology 93, 2698-2709.

Diggle, P., Liang, K.-Y., Zeger, S., 1995. Analysis of longitudinal data. Oxford University Press.

Halaki, M., O'Dwyer, N., Cathers, I., 2006. Systematic nonlinear relations between displacement amplitude and joint mechanics at the human wrist. Journal of Biomechanics 39, 2171-2182.

Hill, D.K., 1968. Tension due to interaction between the sliding filaments in resting striated muscle. The effect of stimulation. Journal of Physiology 199, 637-684.

Horii, E., An, K.N., Linscheid, R.L., 1993. Excursion of prime wrist tendons. Journal of Hand Surgery. American Volume 18, 83-90.

Houk, J.C., Singer, J.J., Goldman, M.R., 1970. An evaluation of length and force feedback to soleus muscles of decerebrate cats. Journal of Neurophysiology 33, 784-811.

Kearney, R.E., Hunter, I.W., 1982. Dynamics of human ankle stiffness: Variation with displacement amplitude. Journal of Biomechanics 15, 753-756.

Kearney, R.E., Stein, R.B., Parameswaran, L., 1997. Identification of intrinsic and reflex contributions to human ankle joint stiffness dynamics. IEEE Transactions on Biomedical Engineering 44, 493-504.

Lieber, R.L., Fazeli, B.M., Botte, M.J., 1990. Architecture of selected wrist flexor and extensor muscles. Journal of Hand Surgery. American Volume 15, 244-250.

Lin, D.C., Rymer, W.Z., 1993. Mechanical properties of cat soleus muscle elicited by sequential ramp stretches: implications for control of muscle. Journal of Neurophysiology 70, 997-1008.

Loram, I.D., Maganaris, C.N., Lakie, M., 2007. The passive, human calf muscles in relation to standing: the non-linear decrease from short range to long range stiffness. Journal of Physiology 584, 661-675.

Matthews, P.B.C., Stein, R.B., 1969. The sensitivity of muscle spindle afferents to small sinusoidal changes of length. Journal of Physiology 200, 723-743.

McFarland, G.B., Krusen, U.L., Weathersby, H.T., 1962. Kinesiology of selected muscles acting on the wrist: electromyographic study. Archives of Physical Medicine and Rehabilitation 43, 165 - 171.

Milner, T.E., Cloutier, C., 1998. Damping of the wrist joint during voluntary movement. Experimental Brain Research 122, 309-317.

Neilson, P.D., 1993. Tonic stretch reflex in normal subjects and in cerebral palsy, in: Gandevia, S., Burke, D., M., A. (Eds.), Science and practice in clinical neurology. Cambridge University Press, Sydney.

Neilson, P.D., McCaughey, J., 1981. Effect of contraction level and magnitude of stretch on tonic stretch reflex transmission characteristics. Journal of Neurology, Neurosurgery and Psychiatry 44, 1007-1012.

Poppele, R.E., 1973. Systems approach to the study of muscle spindles, in: Stein, R.B., Pearson, K.G., Smith, R.B., Redford, J.B. (Eds.), Control of posture and locomotion. Plenum Press, New York, pp. 127-144.

Rack, P.M.H., 1981. Limitations of somatosensory feedback in control of posture and movement, in: Geiger, S.R. (Ed.), Handbook of Physiology: The Nervous System. American Physiological Society, Bethesda, Maryland, pp. 229-256.

Rack, P.M.H., Westbury, D.R., 1974. The short range stiffness of active mammalian muscle and its effect on mechanical properties. Journal of Physiology 240, 331-350.

Sinkjaer, T., Hayashi, R., 1989. Regulation of wrist stiffness by the stretch reflex. Journal of Biomechanics 22, 1133-1140.

Sinkjaer, T., Toft, E., Andreassen, S., Hornemann, B.C., 1988. Muscle stiffness in human ankle dorsiflexors: intrinsic and reflex components. Journal of Neurophysiology 60, 1110-1121.

Wiegner, A.W., 1987. Mechanism of thixotropic behavior at relaxed joints in the rat. Journal of Applied Physiology 62, 1615-1621.

Winter, D.A., 1990. Biomechanics and motor control of human movement. Wiley-Interscience, New York.

