The Effect of Prolonged Walking With Intermittent Standing on Erector Spinae and Soleus Muscle Oxygenation and Discomfort

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Abstract
Prolonged periods of walking have been associated with musculoskeletal discomfort and injuries. Previous research has shown that muscle fatigue is related to decreases in muscle oxygenation during short term walking. The objective of the proposed research is to determine the impact of prolonged walking with intermittent standing on musculoskeletal discomfort and muscle oxygenation measures in young adults. Nine young adults walked for a period of 2 hours. Ratings of perceived discomfort were recorded using a questionnaire. Muscle oxygenation and hemoglobin levels were collected from the lower back erector spinae and soleus muscles using near infrared spectroscopy (NIRS). Subjective discomfort significantly increased throughout the 2 hours. Prolonged walking generally induced increased oxygenation of the erector spinae and soleus across walking periods, within walking periods and across standing periods. These increases were more pronounced at the beginning of the walking session and continued through the second or third periods. Erector spinae and soleus total hemoglobin increased within walking period one and two. Only the soleus total hemoglobin significantly increased after the first walking and standing periods and during all the transitions from walking to standing. Increased oxygenation and total hemoglobin during prolonged walking with intermittent standing are likely a result of the repeated dynamic contractions and exercise-induced blood volume expansion. Increased discomfort was found; however, this was not explained by detrimental changes in oxygenation or total hemoglobin.

Key words Muscle oxygenation, walking, discomfort, NIRS, walk.

Introduction
Many jobs require prolonged periods of standing or walking. In 2016, employees across all occupations spent an average of 61% of their workday, nearly 5 hours, on their feet and at least 75% in many occupations, including food service, healthcare, retail, skilled trades, and teaching. (BLS, 2016). Spending that much time in an upright posture at work results in musculoskeletal discomfort and injuries, decreased productivity, and psychological fatigue (Cham and Redfern, 2001; Chiu and Wang, 2007; Halim and Omar 2011; Halim et al., 2012; Na et al., 2011). Injuries from prolonged exposure to standing or walking at work contribute to $4.2 billion in worker compensation costs in the United States in 2011 (NSC, 2015). These injuries encompass lower extremity and low back pain, degenerative joint damage including osteoarthritis, muscle injury, and circulatory diseases (Cham and Redfern, 2001; Halim and Omar 2011; Halim et al., 2012; Macfarlane et al., 1997; Meijsen and Knibbe, 2007; Na et al., 2011; Redfern and Cham, 2000). Objective measures of muscle fatigue and swelling have been used in an attempt to explain this discomfort. Unfortunately, the results are inconsistent with some finding changes in muscle fatigue and leg swelling after varying periods of time and others reporting no change (Coenen et al., 2017; Halim et al., 2012; Hansen et al., 1998; Redfern and Cham, 2000). Objective measures of muscle fatigue and swelling after varying periods of time and others reporting no change (Coenen et al., 2017; Halim et al., 2012; Redfern and Cham, 2000). Halim et al. (2012) found muscle fatigue, measured as median power frequency, in the legs and low back developing after only 20 minutes. Whereas others identified muscle fatigue onset, measured with median power frequency or muscle force twitch, occurring after two hours or five hours of standing (Gregory and Callaghan, 2008; Garcia, 2015). Regardless of the timing of muscle fatigue onset, most authors conclude that muscle fatigue developed during prolonged standing is associated with elevated risk for leg or low back discomfort and other musculoskeletal disorders.

Changes in blood flow and oxygenation have also been used as an objective measure of muscle fatigue in both static muscle contractions (Albert et al., 2004; Kell and Bhambhani, 2008; Matsuura et al., 2011; McGill et al., 2000) and short term walking/running (Hiroyuki et al., 2002; Lee et al., 2011; Rissanen et al., 2012). Hiroyuki et al. (2002) found that vastus lateralis and gastrocnemius oxygenation increased with the onset of walking, while in increases in speed, or effort, yielded decreases in oxygenation. Total hemoglobin revealed opposite trends. Initially, total hemoglobin decreased at the start of walking and then increased throughout exercise and alongside speed (Hiroyuki et al., 2002). Near infrared spectroscopy (NIRS) is a non-invasive technique that measures changes in absorption of near infrared light by oxyhemoglobin and deoxyhemoglobin to determine the change of total hemoglobin (tHb) and tissue oxygen saturation (SO2) in a muscle over a period of time. SO2 reflects the balance between supply of oxygen and its consumption in the muscle region while...
tHb represents the changes in total volume of hemoglobin in the area (Quaresima et al., 2004).

