INTRODUCTION

Occupations involving frequent flexed trunk postures are associated with a higher incidence of low back pain (LBP) [1]. A single exposure to static trunk flexion leads to creep deformation of trunk viscoelastic tissues, reducing passive trunk stiffness [2]. Such reductions in passive trunk stiffness require neuromuscular compensation to maintain mechanical equilibrium and stability of the spine [2], and may require a longer time for recovery than the initial exposure duration [3]. Repeated static flexion may thus result in an accumulation of disturbances to trunk mechanical and neuromuscular behaviors. In this study, the effects of flexion duration and duty cycle on trunk intrinsic stiffness and reflex response were investigated.

METHODS

Healthy young adults with no self-reported history of LBP participated, after completing an informed consent procedure approved by the Virginia Tech IRB. Participants included six males with mean (SD) age = 23(2) yr, stature = 177.9 (3.7) cm, and body mass = 71.6 (8.4) kg. Respective values for six females were 25.7 (1.7) yr, 163.6 (4.5) cm, and 58.3 (2.9) kg. Trunk posture was monitored using electromagnetic sensors (Xsens, Los Angeles, CA, USA) over the T12 spinous processes, and muscle activity (EMG) was recorded using bipolar surface electrodes over the bilateral erector spinae, external obliques, and rectus abdominis muscles. Participants completed six counterbalanced experimental sessions involving exposure to all combinations of three durations (1, 2, and 4 min) of static flexion, and two flexion duty cycles (33% and 50% exposure relative to exposure + rest durations).

During the experiment, participants stood in a rigid metal frame designed to restrain the pelvis and lower limbs in a fixed, but comfortable posture. Static trunk flexion was achieved by bending forward in a controlled manner until minimal trunk extensor muscle activity was observed, indicating a relaxed flexed posture. This procedure was expected to induce creep deformation of passive posterior tissues. Participants repeated a sequence of static flexion-rest-flexion according to the assigned duration and duty cycle; this was done continuously, for 48-minute periods.

A pseudorandomly-timed sequence of 12 anterior-posterior position perturbations of ±5mm were applied to the trunk at ~T8 via a servomotor (Kollmorgen, Radford, VA), rigid rod, and chest harness (Fig. 1), prior to and immediately following exposure periods. During these perturbations, participants maintained a constant sub-maximal extensor effort of 10% MVC using real-time visual feedback of EMG. Trunk displacements were measured with both the servomotor encoder and a laser displacement sensor (Keyence, Osaka, Japan), while reaction forces were measured using an in-line load cell (Interface SM2000, Scottsdale, AZ, USA).

Figure 1: Experimental set-up demonstrating a participant during neuromuscular measurement.
Neuromuscular behaviors were characterized by several measures. Reflex delay was determined as the time between perturbation onset and reflexive muscle response for each anteriorly directed perturbation (via EMG) [4]. Intrinsic trunk stiffness was quantified by relating measured trunk kinematics to trunk kinetics during the reflex delay [5]. Reflex response was estimated by subtracting the intrinsic response from measured trunk kinetics and was correlated to delayed trunk velocity to estimate reflex gain [5]. The overall effects (i.e., differences between neuromuscular measures at t=0 and t=48 min) of flexion duration, duty cycle, and gender were assessed using mixed-factor analyses of variance (ANOVA). Statistical significance was determined when p < 0.05.

RESULTS AND DISCUSSION

Mean (SD) pre-exposure trunk stiffness was higher (p<0.0001) among males than females, at 8300 (1262) and 6252 (922) N/m, respectively. Intrinsic trunk stiffness decreased significantly with increasing flexion duration (p=0.01) and in the higher duty cycle (p=0.04). Muscle reflex delays were comparable (p=0.19) between males [62(5) ms] and females [65(3) ms], and were unaffected by trunk flexion duration (p=0.37) or duty cycle (p=0.27). These results indicate that a longer duration of static flexion and insufficient rest periods between these increase the severity of changes to intrinsic stiffness. Since background muscle activity during perturbations was maintained at the same level, the decrement in intrinsic stiffness was most likely caused by alterations in passive mechanical trunk properties, indicating accumulated creep deformation following repeated static flexion [6].

Pre-exposure reflex gain did not differ (p=0.73) between genders. Though not significant (p=0.09), reflex gains decreased with increasing duty cycle. Another measure of reflex response (i.e., maximum reflex force) decreased (p=0.03) with increasing duty cycle, but was similar across flexion durations. There was a gender effect (p=0.005) on reflex gain, with females having substantially larger decreases after exposure. These decreased reflex responses are consistent with evidence from feline models, in which a decreased reflex response was found following prolonged cyclic lumbar flexion-extension [6]. Repetitive or cyclic tasks are presumably responsible for developing laxity in the viscoelastic tissues that may diminish the intensity of muscular activation.

CONCLUSIONS

Increased reflexive responses have been suggested as compensatory adaptations to decreases in passive trunk stiffness following prolonged static flexion [2,5]. The present results (Fig. 2), however, suggest that repeated static flexion decreases both passive trunk stiffness and reflexive responses. Specifically, longer flexion durations or shorter rest periods between flexion induce more severe changes. Hence, simultaneous decreases in both passive trunk stiffness and reflexive responses may increase the risk for spinal instability and low-back injury following repeated static flexion tasks.

**Figure 2:** Effects of flexion duration on trunk intrinsic stiffness and reflexive gain following repeated static flexion. Values are normalized to pre-exposure measures

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ACKNOWLEDGEMENTS

This work was supported by Award Number R01 OH004089 from the CDC. Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the CDC.