



Effect of body weight support on muscle activation during walking on a lower body positive pressure treadmill



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ABSTRACT

Following lower limb injury, some patients are not able to walk at full weight bearing and may require body weight support for ambulation during the early stages of rehabilitation. The aim of the present study was to investigate how various degrees of reduced effective body weight in a Lower Body Positive Pressure Treadmill (LBPPT), affects muscle activation levels during walking. Twelve healthy participants were instructed to walk at 2.5 km/h and 3.6 km/h on a LBPT that provided a reduced effective body weight equivalent to 100%, 80%, 60%, 40%, and 20% of their individual body mass. Electromyography data were recorded during 20 gait cycles, from seven lower limb muscles, and segmented into a mean envelope by computing root mean square values. A two-way repeated measures ANOVA was used to test for differences in the highest root mean square value obtained, with walking speed and fractional reduction in effective body weight as factors. Significant decreases in EMG amplitude were identified in the following muscles as a result of reduced effective body weight: Vastus Medialis, Vastus Lateralis, Soleus, Gastrocnemius Medial and Lateral head ($p \leq 0.05$). For Tibialis Anterior, significant reductions in EMG amplitude were only observed when effective body weight was reduced to 40% or less at a walking speed of 2.5 km/h ($p \leq 0.05$). The EMG amplitude for Tibialis Anterior at 3.6 km/h and Biceps Femoris at both speeds remained unaffected at all fractional reductions ($p \geq 0.05$). These findings suggest that the muscles of the lower limb respond differently to the body weight support provided by the LBPT during walking, with the extensor muscles of the knee and ankle displaying decreased muscle activation, and the Tibialis Anterior and Biceps Femoris displaying minimal to no changes in muscle activation.

1. Introduction

Following injury or orthopedic surgery of the lower limb, early mobilization through a rehabilitation intervention is a key component in restoring physical function in the shortest possible amount of time, while simultaneously limiting the negative consequences of immobilization such as muscle atrophy, loss of muscle power and decreased maximal aerobic capacity (Kortebein et al., 2008). However, some patients are not able to walk at full weight bearing and may require body weight support for ambulation in the early stages of rehabilitation. Therefore, assistive devices such as walkers, crutches or parallel bars have commonly been used to help patients reduce force loading on the affected leg. Unfortunately, these devices cannot replicate the joint and gait mechanics of normal walking, and require considerable upper body strength, making them unsuitable for patients with polytrauma or spinal cord injury (Eastlack et al., 2005). Similarly, the use of harness based systems and walking in water (hydrotherapy) has been incorporated to provide body weight support, but this too has

negative drawbacks. For instance, harness based systems can create patient discomfort and impede blood circulation, thereby rendering them inappropriate for long-term rehabilitation use (Grabowski, 2010). Hydrotherapy, has been shown to alter gait kinematics as well as muscle activation when compared to normal overground walking (Barela et al., 2006), and is not appropriate during the immediate postoperative period due to wound considerations.

In recent years a new device, the Lower Body Positive Pressure Treadmill (LBPT), has been developed. The LBPT is an increasingly popular training and rehabilitation modality that offers the opportunity to reduce weight bearing during gait or running in the early phases of rehabilitation, without the negative drawbacks of the aforementioned methods. More precisely, LBPT treadmills have been shown to effectively reduce ground reaction and joint forces in the lower body during gait (Grabowski, 2010; Patil et al., 2013) and running (Grabowski and Kram, 2008; Jensen et al., 2016), while also providing a linear decrease in muscle activation of lower limb muscles in relation to the amount of unloading provided during running (Hunter et al., 2014; Liebenberg

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et al., 2011; Mercer et al., 2013; Sainton et al., 2015). Therefore, the use of a LBPPT may be a valuable alternative to the present methods used to provide reduced weight bearing to patients commencing a rehabilitation program.

Although some research has been published on the effects of body weight support using a LBPPT device on the kinetics, kinematics and muscular activation during running (Farina et al., 2017), there is a clear knowledge gap on the same effects during walking. As the majority of patients recovering from either orthopedic surgery or injury to the lower limb will commence their rehabilitation program by walking, it is imperative that we gain more knowledge on whether or not the use of LBPPT devices is a viable solution for these patients.

Therefore, the aim of the present study was to investigate how various degrees of effective body weight (between 80% and 20%) in a LBPPT, affects muscle activation levels during walking at two different speeds (2.5 km/h and 3.6 km/h). We hypothesized that muscle activation of the extensor muscles of the lower body would decrease while activation of the propulsive muscles, would remain unaffected.