The impact of prolonged upright postures, standing or walking, on changes in muscle SO2 has not been widely investigated. Callaghan et al. (2010) measured change in oxygenation in the right erector spinae while subjects stood for two hours. While perceived lower back discomfort increased throughout standing, no significant changes in SO2 were reported. The authors suggest that perhaps two hours of standing was not enough time to induce fatigue in the lower back (Callaghan et al., 2010). Additionally, other muscles that play a role in supporting the body during standing, such as those of the legs, may undergo greater changes in SO2. For example, Garcia et al. (2018) found increased soleus SO2, throughout, and tHb, only after 4 hours, during a prolonged standing work cycle paradigm. No one has yet to examine changes in muscle SO2 and tHb in the back or legs during prolonged walking and its possible relationship to musculoskeletal discomfort.

The purpose of this study was to investigate changes in muscle SO2 and tHb in the erector spinae (ES) and soleus (SOL) muscles during prolonged walking with intermittent standing. In addition, the study aims to determine any association of these muscle measures with pain and discomfort. The hypothesis is that NIRS could provide an objective measure associated with muscle discomfort and potential injury during prolonged walking.

Methods

A total of nine healthy adults (mean age 22.4 ± 2.3 years, weight 75.86 ± 14.77 kg, height 1.76 m ± 0.07 m, 6M) completed the testing protocol. Study participants were screened for any orthopedic, neurological, pulmonary, or cardiovascular health problems. Before inclusion, all subjects had self-verified their ability to walk normally at a self-selected speed without pain and stand/walk for 2-6 hours. This study was approved by the Institutional Review Board of the University of Pittsburgh. The same brand of shoes and socks were provided to all the subjects to wear throughout the testing session. The flooring surface consisted of a 3 mm thick vinyl tile embedded into testing laboratory.

Instrumentation and discomfort questionnaires

Surveys used to evaluate standing discomfort and pain during prolonged standing (Redfern and Cham, 2000) were administered to subjects throughout the walking protocol to monitor ratings of perceived discomfort (RPDs). The surveys were based on the nonlinear CR10-Borg scale (Borg, 1998) which ranged from 0 (No discomfort at all) – 10 (Extremely uncomfortable). The survey included ratings for overall tiredness, overall leg tiredness and discomfort levels for specific areas of the body (upper and lower back, hips, upper legs, knees, lower legs, ankles, and feet) (Redfern and Cham, 2000).

Muscle SO2 and tHb was measured using a frequency domain multi-distance NIRS system (Imagent, ISS, Champaign, IL). NIRS probes were placed snugly on the leg-dominant side of the lower back ES muscle group at the L3 vertebrae level approximately 2 cm away from the spinal column and the lateral SOL muscle. A black cloth was placed around the trunk and calf, covering the NIRS sensor to block out any additional light. Before each testing session, the probes were calibrated to a phantom with a known absorption coefficient (Imagent, ISS, Champaign, IL). Each probe consisted of 4 light-emitting diodes that operated at 2 wavelengths (690 and 830 nm) and 2 phase-sensitive detectors (Figure 1). Frequency domain NIRS uses amplitude-modulated light sent in at a series of source positions and records the amplitude and phase of this modulated signal at the detector position, ranging from 1.3 cm to 3.2 cm away. Optical absorption at each color is used to estimate hemoglobin and oxygen saturation (see (Quaresima et al.) for details). The near infrared light was modulated at a frequency of 110 MHz and collected at a sampling rate of 1.2 Hz.

