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Training Propulsion via Acceleration of the Trailing Limb

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***Abstract*—Walking function, which is critical to performing many activities of daily living, is commonly assessed by walking speed. Walking speed is dependent on propulsion, which is governed by ankle moment and the posture of the trailing limb during push-off. Here, we present a new gait training paradigm that utilizes a dual belt treadmill to train both components of propulsion by accelerating the belt of the trailing limb during push-off. Accelerations require participants to produce greater propulsive force to counteract inertial effects, and increases extension of the trailing limb through increased belt velocity.**

We hypothesized that one session of training in our paradigm would produce after effects in propulsion mechanics and, consequently, walking speed. We tested our training paradigm on healthy young adults at two acceleration magnitudes–7 m/s^2 (HA) and 2 m/s^2 (LA)–and compared their results to a third control group (VC) that walked at a higher velocity during training.

Results show that the HA group significantly increased walking speed following training (mean $\pm$ s.e.m: 0.073 $\pm$ 0.013 m/s, p $<$ 0.001). The change in walking speed in the LA and VC groups was not significant (LA: 0.032 $\pm$ 0.013 m/s, VC: -0.003 $\pm$ 0.013 m/s). Responder analysis showed that changes in push-off posture and in activation of ankle plantar-flexor muscles contributed to the greater increases in gait speed measured in the HA group compared to the LA and VC groups. The duration of after effects post training suggest that the measured changes in neuromotor coordination are consistent with use-dependent learning.

I. Introduction

Ambulation is critical to performing many activities of daily living, including self-care and community engagement. Walking ability, commonly assessed by walking speed, decreases with age and is affected by numerous neurological conditions [1], [2]. As our aging population increases, there is a critical need for rehabilitation techniques that are effective in retraining walking ability to prolong independent living and quality of life for these individuals.

Walking function is dependent on propulsive force generation in both young and elderly adults [1], [3]. Propulsive force generation is determined by two main factors: posture of the trailing limb at push-off and ankle moment [2], [4]–[6]. Push-off posture that increases the distance between the foot of the trailing limb and the body center of mass increases the component of the ground reaction force that acts in the forward direction, increasing propulsion. Extension of the trailing limb during push-off can be assessed by trailing limb angle and stride length. While postural modification alone can increase propulsion, so can changes in the activation of ankle plantarflexor muscles that generate the ankle moment. Decreased ankle moment is a functional limitation in elderly adults that leads to decreased walking speed, making ankle moment generation a key target for intervention [3].

In this work, we present a novel paradigm for propulsive gait training that utilizes a dual belt treadmill to train both ankle moment and push-off posture. Our paradigm targets both propulsive mechanisms by accelerating the treadmill belt of the trailing limb during the double support phase of gait. The belt acceleration introduces a fictitious inertial force that requires the ankle plantarflexor muscles to generate a greater ankle moment during push-off to maintain a steady position on the treadmill. Assuming no modification in push-off timing, accelerations of the belt increase the velocity of the trailing foot which increases extension of the trailing limb, thereby training advantageous modifications in push-off posture.

Following a single-session of our training paradigm , behavioral after effects could be driven by two independent learning mechanisms: adaptation or use-dependent learning (UDL). Adaptation is a learned response to a change in environmental dynamics that drives a re-calibration of feedforward motor commands that persist when the environmental demands are removed. Without additional reinforcement, after effects of adaptation typically peak immediately after training and follow an exponential decay back to baseline behavior as the central nervous system de-adapts, typically within 150 strides ($\sim$ 2 minutes of walking) [7]–[10]. Locomotor adaptation approaches have been used in rehabilitation to address gait asymmetry [8] and foot clearance [11], but have not been used to directly target propulsion. Instead, UDL is a type of Hebbian learning that is the basis of many high-repetition rehabilitation protocols [12]–[14]. UDL occurs when participants perform numerous consistent movements that bias future movements in the direction of the repeated movement. Repeated movements in our protocol include walking with an increased leg extension during push-off and increased plantarflexor muscle activity. After effects of UDL are more persistent than adaptation and typically last for more than 10 minutes. [14]–[16].

The primary purpose of this study was to test the efficacy of training propulsion using belt accelerations to increase gait speed. We tested our paradigm in healthy individuals at two acceleration magnitudes (High, 7 m/s^2 and Low, 2 m/s^2), and included a velocity control group (VC) that walked at an increased constant velocity during training. The increase in velocity imposed in the VC group was matched to the average change in velocity caused by belt accelerations applied in the high acceleration group. To test for effects in gait speed, our primary outcome measure, we evaluated participants self-selected treadmill walking speed before and after exposure

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to training in a user driven speed condition [17]. To test for change in propulsion, force-plate measurements of the anterior ground reaction force (AGRF) were used to quantify peak AGRF and propulsive impulse during push-off. To test for modifications in push-off posture, trailing limb angle (TLA) and stride length (SL) were measured via 3D kinematic motion tracking. Change in ankle moment was assessed indirectly from EMG data measured from four ankle plantar- and dorsiflexor muscles that contribute to the ankle moment.

We hypothesized that exposure to our training paradigm would produce after effects in propulsion that would increase walking speed in both acceleration groups, but not in the velocity control group. As change in propulsion may be due to changes in ankle moment and/or push-off posture, we tested for effects in both mechanisms. We hypothesized that there would be an increase in TLA, SL, and ankle muscle activation in all groups during training, but sustained change following training only in the acceleration groups.

To determine if after effects of training were driven by adaptation or UDL, we established whether after effects persisted in a five minute long post-training session. We hypothesized that after effects driven by adaptation would result in immediate significant after effects (within the first 20 strides) that decay to baseline behavior due to deadaptation, resulting in no significant effects by the end of the post-training session (5 minutes). For UDL, we hypothesized that after effects would persist for the full post-training session, and be significantly different from baseline at the end of the post-training session.

II. Materials and Methods

A total of 76 healthy young adults, free from neurological or musculoskeletal injury, participated in this study. Of the 76 individuals, eight were excluded due to technical failure, four for self-selected walking speed outside the operational range of our controller (0.6 1.6) m/s, two for excessive cross-over between treadmill belts during walking, two for highly variable walking speed during the user-driven treadmill conditions ($> \pm 0.05$ m/s within a minute), and one for tripping. The high acceleration (HA) group had nineteen participants (9 males, age (mean $\pm$ std) 24 $\pm$ 4 y), the low acceleration (LA) group had twenty participants (10 males, age 23.5 $\pm$ 3.75 y), and the velocity control (VC) group had twenty participants (11 males, age 24.5 $\pm$ 3.5 y). The study was approved by the Institutional Review Board of the University of Delaware (IRBNet ID: 929630-5). Each participant provided written informed consent and received compensation for participation.

