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Active trunk stiffness increases with co-contraction

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Abstract

Trunk dynamics, including stiffness, mass and damping were quantified during trunk extension exertions with and without voluntary recruitment of antagonistic co-contraction. The objective of this study was to empirically evaluate the influence of co-activation on trunk stiffness. Muscle activity associated with voluntary co-contraction has been shown to increase joint stiffness in the ankle and elbow. Although biomechanical models assume co-active recruitment causes increase trunk stiffness it has never been empirically demonstrated. Small trunk displacements invoked by pseudorandom force disturbances during trunk extension exertions were recorded from 17 subjects at two co-contraction conditions (minimal and maximal voluntary co-contraction recruitment). EMG data were recorded from eight trunk muscles as a baseline measure of co-activation. Increased EMG activity confirms that muscle recruitment patterns were different between the two co-contraction conditions. Trunk stiffness was determined from analyses of impulse response functions (IRFs) of trunk dynamics wherein the kinematics were represented as a second-order behavior. Trunk stiffness increased 37.8% ($p < 0.004$) from minimal to maximal co-activation. Results support the assumption used in published models of spine biomechanics that recruitment of trunk muscle co-contraction increases trunk stiffness thereby supporting conclusions from those models that co-contraction may contribute to spinal stability.

Keywords: Co-activation; Spine; Stiffness; Stability

1. Introduction

Muscle stiffness increases with activation as a result of the increased number of activated cross-bridges [25]. Muscle activation has been shown to increase joint stiffness in the elbow [1,35], the ankle [19], and the trunk [28,29]. With co-contraction the activity of the agonist and antagonist muscles increase thereby causing increased joint stiffness [17]. Although biomechanical models of the spine have assumed that recruitment of antagonistic co-contraction causes increased trunk stiffness [8,13] in it has never been empirically demonstrated in the trunk [9].

Trunk stiffness is an important contributor to spinal stability. Stability describes the ability to maintain equilibrium despite the presence of kinematic and/or control disturbances. Although passive tissues contribute to trunk stiffness, the ligamentous spine without active muscular support is unstable [6], therefore trunk stiffness is primarily associated with active muscles of the torso musculature [7]. Research concludes that paraspinal muscle reflexes also contribute to apparent stiffness of a joint by responding to perturbation movements and associated muscle strain with proportional muscle activation [5,14]. The stiffness data reported in the current study is the combined behavior of the active intrinsic muscle stiffness and the reflex response. This is referred

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to as the "effective stiffness" [4]. Effective stiffness can be accurately measured using small amplitude pseudo-random force disturbances and resulting trunk movement [28].

Empirical measurements demonstrate that trunk stiffness increases with exertion effort and associated muscle activity [4,28]. Clearly recruitment of antagonistic co-contraction must cause an increase in trunk muscle activation of both the antagonist and agonist muscle groups [12]. Therefore, we hypothesize that active trunk stiffness must increase with co-activation of the torso musculature due to the associated increase in recruitment. The specific aim of the project was to evaluate trunk dynamics, specifically stiffness, using systems identification analyses from data recorded during trunk extension exertions with minimal and maximal voluntary co-contraction recruitment.

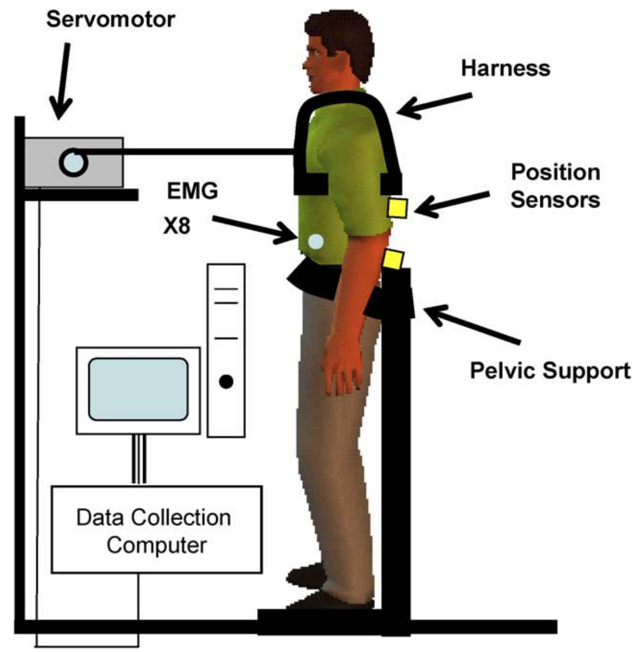

Fig. 1. Experimental setup. Force perturbations in a pseudo-random fashion superimposed on isotonic loads were applied to subjects to elicit small trunk movement during two co-activation conditions (maximal and minimal voluntary recruitment of flexor muscles). Subjects were securely strapped into a rigid pelvic support structure to isolate movement of the trunk for all trials.

2. Methods

2.1. Experiment

Seventeen subjects with no previous history of LBP participated after signing informed consent approved by the institutional review board at Virginia Tech. The mean (standard deviation) height and mass of the subjects were 175.5 (12.0) cm and 74.3 (14.2) kg, respectively.

The experiment consisted of an assessment of trunk stiffness at two recruitment conditions (minimal and maximal voluntary co-contraction) while maintaining constant trunk extension exertions. Subjects were attached to a servomotor (Pacific Scientific, Rockford, IL) via a harness and cable system such that anteriorly directed horizontal loads were applied at the T10 level of the trunk (Fig. 1). The servomotor applied constant isotonic preloads, which the subject was instructed to resist by maintaining an upright posture. Isotonic loads included 15% and 30% of the subject's maximum voluntary exertion (MVE). MVE force was measured in isometric trunk extension prior to the experiment. Subjects were instructed to maximally recruit their trunk flexor muscles as antagonists during maximum co-contraction trials while maintaining an upright posture against the preload. During minimum co-contraction trials subjects were instructed to relax their trunk flexor muscles while maintaining an upright posture. Recruitment and preload conditions were presented in random order.

During the exertions pseudorandom binary (PRB) perturbations of ± 70 N were superimposed on the force preload and were measured with a force transducer (Omega, Stamford, CT) attached to the motor. The force perturbations produced small flexion and extension movements of the trunk that were recorded with electromagnetic position sensors (Ascension Technology Corp., Burlington, VT; position resolution = 0.8 mm). Two six degree-of-freedom position sensors were taped to the skin over the spinous process at S1 and T10 and sampled at 100 Hz. Two trials with duration of 20 s were performed in each condition. Subjects were instructed to maintain the desired co-contraction effort throughout the trial. Fatigue was minimized by experimental design of low exertion levels, i.e., 15% and 30% MVC and also by requiring at least 1 min rest between each trial.

EMG data were collected during each trial to document co-active recruitment. EMG signals were collected from bipolar surface electrodes (Delsys, Boston, MA) on the left and right rectus abdominus (RA), lumbar paraspinal (LP), internal oblique (IO), and external oblique (EO) as described in Granata [11]. All EMG data were band-pass filtered in hardware between 20 and 450 Hz and sampled at 1000 Hz. The EMG signals were rectified and filtered using a 15 Hz, low-pass, seventh-order Butterworth filter. EMG were normalized to their corresponding peak EMG values recorded during maximum isometric flexion, extension, and lateral twisting exertions. Reported EMG recruitment data represents the average isometric baseline value from the first 250 ms of recorded data i.e., during steady state preload and prior to perturbations. All trunk extension exertions were sagitally symmetric. Preliminary results indicated that there was no significant