Protocol

The walking sessions began with a 2 minute seated baseline (B) followed by five minute standing baseline (S0) then a 30 minute walking period (W1). This patterned continued, resulting in five periods of five minutes of standing (S0, S1, S2, S3, S4, S5) and four periods of 30 minutes of walking (W1, W2, W3, W4).
of walking (denoted W1, W2, W3, W4) (Figure 2). Subjects walked in a gait laboratory setting in a figure eight pattern at a self-selected speed along a path that was 10.5 m long and 3 m wide, resulting in a total loop length of 25 m. At the end of each 30 minute walking period, subjects were instructed to stop walking and remain standing for 5 minutes while maintaining ground contact with both feet (S1, S2, S3, S4). Surveys were administered during the 5 minute standing baseline and the following 5 minute standing times for a total of 5 ratings. ES and SOL SO2 and tHb were continuously recorded throughout the testing session.

### Data analysis

The RPDs were transformed to a linear scale that ranged from 6-23, where a rating of 6 represented no discomfort at all (Borg, 1998). The responses were normalized to the first survey taken at baseline. Muscle SO2 and tHb were calculated as the ratio of oxy- and total-hemoglobin and the sum of oxy- and deoxyhemoglobin, respectively. The diffusion approximation solution described in Fantini et al. (1994) was used to estimate absorption and phase components of the NIRS signal. Optical absorption was then used to compute hemoglobin levels using the Beer-Lambert law with extinction coefficients for hemoglobin given in Prahl (1998). The resulting time courses for muscle SO2 and tHb were calculated for both detectors on each of the two probes. Time series were filtered using a phaseless digital Butterworth filter with a low-pass cutoff of 0.15 Hz. Due to technical issues with NIRS equipment, 8 subjects were included in SOL muscle analysis and 7 subjects were included in ES analysis. NIRS parameters were normalized to baseline sitting values and are reported as the change in total hemoglobin and SO2 relative to this seated baseline period. Muscle SO2 and tHb were averaged every five minutes during both the standing and walking periods resulting in one value per standing period (five total standing periods) and six values per walking period (four total walking periods with six points within each walking period). Data was verified for normality. Analysis of variance (ANOVA) was used to evaluate the effect of walking period (W1, W2, W3, W4), time segment within walking period and then gradually increased within the later walking periods (Figure 3). During the first standing period, S1 the ES and SOL SO2 tend to decreased. When walking commenced in period W2, ES and SOL SO2 increased, though not as rapidly as in W1. This pattern continued throughout the remainder of the protocol. Throughout each walking period, tHb generally increased in the ES and SOL muscles. A dramatic increase in SOL tHb was observed during the standing periods, but was not observed with the same magnitude in the ES.

In general, ES and SOL SO2 increased throughout the walking session. Walking period (F = 35.14; p < 0.0001), within walking period (F = 19.60; p < 0.0001), and their interaction (F = 7.39; p < 0.0001) had a significant impact on ES muscle SO2. Similarly, walking period (F = 34.83; p < 0.0001), within walking period (F = 33.84; p < 0.0001), and their interaction (F = 21.49; p < 0.0001) also had a significant impact on SOL muscle SO2 (Figure 4). Post-hoc analysis revealed that W1 was significantly less than later walking periods for both muscles’ SO2. Within W1, W2, and W3 there was a significant increase in ES and SOL muscle SO2 from the start of walking period until the end of that same period. In comparing across walking

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### Table 1. Mean (SD) of ratings of perceived tiredness or discomfort during prolonged walking.