2. Methods

2.1. Participants

Twelve healthy participants (7 male and 5 females, age 27.0 ± 4.2 years (mean \pm standard deviation (SD)), height 178.1 ± 9.9 cm, BMI 23.13 ± 3.09 , body mass 74.13 ± 15.41 kg) volunteered for participation in the study after having been informed both verbally and in writing about the experimental risks of the study. Participants were deemed eligible for participation if they were free of any acute or chronic musculoskeletal injuries at the time of data collection. The number of participants were determined using an α level of 0.05, a β level of 0.95 and an effect size of 1.1. The effect size estimation was based on previous results (Jensen et al., 2016; Mercer et al., 2013). Participants were recruited from students enrolled on the Clinical Science and Technology Master education at Aalborg University. All participants provided written informed consent and the experimental procedures performed were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments. None of the participants had any prior experience in walking with bodyweight support.

2.2. Study protocol

Participants were instructed to walk at 2.5 km/h and 3.6 km/h in a LBPPT (AlterG Anti-Gravity Treadmill®, model M320/F320, Fremont, CA, USA) that provided reduced effective body weight during gait. Participants were tested at 100%, 80%, 60%, 40%, and 20% of their individual body mass. All participants wore AlterG neoprene shorts, and their own indoor running or gymnastic shoes. Prior to the initiation of the testing protocol, a calibration was performed with the participant standing on the treadmill to adjust the chamber pressure. To familiarize the participants to the specific treadmill conditions, the study protocol commenced by walking at 2.5 km/h and 100% of effective body weight for two minutes as a warm-up period. Next, participants continued walking at the same speed and effective body weight for two minutes. This served as the baseline measurement. Following this, two minutes of walking was completed at either 80%, 60%, 40% or 20% of effective body weight and followed by another two minutes walking at 100% of effective body weight to regain a normal gait pattern (Fig. 1). This continued until two minutes of walking had been completed on all fractions of the reduced effective body weight. The order of the reduced fractions was randomized. Following a five minute break, the protocol was repeated at a walking speed of 3.6 km/h. Data were collected during the last 30 s of each two-minute interval. The speed of 2.5 km/h was chosen to represent the speed of persons with a physical disability, while the speed of 3.6 km/h was chosen to represent normal walking

speed in healthy adults.

2.3. Measurements

Prior to the experiment, the skin surface of all participants was shaved, abraded and cleaned with alcohol before placing surface electromyography electrodes (EMG) (Ambu Neuroline 720 01-K/12, Ag/AgCl, inter electrode distance 20 mm, Ambu A/S, Ballerup, Denmark) over the following seven muscles of the dominant lower limb: biceps femoris (BF), vastus medialis (VM), vastus lateralis (VL), medial and lateral head of gastrocnemius (GM and GL), soleus (SOL) and tibialis anterior (TA). The electrodes were mounted in accordance to the SENIAM guidelines (Hermens et al., 2000). EMG data were collected using a wireless EMG amplifier (TeleMyo 2400 G2 Telemetry System, Noraxon U.S.A. Inc., Arizona, USA) at a sampling rate of 1500 Hz and with an individual specific gain factor (500–1000). Further, a footswitch (DTS Footswitch, Noraxon U.S.A. Inc., Arizona, USA) was mounted under the right foot, enabling the identification of heel strike during the gait cycle. All data were recorded from October through November in 2016.

2.4. Signal processing

Following data acquisition, all EMG data were band pass filtered [10–500 Hz] before being segmented into approximately 17–20 gait cycles using the data obtained from the footswitch. A gait cycle was defined as the period between two successive heel strikes. The initial (from start of recording to first heel strike) and terminating part (from last heel strike to the end of recording) of each recording was excluded from the analysis to avoid noise interference. The linear envelopes of the EMG signals across each of the 17–20 gait cycles were obtained by calculating root mean square values in 100 ms non-overlapping epochs. Each of the EMG envelopes was then interpolated into 20 time points, and the average for each time point across the 17–20 repetitions was calculated for each participant, yielding a mean EMG envelope for each muscle. As a measure of peak muscle activation during the cycle, peak EMG amplitude was computed as the highest EMG amplitude value (Fig. 2). All peak EMG amplitude values obtained during walking at a reduced effective body weight was then normalized with respect to the normalization factor defined as the peak EMG amplitude obtained at 100% of effective body weight, and used for the statistical analysis.

