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Effects of Using a Powered Compared to Passive-Elastic Ankle-Foot Prosthesis During Level Ground Walking on Leg Muscle Activation Patterns in Persons With a Unilateral Transtibial Amputation

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Effects of Using a Powered Compared to Passive-Elastic Ankle-Foot
Prosthesis During Level Ground Walking on Leg Muscle Activation Patterns in Persons
With a Unilateral Transtibial Amputation

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Abstract

In healthy adults walking on level ground at a steady speed, the ankle plantar-flexors provide the majority of the push off work necessary to redirect and accelerate the CoM during the step-to-step transition. People with a transtibial amputation lack these muscles, compensate for the lack of powered plantarflexion with more proximal muscles, and exhibit biomechanical asymmetries when using a passive-elastic prosthetic foot. Use of a biomimetic powered ankle-foot prosthesis has resulted in normative metabolic costs and biomechanics during walking in people with a transtibial amputation compared to non-amputees. Therefore, use of a powered ankle-foot prosthesis likely affects leg muscle activity, but these effects are currently unknown. We investigated changes in activation of the gluteus maximus, gluteus medius, rectus femoris, vastus lateralis, biceps femoris, tibialis anterior, soleus, and lateral gastrocnemius muscles of the affected and unaffected legs of 10 people with a unilateral transtibial amputation using a passive-elastic and powered ankle-foot prosthesis during level-ground walking at 1.25m/s. We hypothesized that peak muscle activation and integrated electromyography (iEMG) would decrease in the unaffected leg, and increase in the affected leg. Peak muscle activation was greater in unaffected leg gluteus maximus, rectus femoris, lateral gastrocnemius, and soleus while using a powered prosthesis compared to passive-elastic prosthesis (p<0.05). There were no changes in affected leg peak muscle activation or iEMG in either leg with use of a powered compared to passive-elastic prosthesis. Though use of the powered prosthesis did not reduce unaffected leg peak or iEMG, or increase affected leg peak or iEMG, other biomechanical changes may be responsible for overall increases in leg muscle activation.

1. Introduction

Level-ground walking can be mechanically characterized by three phases: 1) single support, when one foot is in contact with the ground, 2) double support, when both feet are in contact with the ground, and 3) leg swing, when one foot is off the ground and being moved forward. The legs must support body weight and redirect and accelerate the center of mass (CoM) into the next step to achieve steady speed, level-ground walking [1-5]. To accomplish these mechanical tasks, lower limb muscles must be active at different points in the gait cycle to generate force and perform work [6].

Specifically, in healthy adults walking at a steady speed over level ground, the hip extensors, knee extensors, and ankle dorsi-flexors are most active at the end of the leg swing phase, to decelerate the leg in preparation for heel strike, and during the beginning of single support [6]. The ankle plantar-flexors are primarily active during mid to late stance and produce propulsive power to move the CoM into the next step [6, 7]. Specifically, the medial gastrocnemius and soleus provide the majority of the push off work necessary to redirect and accelerate the CoM prior to and during the step-to-step transition [7, 8].

Providing ankle push off power accounts for approximately 41% of the total positive individual leg mechanical power required to walk on level ground across a range of speeds [9]. Similarly, the propulsive work provided by the trailing leg prior to and during the step-to-step transition to redirect and accelerate the CoM into the next step accounts for approximately half of the net

metabolic power required to walk on level ground [7]. Persons with a transtibial amputation using a passive-elastic prosthesis lack the ability to generate prosthetic ankle push off work anew during walking. Previous studies have shown that passive-elastic prostheses perform some of the work normally done by the uniarticular soleus, but they do not deliver enough power to accelerate the leg into swing or to compensate for the missing biarticular gastrocnemius [10]. In fact, passive-elastic prostheses provide only half the mechanical work and one-eighth the mechanical power of that provided by a biological ankle when walking on level ground [10-12]. The inability to generate push-off power by the prosthesis also contributes to biomechanical asymmetries during walking. Such asymmetries, due to asymmetrical force production from the legs and stance time differences between the unaffected and affected legs, likely contribute to the higher incidences of joint disorders, chronic residual limb pain, and musculoskeletal disorders in people with transtibial amputations compared to non-amputees [13-15]. Furthermore, people with a transtibial amputation using a passive-elastic prosthesis during walking must compensate for a lack of prosthetic power and work with proximal muscles in the affected leg [10, 16]. Musculoskeletal modeling and simulation studies predict that the unaffected leg rectus femoris, gluteus maximus, and soleus deliver more energy than the same muscles of non-amputees during the first half of stance to compensate for the lack of prosthetic power from the trailing leg, while the iliopsoas increases activation during early swing [10]. Musculoskeletal models also predict that the primary mechanism of compensation is an increase in energy output from the muscles surrounding the hip joint of the affected leg in early stance [16]. Therefore, it is important to examine muscle activity differences in people with a unilateral transtibial amputation using different types of prostheses, to determine which devices minimize asymmetries and/or normalize muscle activation.