<table>
<thead>
<tr>
<th>Walking Time [min]</th>
<th>S0 0 min</th>
<th>S1 35 min</th>
<th>S2 75 min</th>
<th>S3 105 min</th>
<th>S4 140 min</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tiredness</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall Tiredness</td>
<td>6.0 (0.0)</td>
<td>7.4 (1.9)</td>
<td>8.9 (3.7)</td>
<td>10.1 (4.0)</td>
<td>11.7 (3.6)</td>
</tr>
<tr>
<td>Overall Leg Tiredness</td>
<td>6.0 (0.0)</td>
<td>7.8 (2.0)</td>
<td>10.7 (2.3)</td>
<td>11.9 (2.6)</td>
<td>12.8 (3.3)</td>
</tr>
<tr>
<td><strong>Discomfort</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upper Back</td>
<td>6.0 (0.0)</td>
<td>8.6 (2.7)</td>
<td>10.2 (4.0)</td>
<td>10.1 (3.5)</td>
<td>11.1 (4.5)</td>
</tr>
<tr>
<td>Lower Back</td>
<td>6.0 (0.0)</td>
<td>7.8 (2.3)</td>
<td>10.7 (3.0)</td>
<td>11.3 (3.6)</td>
<td>11.8 (4.1)</td>
</tr>
<tr>
<td>Hips</td>
<td>6.0 (0.0)</td>
<td>7.0 (1.8)</td>
<td>8.3 (2.7)</td>
<td>10.2 (3.3)</td>
<td>10.2 (3.6)</td>
</tr>
<tr>
<td>Upper Legs</td>
<td>6.0 (0.0)</td>
<td>7.4 (1.8)</td>
<td>9.8 (2.5)</td>
<td>10.8 (2.7)</td>
<td>11.9 (2.7)</td>
</tr>
<tr>
<td>Knees</td>
<td>6.0 (0.0)</td>
<td>7.1 (1.8)</td>
<td>9.7 (2.6)</td>
<td>10.9 (3.1)</td>
<td>11.7 (3.0)</td>
</tr>
<tr>
<td>Lower Legs</td>
<td>6.0 (0.0)</td>
<td>6.7 (3.3)</td>
<td>9.9 (3.5)</td>
<td>11.0 (3.8)</td>
<td>11.4 (3.7)</td>
</tr>
<tr>
<td>Ankles</td>
<td>6.0 (0.0)</td>
<td>8.8 (2.4)</td>
<td>9.4 (3.2)</td>
<td>11.9 (3.7)</td>
<td>12.2 (3.3)</td>
</tr>
<tr>
<td>Feet</td>
<td>6.0 (0.0)</td>
<td>9.2 (2.4)</td>
<td>10.8 (2.7)</td>
<td>11.3 (2.9)</td>
<td>13.2 (3.2)</td>
</tr>
</tbody>
</table>

Rating of perceived overall tiredness and overall leg tiredness increased with time walking (p < 0.0001, Table 1). Similarly, RPDs for all body regions surveyed increased with time (p < 0.0001, Table 1). The RPDs were transformed to a linear scale that ranged from 6-23, where a rating of 6 represented no discomfort at all. All measures significantly increased with time (p < 0.0001).

A consistent pattern in SO2 was observed across subjects in both ES and SOL during walking. In general, ES and SOL SO2 increased during the first 30 minute walking period and then gradually increased within the later walking periods (Figure 3). During the first standing period, S1 the ES and SOL SO2 tend to decreased. When walking commenced in period W2, ES and SOL SO2 increased, though not as rapidly as in W1. This pattern continued throughout the remainder of the protocol. Throughout each walking period, tHb generally increased in the ES and SOL muscles. A dramatic increase in SOL tHb was observed during the standing periods, but was not observed with the same magnitude in the ES.
periods, there was a significant increase in the starting value of SO2 from W1 to W2, and from W2 to W3. Additionally, the end SO2 value for W1 of both muscles was significantly less than the ending value of the periods following.

The transition from walking to standing resulted in a significant decrease in SO2 throughout testing for both the ES and SOL (F = 13.73; p < 0.0001, F = 6.85; p = 0.008, respectively). In addition, muscle oxygenation was significantly different between the standing periods for the ES and the SOL (F = 18.07; p < 0.0001, F = 61.26; p < 0.0001, respectively). Post-hoc testing revealed that the average muscle SO2 of the baseline standing period, S0, was significantly less than all of the following standing periods. Similarly, the average muscle SO2 of the first standing period following walking, S1, was significantly less than all later standing periods.