2.5. Statistical analysis

For the EMG data, outliers were identified as any data point above or below an upper (3rd quartile + (interquartile range \cdot 1.5)) or lower threshold (1st quartile – (interquartile range \cdot 1.5)), and removed before further statistical analysis. A two-way ANOVA with repeated measures was used to test for differences in EMG amplitude (dependent variable) across the five levels of effective body weight reduction (independent variable, effective body weight reduction levels being 100%, 80%, 60%, 40%, and 20%) and across the two levels of walking speed (independent variable, walking speed being 2.5 and 3.6 km/h). If a significant interaction was detected, separate one-way ANOVA's were run across all levels of the two independent variables to analyze simple main effects. If no interaction were present the main effects of the two-way repeated measures ANOVA were reported. All post hoc testing was carried out using the Bonferroni correction, in which a step-down Holm-Bonferroni adjustment was applied to all obtained post hoc p -values, to retain the familywise error rate for multiple comparisons (Holm, 1979). Normal distribution of the data were validated with a Shapiro-Wilks test of normality, combined with visual inspection of histograms and QQ-plots. Mauchly's test of sphericity was used to test if the variance of the differences between levels were equal. In the case that the assumption of sphericity was violated a Greenhouse-Geisser correction was applied. SPSS Version 25.0 (IBM Corp; Armonk, NY,

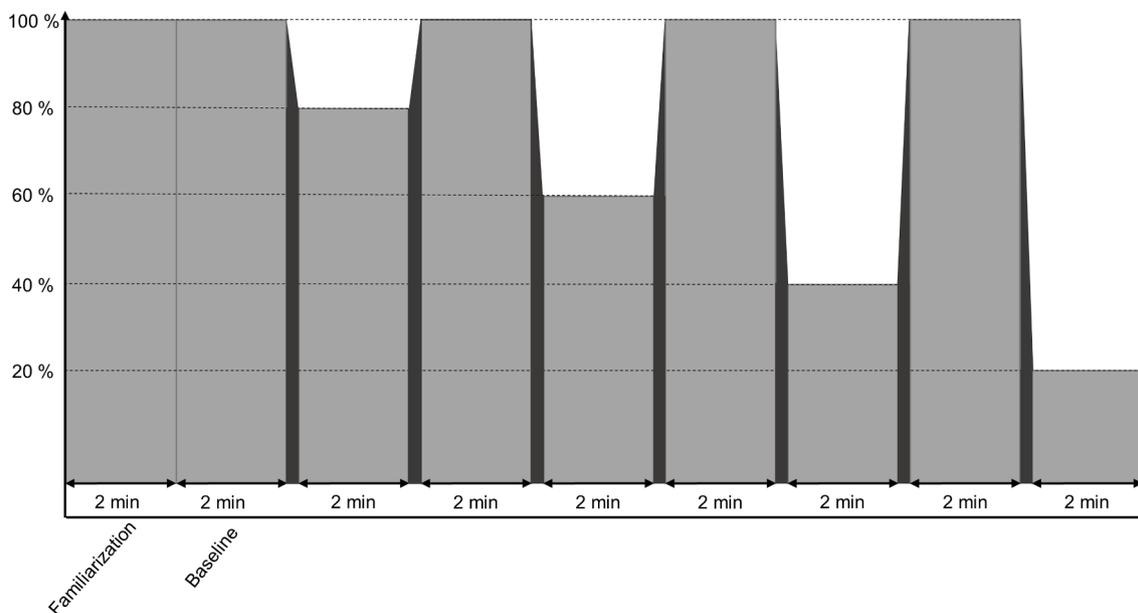


Fig. 1. Experimental protocol. Illustrates the different fractions of effective body weight at which data were acquired. Each session commenced by a familiarization phase consisting of two minutes walking at 100% of effective body weight, followed by a baseline recording that also consisted of two minutes of walking at 100% of effective body weight. Then the effective body weight was reduced to either 80%, 60%, 40%, or 20% in a randomized order. Each two minute walk performed at a reduced effective body weight was followed by two minutes of walking at 100% of effective body weight.

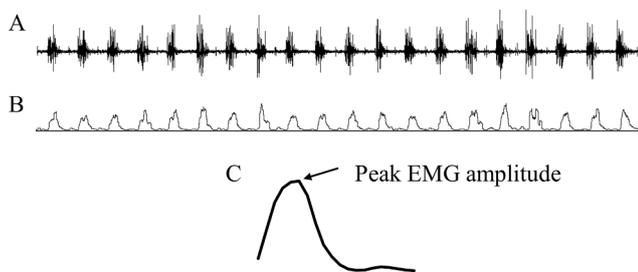


Fig. 2. Processing of the recorded EMG signal. A: Depiction of the raw EMG signal recorded from Vastus Medialis for 20 cycles during walking at 3.6 km/h. B: Linear envelopes were created by calculating root mean square values in 100 ms non-overlapping epochs. C: A single linear envelope was computed, consisting of the average of the 20 cycles. Then the peak EMG value was identified and used for statistical analysis.

USA) was used for all statistical analyses. Statistical significance was accepted at $p \leq 0.05$. Results are presented as mean \pm standard deviation (SD).

3. Results

The mean effect of reducing effective body weight, at a walking speed of either 2.5 km/h or 3.6 km/h, is illustrated in Fig. 3 and Fig. 4, respectively.

