Abstract

Background and Purpose—Previous studies suggest individuals post-stroke can achieve substantial gains in walking function following high-intensity locomotor training (LT). Recent findings also indicate practice of variable stepping tasks targeting locomotor deficits can mitigate selected impairments underlying reduced walking speeds. The goal of this study was to investigate alterations in locomotor biomechanics following three different LT paradigms.

Methods—This secondary analysis of a randomized trial recruited individuals 18–85 years old and >6 months post-stroke. We compared changes in spatiotemporal, joint kinematics and kinetics following up to 30 sessions of high-intensity (>70% heart rate reserve [HRR]) LT of variable tasks targeting paretic limb and balance impairments (high-variable, HV), high-intensity LT focused only on forward walking (high-forward, HF), or low-intensity LT (<40% HRR) of variable tasks (low-variable, LV). Sagittal spatiotemporal and joint kinematics, and concentric joint powers were compared between groups. Regressions and principle component (PC) analyses were conducted to evaluate relative contributions or importance of biomechanical changes to between and within groups.

Results—Biomechanical data were available on 50 participants who could walk ≥0.1 m/s on a motorized treadmill. Significant differences in spatiotemporal parameters, kinematic consistency, and kinetics were observed between HV and HF vs LV. Resultant PC analyses were characterized by paretic powers and kinematic consistency following HV, while HF and LV were characterized by non-paretic powers.

Conclusion—High-intensity LT results in greater changes in kinematics and kinetics as compared to lower-intensity interventions. The results may suggest greater paretic-limb
contributions with high-intensity variable stepping training that targets specific biomechanical
deficits.

Clinical Trial Registration-URL—https://clinicaltrials.gov/. Unique Identifier: NCT02507466

Keywords
gait; rehabilitation; biomechanics

Introduction
Restoration of locomotor function post-stroke is a primary goal of rehabilitation, although reduced walking speeds and abnormal gait patterns often persist for years post-injury. Previous studies suggest specific locomotor training (LT) parameters, including the amount and cardiovascular intensity of task-specific (stepping) practice, can enhance walking speeds\(^1\),\(^2\). Additional research also demonstrated that LT focused on practice of variable stepping tasks directed towards the primary biomechanical subcomponents of upright locomotion that contribute to forward progression (limb-swing, propulsion, stance control or dynamic stability\(^3\)–\(^6\)) results in greater gains in gait speed and endurance than typical interventions\(^7\),\(^8\). A recent study\(^9\) indicated that high-intensity LT performed in variable contexts with focus on these biomechanical subcomponents, but without attempts to normalize kinematics, resulted in gains in gait speed or dynamic stability as compared to variable, low-intensity LT, or high-intensity LT with limited variability, respectively.

Despite these gains, the biomechanical strategies used by patients post-stroke to achieve faster speeds can be concerning for clinicians and patients. For example, asymmetrical limb-loading or altered kinematic patterns are often used to compensate for paretic-limb deficits, which can be energetically inefficient\(^10\) and precipitate musculoskeletal injury\(^11\). Traditional rehabilitation theories\(^12\),\(^13\) discourage use of compensatory patterns during rehabilitation, and conventional interventions often focus on minimizing neurological deficits (i.e., strength and balance), and normalizing kinematics using therapist\(^14\) or robotic-assistance\(^15\). Such therapeutic activities are thought to enhance neurological recovery, defined as restoration of previous neurological function to allow movement patterns similar to able-bodied individuals\(^16\). However, the efficacy of these strategies to improve specific measures of function or neurological recovery are limited. Nonetheless, a major concern of performing high-intensity LT without focus on kinematic patterns is that such practice may result in and reinforce compensatory strategies with repeated training\(^12\),\(^13\), or alternative movement strategies in presence of residual neurological recovery.

An alternative theory is that practice of challenging stepping tasks that require altered volitional commands may result in neuromuscular adaptations that can contribute to enhanced functional and neurological recovery. A large body of literature indicates that individuals with or without neurological injury adapt their neuromuscular strategies in response to various biomechanical or environmental demands that perturb their typical movement patterns\(^17\),\(^18\). For example, split-belt treadmill walking paradigms have been utilized in individuals with step length asymmetry post-stroke to induce perturbations that increase this asymmetry (i.e., magnify errors), although immediate and long-term

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adaptations result in improved spatiotemporal symmetry with removal of the perturbation. However, patients post-stroke demonstrate deficits beyond gait asymmetry that contribute to reduced locomotor function, including difficulty with propulsion, limb swing, stance control and dynamic stability. Pilot studies of high-intensity LT in variable contexts that attempt to address these deficits during practice of challenging stepping tasks has resulted in gains in paretic and non-paretic limb kinematics and kinetics that could be characterized as indicator of recovery and compensation. Whether such training elicits differential changes in movement strategies as compared to other LT strategies, including interventions not focused on specific biomechanical subcomponents of walking or practice at lower intensities, is not clear.