When people with a unilateral transtibial amputation walk on level ground using a passive-elastic prosthesis, they prefer to walk at slower speeds [17] and exhibit asymmetric muscle activation patterns – magnitude and duration of activation –between their affected and unaffected legs [18, 19]. The use of a biomimetic powered ankle-foot prosthesis has resulted in normative metabolic costs and biomechanics (i.e. preferred walking velocity, trailing prosthetic leg mechanical work, and leading biological leg mechanical work) in people with a transtibial amputation compared to non-amputees walking over level ground [20]. To perform the mechanical tasks of walking, muscles must generate force and perform work, which requires metabolic energy, thus it is likely that use of a powered prosthesis also affects the muscle activation patterns of both the affected and unaffected legs. However, the effects of using a powered ankle-foot prosthesis on muscle activation are not yet known.

We investigated changes in muscle activation of both the affected and unaffected legs of people with a unilateral transtibial amputation using a passive-elastic and powered ankle-foot prosthesis during level-ground walking. We hypothesized that with use of the powered ankle-foot compared to a passive-elastic prosthesis: 1) peak muscle activation (peakEMG) would be lower in all muscles of the unaffected leg, 2) peakEMG would be higher in all muscles of the affected leg, 3) integrated EMG (iEMG) would be lower in all muscles of the unaffected leg and, 4) iEMG would be higher in all muscles of the affected leg. Finally, because iEMG is influenced by both the amplitude and duration of activation, we hypothesized that the unaffected leg muscles would be active for a shorter duration and affected leg muscles would be active for a longer duration with use of the powered compared to passive-elastic prosthesis.

2. Methods

2.1 Subject Recruitment

Ten adults (6M, 4F, average ± SD: age 42 ± 11 yrs., height 1.7 ± 0.08 m, mass without a prosthesis 77.3 ± 14.8 kg) with a unilateral transtibial amputation who were at or above a Medicare Functional Classification of K3 and free of neurological, cardiovascular, or musculoskeletal disease other than that associated with amputation participated. All subjects gave written informed consent prior to participating according to a protocol approved by the United States Department of Veteran Affairs' Human Subjects Institutional Review Board and in accordance with the principles expressed in the Declaration of Helsinki.

2.2 Experimental Protocol

A certified prosthetist from BionX Medical Technologies aligned subjects to the BiOM T2 powered prosthesis (BiOM, BionX Medical Technologies Inc., Bedford, MA). We then placed reflective markers bilaterally on subjects' first and fifth metatarsal heads, posterior calcanei, medial and lateral malleoli, medial and lateral femoral condyles, greater trochanters, iliac crests, anterior superior iliac spines, and posterior superior iliac spines. For the prosthetic feet, we placed reflective markers at the approximate locations of the first and fifth metatarsal heads, and the posterior of the calcaneus based on the locations for the unaffected leg. Malleolus markers for the powered prosthesis were placed on the encoder, which coincided with the center of rotation in the sagittal plane (Figure 1A). Malleolus markers for the passive prosthesis were placed on the medial and lateral edges of the carbon fiber prosthesis at the most dorsal point of

the keel (Figure 1B). Clusters of four markers placed over the thigh and shank segments tracked segment movement.