3.1. VM

The two-way interaction between effective body weight and walking speed was not significant for VM, $F(4, 28) = 1.757, p = 0.166$. The main effect of walking speed did not result in significant differences in normalized EMG amplitude ($p = 0.282$). The main effect of reducing effective body weight resulted in the normalized EMG amplitude values being significantly lower at 60% ($58.8 \pm 16.2\%, p = \leq 0.001$), 40% ($45.5 \pm 17.4\%, p = \leq 0.001$), and 20% ($44.2 \pm 18.9\%, p = \leq 0.001$) compared to the baseline measurement of 100%. Similarly, the normalized EMG amplitude values were significantly lower at 40% ($p = 0.007$) and 20% ($p = 0.030$) compared to the measurement at

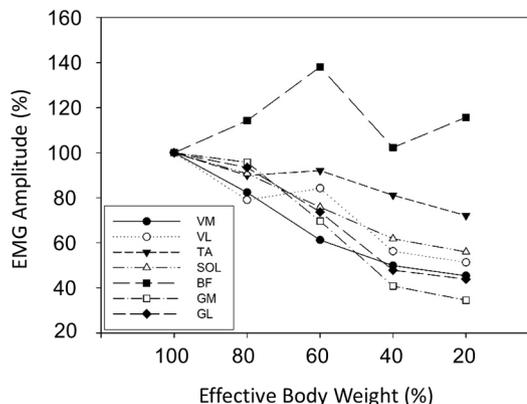


Fig. 3. Effect of reduced effective body weight on EMG amplitude during walking at 2.5 km/h. Graphical illustration of the mean normalized EMG amplitude obtained for each muscle at 100%, 80%, 60%, 40%, and 20% of effective body weight during walking at 2.5 km/h. Black filled circles represent Vastus Medialis (VM). Open circles represent Vastus Lateralis (VL). Black filled triangles represent Tibialis Anterior (TA). White open triangles represent Soleus (SOL). Black filled squares represent Biceps Femoris (BF). White open squares represent Gastrocnemius Medial head (GM). Black filled diamonds represent Gastrocnemius Lateral head (GL). N = 12.

80% ($76.9 \pm 20.3\%$) (Table 1).

3.2. VL

The two-way interaction between effective body weight and walking speed was not significant for VL, $F(4, 32) = 2.397, p = 0.071$. The main effect of walking speed did not result in significant differences in normalized EMG amplitude ($p = 0.167$). The main effect of reducing effective body weight resulted in the normalized EMG amplitude values being significantly lower at 80% ($75.4 \pm 19.8\%, p = \leq 0.001$), 40% ($57.9 \pm 35.4\%, p = 0.028$), and 20% ($50.4 \pm 30.1\%, p = \leq 0.001$) compared to the baseline measurement of 100%. Further, there were significant differences between the normalized EMG amplitudes obtained at 20% compared with that at 80% ($p = \leq 0.001$) and 60%

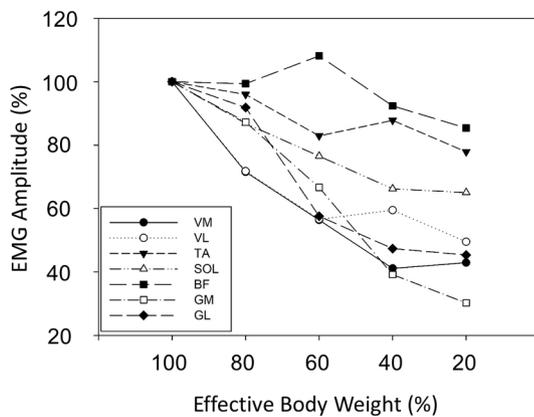


Fig. 4. Effect of reduced effective body weight on EMG amplitude during walking at 3.6 km/h. Graphical illustration of the mean normalized EMG amplitude obtained for each muscle at 100%, 80%, 60%, 40%, and 20% of effective body weight during walking at 3.6 km/h. Black filled circles represent Vastus Medialis (VM). Open circles represent Vastus Lateralis (VL). Black filled triangles represent Tibialis Anterior (TA). White open triangles represent Soleus (SOL). Black filled squares represent Biceps Femoris (BF). White open squares represent Gastrocnemius Medial head (GM). Black filled diamonds represent Gastrocnemius Lateral head (GL). $N = 12$.

($70.4 \pm 30.2\%$, $p = 0.048$) (Table 1).

