NEUROMOTOR HABITUATION AS A MECHANISM FOR VIBRATION INDUCED LOW BACK PAIN

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Introduction

Occupational exposure to whole body vibration has long been associated with increased incidence of low back pain and low back injuries[1]. A number of studies have investigated transmissibility of seat pan vibration [5, 6]. While transmissibility has been well researched, the mechanism by which vibration may induce injury has not been thoroughly studied. Winter et al. identified increased reflex response delay after vibration exposure and speculated that muscular fatigue may be the cause of this increase[9]. However, a mechanism has yet to be demonstrated completely.

A potential mechanism that may explain the increased risk is neuromotor habituation. Muscle spindle organs have been shown in the extremities to be sensitive to muscle and tendon vibration. Rapid length changes in muscle have been shown to result in kinesthetic illusions as the regular firing of the muscle spindles is interpreted as muscle lengthening[4, 7]. These illusions have also been demonstrated in the paraspinal musculature[2]. With removal of vibration, research in the extremities has demonstrated increased positioning errors, probably due to neuromotor habituation[8].

In this research, it has been hypothesized that neuromotor habituation after exposure to occupational vibration will increase positioning errors. It is further hypothesized that these errors can be shown to be linked to increased reflex response time. Such increased reflex response time could, in turn, decrease spinal stability and increase low back injury risk.

Methods

Both positioning error and sudden load response were measured before and after exposure to 20 minutes of 5 Hz, 0.223 m/s^2 RMS seat pan vibration. Subjects were asked to sit on an unpadded seat without a backrest. Throughout the whole body vibration period, subjects were instructed to put their hands on a stable hand rest and feet on an adjustable stable footrest. The subjects were instructed to assume a comfortable and relax sitting posture for the duration of the exposure.

Positioning error was measured using an active-active reposition sense protocol. Electromagnetic markers (Motionstar, Ascension Tech, Burlington, VT) were used to track trunk motion. With markers attached to the skin at the T10 vertebra, the S1 vertebra and manubrium, trunk flexion (the angle from vertical of the line connecting T10 and S1) and lumbar curvature (the difference in inclination of the T10 and S1 markers) were tracked. In the reposition sense protocol, subjects were asked to maintain an upright trunk flexion and to rotate their pelvis and lumbar curvature to assume a target lumbar curvature. In the protocol subjects completed training trials, where they were asked to match their lumbar curvature using a visual display, and assessment trials, where they were asked to reproduce the lumbar curvature from memory. After two initial training trials, training trials and assessment trials were alternated for a total of 3 assessment trials. Reposition error was defined as the absolute difference between the target lumbar curvature and the lumbar curvature the subject assumed during the assessment trials.

For sudden loading trials, subjects were asked to stand on a force plate with their pelvis fixed with a belt. A sudden impulse load was applied by dropping a weight of 4.5 kg a height of 10 cm. The weight applied a sudden flexion moment through a chest harness. Electromyographic (Delsys, Boston, MA) data was recorded from the erector spinae, rectus abdominus and internal and external oblique muscle groups. Trunk motion was collected with the electromagnetic sensors.

A simulink model (MATLAB, Natick MA) was created in which the trunk was modeled as an inverted pendulum and muscle reflex response was modeled as a feedback with a detection threshold, a fixed time delay, and a linear gain. Overall trunk stiffness and trunk inertia from Cholewicki et al. were used[3]. An increase in positioning errors was modeled as an increase in detection threshold.

Results

Both reposition error and erector spinae muscle activity delay were found to increase significantly after exposure to vibration, returning close to baseline after approximately 20 minutes. This pattern was also reflected in the significant increase after vibration in trunk flexion in response to sudden loading.

By increasing detection threshold for reflex response in the model, it was possible to show that changes in the detection threshold (position error) would indeed increase response delays and increase trunk flexion. It was shown that altering gain did not change these delays suggesting that muscular fatigue may not explain the data.

Discussion

From the model, it can be predicted that loss in proprioception (position sense) can lead to increased muscle response times and increased trunk flexion in response to a sudden load. This was also demonstrated experimentally. This association supports the hypothesis that neuromotor habitation from vibration can lead to loss in proprioception and in turn alter low back stabilization. Future work will examine occupational factors such as seating configuration and vibration frequency on these neuromotor changes.

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