We tuned the BiOM iteratively until the prosthetic ankle range of motion, peak ankle power, peak ankle moment, and net ankle work normalized to body mass including the prosthesis matched ankle data of non-amputees and the unaffected leg within one standard deviation [21]. To acclimate to using the powered prosthesis during walking prior to data collection for analysis, subjects walked while using the powered prosthesis for approximately 10 hours over 5 experimental sessions on a dual-belt force-measuring treadmill at 1.25 m/s and different slopes ($0°$, $\pm3°$, $\pm6°$, $\pm9°$) prior to the session where we measured surface EMG for analyses. All experimental sessions were at least 24 hours and no more than 2 weeks apart. The first two sessions were each 2-3 hours long and were dedicated to tuning the powered prosthesis. During the third through fifth sessions (each 1.5 hours in length) we collected metabolic energy expenditure data during walking using the powered prosthesis tuned for level ground, the powered prosthesis tuned for each slope, and a passive-elastic prosthesis. Then, in the sixth session (approximately 2.5 hours in length), we measured surface electromyography (EMG).
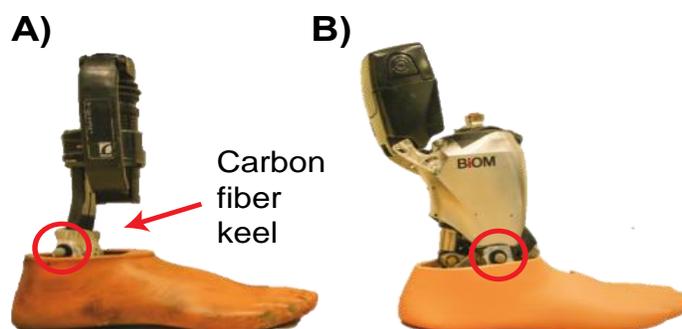

Figure 1. "Malleolus" marker placement on A) a passive-elastic prosthesis and B) the BiOM powered prosthesis.

Subjects with an amputation used their own passive-elastic, energy storage and return (ESAR) prosthesis (Table 1) and the BiOM T2 powered prosthesis. Subjects walked 1.25 m/s on a dual-belt force-measuring treadmill (Bertec Corp., Columbus, OH) for at least 30 seconds while we collected ground reaction forces (GRFs) and surface electromyography (EMG) data at 1000 Hz (Noraxon, Scottsdale, AZ) bilaterally from the biceps femoris long head, gluteus maximus, gluteus medius, vastus lateralis, rectus femoris, lateral gastrocnemius, soleus, and tibialis anterior. We only collected surface EMG data for the lateral gastrocnemius, soleus, and tibialis anterior on the unaffected leg. We placed electrodes on the muscles of interest using visualization and palpation to locate the muscle belly [21]. Prior to electrode placement, we shaved, cleaned, and abraded the skin using Nuprep gel (Weaver and Company, Aurora, CO).

Table 1. Subject anthropometrics and passive-elastic prosthetic feet.

Sex	Height (m)	Mass w/ BiOM (kg)	Mass w/ ESAR (kg)	Passive-elastic foot type
F	1.66	59.5	58.0	Freedom Innovations Renegade
F	1.66	65.3	61.7	Ottobock Triton IC60
F	1.68	69.4	68.5	Össur Pro-flex XC
M	1.75	72.1	70.3	Freedom Innovations Renegade
M	1.71	78.0	77.0	Össur Vari-flex
F	1.71	84.1	81.8	Össur Vari-flex XC
M	1.82	89.4	88.9	College Park Soleus
M	1.85	96.2	95.3	Össur Proflex
M	1.83	97.1	95.5	Ability Dynamics Rush 81
M	1.82	102.3	100.2	Ability Dynamics Rush 87
AVG (SD)	1.70 (0.08)	81.3 (14.72)	79.7 (14.98)	-

2.3 Data Analysis

We filtered GRFs using a 4th order recursive butterworth filter with a 30 Hz cutoff and determined heel strike and toe off events with a 20 N threshold of the vertical GRF.

Using a custom Matlab (Mathworks, Natick, MA) script, we band-pass filtered at 10-495 Hz, rectified, and RMS-averaged with a 50ms window the raw EMG data. We then normalized EMG to the maximum signal magnitude for each muscle over a stride while subjects used the ESAR prosthesis. We determined the peak EMG value from the smoothed and normalized EMG for a stride (peakEMG). We integrated the smoothed and normalized EMG (iEMG) data for each muscle over time for an entire stride ($iEMG_{stride}$) and over time for the stance phase ($iEMG_{stance}$). Because iEMG depends on the magnitude and duration of muscle activation, we determined muscle onset and offset times by finding the point within a stride (heel strike to heel strike of the same foot) that the smoothed and normalized EMG crossed a threshold set at 10% of the maximum signal magnitude for each muscle using the ESAR prosthesis. We chose 10% of the maximum signal using trial and error, because it accurately showed muscle activation onset and offset, without including extra signal noise. We averaged EMG data for at least five strides per subject.

