



Persons with lower-limb amputation have impaired trunk postural control while maintaining seated balance

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ABSTRACT

Abnormal mechanics of movement resulting from lower-limb amputation (LLA) may increase stability demands on the spinal column and/or alter existing postural control mechanisms and neuromuscular responses. A seated balance task was used to investigate the effects of LLA on trunk postural control and stability, among eight males with unilateral LLA (4 transtibial, 4 transfemoral), and eight healthy, non-amputation controls (matched by age, stature, and body mass). Traditional measures derived from center of pressure (COP) time series, and measures obtained from non-linear stabilogram diffusion analyses, were used to characterize trunk postural control. All traditional measures of postural control (95% ellipse area, RMS distance, and mean velocity) were significantly larger among participants with LLA. Non-linear stabilogram diffusion analyses also revealed significant differences in postural control among persons with LLA, but only in the antero-posterior direction. Normalized trunk muscle activity was also larger among participants with LLA. Larger COP-based sway measures among participants with LLA during seated balance suggest an association between LLA and reduced trunk postural control. Reductions in postural control and spinal stability may be a result of adaptations in functional tissue properties and/or neuromuscular responses, and may potentially be caused by repetitive exposure to abnormal gait and movement. Such alterations could then lead to an increased risk for spinal instability, intervertebral motions beyond physiological limits, and pain.

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1. Introduction

Low back pain (LBP) represents a substantial secondary impairment among persons with lower-limb amputation (LLA) [1]. LBP prevalence is substantially higher among persons with LLA (52–71%) compared to the general population (6–33%) [2,3]. More than half (52%) of persons with LLA report experiencing at least one back pain episode in the prior month, 25% describe their LBP as constant, and 31% describe their pain as severe [2]. LBP may even be more bothersome than residual limb and phantom limb pain, two pain sources considered major contributors to post-amputation morbidity [2,3]. While the problem of LBP among persons with LLA has been well documented, the underlying causes in this population remain largely unknown. Given the high prevalence and debilitating nature of LBP among persons with LLA, it is important to understand the mechanisms of LBP onset and recurrence in this population.

Notable alterations and asymmetries in gait following LLA have been described, including larger or more prolonged forces generated by and transmitted through the contralateral limb during the stance phase of gait [4]. Such altered and asymmetric movement patterns have been associated with increased three-dimensional trunk kinematics compared to able-bodied controls [e.g., 5], suggesting that abnormal mechanics of movement resulting from LLA may increase stability demands on the spinal column and/or alter existing postural control mechanisms and neuromuscular responses. Further, changes in spinal posture (e.g., lordosis, scoliosis) and/or muscle architecture, sometimes observed in persons with LLA [e.g., 6,7], may also influence such responses. Despite these alterations/adaptations in gait and posture, the effects of LLA and subsequent prosthetic use on trunk postural control and spinal stability are not well understood.

Maintaining spinal stability requires efficient and synergistic responses from passive structures and active neuromuscular control [8]. The aforementioned alterations in gait and movement with LLA may result in new spinal loading patterns, rates, and magnitudes, which could chronically alter motor control strategies and functional properties of the passive spine. Numerous studies have demonstrated disturbances in passive and active trunk behaviors of healthy persons following acute exposure to

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mechanical loading and atypical postures, suggesting an association between altered trunk behaviors, reduced postural control, and the occurrence of LBP. For example, prolonged static or dynamic trunk flexion reduces passive support of the spine [9], and whole-body vibration exposure can compromise trunk proprioception [10]. Mechanical or sensory deficits in passive tissues can also lead to decreased muscle force output, reduced proprioception, and inhibition of stretch reflexes [11]. Thus, repeated exposures to abnormal mechanics of motion could lead to similar, but chronic, alterations in trunk behaviors, and subsequent reductions in trunk postural control and spinal stability among persons with LLA.

Measures of the center of pressure (COP) during seated balance have been used to assess trunk postural control in both healthy individuals and LBP patients, and impaired trunk postural control has been associated with spinal instability and LBP [e.g., 12]. While LLA is associated with deficits in whole-body postural control during quiet standing [13], these are likely a result of lost musculature in the lower limb (e.g., at the ankle joint). During quiet upright stance, postural adjustments can be made using a variety of responses through the ankle, knee, hip, and lumbosacral joints. It was therefore anticipated that performing a task not requiring the lower limbs, where inherent differences and asymmetries are present among persons with LLA, a better understanding of the effects of LLA on trunk postural control and spinal stability could be obtained. Therefore, the goal of the present work was to investigate trunk postural control among persons with LLA during a seated stability task. It was hypothesized that persons with LLA would have impaired trunk postural control compared to non-amputation controls, evidenced by increases in COP-based seated sway measures, and suggesting a decrease in spinal stability and the potential for increased risk of low back injury.