A. Experimental Set-Up

Our experimental protocol is shown in Fig. 1. Participants walked for 5 minutes at a self-selected speed (Baseline), followed by 10 minutes in the training condition (Training), followed by 5 minutes at a self-selected speed (Post-training). In Baseline and Post-training, the treadmill speed was set via a user-driven speed controller described in Sec. II-C. Participants walked on an instrumented dual-belt treadmill (Bertec Corp., Columbus OH, USA), while wearing four reflective spherical markers (one per each greater trochanter and lateral malleolus),

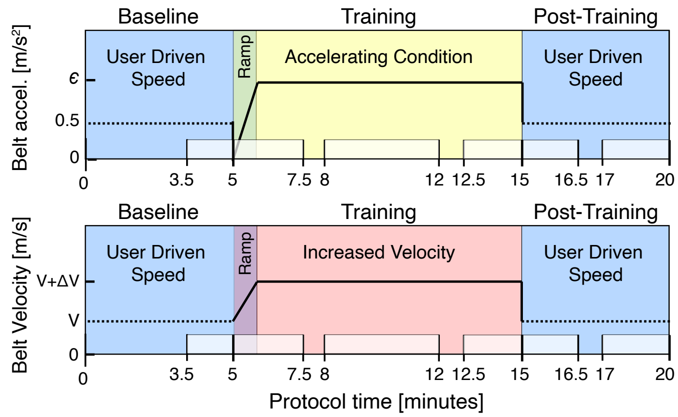

Fig. 1. Experimental Protocol. Highlighted phases at the bottom signify periods in which kinematic marker data were collected. In all Baseline and Post-Training conditions, a user-driven treadmill controller allowed participants to walk at a self-selected speed. Each training condition included an initial one minute long ramp phase to gradually introduce belt accelerations or change in velocity. Top: Acceleration training. Belt accelerations are shown on the y-axis, where ϵ signifies the magnitude of accelerations applied during training (2 m/s^2 or 7 m/s^2). Bottom: Velocity Control training. Belt velocity is shown on the y-axis. During training an increase of ΔV = 0.05 m/s was applied over the final velocity achieved in Baseline.

and eight bipolar EMG electrodes (bilaterally on the tibialis anterior, lateral gastrocnemius, medial gastrocnemius, and soleus muscles). A ten camera Vicon T40-S passive motion capture system (Oxford Metrics, Oxford, UK) was used to measure marker position in 3D space and an OT Bioelettronica amplifier and software were used to acquire EMG. Due to system limitations, marker data were acquired only during the four periods highlighted in Fig. 1, at 100 Hz. EMG and treadmill force/torque data were acquired continuously throughout all experimental conditions at 10,240 Hz and 500 Hz respectively. A 24-in screen placed at eye level approximately 3 m in front of the treadmill provided a visual target to keep participants from looking down at the treadmill. The screen displayed a slideshow of nature scenes that changed every minute and a half. Text appeared at the 6, 12 and 18 minute marks to provide feedback on protocol duration. Participants wore noise canceling headphones (COWIN E7) that played white noise to eliminate environmental distractions.

The high acceleration magnitude was set to 7 m/s^2, which was highly noticeable yet safe for healthy individuals. The low acceleration magnitude was set to 2 m/s^2, the lower bound of the perceptible range for belt acceleration magnitudes [18]. For the VC group, an increase in velocity of 0.05 m/s was imposed over the final steady state walking speed achieved in Baseline. The change in velocity was chosen to match the average change in velocity experienced in the HA training condition, calculated as the average change in velocity across the gait cycle due to belt accelerations applied during push-off measured in 9 participants in our previous study [18]. Data from eight participants in the "imperceptible" group in our previous study were included in the LA group in this study.

B. Self-selected walking speed

A preliminary set of trials were conducted to determine participants self-selected walking speed immediately prior to our protocol. Participants walked on the treadmill at an initial speed of 0.5 m/s that was increased by the experimenter in intervals of 0.02 m/s until the participant verbally indicated the treadmill had reached their fastest comfortable walking speed. The treadmill was then returned to 0.5 m/s and the same ramp-up procedure was repeated to find participants' self-selected walking speed. A ramp-down procedure was then conducted with the treadmill starting at the participants fastest comfortable walking speed and decreased in increments of 0.02 m/s until the participant indicated their self-selected walking speed had been reached. The ramp-up and ramp-down procedures to determine self-selected walking speed were each repeated twice, and the average of the four measured velocities was taken as the participants' self-selected walking speed [19].

C. User-Driven Speed Controller

In standard treadmill walking, walking speed is restricted to the constant velocity imposed by the treadmill. Because our training paradigm seeks to modify participants walking speed, a treadmill that operates at a constant velocity–and thus restricts changes in walking speed–is impractical, and likely to eliminate any after effects due to training. To address this issue, we used a user-driven treadmill controller (UDTC) that changes the velocity of the treadmill in response to changes in the participants walking behavior, and more readily mirrors over-ground walking conditions [17].

The UDTC changes speed based on an empirically weighted combination of the following three gait parameters: change in AGRF, step length, and position of participants' center of mass relative to the center of the treadmill. For example, if participants produce greater AGRF, increase their step length, or walk further forward on the treadmill, the treadmill speed increases. Conversely, decreases in AGRF, step length, or movement to the back of the treadmill would decrease speed. The maximum belt acceleration was set to 0.5 m/s^2, and was previously tested to ensure participant safety and comfort [18].

Prior to our protocol, participants were given a brief training in the UDTC condition. Participants were started at their self-selected walking speed and given up to 5 minutes to familiarize themselves with the UDTC. In line with previous results, some participants increased their walking speed on the UDTC from the self-selected walking speed they chose in the fixed speed condition [17]. Consequently, for our experimental protocol, the baseline period was set to at least 5 minutes, but was continued for a maximum of 10 minutes until the peak change in velocity within one minute was smaller than 0.05 m/s. The Baseline (Fig. 1) condition of our protocol was then defined as the period spanning the one minute of steady state walking, and the previous four minutes prior to achieving a steady state.

D. Belt Acceleration Controller

To train increases in propulsion, we developed a controller that accelerates the treadmill belt of the trailing limb during the double support phase of gait and returns it to its previous speed during the swing phase of gait (Fig. 2). The rationale behind the use of this dynamic distortion (acceleration) was to attenuate the ground reaction force, requiring participants

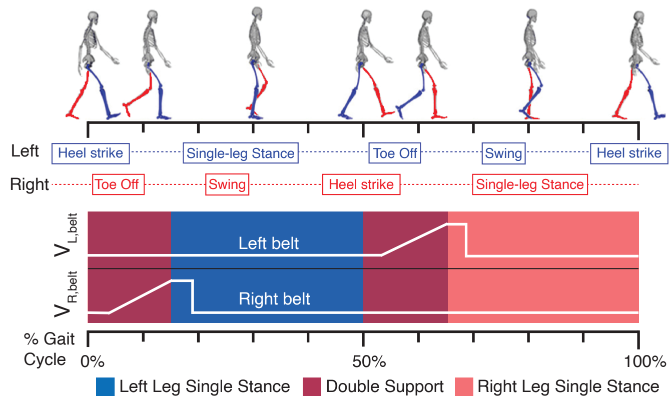

Fig. 2. Timing diagram of belt velocities used for the acceleration groups. When the belt was being accelerated, belt velocity ($V_{R/Lbelt}$) was set to the steady state velocity measured at the end of the baseline condition.

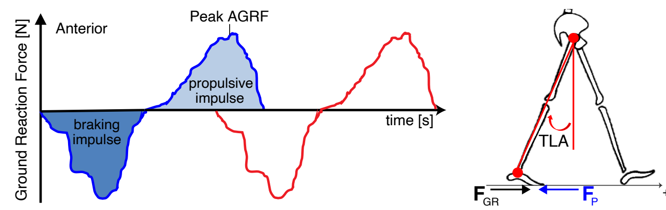

Fig. 3. Definition of gait parameters propulsive impulse (PI), Peak AGRF and TLA. When the belt is not accelerated, the propulsive force generated by the participant ($\mathbf{F}_P$, blue arrow) is equal and opposite to the ground reaction force ($\mathbf{F}_{GR}$, black arrow), i.e. the external force applied to the participant. The anterior portion of the ground reaction force measured by the force plates (shown on the left) corresponds to the forward directed $\mathbf{F}_{GR}$ depicted in the right diagram. If the belt (and leg) is accelerated in the direction of $\mathbf{F}_P$, the inertial effects attenuate the external force applied to the participant ($|\mathbf{F}_{GR}| < |\mathbf{F}_P|$), requiring participants to generate a greater $\mathbf{F}_P$.

to push harder to overcome the inertial effects introduced by the belt acceleration (Fig. S1 of the Supplementary Materials). Moreover, assuming that participants do not modify their push-off timing, the foot on the accelerated belt will move at a larger average speed, causing TLA and SL to increase. In this way, we aimed to target both gait mechanisms (ankle moment and push-off posture) that modulate propulsive force generation.