2.4 Statistical Analysis

We used paired dependent t-tests to determine the effects of using different prosthetic feet on peakEMG$_{stride}$, peakEMG$_{stance}$, iEMG$_{stride}$, iEMG$_{stance}$, and the onset/offset of muscle activation for all 13 muscles with RStudio (RStudio, Boston, MA). We set significance at $p = 0.05$.

3. Results

We found an effect of prosthetic foot type on unaffected leg peakEMG$_{stride}$ for gluteus maximus, rectus femoris, lateral gastrocnemius, and soleus. Unaffected leg gluteus maximus, and rectus femoris peakEMG$_{stride}$ increased 105% and 42%, respectively, with use of the BiOM compared to an ESAR prosthesis ($p<0.05$, Fig. 2). Similarly, unaffected leg lateral gastrocnemius and soleus peakEMG$_{stride}$ increased 71% and 50%, respectively, with use of the BiOM compared to an ESAR prosthesis ($p<0.005$, Fig. 2). We found no effect of prosthetic foot type on affected leg peakEMG$_{stride}$ for any of the muscles ($p>0.05$, Fig. 2).
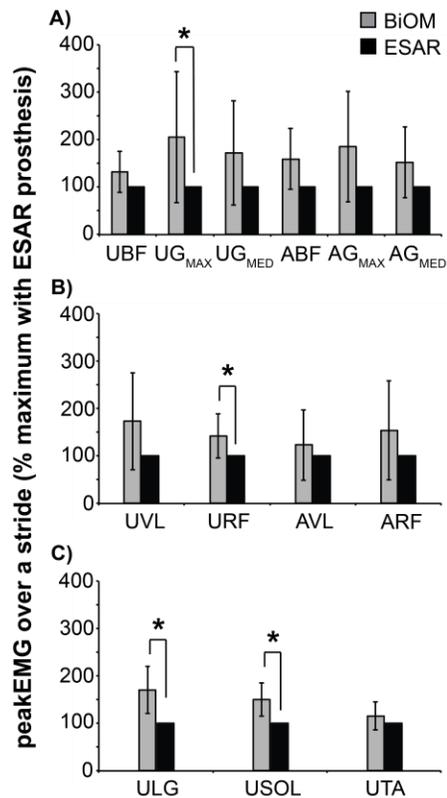
Figure 2. Average peakEMGstride ±S.D. for A) hip extensors, B) knee extensors, and C) ankle flexors/extensors. U represents the unaffected leg and A represents the affected leg. BF is bicep femoris, G_{MAX} is gluteus maximus, G_{MED} is gluteus medius, VL is vastus lateralis, RF is rectus femoris, LG is lateral gastrocnemius, SOL is soleus, and TA is tibialis anterior. *indicates significant difference in peakEMG between prostheses.