3.3. TA

A significant two-way interaction was present between effective body weight and walking speed for TA, $F(4, 32) = 22.743$, $p \leq 0.001$ (Fig. 5). The simple main effects analysis showed no significant differences in normalized EMG amplitude due to speed when compared at an effective body weight of 80% ($p = 0.432$), 60% ($p = 0.143$), 40% ($p = 0.090$), and 20% ($p = 0.423$). When walking at 2.5 km/h the normalized EMG amplitude values were significantly lower at 40% ($81.1 \pm 13.2\%$, $p = 0.042$) and 20% ($72.1 \pm 17.5\%$, $p \leq 0.001$) compared to the baseline measurement of 100%. Further, there were significant differences between the normalized EMG amplitudes obtained at 20% compared with that at 80% ($90.0 \pm 15.5\%$, $p = 0.040$) and 60% ($92.1 \pm 15.9\%$, $p = 0.009$) (Table 1). When walking at 3.6 km/h, the simple main effects analysis showed no significant differences, indicating that muscle activation of the TA muscle is only significantly reduced during walking at 2.5 km/h (Table 1).

3.4. SOL

A significant two-way interaction was present between effective body weight and walking speed for SOL, $F(4, 36) = 5.024$, $p = 0.003$ (Fig. 5). The simple main effects analysis showed no significant differences in normalized EMG amplitude due to speed when compared at an effective body weight of 80% ($p = 0.258$), 60% ($p = 0.620$), 40% ($p = 0.390$), and 20% ($p = 0.183$). When walking at 2.5 km/h the normalized EMG amplitude values were significantly lower at 60% ($75.8 \pm 8.2\%$, $p \leq 0.001$), 40% ($61.8 \pm 11.7\%$, $p \leq 0.001$), and 20% ($55.9 \pm 17.4\%$, $p \leq 0.001$) compared to the baseline measurement of 100%. Similarly, the normalized EMG amplitude values were significantly lower at 40% ($p = 0.005$) and 20% ($p = 0.002$) compared to the measurement at 80% ($90.8 \pm 12.0\%$), and at 20% compared to 60% ($p = 0.007$) (Table 1). When walking at 3.6 km/h the normalized EMG amplitude values were significantly lower at 60% ($76.6 \pm 14.5\%$, $p = 0.018$), 40% ($66.3 \pm 16.0\%$, $p \leq 0.001$), and 20% ($65.1 \pm 23.5\%$, $p = 0.003$) compared to the baseline measurement of 100%. Similarly, the normalized EMG amplitude values were significantly lower at 40% ($p = 0.002$) compared to the measurement at 80% ($86.9 \pm 14.3\%$) (Table 1).

3.5. BF

The two-way interaction between effective body weight and walking speed was not significant for BF, $F(1.4, 12.5) = 1.139$, $p = 0.329$. The main effect of reducing effective body weight did not show a significant difference in normalized EMG amplitude ($p = 0.814$). The main effect of walking speed resulted in a significant difference ($p = 0.07$), indicating that the normalized EMG amplitude was lower when walking at 3.6 km/h compared to 2.5 km/h.

3.6. GM

The two-way interaction between effective body weight and walking speed was not significant for GM, $F(1.9, 15.3) = 2.803$, $p = 0.093$. The main effect of reducing effective body weight resulted in a significant difference in normalized EMG amplitude ($p \leq 0.001$). More specifically, the normalized EMG amplitude values were significantly lower at 80% ($91.5 \pm 22.2\%$, $p = 0.042$), 40% ($40.0 \pm 14.9\%$, $p \leq 0.001$), and 20% ($32.4 \pm 14.7\%$, $p \leq 0.001$) compared to the baseline measurement of 100%. Similarly, the normalized EMG amplitude values were significantly lower at 20% ($p = 0.024$) compared to the measurement at 80% (Table 1). The main effect of walking speed resulted in the normalized EMG amplitude being significantly lower when walking at 3.6 km/h compared to 2.5 km/h ($p \leq 0.001$).

3.7. GL

A significant two-way interaction was present between effective body weight and walking speed for GL, $F(4, 40) = 2.678$, $p \leq 0.001$ (Fig. 5). The simple main effects analysis, showed no significant differences in normalized EMG amplitude due to speed when compared at an effective body weight of 80% ($p = 0.586$), 60% ($p = 0.056$), 40% ($p = 0.950$), and 20% ($p = 0.742$). When walking at 2.5 km/h the normalized EMG amplitude values were significantly lower at 40% ($47.8 \pm 16.8\%$, $p \leq 0.001$), and 20% ($43.9 \pm 16.7\%$, $p \leq 0.001$) compared to the baseline measurement of 100%. Similarly, the normalized EMG amplitude values were significantly lower at 40% ($p = 0.014$) and 20% ($p = 0.014$) compared to the measurement at 80% ($93.4 \pm 20.7\%$) (Table 1).

When walking at 3.6 km/h the normalized EMG amplitude values were significantly lower at 60% ($57.6 \pm 23.3\%$, $p = 0.009$), 40% ($47.4 \pm 28.7\%$, $p = 0.009$), and 20% ($45.4 \pm 25.8\%$, $p \leq 0.001$) compared to the baseline measurement of 100%. Similarly, the normalized EMG amplitude values were significantly lower at 20% ($p = 0.014$) compared to the measurement at 80% ($91.9 \pm 25.5\%$) (Table 1).

