

# **FLEXION-RELAXATION INDUCED CREEP DEFORMATION ALTERS PARASPINAL REFLEX RESPONSE AND RECOVERY**

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## **INTRODUCTION**

Trunk flexion postures are a well recognized risk factor for low back pain.<sup>1,2</sup> Recent investigations link prolonged and/or cyclic trunk flexion to impaired neuromuscular function. Specifically, animal models show that the reflex response in the paraspinal muscles is disturbed following spine flexion.<sup>3</sup> This flexion induced neuromuscular disturbance was attributed to creep deformation of the passive tissue in the spine.<sup>4</sup> During prolonged static and cyclic lumbar flexion, viscoelastic tissues in the spine may provide resistance against flexion loading. This allows the trunk muscles to become deactivated, i.e. flexion-relaxation. The passive tissue load produces tissue laxity and creep deformation. It is indicated by an increase in relative trunk angle over time. Laxity in viscoelastic tissues may cause mechanoreceptors in the ligaments to become desensitized. As a result, their ability to monitor vertebral movements and initiate reflexive muscular action is reduced.<sup>4</sup>

The goal of this investigation was to determine the effect of prolonged flexion-relaxation and recovery time on paraspinal reflex behavior in human subjects.

## **METHODS**

A total of 25 human subjects with no history of lower back pain participated in the study. In order to record trunk muscle response and kinematics behavior, subjects were seated without trunk support in an upright posture.

A restraining belt was strapped around the subject's waist to immobilize the pelvis. A harness and cable system attached the subject to the servomotor at the T10 level of the torso.

The subjects resisted a constant isotonic flexion preload by maintaining an upright posture. Pseudorandom stochastic force perturbations ( $\pm 70\text{N}$ ) were superimposed on the preload to elicit reflexes in the paraspinal muscles. The applied forces were measured by a force transducer and electromyographic (EMG) response was recorded from surface electrodes over the right and left lumbar paraspinal muscles. A trial consisted of two ten-second force perturbation sequences during which reflexes and recorded.

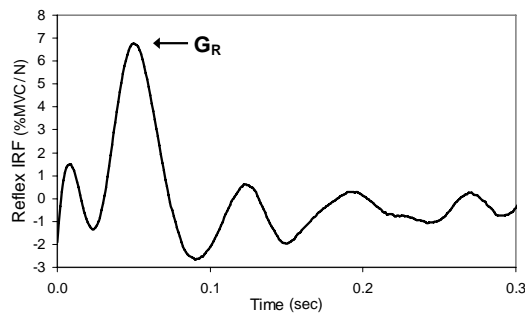
Paraspinal reflexes were recorded prior to beginning static flexion-relaxation. Spinal ligament stretch was induced by having subjects lean forward to a flexion-relaxation posture while remaining seated within the pelvic restraint. This posture was held for four minutes while the trunk angle was recorded using EMG sensors. Then, the subjects returned to the upright posture and a reflex trial was recorded. The cycle was repeated four times for a total of 16 minutes of static flexion-relaxation.

A nonparametric impulse response function (IRF) was calculated from the pseudorandom force input and the rectified EMG output of the erector spinae muscles. Calculation of the IRF was based on

deconvolution techniques for a time delayed linear system response.

$$y(t) = \int_{T_1}^{T_2} IRF(\tau)x(t - \tau)dt = IRF(t) * x(t)$$

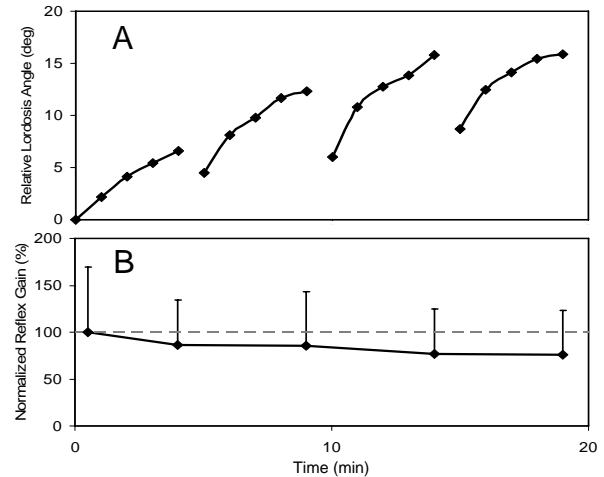
Reflex gain,  $G_R$ , was calculated from the peak of the IRF (Figure 1). Reflex gain characterizes the magnitude of muscle reflex response scaled with respect to the time dependent amplitude of the force distribution. High  $G_R$  indicates a large reflex response. The kinematics gain,  $G_K$ , was estimated from a similar analysis relating force input to torso movement.



**Figure 1:** Typical IRF computed from applied trunk force (input) and rectified EMG (output). Reflex gain was computed from the peak of the IRF.

## RESULTS AND DISCUSSION

Results showed that prolonged static flexion-relaxation influenced the function of the neuromuscular system in human beings. Relative lordosis angle increased during each four minute flexion-relaxation cycle (Figure 2A) indicating creep deformation of the passive lumbar spine. In addition, lumbar paraspinal reflex gain decreased throughout the flexion-relaxation trials. Significant, but incomplete recovery occurred during the one minute between trials. However, reflex gain declined significantly after 16 minutes of flexion-relaxation (Figure 2B). The decrease in reflex gain follows the change in spinal tissue laxity. Tissue laxity can result in desensitization of neurosensors and explain the noted reduction in reflex gain.



**Figure 2:** Lordosis angle increased during flexion-relaxation with significant, but incomplete recover occurring between trials (A). Reflex gain declined significantly following static flexion-relaxation (B).

## SUMMARY/CONCLUSIONS

Results confirm that prolonged static flexion-relaxation in humans results in passive tissue creep and neuromuscular changes. Reduced reflexes suggest that the spine was less stable following prolonged flexion-relaxation, and therefore, more susceptible to lower back pain and injury. Thus, extended rest may be required for full recovery. Inhibited paraspinal reflexes may contribute to the risk of lower back injury in workers using a flexed posture due to the inability of the neuromuscular system to coordinate an appropriate muscle response to an unexpected load.

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