Push-off occurs at the end of the double support phase of gait that typically lasts for 100-150 ms. While double support can be detected in real time using force-plate data, the delay between detection of dual support and the execution of an acceleration command exceeded 150 ms, making the use of real-time detection impracticable. As such, we developed a simple algorithm to predict when push-off would occur based on the prior gait cycle. Using this algorithm, the controller sends an acceleration signal at a time t, when

$$t > t_{HS} + \alpha{\cdot}\Delta T_{prior} - \beta \qquad (1)$$

where t is the current time, t_{HS} is the time instant of heel strike of the leg currently in stance, ΔT_{prior} is the time between the previous left and right heel strikes that provides a prediction of when double support will occur, and β is an anticipation factor included to account for system delays. Based on this logic, when the amount of time elapsed following heel strike exceeds $\alpha{\cdot}\Delta T_{prior} - \beta$, the acceleration signal is

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sent. Parameter values of $\alpha = 1.175$ and $\beta = 0.185$ s were determined empirically on a separate group of 10 individuals to confirm that accelerations were appropriately timed to occur during push-off for a reasonable range of walking speeds (0.6 - 1.6 m/s). Accelerations were saturated by a speed increase limit of 0.7 m/s to ensure participants safety. 100 ms after detection of toe-off, during the swing phase, the controller decelerates the belt at 10 m/s^2 back to its prior speed (Fig. 2).

1) Data Preprocessing: EMG, kinematic marker data, and gait speed data were acquired on three separate systems and time synced via a common force-plate data signal. VICON marker position data were fed into a standard Visual3D pre-processing pipeline that included i) manual labeling of markers, ii) interpolation of missing marker data with a third order polynomial fit for a maximum gap size of five samples, and iii) low-pass filtering at 6 Hz with a 4th order zero-shift Butterworth filter [19]. Force-plate data were low-pass filtered at 25 Hz with a 4th order zero-shift Butterworth filter [19]. EMG data were bandpass filtered at 20-500 Hz, rectified, and the envelope was taken via a lowpass zero-shift 4th order Butterworth filter with a 10 Hz cut off frequency [5].

2) Data Analysis: Force-plate data were used to define heel strike and toe off events. Heel strike events were determined as the instants at which the vertical ground reaction force exceeded 5% max force and remained above 5% max force for at least 200 ms. Toe off events were determined as the instants at which the vertical ground reaction force fell below 5% max force and remained below for at least 150 ms. EMG data of each muscle (tibialis anterior (TA), lateral and medial gastrocnemius (LG, MG), and soleus (SO)) were segmented and linearly resampled to [0 - 100] percent of gait cycle, defined by each heel strike to heel strike event. Each gait cycle was further subdivided into periods of single and double support using heel-strike and toe-off events.

Gait speed (GS) was sampled at each heelstrike event, when the UDTC updates the belt velocity. Propulsive impulse (PI) which quantifies change in momentum, was calculated as the area under the positive (anterior) portion of the ground reaction force (Fig. 3) [2]. Peak AGRF, which better coincides with changes in GS [20], was taken as the maximum anterior ground reaction force within each gait cycle. Because belt accelerations cause distortions in force-plate measurements [21], PI and Peak AGRF measures during training were excluded from analysis for both acceleration groups.

TLA was defined as the maximum angle between the straight line connecting the greater trochanter and the lateral malleolus of the trailing limb for each stride cycle (Fig. 3). While TLA is typically measured at time of peak AGRF [2], our alternative definition of TLA [22] enabled measurement of TLA during training in all groups, and was highly correlated (R^2 =0.98) with TLA measured at peak AGRF in the baseline condition for all groups. Stride length (SL) was calculated as the anterior-posterior excursion of the lateral malleolus ankle marker from heel strike to heel strike [23].

EMG activation related to propulsion was defined as the max EMG signal measured in plantar-flexor muscles during the single support phase of gait (12-50% GC) [5], [6]. EMG activation related to weight acceptance (breaking) was defined as the max EMG signal measured in dorsiflexor muscles in the interval around heel strike (0-12, 80-100% GC) [5], [6]. EMG data were normalized by the median peak activation measured during the last minute of baseline walking when participants had reached a steady state velocity. One participant in each acceleration group was excluded due to excessive noise caused by EMG cable motion during walking.

E. Statistical Analysis

We used a mixed model analysis to evaluate the effects of training and training group on gait parameters (GS, PI, Peak PI, TLA, SL, and Peak EMG). Training group (HA, LA, VC) was the between participants factor. Experimental phase was the within-participants factor, and included the levels: baseline (BL), Early and Late Training (Early TR, Late TR), and Early and Late post-training (Early PT, Late PT). BL was calculated as the mean of the last minute of baseline walking. Early and Late TR were calculated as the mean values measured in the first and last 20 strides in the training condition (not including the ramp phase), and were included to determine the **effects of training** for valid gait measures (TLA, SL, and EMG). Early and Late PT were calculated as the mean values measured in strides 5-24 and the last 20 strides in the post-training condition, and were included to determine the **after effects** of training on all gait parameters. Post-training strides 1-4 were excluded due to transient effects of participants taking “stutter steps” as a result of changing the treadmill behavior midstream (Fig. S2 of the Supplementary Materials) [23]. All strides were defined as the average between left and right leg data, as the factor leg, and interaction between leg and experimental phase was not significant when included in the mixed model analysis for all gait parameters. When the mixed model returned a significant effect ($p < 0.05$), Tukey HSD post-hoc testing was used to quantify the effect on the measured gait parameters. We additionally report the significant within group effects of experimental phase for all gait parameters.

To investigate efficacy of training at the individual level, we calculated z-scores of the within participant change from baseline in early and late post-training. Participant specific z-scores were defined as the change in outcome measure from baseline, divided by the standard deviation of the outcome measure in baseline. Participants were classified as positive responders if their z-score was greater than zero, and negative responders if their z-score was less than zero. We determined the number of positive and negative responders in each training group for all gait parameters, and their respective median effect size and range. Participants identified as positive responders in GS largely overlapped with positive responders in other gait parameters reported (Average overlap: 92.4% HA group, 77.3% LA group, 59.15% VC group).

To investigate the learning mechanisms responsible for observed after effects, we compared immediate after effects measured in Early PT to Late PT. Immediate, significant after effects in Early PT that decay to baseline behavior (become non-significant) by Late PT were taken as evidence for adaptation. Significant after effects in both Early and Late PT were taken as evidence for UDL. MATLAB and JMP Pro software were used for all statistical analyses.

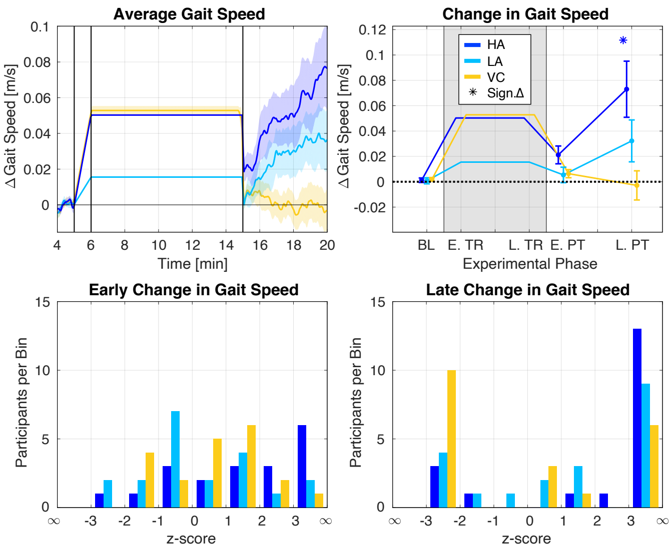

Fig. 4. Wiithin participant change in gait speed from baseline walking, broken down by group. Representation of within participant change is provided for easier graphical display of the effects of training, however statistical analysis was performed on raw data. Top Left: Group average change in GS across the experimental protocol, resampled in time. Shaded areas depicts standard error. Estimated change in gait speed is reported for acceleration groups based on duration of applied accelerations. Top Right: Mean and standard error of group average change in GS. Asterisks denote significant change from baseline from post-hoc Tukey HSD analysis. Bottom: Histogram from responder analysis of after effects measured in the Early (right) and Late (left) post-training session.