2. Methods

2.1. Participants

Eight males with unilateral LLA (4 transtibial, 4 transfemoral) and eight male, non-amputation controls participated (Table 1). The most frequent reason for amputation was trauma (5), followed by congenital deformity or abnormality (2), and cancer (1). The mean (SD) duration of prosthetic use among the LLA group was 12.1 (10.1) years. Members of the control group were recruited to match participants with LLA, at the individual level, in terms of age, stature, and body mass (within <8 year, <5 cm, and <5 kg, respectively). Inclusion criteria for participants with LLA, consistent with previous biomechanical studies [14,15], were: (1) adults with a unilateral above- or below-knee amputation; (2) regular/daily use of prosthesis (≥ 1 year post-amputation/rehabilitation); and, (3) independent locomotion without the use of walking aids. All participants completed the short, self-administered version of the International Physical Activity Questionnaire (IPAQ) [16], and were categorically identified as moderately active. Potential participants (in both groups) were excluded if they had any recent history (6 months) of falls, neurologic deficits, or any underlying musculoskeletal disorders (not including amputation)

that could confound the results. In particular, none of the participants in the study had low back pain at the time of testing. Each participant completed initial informed consent procedures approved by the Virginia Tech Institutional Review Board. Participants with LLA wore their prosthetic device during all testing procedures.

2.2. Experimental design and procedures

Seated balance was tested using an unstable chair (Fig. 1A) that pivots on a low-friction ball-and-socket joint. Adjustments to the seat allow for the participant's center of mass to be centered over the ball-and-socket. Four springs are placed circumferentially, in each cardinal direction, to provide supplemental support [17]. These springs can be adjusted inward/outward (7–22 cm) from the center, thereby facilitating control of task difficulty by altering the resistance of the chair to rotation (Fig. 1B). Following calibration procedures [17,18], the spring positions were converted to a percentage of the gravitation gradient (∇G) for each participant seated on the chair. The value of ∇G determines the mass (or weight) distribution of the participant on the chair, with 100% ∇G specifying spring positions that will fully equilibrate the gravitational gradient (i.e., facilitate seated stability with no need for participant compensation). Here, the task difficulty (i.e., spring positions) was standardized to 60% ∇G for all four springs. A similar setting has been used previously, and is a difficulty level sufficient for discerning differences in seated sway measures between groups or exposure conditions [17,19]. It is slightly conservative, however, since it was not known a priori to what degree postural control would be degraded among persons with LLA. Pelvic motions were minimized using a belt placed across the hips, and an adjustable footrest limited motion of the lower-limbs and kept the knees and hips at $\sim 90^\circ$ angles.

Initially, participants performed seated maximum voluntary contractions (MVC) in trunk flexion, extension, and left/right lateral bending. These were done in a separate fixture, with a custom chest harness connected to a fixed anchor via a rigid rod, allowing participants to make maximal efforts in the desired direction. During MVCs, electromyographic (EMG) activities of the bilateral lumbar (L3) erector spinae, rectus abdominis, and external oblique muscles were recorded using bipolar Ag/AgCl surface electrodes, and following existing electrode placement protocols [20]. Initially, the skin was prepared using abrasion and cleaned with alcohol, and inter-electrode impedance was maintained below 10 K Ω . Raw EMGs were preamplified (100 \times) near the collection site, bandpass filtered (10–500 Hz), amplified, and converted to RMS in hardware (Measurement Systems Inc., Ann Arbor, MI, USA), then sampled at 1000 Hz. Peak EMG-RMS values were identified, and used subsequently for normalization (see below).

Participants were given five initial practice trials to reduce learning effects and acclimate to the task [18], which involved maintaining seated balance on the chair using (primarily) lumbar spine motion. Participants then completed three seated balance trials, and were instructed to keep the chair surface as level as possible while sitting with an upright posture (no slouching), eyes open and looking straight ahead, and arms folded across their

Table 1

Mean (SD) participant characteristics in the lower-limb amputation (LLA) and control groups. Reported *p* values represent group comparisons (pooled transtibial + transfemoral vs. control) from unpaired *t* tests. TTA: transtibial, TFA: transfemoral.

	TTA (<i>n</i> =4)	TFA (<i>n</i> =4)	LLA (<i>n</i> =8)	Control (<i>n</i> =8)	<i>p</i>
Age (year)	43.0 (26.1)	39.0 (12.0)	41.1 (18.7)	36.9 (13.4)	0.61
Stature (cm)	174.5 (6.4)	175.8 (4.4)	175.0 (5.0)	174.2 (3.8)	0.74
Body mass (kg)	73.1 (12.9)	79.1 (7.0)	76.6 (10.2)	80.3 (11.4)	0.50

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