4. Discussion

The aim of this study was to investigate how various fractions of reduced effective body weight affects muscle activation levels during walking at two different speeds (2.5 and 3.6 km/h) in a LBPT device. In general, all muscles exhibited significantly decreased EMG amplitudes, reflecting the fractional reduction in effective body weight during both walking speeds, except for TA and BF. The EMG amplitude was only significantly decreased in TA when effective body weight was reduced to either 40% or 20% at a walking speed of 2.5 km/h. However, no significant reductions occurred in TA when walking at 3.6 km/h. This was similar for BF where no decrease occurred at all. These results generally supported our initial hypothesis that muscle activation of the extensor muscles of the lower body would decrease while activation of the propulsive muscles, would remain unaffected.

The fact that all the predominant extensor muscles of the knee and ankle joint exhibited reduced muscle activation, when effective body weight was reduced is similar to results on muscle activation during

Table 1

The mean normalized EMG amplitude at the fractional reductions in effective body weight and the results of the two repeated measures ANOVA showing the *p*-values of the interaction, main effects of effective body mass and speed, and all post hoc comparisons. When a significant interaction was present for a muscle, the results of the simple main effects analysis of reduced body weight is shown for each of the two walking speeds, 2.5 km/h and 3.6 km/h. VM, Vastus Medialis; VL, Vastus Lateralis; TA, Tibialis Anterior; SOL, Soleus; BF, Biceps Femoris; GM, Gastrocnemius Medial head; GL Gastrocnemius Lateral head. N = 12. † = Greenhouse-Geisser correction applied.

VM	100	80	60	40	20	ANOVA
EMG amplitude	76.9%	58.8%	45.5%	44.2%	Interaction 0.166	
100		0.070	≤ 0.001	≤ 0.001	≤ 0.001	Speed 0.282
80			0.070	0.007	0.030	Effective body mass
60				0.070	0.182	≤ 0.001
40					1.000	
VL	100	80	60	40	20	ANOVA
EMG amplitude	75.4%	70.4	57.9%	50.4%	Interaction 0.071	
100		≤ 0.001	0.135	0.028	≤ 0.001	Speed 0.167
80			1.000	0.152	≤ 0.001	Effective body mass
60				1.000	0.048	≤ 0.001 †
40					1.000	
TA (2.5 km/h)	100	80	60	40	20	ANOVA
EMG amplitude	90.0%	92.1%	81.1%	72.1%	≤ 0.001	
100		1.000	0.804	0.042	≤ 0.001	Interaction 0.001
80			1.000	0.695	0.040	
60				1.000	0.009	
40					0.384	
TA (3.6 km/h)	100	80	60	40	20	ANOVA
EMG amplitude	96.0%	82.9%	87.8%	77.9%	≤ 0.001	
100		1.000	1.000	0.423	0.280	Interaction 0.001
80			1.000	1.000	0.464	
60				1.000	1.000	
40					1.000	
SOL (2.5 km/h)100	80	60	40	20	ANOVA	
EMG amplitude	90.8%	75.8%	61.8%	55.9%	≤ 0.001	
100		0.172	≤ 0.001	≤ 0.001	≤ 0.001	Interaction 0.003
80			0.420	0.030	0.014	
60				0.294	0.035	
40					0.451	
SOL (3.6 km/h)100	80	60	40	20	ANOVA	
EMG amplitude	86.9%	76.6%	66.3%	65.1%	≤ 0.001	
100		0.395	0.018	≤ 0.001	0.021	Interaction 0.003
80			0.395	0.018	0.198	
60				0.395	0.614	
40					1.000	
BF	100	80	60	40	20	ANOVA
EMG amplitude	106.8%	123.1%	97.4%	100.6%	Interaction 0.329†	
100		1.000	1.000	1.000	1.000	Speed 0.007
80			1.000	1.000	1.000	Effective body mass
60				1.000	1.000	0.814
40					1.000	
GM	100	80	60	40	20	ANOVA
EMG amplitude	91.5%	68.1%	40.0%	32.4%	Interaction 0.093†	
100		0.042	0.708	≤ 0.001	≤ 0.001	Speed ≤ 0.001
80			1.000	0.192	0.024	Effective body mass
60				0.813	0.290	≤ 0.001 †
40					0.813	
GL (2.5 km/h)	100	80	60	40	20	ANOVA
EMG amplitude	93.4%	73.6%	47.8%	43.9%	≤ 0.001	
100		1.000	0.108	≤ 0.001	≤ 0.001	Interaction 0.045
80			1.000	0.021	0.008	
60				1.000	0.895	
40					1.000	
GL (3.6 km/h)	100	80	60	40	20	ANOVA
EMG amplitude	91.9%	57.6%	47.4%	45.4%	≤ 0.001	
100		1.000	0.009	0.009	≤ 0.001	Interaction 0.045
80			0.445	0.150	0.014	
60				0.996	1.000	
40					1.000	

Bold indicates that a significant *p*-value is present.