TABLE I
MIXED MODEL RESULTS: FIXED EFFECTS FOR ALL GAIT PARAMETERS.

Gait Speed	Nparm	DF Den.	F Ratio	Prob > F
Group	2	59.80	0.608	0.548
Exp. Phase	2	112.00	10.552	**< 0.001**
Group*Exp. Phase	4	112.00	4.621	**0.002**
Propulsive Impulse	Nparm	DF Den.	F Ratio	Prob > F
Group	2	58.50	0.12	0.890
Exp. Phase	2	112.00	34.189	**< 0.001**
Group*Exp. Phase	4	112.00	2.091	0.087
Peak AGRF	Nparm	DF Den.	F Ratio	Prob > F
Group	2	59.10	0.292	0.748
Exp. Phase	2	112.00	18.603	**< 0.001**
Group*Exp. Phase	4	112.00	4.177	**0.003**
TLA	Nparm	DF Den.	F Ratio	Prob > F
Group	2	59.00	0.726	0.488
Exp. Phase	4	223.00	20.583	**< 0.001**
Group*Exp. Phase	8	223.00	1.795	0.079
Stride Length	Nparm	DF Den.	F Ratio	Prob > F
Group	2	61.20	1.299	0.280
Exp. Phase	4	223.00	10.192	**< 0.001**
Group*Exp. Phase	8	223.00	1.863	0.067
Lateral Gastrocnemius	Nparm	DF Den.	F Ratio	Prob > F
Group	2	203.30	0.000	1.000
Exp. Phase	4	216.00	15.480	**< 0.001**
Group*Exp. Phase	8	216.00	2.775	**0.006**
Medial Gastrocnemius	Nparm	DF Den.	F Ratio	Prob > F
Group	2	213.40	0.000	1.000
Exp. Phase	4	216.00	11.269	**< 0.001**
Group*Exp. Phase	8	216.00	0.855	0.555
Soleus	Nparm	DF Den.	F Ratio	Prob > F
Group	2	190.40	0.000	1.000
Exp. Phase	4	216.00	11.691	**< 0.001**
Group*Exp. Phase	8	216.00	2.434	**0.015**
Tibialis Anterior	Nparm	DF Den.	F Ratio	Prob > F
Group	2	232.70	0.000	1.000
Exp. Phase	4	216.00	8.301	**< 0.001**
Group*Exp. Phase	8	216.00	2.937	**0.004**

III. RESULTS

A. Gait Speed

We hypothesized that exposure to our training paradigm would produce after effects that would increase post-training gait speed in both acceleration groups, but not in the velocity control group. Full mixed model results are reported in Tbl. S1 of the Supplementary Materials.

The model returned a significant fixed effect of experimental phase, and an interaction between training group and experimental phase (Fig. 4, Tbl. I). Across groups, gait speed in Late PT was significantly greater than both BL and Early PT (mean $\pm$ s.e.m: 0.034 $\pm$ 0.008 m/s, 0.023 $\pm$ 0.008 m/s, respectively). The interaction was driven by a greater increase in Late PT from BL in the HA group compared to the LA and VC groups (**HA**: 0.073 $\pm$ 0.013 m/s, **LA**: 0.032 $\pm$ 0.013 m/s, **VC**: -0.003 $\pm$ 0.013 m/s, **HA vs LA**: $p = 0.031$, **HA vs VC**: $p < 0.001$), and in Late PT from Early PT in both acceleration groups compared to the VC group (**HA**: 0.052 $\pm$ 0.013 m/s, **LA**: 0.027 $\pm$ 0.013 m/s, **VC**: -0.009 $\pm$ 0.013 m/s, **HA vs VC**: $p = 0.001$, **LA vs VC**: $p = 0.050$). Post-hoc testing showed that the increase in gait speed in Late PT over BL and Early PT was significant in the HA group.

Responder analysis (Tbl. II, Fig. 4) showed a greater number of positive responders and larger median effects across post-training in the HA group, and in Late PT in the LA group compared to the VC group (**HA:** Early: 14 positive responders (median z-score: 2.39, range: [0.59 6.25]), Late: 15 (8.36 [1.36 20.91]); **LA:** Early: 9 (1.74 [0.56 8.69]), Late: 14 (4.48 [0.03 25.52]); **VC:** Early: 14 (1.22 [0.01 3.05]), Late: 10 (3.09 [0.08 16.56])).

In agreement with our hypothesis, both acceleration groups had greater increases in post-training gait speed than the velocity control group, which had no change in post-training gait speed. The high acceleration group had the largest, most consistent increase in gait speed compared to both other groups.

B. Propulsion

We hypothesized that post-training increases in walking speed would be driven by increases in propulsion, as quantified by propulsive impulse and peak anterior ground reaction force, that would be present in both acceleration groups, but not in the velocity control group. The full mixed model results are provided in the Supplementary Materials (Tbl. S2-S3).

1) Propulsive Impulse: The model returned a significant fixed effect of experimental phase (Fig. 5, Tbl. I). Across groups there was a significant increase in propulsive impulse in Early and Late PT over BL (Early PT: 0.74 $\pm$ 0.15 Nm·s, Late PT: 1.24 $\pm$ 0.15 Nm·s), and in Late PT over Early PT (0.50 $\pm$ 0.15 Nm·s). Compared to the average effect across groups, the VC group had a smaller increase in Late PT over BL, that was significantly less than the HA group (**HA**: 1.63 $\pm$ 0.26 Nm, **VC**: 0.81 $\pm$ 0.26 Nm, **HA vs VC**: $p < 0.029$), while the HA group had a larger increase in Late PT over Early PT that was significantly greater than the VC group (**HA**: 1.03 $\pm$ 0.26 Nm, **VC**: 0.09 $\pm$ 0.26 Nm, **HA vs VC**: $p < 0.012$). Post-hoc testing revealed significant increases in propulsive impulse in