Table 1: The mean (standard error) of the exponents (alpha) and mean (range) of the intercepts (beta) in the mathematical relations between stretch amplitude ($alpha_1$, $beta_1$) and joint mechanics as previously reported (Halaki et al., 2006), between stretch amplitude ($alpha_{2RG}$) and neural reflex gain and between neural reflex gain ($alpha_{3RG}$, $beta_{3RG}$) and joint mechanics. The product of $alpha_{2RG}$ and $alpha_{3RG}$ is also shown for comparison with $alpha_1$.

Mechanical parameter	α_1	β_1	α_{2RG}	α_{3RG}	β_{3RG}	$\alpha_{2RG} \cdot \alpha_{3RG}$
Relation	**(mechanics – amplitude)**		**(neural reflex gain – amplitude)**	**(mechanics – neural reflex gain)**		
K (Nm rad^{-1})	-0.11 (0.02)	6.94 (4.17 – 13.25)	-0.76 (0.02)	0.15 (0.02)	5.89 (3.29 – 10.38)	-0.11
B (Nm s rad^{-1})	-0.47 (0.02)	0.08 (0.05 – 0.14)		0.60 (0.03)	0.03 (0.01 – 0.04)	-0.46
Damping ratio	-0.48 (0.03)	0.32 (0.20 – 0.39)		0.61 (0.04)	0.11 (0.07 – 0.14)	-0.46
Wn (Hz)	-0.12 (0.01)	9.26 (7.27 – 10.83)		0.16 (0.01)	7.07 (5.65 – 8.28)	-0.12

Table 2: The product of alpha$_{2RG}$ and alpha$_{3RG}$ ($\alpha_{2RG} \cdot \alpha_{3RG}$) when the neural reflex gain was analysed separately at each frequency

Frequency	3 Hz	4 Hz	5 Hz	6 Hz	7 Hz	8 Hz	9 Hz	10 Hz	11 Hz	12 Hz
K (Nm rad^{-1})	-0.10	-0.10	-0.11	-0.11	-0.11	-0.11	-0.11	-0.11	-0.11	-0.11
B (Nm s rad^{-1})	-0.38	-0.38	-0.43	-0.43	-0.44	-0.45	-0.45	-0.45	-0.45	-0.45
Damping ratio	-0.38	-0.39	-0.43	-0.44	-0.45	-0.45	-0.46	-0.46	-0.46	-0.45
Wn (Hz)	-0.06	-0.11	-0.12	-0.12	-0.13	-0.14	-0.13	-0.13	-0.14	-0.12

Figure Captions

Figure 1: Raw data from Subject 1 showing torque, displacement, velocity, raw EMG and IEMG from FCR and ECR for the 0.4° RMS and 6° RMS stretches at 5 Hz. Upward displacement is extension of the wrist. The calibrations show amplitude only and do not indicate signal offsets.

Figure 2: Group mean (a) coherence square, (b) gain and (c) phase of the neural reflex response for FCR vs. frequency for all stretch amplitudes.

Figure 3: Mean (± standard error) across all subjects and frequencies of (a) coherence square, (b) gain and (c) phase of the neural reflex response for FCR vs. the six stretch amplitudes, (d) log (1 - average coherence square of the reflex response for FCR), (e) log (average gain of the reflex response for FCR) vs. log (stretch amplitude) and (f) phase of the reflex response for FCR vs. stretch amplitude and regression lines for 3 subjects. The r^2 values show the variance accounted for by the random subject factor model for 10 subjects. For each variable, the intercept is different for each subject but the slope of the regression line is the same for every subject.

Figure 4: (a) Log (stiffness), (b) log (viscosity), (c) log (damping ratio) and (d) log (natural frequency) vs log (neural reflex gain) and regression lines for three subjects. The r^2 values show the variance accounted for by the random subject factor model for 10 subjects. For each variable, the intercept is different for each subject but the slope of the regression line is the same for every subject.

Figure 5: Paths between stretch amplitude and joint mechanics indicating the mathematical relations (1) between stretch amplitude and joint mechanics, (2) between

stretch amplitude and neural reflex gain and (3) between neural reflex gain and joint mechanics.

Figure 6: The gain of the neural reflex response plotted against the average velocity of stretch for all signals for a typical subject (subject 6).