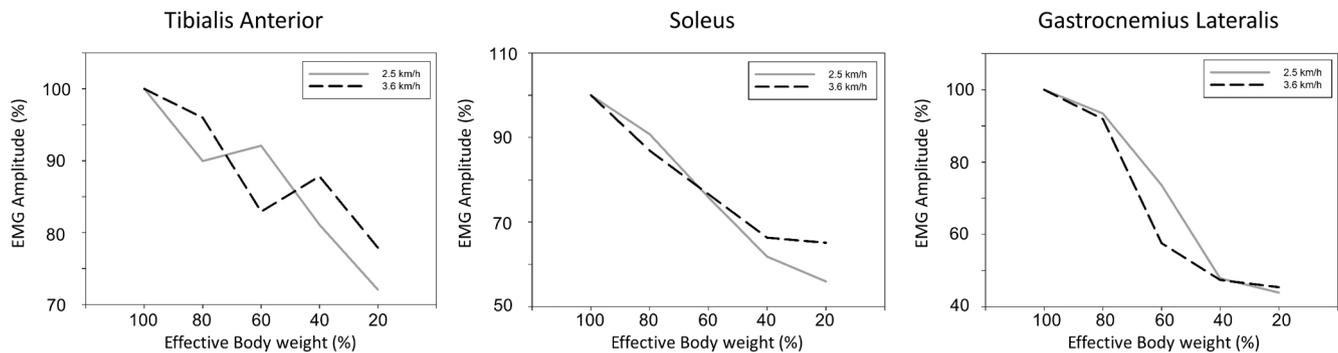


Fig. 5. Interaction effect of Tibialis Anterior, Soleus and Gastrocnemius Lateralis. A significant interaction effect was present for Tibialis Anterior ($p = 0.001$), Soleus ($p = 0.003$) and Gastrocnemius Lateralis ($p = 0.045$). In the figure, the effect of reducing effective body weight is shown for each of the walking speeds to illustrate their interaction.

running on a LBPPT device (Hunter et al., 2014; Jensen et al., 2016; Mercer et al., 2013). A general trend was that the reduction in EMG amplitude first reached significance when effective body weight was reduced to 60% or less, except in the VL and GM muscles. As the positive pressure is increased within the LBPPT chamber, a vertical force is created, thereby alleviating the forces that the extensor muscles act to oppose. The vertical force has similarly been shown to reduce knee joint forces (Patil et al., 2013), ground reaction forces (Grabowski and Kram, 2008; Grabowski, 2010; Jensen et al., 2016) and model-estimated knee and ankle forces (Jensen et al., 2016) during running.

The same reductions in muscle activation were not present in BF. The role of this bi-articular muscle during walking is not easily discerned. The BF muscle could act as a propulsive muscle during walking by providing hip extension during the stance phase. Further, it may create flexion of the knee during the swing phase to prevent the foot from hitting the ground. There are, however, discrepancies in the literature as some studies indicate that the BF contributes to propulsion (Ellis et al., 2014; Franz and Kram, 2012; Liu et al., 2008; Neptune et al., 2004; Sasaki and Neptune, 2006), while others do not (Lay et al., 2007; Liu et al., 2006; McGowan et al., 2009; Neptune et al., 2008; Yang and Winter, 1985). If hip extension and knee flexion are performed by the BF during walking, these tasks most likely remain unaffected by the vertical force created by the LBPPT, and therefore no changes are seen in muscle activation of this muscle. These results are similar to the studies that have documented no or very limited changes in the BF muscle activation during running on a LBPPT (Hunter et al., 2014; Jensen et al., 2016; Mercer et al., 2013). One interesting result was the significant main effect of reduced normalized EMG amplitude of the BF when walking at 3.6 km/h compared to 2.5 km/h. This result, which is in contrast to the findings of Jensen et al. (2016), may be explained by the different normalization procedures applied in the two studies. In the study by Jensen et al. the EMG amplitude obtained during a maximum isometric knee flexion test was used as the normalization factor, whereas the EMG amplitude obtained at 100% of effective body weight at the specific walking speed was used in the present study. Since muscle activation has been shown to increase in general in relation to movement velocity, even when body weight is supported (Liebenberg et al., 2011; Mercer et al., 2013), the absolute values of the normalization factor most likely was higher at a walking speed of 3.6 km/h compared to 2.5 km/h. This may in turn explain why the normalized EMG amplitude was lower at 3.6 km/h, as the possibility of detecting reductions are far less when the absolute values of the normalization factor is low.