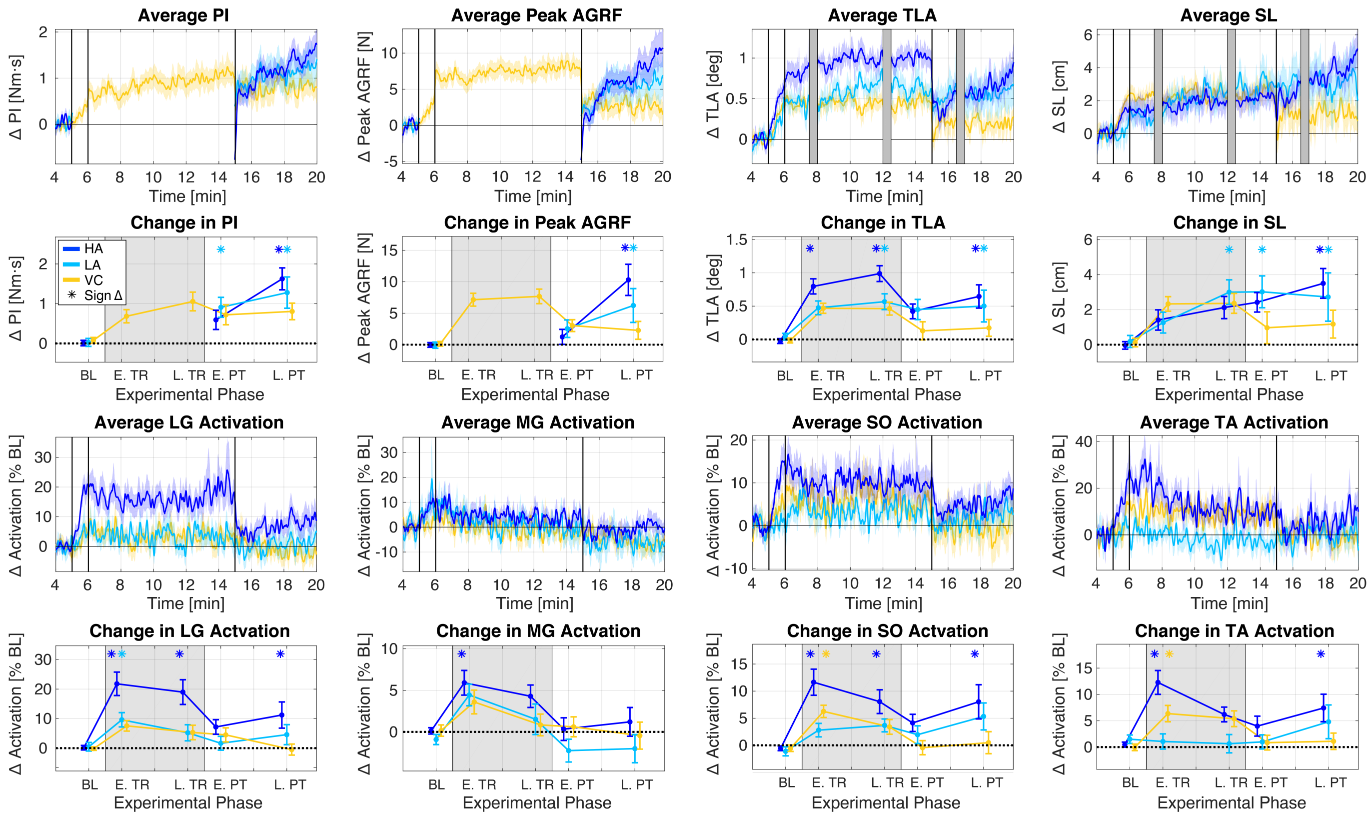

Fig. 5. Within participant change in all gait parameters broken down by group. Rows 1 and 3: Group average change in gait parameters across the experimental protocol, resampled in time. Representation of within participant change from baseline behavior is provided for easier graphical display of the effects of training. Statistical analysis was performed on raw data. Rows 2 and 4: Mean and standard error of group average change in gait parameters from our mixed model analysis. Asterisks denote significant change from BL from post-hoc Tukey HSD analysis.

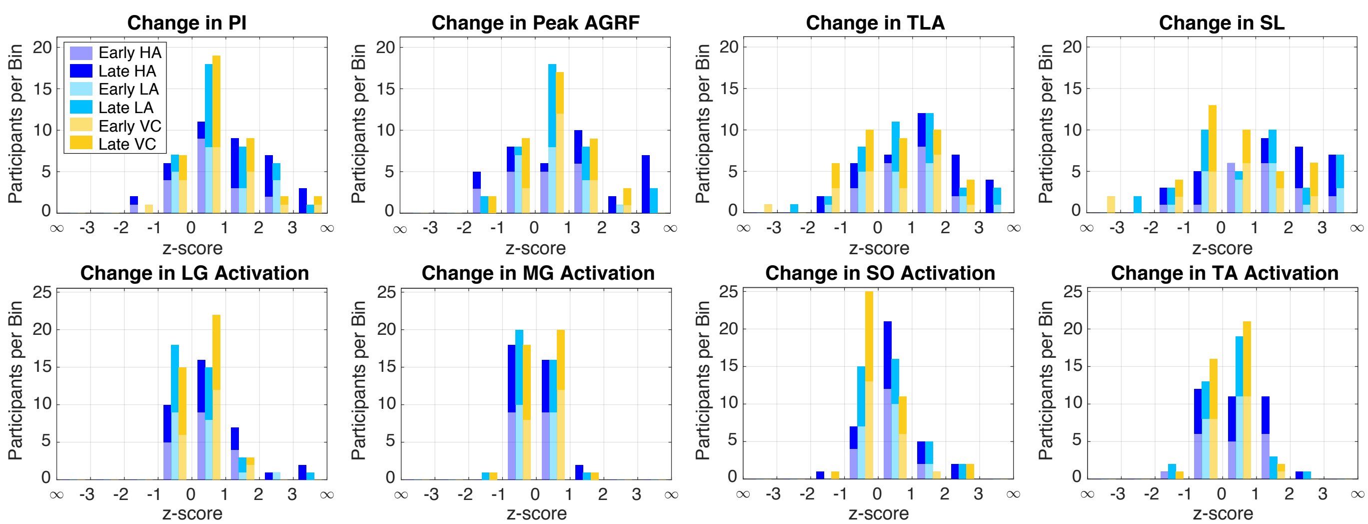

Fig. 6. Histogram of responder analysis for after effects measured in early (strides 5-24) and late (final 20 strides) post-training for all gait parameters.

Late PT over Early PT and BL in the HA group, and in Early and Late PT over BL in the LA group (0.90 ± 0.26 Nm·s, 1.27 ± 0.26 Nm·s, respectively).

Responder analysis (Tbl. II, Fig. 6) showed a similar number of positive responders between groups, but a greater effect size in Late PT in the HA group (**HA:** Early: 14 (0.69 [0.06 2.36]), Late: 16 (2.06 [0.31 4.46]); **LA:** Early: 15 (0.98 [0.07 2.99]), Late: 18 (0.83 [0.01 5.48] z-score); **VC:** Early: 15 0.83 [0.10 3.72]), Late: 17 (0.75 [0.23 3.34])).

2) Peak AGRF: The model returned a significant fixed effect of experimental phase, and a significant interaction between training group and experimental phase (Fig. 5, Tbl. I). Across groups there was a significant increase in peak AGRF in Early and Late PT over BL (Early PT: 2.27 ± 1.04 N, Late PT: 6.27 ± 1.04 N), and in Late PT over Early PT (3.99 ± 1.04 N). The interaction was driven by a greater increase in

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Late PT over Early PT in the HA group compared to the LA and VC groups (**HA**: 9.05 $\pm$ 1.83 N, **LA**: 3.68 $\pm$ 1.79 N, **VC**: 0.76 $\pm$ 1.79 N, **HA vs LA**: $p = 0.038$, **HA vs VC**: $p < 0.001$), and a greater increase in Late PT over BL in the HA group compared to the VC group (**HA**: 10.30 $\pm$ 1.83 N, **VC**: 2.28 $\pm$ 1.79 N, **HA vs VC**: $p = 0.002$). Post-hoc testing revealed significant increases in peak AGRF in Late PT over Early PT and BL in the HA group, and in Late PT over BL in the LA group (6.22 $\pm$ 1.79 N).

Responder analysis (Tbl. II, Fig. 6) showed a greater number of positive responders in the HA and LA groups in Late PT compared to the VC group. Positive responders in the HA group had the largest median effect size across post-training (**HA:** Early: 11 (1.13 [0.00 1.62]), Late: 14 (2.98 [0.66 5.35]); **LA:** Early: 13 (0.92 [0.04 2.34]), Late: 17 (0.72 [0.12 5.65]); **VC:** Early: 17 (0.75 [0.06 2.29]), Late: 12 (1.23 [0.06 2.55])).

In sum, both acceleration groups had significant increases in both propulsive measures following training, while the VC group had no significant change. Across the post-training session, the HA group increased their propulsion to a greater extent than both the LA and VC groups, in line with their measured change in gait speed. In the responder analysis, the acceleration groups exhibited the largest effects, although modest increases were seen in the VC group.