We found no significant effect of prosthetic foot type on unaffected or affected leg $iEMG_{stride}$ or $iEMG_{stance}$ ($p > 0.05$, Fig. 3), although there was a trend for increased iEMG in the unaffected and affected legs with use of the BiOM compared to an ESAR prosthesis.
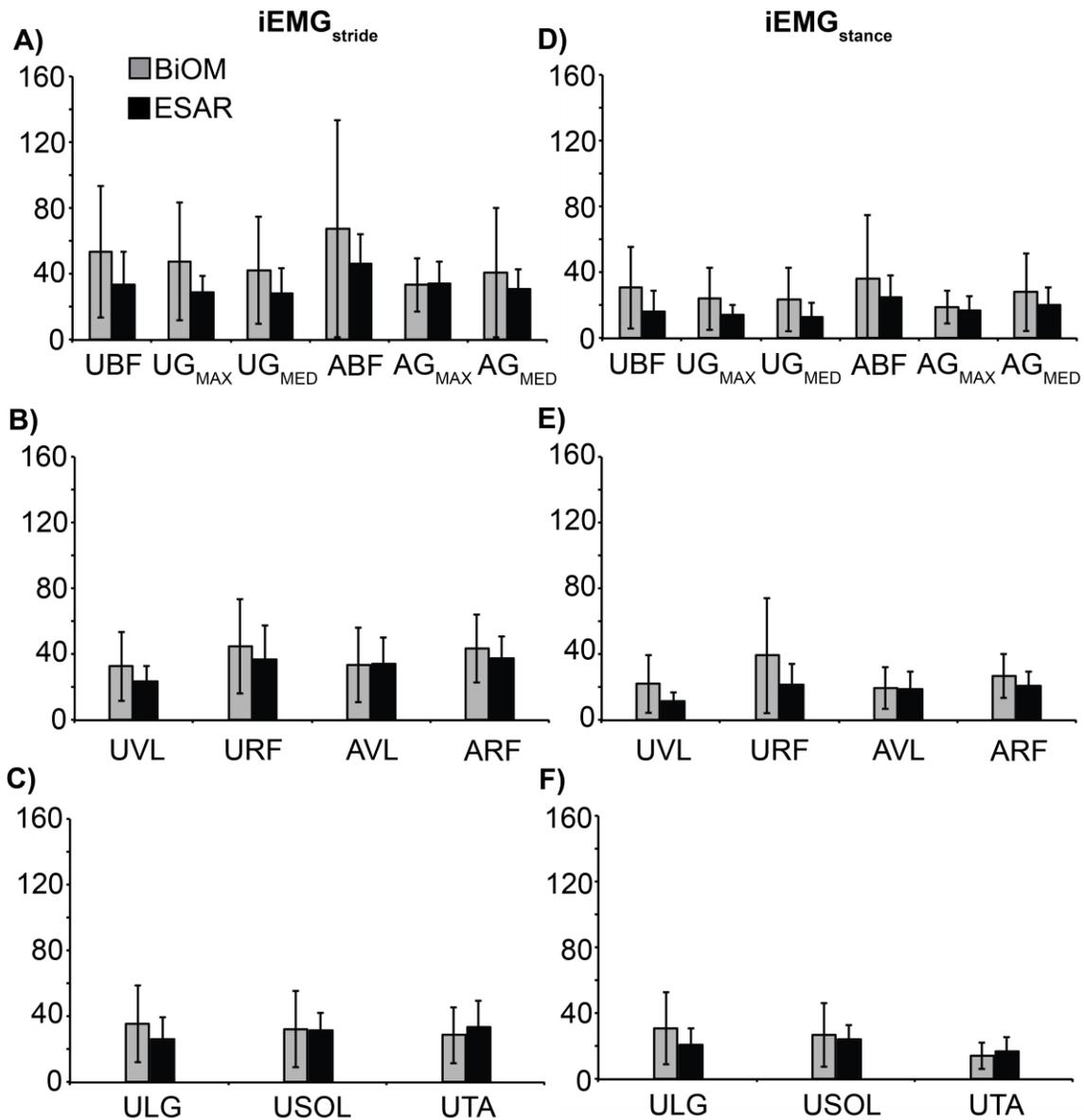
Figure 3. Average iEMG$_{stride}$ (A-C) and iEMG$_{stance}$ (D-F) ± S.D. for hip extensors (top row), knee extensors (middle row), and ankle flexors/extensors (bottom row). U represents the unaffected leg and A represents the affected leg. BF is bicep femoris, G$_{MAX}$ is gluteus maximus, G$_{MED}$ is gluteus medius, VL is vastus lateralis, RF is rectus femoris, LG is lateral gastrocnemius, SOL is soleus, and TA is tibialis anterior. There were no significant differences in iEMG between prostheses.