During walking, the role of the TA muscle is to prevent rapid plantar flexion of the ankle during initial stance, to ensure the forefoot is

clearing the ground during the swing phase, and to position the ankle joint for initial ground contact (Perry and Davids, 1992). As such, the primary activation of the TA muscle has been reported to occur during the stance-to-swing transition and during the swing phase (Di Nardo et al., 2013). The results of the present study indicated that effective body weight must be reduced to 40% or less before a significant reduction in TA muscle activation is seen during walking at 2.5 km/h. It is possible that the activation of the TA muscle during the swing phase, remains relatively unaffected from the vertical force created by the LBPPT. And similarly, that during the stance-to-swing transition, the TA muscle mainly performs dorsiflexion of the ankle, which is also unaffected from the vertical force created by the LBPPT. This is most likely the reason that effective body weight must be reduced to 40% or less before a significant reduction in TA muscle activation is seen. On the contrary, there was a significant interaction present, revealing that the TA muscle did not exhibit any significant reductions in EMG amplitude when walking at 3.6 km/h, thus indicating that during faster walking speeds the activation of this muscle is upregulated. It therefore seems that the requirements for the TA muscle to prevent rapid plantar flexion during initial stance during faster walking speeds, could result in increased EMG amplitude despite the effective body weight being reduced severely.

A significant interaction effect was also present for GL and SOL. While both muscles exhibited reduced EMG amplitude in response to effective body weight being decreased, the interaction revealed that the normalized EMG amplitude decreased more toward the lower fractions of effective body weight when walking at 2.5 km/h compared to when walking at 3.6 km/h. This is in accordance with previous studies, having shown increased muscle activation in relation to movement velocity, (Liebenberg et al., 2011; Mercer et al., 2013). It must be noted though, that the simple main effects analysis did not result in any significant differences when EMG amplitude from the two different walking speeds were compared across levels of reduced effective body weight. As a result, the interaction effect must be interpreted to be modest at best.

To the best of our knowledge this is the first study to assess muscle activation of multiple muscles during walking in a LBPPT device, as most studies have investigated this during running. The results are particularly relevant to clinicians utilizing the LBPPT in a rehabilitation setting, as this could render the use of the LBPPT, as a viable rehabilitation device for patients suffering from pathologies involving the muscles and tendons used for extension of the knee and ankle joint, such as patellar tendinitis or achilles tendon rupture. However, since muscle activation levels are not significantly reduced for BF, it is hypothesized that the LBPPT is most likely not appropriate for stress relief

in for example hamstring tendinitis, although further research is needed to clarify this specifically. While muscle activation is not significantly reduced in BF, it is in VM and VL. Therefore, as has been previously pointed out (Jensen et al., 2016), the LBPPT device facilitates antagonist (BF) activation compared to agonist (VM and VL) activation, resulting in the tensile forces on the anterior cruciate ligament being reduced. As such, the LBPPT device may pose an advantage in the rehabilitation of patients having undergone anterior cruciate ligament surgery.

Further, the current results suggest that at least 40% reduction in effective body weight should be applied in combination with a slow walking speed (2.5 km/h), if the TA muscle is to display a significant reduction in activation. This should be incorporated if the LBPPT is to be used in the treatment of for instance shin splints. It is possible that the LBPPT device can be used in various other rehabilitation settings, but considerations should be made that the lower limb muscles are not relieved in a similar fashion.

The present study is limited by the fact that only EMG amplitudes are reported as a measure of differences in muscle activation due to reduced effective body weight. The addition of an analysis of the timing of activation would have enhanced the interpretation of the results. Similarly, the addition of a kinematics analysis would have yielded valuable information of the movement consistency and changes following reductions in effective body weight, but this was not deemed possible due to the design of the machine, whereby the participant is encapsulated inside the positive pressure chamber. It has previously been shown that the accuracy of the AlterG Anti-Gravity Treadmill in providing precise reductions in effective body weight is questionable (McNeill et al., 2015). This is an inherent limitation of the current study and may help to explain why a significant reduction is seen in EMG amplitude for the VL and GM muscles at 80%, 40%, and 20%, but not at 60%.

In conclusion, walking with body weight support in a LBPPT, significantly decreased muscle activation of the muscles VM, VL, GM, GL, and SOL. Greater reductions in effective body weight ($\geq 40\%$) and a slow walking speed (2.5 km/h) were needed to significantly decrease the activation of the TA muscle. The muscle activation of BF remained unaffected across all fractional reductions in effective body weight and walking speeds. These findings suggests that the muscles of the lower limb respond differently to the body weight support provided by the LBPPT during walking, thus making the LBPPT a possible rehabilitation solution for some, but not all injuries to the lower limbs.

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Declaration of Competing Interest

The authors report no conflicts of interest.

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Appendix A. Supplementary material

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jelekin.2019.05.021>.

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