C. Push-off Posture

As both push-off posture and ankle moment can contribute to changes in propulsion, we tested for effects in both propulsive mechanisms [2]. For push-off posture, we hypothesized that there would be an increase in trailing limb angle and stride length in all groups during training, but sustained change post-training only in the acceleration groups. The full model results are reported in the Supplementary Materials (Tbl. S4-S5).

1) Trailing Limb Angle: The model returned a significant fixed effect of experimental phase (Fig. 5, Tbl. I). Across groups, there was a significant increase in trailing limb angle in all experimental phases over BL (Early TR: 0.58 $\pm$ 0.08 deg, Late TR: 0.67 $\pm$ 0.08 deg, Early PT: 0.33 $\pm$ 0.08 deg, Late PT: 0.44 $\pm$ 0.08 deg). Compared to the average effect across groups, the HA group had a larger increase in Late TR over BL, that was significantly larger than both other groups (**HA**: 0.99 $\pm$ 0.14 deg, **LA**: 0.57 $\pm$ 0.14 deg, **VC**: 0.46 $\pm$ 0.14 deg, **HA vs LA**: $p = 0.036$, **HA vs VC**: $p < 0.009$). Compared to the average effect across groups, the VC group had a smaller increase in Late PT over BL, that was significantly less than the HA group (**HA**: 0.64 $\pm$ 0.14 deg, **VC**: 0.17 $\pm$ 0.14 deg, **HA vs VC**: $p = 0.019$). Post-hoc testing revealed significant increases in trailing limb angle in Early TR, Late TR, and Late PT over BL in the HA group (Early TR: 0.80 $\pm$ 0.14), and in Late TR and Late PT over BL in the LA group (Late PT: 0.50 $\pm$ 0.14 deg).

Responder analysis (Tbl. II, Fig. 6) showed a greater number of positive responders and larger median effects in the HA and LA groups across post-training compared to the VC group (**HA:** Early: 16 (1.27 [0.13 2.80]), Late: 14 (2.24 [0.32 5.03]); **LA:** Early: 14 (1.42 [0.06 3.41]), Late: 15 (1.14 [0.05 5.08]); **VC:** Early: 11 (1.08 [0.16 2.24]), Late: 12 (1.00 [0.04 2.30])).

TABLE II
RESPONDER ANALYSIS: MEDIAN EFFECT SIZE AND RANGE.

		High Acceleration		Low Acceleration		Velocity Control	
		Num	Median [range]	Num	Median [range]	Num	Median [range]
GS	Early +	14	2.39 [0.59 6.25]	9	1.74 [0.56 8.69]	14	1.22 [0.01 3.05]
	Early –	5	-0.83 [-2.31 -0.10]	11	-0.72 [-5.29 -0.01]	6	-1.08 [-1.93 -0.14]
	Late +	15	8.36 [1.36 20.91]	14	4.48 [0.03 25.52]	10	3.09 [0.08 16.56]
	Late –	4	-4.40 [-6.88 -1.63]	6	-2.76 [-7.08 -0.81]	10	-4.15 [-7.12 -2.15]
PI	Early +	14	0.69 [0.06 2.36]	15	0.98 [0.07 2.99]	15	0.83 [0.10 3.72]
	Early –	5	-0.69 [-1.75 -0.31]	5	-0.21 [-0.42 -0.01]	5	-0.72 [-1.07 -0.12]
	Late +	16	2.06 [0.31 4.46]	18	0.83 [0.01 5.48]	17	0.75 [0.23 3.34]
	Late –	3	-0.11 [-1.10 -0.00]	2	-0.37 [-0.52 -0.22]	3	-0.25 [-0.73 -0.12]
P. AGRF	Early +	11	1.13 [0.00 1.62]	13	0.92 [0.04 2.34]	17	0.75 [0.06 2.29]
	Early –	8	-0.61 [-1.91 -0.04]	7	-0.49 [-0.87 -0.20]	3	-0.82 [-0.94 -0.55]
	Late +	14	2.98 [0.66 5.35]	17	0.72 [0.12 5.65]	12	1.23 [0.06 2.55]
	Late –	5	-0.87 [-1.53 -0.10]	3	-1.18 [-1.71 -0.58]	8	-0.66 [-1.48 -0.05]
TLA	Early +	16	1.27 [0.13 2.80]	14	1.42 [0.06 3.41]	11	1.08 [0.16 2.24]
	Early –	3	-0.69 [-0.78 -0.65]	6	-0.20 [-1.15 -0.06]	9	-0.64 [-3.68 -0.09]
	Late +	14	2.24 [0.32 5.03]	15	1.14 [0.05 5.08]	12	1.00 [0.04 2.30]
	Late –	5	-0.80 [-1.39 -0.15]	5	-0.83 [-2.07 -0.08]	8	-0.86 [-1.50 -0.45]
SL	Early +	17	1.23 [0.24 3.36]	14	1.38 [0.05 5.09]	11	0.72 [0.07 2.47]
	Early –	2	-0.92 [-1.56 -0.29]	6	-0.16 [-1.32 -0.01]	9	-0.45 [-3.24 -0.14]
	Late +	13	2.78 [1.13 5.37]	11	2.68 [0.27 9.53]	10	1.16 [0.46 2.73]
	Late –	6	-0.63 [-1.12 -0.07]	9	-0.73 [-2.12 -0.07]	10	-0.77 [-1.64 -0.07]
LG	Early +	13	0.74 [0.10. 1.70]	10	0.39 [0.09 2.23]	14	0.40 [0.02 1.69]
	Early –	5	-0.44 [-0.53 -0.11]	9	-0.27 [-0.65 -0.05]	6	-0.30 [-0.91 -0.11]
	Late +	14	0.93 [0.02 3.75]	10	0.55 [0.20 3.03]	11	0.15 [0.01 1.25]
	Late –	5	-0.46 [-0.76 -0.23]	9	-0.30 [-0.83 -0.03]	9	-0.42 [-0.87 -0.04]
MG	Early +	9	0.36 [0.03 0.89]	9	0.18 [0.01 0.82]	12	0.22 [0.01 0.72]
	Early –	9	-0.42 [-0.96 -0.07]	10	-0.58 [-0.75 -0.11]	8	-0.33 [-0.84 -0.11]
	Late +	9	0.30 [0.01. 1.65]	8	0.22 [0.02 1.15]	9	0.36 [0.03 1.01]
	Late –	9	-0.23 [-0.69 -0.02]	11	-0.34 [-1.29 -0.02]	11	-0.37 [-1.35 -0.10]
SO	Early +	14	0.50 [0.06 1.38]	12	0.30 [0.09 1.46]	7	0.57 [0.05 1.20]
	Early –	4	-0.06 [-0.74 -0.01]	7	-0.15 [-0.70 -0.06]	13	-0.23 [-0.84 -0.01]
	Late +	14	0.63 [0.10. 2.95]	11	0.88 [0.15 2.82]	7	0.34 [0.05 2.17]
	Late –	4	-0.14 [-1.45 -0.03]	8	-0.05 [-0.72 -0.00]	13	-0.29 [-1.13 -0.02]
TA	Early +	11	1.00 [0.27 1.21]	11	0.26 [0.10. 0.93]	12	0.31 [0.11 1.24]
	Early –	7	-0.33 [-1.70 -0.12]	8	-0.18 [-0.89 -0.03]	8	-0.24 [-0.78 -0.02]
	Late +	12	0.97 [0.08 2.82]	12	0.45 [0.12 2.22]	11	0.27 [0.04 1.56]
	Late –	6	-0.22 [-0.87 -0.01]	7	-0.35 [-1.47 -0.02]	9	-0.26 [-1.16 -0.05]

2) Stride Length: The model returned a significant fixed effect of experimental phase (Tbl. I, Fig. 5). Across groups, there was a significant increase in stride length in all experimental phases compared to BL (Early TR: 1.63 $\pm$ 0.46 cm, Late PT: 2.49 $\pm$ 0.46 cm, Early PT: 2.14 $\pm$ 0.46 cm, Late PT: 2.47 $\pm$ 0.46 cm). Compared to the average effect across groups, the VC group had a smaller increase in Late PT over BL, that was significantly less than the HA group (**HA**: 3.50 $\pm$ 0.81 cm, **VC**: 1.18 $\pm$ 0.79 cm, **HA vs VC**: $p = 0.040$). Post-hoc testing revealed significant increases in stride length in Late PT over BL in the HA group, and in Late TR, Early PT and Late PT compared to BL in the LA group (3.00 $\pm$ 0.79 cm, 3.02 $\pm$ 0.79 cm, 2.72 $\pm$ 0.79 cm, respectively).