The timing of muscle activation onset within a stride was not significantly different with use of the BiOM compared to an ESAR prosthesis. However, the muscle activation offset was 6% later in the stride for the unaffected leg lateral gastrocnemius (p<0.05, Fig. 4), and 29% later in the stride for affected leg rectus femoris with use of the BiOM compared to an ESAR prosthesis (p<0.005, Fig. 4).
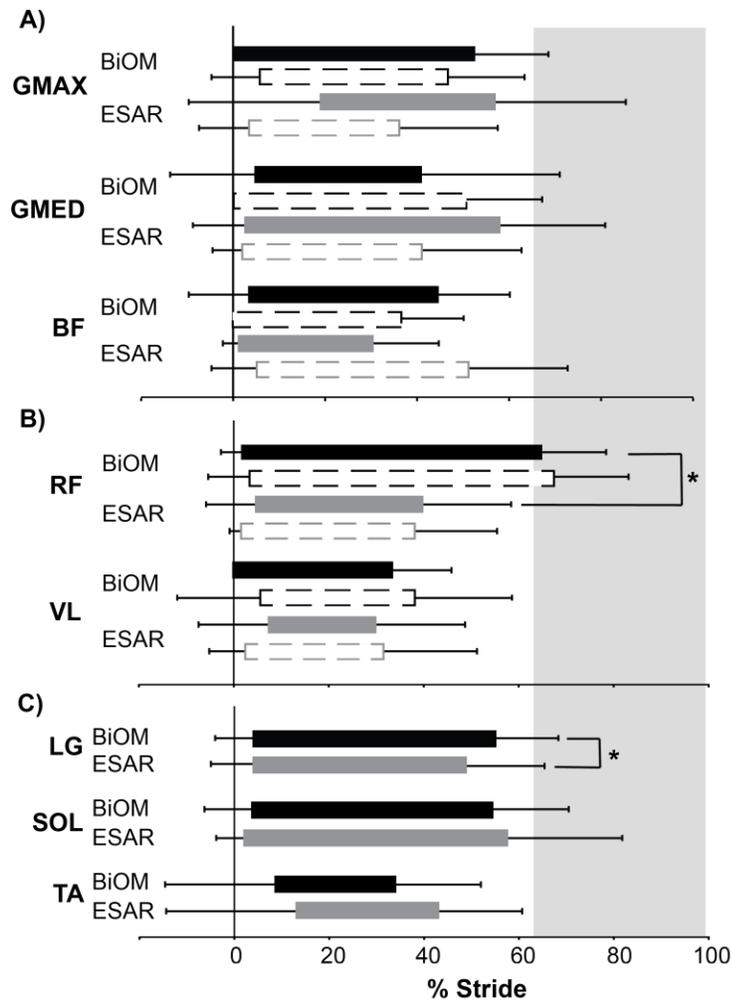

Figure 4. Average ± S.D. muscle activation onset and offset (duration) for A) hip extensors, B) knee extensors, and C) ankle flexors/extensors. Solid outlines are the unaffected leg and dashed outlines are the affected leg. Black bars represent BiOM, grey bars represent ESAR. Shaded region indicates the leg swing phase. *indicates significant difference between BiOM and ESAR.

4. Discussion

In contrast to our first hypothesis, we found that peakEMG$_{stride}$ was greater in the unaffected leg gluteus maximus, rectus femoris, lateral gastrocnemius, and soleus muscles with use of the BiOM compared to an ESAR prosthesis (Figure 2). Our second hypothesis was unsupported because we found no differences in peakEMG between prostheses for any of the affected leg muscles. However, while no changes were statistically significant, peakEMG trended higher in all affected and unaffected leg muscles with use of the BiOM compared to an ESAR prosthesis (Figure 2).

Our third hypothesis was unsupported because we found no changes in unaffected leg iEMG with use of the BiOM compared to an ESAR prosthesis. Similarly, though iEMG$_{stride}$ trended higher in all affected leg muscles with use of the BiOM compared to an ESAR prosthesis, there were no significant increases in affected leg iEMG$_{stride}$ (Figure 3), thus refuting our fourth hypothesis. Similar to iEMG$_{stride}$, we found no significant effect of prosthetic foot type on affected or unaffected leg iEMG$_{stance}$ (Figure 3). Our fifth and final hypothesis was partially unsupported because we found that with use of the BiOM compared to an ESAR prosthesis, muscle activation offset was 6% later in the stride for the unaffected leg lateral gastrocnemius. Conversely, our hypothesis was partially supported because muscle activation offset was 29% later in the stride for affected leg rectus femoris with use of the BiOM compared to an ESAR prosthesis.