Walking period, within walking period, and their interaction also impacted ES tHb (F = 2.99; p = 0.058, F = 4.97; p = 0.002, F = 2.38; p = 0.006, respectively) and SOL muscle tHb, (F = 9.11; p < 0.001, F = 11.19; p < 0.0001, F = 3.86; p < 0.0001, respectively, Figure 4). For SOL tHb only, W1 was significantly different than later periods. Within W1 and W2 there was a significant increase in ES and SOL muscle tHb. The starting value of W1 was significantly different than that of all other walking periods for SOL tHb.

In only SOL, the transition from walking to standing resulted in a significant increase in tHb (F = 3.05; p < 0.0001). Additionally, SOL tHb was significantly different between the standing periods (F = 10.66; p < 0.0001). Post-hoc testing found that the average muscle tHb of the baseline standing period, S0, was significantly less than all of the following standing periods.

A standard least squares fit was performed to investigate the relationship between muscle parameters (SO2 and tHb) and RPDs during prolonged walking. SOL and ES SO2 were associated with RPDs for the lower leg and lower back regions, respectively. Discomfort was significantly associated with SO2 but not tHb (tHb ES R² = 0.511; p = 0.094, tHb SOL R² = 0.491; p = 0.247). Specifically, the relationship between ES SO2 and lower back discomfort was significant with an increase in ES SO2 corresponding to an increase in lower back discomfort (R² = 0.701; p < 0.0001). Similarly, an increase in SOL SO2 was related to an increase in lower leg discomfort (R² = 0.643; p = 0.002).

**Discussion**

In general, ES and SOL SO2 increased during the first three walking periods, both across walking periods and within walking periods. When subjects took a standing break from walking, oxygenation levels decreased. SO2 was also significantly higher during the last three standing periods, compared to the first two standing periods. For both the ES and SOL, tHb increased within walking period for W1 and W2. Interestingly, only the SOL was observed to have a significant increase in tHb after the first walking period and first standing period before reaching a steady state during later periods. Additionally, SOL tHb was significantly increased during all the transitions from walking to standing.

![Figure 3. Response changes relative to the seated baseline for one subject in ES and SOL SO2 (top) and tHb (bottom) during the trial. The 5 minutes between dotted lines denote the standing periods.](image)
The response in muscle SO2 to prolonged walking found in this study was similar to previously reported lower exertion occupational tasks and short-term walking, where maximal effort was not required (Calaghan et al., 2010; Yang et al., 2007). During two hours of standing, lumbar ES NIRS measures were not significantly altered but did have average oxygenation levels above baseline resting values (Callaghan et al., 2010). Yang et al. (2007) investigated ES SO2 during a continuous occupational lifting cycle finding the largest increase in oxygenation occurred during the first two hour block. This increase in oxygenation was explained to be caused by an increase in blood flow to the muscle during the lifting task, which likely did not induce muscle fatigue (Yang et al., 2007). Behaving in a similar manner, ES SO2 increased in the beginning of the walking protocol, both across walking periods and within walking periods (W1, W2, W3). Additionally, increased tHb, indicating that total blood flow inflow to the muscle was larger than blood outflow, was noted within first walking periods (W1, W2). Both of these parameters did not continue to significantly increase in the last walking period. The walking task resulted in an increase in blood flow to the low back and since the ES was not utilizing the oxygen that was readily available, an increase in SO2 was also seen during the first 90 minutes.

Similarly, the greatest increase in SOL oxygenation was across and within W1, W2 and W3. Blood volume, tHb, was observed to increase in the SOL within W1 and W2 six times more than ES. Increased muscle SO2 during constant exercise, such as walking, may be caused by increased oxygen delivery due to exercise-induced blood volume expansion (Kime et al., 2013). Considering its greater involvement in gait, it is likely that the SOL was utilizing the oxygen that was readily available and prompting exercise-induced blood volume expansion. It has previously been reported that intramuscular pressure is lower during dynamic muscle contractions compared to static contractions (Vedsted et al., 2006). Though intramuscular pressure was not measured in the current study, it is predicted that it is decreased during the walking periods and blood volume increased as a result. During the transition from walking to standing, SOL SO2 significantly increased and SOL SO2 decreased. It is likely that with the decrease in SOL contractions during standing, venous blood flow slowed, resulting in increased blood volume, especially deoxygenated, and subsequently decreased SOL SO2. During the transition from standing to walking muscle contractions began, causing blood vessel compression and venous return which explains the dramatic decrease in SOL tHb. Inflow of blood volume to the muscle was then facilitated by the repeated muscle contractions experienced during the walking periods (Hiroyuki et al., 2002). In environments
where prolonged walking is required followed by intermittent standing, it is possible longer standing period could result in detrimental muscle parameters. Additional research is necessary to determine if increased volume and decreased SO2 would remain present in the SOL without the commencement of another walking period.