Responder analysis (Tbl. II, Fig. 6) showed a greater number of positive responders and larger median effects in the HA and LA groups across post-training compared to the VC group (**HA:** Early: 17 (1.23 [0.24 3.36]), Late: 13 (2.78 [1.13 5.37]); **LA:** Early: 14 (1.38 [0.05 5.09]), Late: 11 (2.68 [0.27 9.53]); **VC:** Early: 11 (0.72 [0.07 2.47]), Late: 10 (1.16 [0.46 2.73])).

In line with our hypothesis, all groups had significant increases in trailing limb angle and stride length during training, but only the acceleration groups had significant, consistent after effects in push-off posture. As such, modulation in push-off posture in the acceleration groups likely contributed to the measured change in propulsion in both groups.

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D. Muscle Activation

For the ankle-moment component of propulsion, quantified indirectly by EMG activation measured in ankle plantar- and dorsiflexor muscles, we hypothesized that muscle activation would increase in all groups during training, but only the acceleration groups would have sustained change following training. The change in muscle activation across the resampled gait cycle is shown in Fig. S3 in the Supplementary Materials, and full model results are provided in Tbl. S6-S9.

1) Lateral Gastrocnemius: The model returned a significant fixed effect of experimental phase, and a significant interaction between training group and experimental phase (Tbl. I, Fig. 5). Across groups, there was a significant increase in activation in all experimental phases over BL (Early TR: 14.48 $\pm$ 1.89%, Late TR: 11.14 $\pm$ 1.89%, Early PT: 6.00 $\pm$ 1.89%, Late PT: 6.66 $\pm$ 1.89%). The interaction was driven by a greater increase in the HA group compared to both other groups in Early TR over BL (**HA**: 23.11 $\pm$ 3.37%, **LA**: 11.38 $\pm$ 3.28%, **VC**: 8.96 $\pm$ 3.19%, **HA vs LA**: $p = 0.013$, **HA vs VC**: $p = 0.003$), and in Late TR over BL (**HA**: 19.94$\pm$ 3.37%, **LA**: 6.83 $\pm$ 3.28%, **VC**: 6.65 $\pm$ 3.19%, **HA vs LA**: $p = 0.006$, **HA vs VC**: $p = 0.005$), as well as a significantly greater increase in Late PT over BL in the HA group compared to the VC group (**HA**: 12.55 $\pm$ 3.37%, **VC**: 0.67 $\pm$ 3.19%, **HA vs VC**: $p = 0.012$). Post-hoc testing showed significant increases in activation in Early TR, Late TR, and Late PT over BL in the HA group, and in Early TR over BL in the LA group.

Responder analysis (Tbl. II, Fig. 6) showed a similar number of positive responders in all groups, but a larger median effect size in the HA group in Early PT, and larger median effects in both acceleration groups compared to VC in Late PT (**HA:** Early: 13 (0.74 [0.10 1.70]), Late: 13 (0.93 [0.02 3.75]); **LA:** Early: 10 (0.39 [0.09 2.23]), Late: 10 (0.55 [0.20 3.03]); **VC:** Early: 13 (0.43 [0.02 1.69]), Late: 10 (0.20 [0.01 1.25])).

2) Medial Gastrocnemius: The model returned a significant fixed effect of experimental phase (Tbl. I, Fig. 5). Across groups there was a significant increase in activation in Early and Late TR over BL (Early TR: 5.99 $\pm$ 0.99%, Late TR: 3.55 $\pm$ 0.99%). Post-hoc testing showed a significant increase in Early TR over BL in the HA group (7.43 $\pm$ 1.76%).

Responder analysis (Tbl. II, Fig. 6) showed small, equivocal changes in MG activation across groups (**HA:** Early: 9 (0.36 [0.03 0.89]), Late: 9 (0.30 [0.01 1.65]); **LA:** Early: 9 (0.18 [0.01 0.82]), Late: 8 (0.22 [0.02 1.15]); **VC:** Early: 12 (0.22 [0.01 0.72]), Late: 9 (0.36 [0.03 1.02])).

3) Soleus: The model returned a significant fixed effect of experimental phase, and a significant interaction between training group and experimental phase (Tbl. I, Fig. 5). Across groups there was a significant increase in activation in all experimental conditions over BL (Early TR: 7.93 $\pm$ 1.16%, Late TR: 5.99 $\pm$ 1.16%, Early PT: 2.96 $\pm$ 1.16%, Late PT: 5.78 $\pm$ 1.16%). The interaction was driven by a significantly greater increase in Early TR over BL in the HA group compared to the LA group (**HA**: 12.57 $\pm$ 2.06, **LA**: 4.01 $\pm$ 2.01%, **HA vs LA**: $p = 0.003$), and a significantly greater increase in Late PT over BL in the HA group compared to the VC group (**HA**: 9.09 $\pm$ 2.06%, **VC**: 1.38 $\pm$ 1.96%, **HA vs VC**: $p = 0.008$). Post-hoc testing showed significant increases in activation in Early TR, Late TR, and Late PT over BL in the HA group (Late TR: 8.83 $\pm$ 2.06%), and in Early TR over BL in the VC group (7.21 $\pm$ 1.96%).

Responder analysis (Tbl. II, Fig. 6) showed a greater number of positive responders in the HA and LA groups compared to the VC group across post-training (**HA:** Early: 14 (0.50 [0.06 1.38]), Late: 14 (0.63 [0.10 2.95]); **LA:** Early: 12 (0.30 [0.09 1.46]), Late: 11 (0.88 [0.15 2.82]); **VC:** Early: 6 (0.59 [0.05 1.20]), Late: 7 (0.34 [0.05 2.17])).

4) Tibialis Anterior: The model returned a significant fixed effect of experimental phase, and a significant interaction between training group and experimental phase (Tbl. I, Fig. 5). Across groups there was a significant increase in activation in all experimental conditions over BL (Early TR: 7.66 $\pm$ 1.27%, Late TR: 5.09 $\pm$ 1.27%, Early PT: 3.12 $\pm$ 1.27%, Late PT: 5.49 $\pm$ 1.27%). The interaction was driven by a greater increase in Early TR over BL in the HA group compared to the LA group (**HA**: 13.06 $\pm$ 2.25%, **LA**: 2.0 $\pm$ 2.19%, **HA vs LA**: $p = 0.001$), and a greater increase in Late PT over BL in the HA group compared to the VC group (**HA**: 8.49 $\pm$ 2.25%, **VC**: 2.07 $\pm$ 2.13%, **HA vs VC**: $p = 0.042$). Post-hoc testing showed significant increases in Early TR and Late PT over BL in the HA group, and in Early TR over BL in the VC group (7.92 $\pm$ 2.13%).

Responder analysis (Tbl. II, Fig. 6) showed a similar number of positive responders in all groups, but the largest median effect in the HA group in Early and Late PT (**HA:** Early: 11 (1.00 [0.27 1.21]), Late: 12 (0.97 [0.08 2.82]); **LA:** Early: 11 (0.26 [0.10 0.93]), Late: 12 (0.45 [0.12 2.22]); **VC:** Early: 12 (0.31 [0.11 1.24]), Late: 11 (0.27 [0.04 1.56])).