The increases in magnitude of peakEMG and in muscle activation duration could be due to the weight of the BiOM prosthesis. iEMG is representative of both the peak EMG amplitude and the

duration of activation. Omuro et al. found that adding 2.5 kg at the ankle for non-amputees increased iEMG amplitude 20% during walking [22]. Similarly, Browning et al. found that the muscles involved with initiating, propagating, and ending the swing phase had 19-81% higher mean EMG amplitude with added masses of 4-8 kg at the ankle for non-amputees walking on level ground [23]. For people with a leg amputation walking on level ground, net metabolic cost (J/s) increased 7-12% when 1.7-2.36 kg was added at the prosthetic ankle to match the mass of the unaffected leg [24, 25]. In our study, prosthetic mass was, on average, 1.6 kg greater when subjects used the BiOM compared to an ESAR prosthesis. Thus, it is possible that this added mass accounted for some of the increases in the unaffected and affected leg peakEMG with use of the BiOM compared to an ESAR prosthesis, and thus could increase the metabolic cost of walking. However, Browning et al. [23] found an increased mean EMG amplitude during walking in the rectus femoris of non-amputees only when 10-12% of the subject's body mass was added distally. Subjects in the present study had a mass of 77.3 ±14.8 kg, so a 1.6 kg increase comprised 1.7-2.6% of their body mass.

Subjects increased stride length by 5%, or 0.07m, in the affected leg (affected leg heel-strike to affected leg heel-strike) and 1%, or 0.02m, in the unaffected leg (unaffected leg heel-strike to unaffected leg heel-strike) when using the BiOM compared to an ESAR prosthesis during walking at 1.25 m/s. In previous studies, increases in stride length in both legs, compared to preferred for non-amputees walking on level ground, led to an increased metabolic cost [26]. This indicates that longer strides compared to preferred likely require more metabolic energy, and thus presumably correlate with increased muscle activation. Therefore, it is possible that the

increase in peakEMG with use of the BiOM prosthesis may have been due in part to the increase in stride length.

In addition to increases in distal weight and stride length of the affected leg, it is possible that insufficient acclimation time to walking using the powered prosthesis contributed to increases in peakEMG. Previous studies have shown that people with transtibial amputations exhibit more co-contraction of residual muscles (remaining tibialis anterior and medial gastrocnemius) and affected leg knee muscles during level ground walking using an ESAR prosthesis [27]. A past study also found that leg muscle co-contraction decreases during 8 weeks of isometric resistance training [28]. In our study, subjects were given approximately 10 hours to acclimate to using the BiOM prosthesis during walking before surface EMG data were collected for analyses. All of our subjects had been using a passive prosthesis for at least 5 years post-amputation, so subjects may not have been familiar enough with using the BiOM after 10 hours of walking to efficiently recruit the muscles and minimize co-contraction during walking. Future studies are needed to establish appropriate acclimation times for use of the powered ankle-foot prosthesis, measure co-contraction, and determine if EMG changes remain after using a powered prosthesis for a prolonged amount of time.

Our study determined the changes in muscle activation associated with walking on level ground using a powered ankle-foot compared to a passive-elastic prosthesis. Future studies should investigate these effects when walking over a wide range of speeds and slopes. Further, while we saw a trend toward increased peakEMG with use of the powered prosthesis, these changes could be due to biomechanical changes such as an increase in stride length. Future studies should be

performed to understand the effect of biomechanical changes observed using the powered prosthesis during walking and corresponding changes in muscle activation magnitude and duration.

5. Conclusion

Use of the BiOM powered prosthesis did not lower peakEMG, $iEMG_{stride}$, or $iEMG_{stance}$ in the unaffected leg, nor did it significantly increase peakEMG, $iEMG_{stride}$, or $iEMG_{stance}$ in the affected leg compared with use of a passive-elastic prosthesis for people with a transtibial amputation. Rather, peakEMG was greater in the unaffected leg gluteus maximus, rectus femoris, lateral gastrocnemius, and soleus while using the BiOM compared to a passive-elastic prosthesis. While not significant, $iEMG_{stride}$, $iEMG_{stance}$, and peakEMG trended greater in all affected leg muscles using the BiOM compared to a passive-elastic prosthesis. Though use of the BiOM powered prosthesis did not reduce unaffected leg peak or integrated EMG or increase affected leg peak or integrated EMG, other biomechanical changes may be responsible for overall increases in leg muscle activation.

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