RPDs increased across all body parts as the walking sessions progressed, in agreement with previous work (Hansen et al., 1998). Increased discomfort in the lower leg and lower back were associated with increased muscle SO2 but not tHb. However, it is not likely that a lack of SO2 was an issue in the development of discomfort during prolonged walking because SO2 increased. A decreased in muscle oxygenation has been cited as a possible source of fatigue during static muscle contractions (Albert et al., 2004; Kell and Bhambhani, 2008; Matsuura et al., 2011; McGill et al., 2000) and short term walking/running (Bhambhani, 2004; Hiroyuki et al., 2002; Lee et al., 2011; Rissann et al., 2012). Previous research has shown poor agreement between subjective discomfort and muscle SO2 during prolonged standing (Callaghan et al., 2010; Garcia et al., 2018). No one has examined a possible vascular origin of discomfort during prolonged walking with intermittent standing. In many occupational that require periods of walking with intermittent standing, workers report musculoskeletal fatigue and discomfort (Cham and Redfern, 2001; Chiu and Wang, 2007; Halim and Omar, 2011; Halim et al., 2012; Na et al., 2011). Though differences in muscle fatigue, as measured by muscle twitch force, have been found during the initial period of a prolonged walking period. Garcia et al. (2016) found that walking initially leads to muscle potentiation in the calf muscles. They hypothesized that as walking continued, fatigue eventually developed in the muscles. Though the development of muscle fatigue was slowed compared to the static contractions of standing, likely due to the cyclic activity of the lower-leg muscles during walking (Garcia et al., 2016). It is likely that lower intensity repeated contractions during prolonged walking with intermittent standing are improving blood flow and SO2, thus delaying the vascular origins of discomfort seen during static contractions and short-term fatigue protocols. It is possible that prolonged walking with intermittent standing may result in different muscular parameters.

There were a few limitations to this study. A limited number of subjects were tested. The subjects were young healthy adults which should represent the subcutaneous tissue depth was within normal ranges and excess adipose tissue was not present at the recorded sites, although it was not measured. Activity level of the participants was not recorded though they were screened for overall health and ability to walk. Future studies should include a greater number of subjects from different populations, especially those with muscle issues or increased discomfort. The instructions to walk at a self-selected speed throughout the testing session may have also affected the results. Subjects may have varied walking speed during the two hour walking protocol and the self-selected walking speeds are likely to be varied across subjects and could impact changes in muscle parameters.

**Conclusion**

Prolonged walking with intermittent standing generally induced increased SO2 of the ES and SOL across walking periods, within walking periods and across standing periods. These increases were more pronounced at the beginning of the walking session and continued through the second or third periods. ES and SOL tHb increased within walking periods for W1 and W2. Only the SOL tHb significantly increased after the first walking and standing periods and during all the transitions from walking to standing. Increased SO2 and tHb during prolonged walking with intermittent standing are likely a result of the repeated dynamic contractions and exercise-induced blood volume expansion. Increased discomfort was measured; however, this was not explained by detrimental changes in SO2 or tHb. The results suggest that the cause of discomfort during prolonged walking with intermittent standing is not directly related to changes in muscle SO2 or tHb in healthy young adults.

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**References**


**Key points**

- Back and leg muscle oxygenation and total hemoglobin were collected during walking.
- Prolonged walking increased muscle oxygenation during first 90 minutes.
- Prolonged walking increased muscle total hemoglobin during first 60 minutes.
- Increased discomfort was not explained by changes in oxygenation or total haemoglobin.

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