Across groups there were significant increases in activation of ankle plantarflexor muscles (LG, MG, SO) during training. High belt accelerations (7 m/s^2) stimulated greater increases in plantarflexor muscle activation during training, that translated to significantly greater increases in post-training plantarflexor activation (LG, SO). As such, increased ankle moment generation likely contributed to the greater increases in post-training propulsion in the HA group. During training, the velocity matched HA and VC groups had significant increases in dorsiflexor activation (tibialis anterior), but only the HA group had significant post-training effects, consistent with the measured changes in velocity for each group [5].

IV. Discussion

We presented a novel paradigm used to train two components of propulsion during walking (push-off posture and ankle moment) to increase gait speed, based on the application of belt accelerations to the trailing limb during the double support phase of gait. In our protocol, we exposed two groups of healthy, young adults to belt accelerations at different magnitudes, high (7 m/s^2) and low (2 m/s^2), and included a velocity control group that was matched to the change in speed experienced in the high acceleration group.

We hypothesized that our training paradigm would produce after effects in propulsion that increase walking speed in both acceleration groups, but not in the velocity control group. Analysis of self-selected walking speed in a user-driven treadmill condition showed that following training, the HA group

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had an increase in gait speed (0.73 $\pm$ 0.013 m/s) that was significantly greater than both other groups. The LA group had a non-significant increase in gait speed (0.32 $\pm$ 0.013 m/s) that was significantly greater than the VC group. The VC group had no significant change in velocity, and a 50-50 split between positive and negative responders. For both acceleration groups, there was a significant and sustained change in propulsion over the course of the post-training session, while the VC group exhibited no significant change in propulsion. These results support our primary hypothesis that belt accelerations train increases in propulsion that drive increased walking speed.

Change in propulsion could be due to changes in push-off posture and/or ankle moment [2], [4]. We hypothesized that *all groups* would have increased extension of the trailing limb and plantarflexor muscle activation during training, but that *only the acceleration groups* would have sustained change in these propulsive mechanisms post-training. In support of this hypothesis, our analysis of gait kinematics (TLA and SL) showed that all groups increased extension of the trailing limb during training, but significant after effects were present only in the acceleration groups. Analysis of EMG data showed increases in plantarflexor muscle activation during training across all groups, with significantly larger increases in activation in the HA group. Following training, significant increases in plantarflexor muscle activity were sustained only in the HA group. These results suggest that belt accelerations stimulate greater activation of the plantarflexor muscles compared to a matched increase in walking speed. Together, these findings suggest that changes in push-off posture contributed to the increases in propulsion in both acceleration groups, and that the larger increase in propulsion in the HA group may be due to sustained increases in ankle moment generation that were significantly greater in the HA group than the LA group.

Our responder analysis elucidated between group differences in participants' early response to training, and identified gait parameters that may contribute to late changes in GS and propulsion. In the acceleration groups, a greater number of participants had early increases in TLA and Soleus activation immediately following training that were sustained across the post-training session. While a similar number of participants had early increases in lateral gastrocnemius activation across groups, increases in the HA group were almost twice as large as those measured in the other two groups. These results suggest that learned changes in gait kinematics paired with sustained change in the neuro-motor commands sent to plantarflexor muscles contributed to the larger increases in gait speed and propulsion measured at the end of training in our acceleration groups compared to the VC group.

To determine the learning mechanisms driving after effects in our paradigm, we compared gait parameters measured in early and late post-training. After effects of adaptation should manifest as large, immediate effects that decay to baseline behavior before the end of the post-training session as the participant de-adapts [8], [9], [23]. For UDL, we expect to see immediate after effects that are sustained for the duration of the post-training session [14]–[16]. For all gait parameters tested, no immediate after effects reached significance in early PT except for SL in the LA group. Significant change in late PT was measured for PI, peak AGRF, TLA, and SL for both acceleration groups, and in GS and EMG activation for the HA group.

We find both the lack of immediate, significant after effects and the duration of after effects to be incompatible with adaptation. In a split belt locomotor adaptation protocol of similar duration (10 minutes of training, and 6 minutes post-training), after effects in step length, hip extension and EMG activation were all significant immediately following training, but decayed to baseline behavior before the end of the post-training session [9]. As the protocol in [9] used a similar training duration and evaluated after effects in similar gait parameters at similar time points as our study, the discrepancy in observed after effects with our results indicate that behavior in our study is unlikely attributable to adaptation.

Instead we find our results to be most in line with UDL as all after effects 1) occurred in the same direction as the change induced during training, 2) scaled with the magnitude of the repeated movements made during training, and 3) persisted for the duration of the post-training session. The percentage of positive responders in our acceleration groups (70-79%) is in line with previous UDL studies, which have shown the number of movement repetitions influences response rates, and predict response rates between 50-100% given the movement repititions (strides) performed in our study [14], [24]. Additionally, an increase in after effects across the post-training session, as measured in both acceleration groups, has been reported for UDL [15], but not for adaptation.

Typically, immediate after effects of UDL are significant. The lack of immediate after effects following our protocol, and the gradual increase over the course of the post-training session, could be influenced by the design of the UDTC, which limits stride-to-stride changes in gait speed for participant safety. Another possibility is that adaptation during training resulted in after effects in the opposite direction of UDL. While adaptation and UDL are independent learning processes, they are not mutually exclusive [12]. If adaptation resulted in oppositely directed after effects, then the combined after effects of both learning processes would be a gradual increase over the post-training session as the after effects of adaptation decay and unmask the persistent after effects of UDL [12]. Finally, as UDL has been studied primarily in upper extremity tasks [14], [16] it is unclear if the immediacy of after effects reported in these studies should manifest in the same way in locomotion, which relies on different neural circuits [22].

Our current findings are limited by a lack of catch trials within the training session, and by the duration of our training protocol. To definitively determine if UDL is the primary driver of observed after effects, future work could include a pharmacological intervention that blocks UDL [15], a longer post-training duration to better characterize persistence of behavior, and a longer training duration to determine if increased movement repetition increases the rate of positive responders. To determine if adaptation is a contributing factor to measured after effects, future work should incorporate catch trials during training to enable measurement of ongoing adaptation and the expected direction of after effects following training.

Following a single session of training in our protocol, the change in gait speed in the HA group was 0.073 m/s. Current gait training strategies for stroke survivors achieve an average change in gait speed of 0.06 m/s [25]. Minimal clinically significant change in gait speed is 0.16 m/s, and can take up to 36 sessions of training to achieve [22]. While the effects in our study in healthy participants do not directly translate to a patient population, the magnitude of our effects after just one training session are encouraging for future research in a patient population, especially considering that healthy participants are subject to ceiling effects as their baseline is assumed to be roughly optimal. Moreover, previous studies have shown that strength training does not directly translate to functional increases in ankle moment during walking, and have called for rehabilitation methods that target propulsive deficits in the context of walking [1]. Our paradigm has the potential to do just that, by training favorable biomechanical changes in both posture and ankle plantar-flexor muscles during walking, and may provide greater translation to walking speed in the community setting than muscle strengthening alone, or gait therapies that utilize fixed speed treadmills [1], [3], [17], [25].

V. Conclusion

Our paradigm significantly increased gait speed both acceleration groups compared to the velocity control group. Increases in gait speed were driven by increases in propulsion, quantified by propulsive impulse and peak anterior ground reaction force. Changes in push-off posture contributed to increased propulsion in both acceleration groups, while significant increases in ankle moment were observed only in the high acceleration group. After effects were largest in the high acceleration group, while the low acceleration group had smaller, qualitatively similar effects, suggesting that the magnitude of after effects scales with the magnitude of accelerations applied during training. The duration of after effects indicate that change in behavior is likely driven by use dependent learning. Future work will focus on further evaluation of the learning mechanism engaged by our paradigm, and the extension of our work to a patient population or healthy elderly adults.

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