The goal of this study was to investigate training-induced changes in locomotor kinematics and kinetics following three different LT paradigms. In this secondary analysis from a previous randomized clinical trial, we compared changes in spatiotemporal, kinematic and kinetic variables during walking trials following up to 30 sessions of high-intensity variable (HV) training, low-intensity training in variable contexts (low-variable, LV) or high-intensity training focused only on forward walking (high-forward, HF). Gains were analyzed between groups using standard ANOVAs and within groups using principal component (PC) analyses to ascertain the subset of biomechanical variables that best explain the variance of in each training group. With differences in gains in gait speeds with high-intensity training, we anticipated greater improvements in kinetics following either HV or HF vs LV due to greater neuromuscular activation strategies required during high-intensity training. Further, given the attention to specific biomechanical deficits targeted during variable training paradigms, we hypothesized greater changes in specific measures of paretic-limb kinematics and kinetics following HV vs HF training. The result from this study may provide insight into whether attention to specific biomechanical deficits during training may ameliorate abnormal movement strategies in patients post-stroke.

**Methods**

**Participants**

Individuals with chronic (>6 months) hemiparesis post-stroke were recruited, with specific inclusion criteria as follows: 18–85 years old; lower-extremity Fugl-Meyer < 34; overground self-selected velocity (SSV) < 1.0 m/s; and medical clearance to participate. Exclusion criteria included: presence of lower extremity contractures that significantly limited range of motion, significant osteoporosis, cardiovascular, respiratory or metabolic instability, inability to ambulate >150 feet prior to stroke, previous history of peripheral or central nervous system injury, and inability to adhere to study requirements. An additional requirement for evaluation of gait biomechanics during graded treadmill (TM) testing was the ability to walk for ≥1 min at 0.1 m/s with the use of handrails as needed. All participants gave written informed consent and all procedures were approved by the local institutional review board.

**Experimental intervention**

Participants were randomized to receive up to 30 1-hr sessions of either HV, HF or LV training over 2 months, with up to 40 min of stepping per session. Primary training goals for
all groups were to: 1) maximize the amount of successful stepping practice; 2) achieve targeted cardiovascular intensities; and, 3) increase difficulty of walking tasks as tolerated. Targeted HR ranges were determined using age-predicted maximum \[208-(0.7\times \text{age})\] \(^{23}\), with HV or HF targeting 70–80% HR reserve, and LV using 30–40% HR reserve\(^9\).

For HV training, sessions were divided into ~10-min bouts including speed-dependent treadmill training, skill-dependent treadmill training, over-ground training, and stair climbing. Speed-dependent treadmill training was performed with an overhead harness system in case of loss of balance, with goals to increase speeds to reach targeted intensities. Criteria for successful stepping included positive bilateral step lengths, minimal limb collapse, and maintaining upright posture in the sagittal and frontal planes \(^7\). Skill-dependent treadmill training was performed by applying perturbations to challenge postural stability, propulsion, and limb swing, and included walking in multiple directions, over inclines and obstacles, with resisted propulsion provided with elastic bands, weighted vests and leg weights on the paretic limb with limited handrail use as tolerated, or reduced handrail use or body weight support and advance with multi-directional walking to challenge postural stability. Two to 5 different tasks/perturbations were randomly alternated and repeated within 10-minute bouts, with difficulty increased as tolerated. Attention was directed towards task completion rather than normalizing kinematic strategies. Overground training focused on achieving fastest possible speeds or performing variable tasks as described above, with use of a gait belt or overhead mobile or rail suspension system. Stair climbing was performed over static or rotating stairs (Stairmaster, Vancouver, WA) using reciprocal gait patterns and progression to faster speeds and reduced handrail use, use of leg weights or weighted vests to target biomechanical deficits. If specific tasks were not practiced during individual sessions, subsequent sessions focused on missed tasks. For the HF paradigm, intensity was also set to 70–80% HR reserve, although was limited only to forward walking on a TM or overground. Task difficulty was increased by increasing walking speeds within targeted intensities. For the LV paradigm, training sessions were similar to HV training described above but with targeted intensities set to 30–40% HR reserve\(^9\). Across all groups, participants performed 1800–3500 steps/sessions (HF>HV>LV, p<0.01) over 32–38 minutes/session (LV > HV and HF, p<0.01) over 25–29 sessions (p=0.79)\(^9\).

Data collection

Participants performed both overground testing of SSV (ProtoKinetics LLC, PA) and graded TM assessments at baseline (BSL) and post-training (POST). Graded TM testing was performed on a motorized TM with speeds starting at 0.1 m/s for 1 min and increased in 0.1 m/s increments every min with simultaneous 12-lead ECGs and oxygen consumption measures. Testing speed was increased until ACSM criteria for test termination was reached, including significant ECG abnormalities, evidence of gait instability, or the participant refused to continue. The fastest TM speed that participants could walk for 1 min was considered peak TM speed.

Biomechanical data at BSL and POST were collected on an instrumented split-belt motorized TM with speed adjusted to participants’ peak speed achieved during testing. During POST, participants also walked at TM speeds matched to peak speeds achieved at
BSL (MATCH) to control for differences in speed. Data were collected for 30 seconds, beginning ~15 sec after the start of the test to allow for accommodation. The split-belt TM was embedded with bilateral 6 degree of freedom force plates (Bertec Corporation, Columbus, OH) and surrounded by an 8-camera motion capture system (Motion Analysis Corporation, Santa Rosa, CA). Thirty-two reflective markers were placed on bilateral lower limbs using a modified Cleveland Clinic marker set, and kinematic and kinetic data were sampled at 100 Hz.

**Data Analysis**

Marker and force data were processed using Cortex software (Motion Analysis Corp, Santa Rosa, CA), and further analyzed using custom software in Visual3D (C-Motion Inc, Germantown, MD) and MATLAB (Mathworks, Natick, MA). A bilateral 6 degree-of-freedom model of each subject’s lower limbs was created from marker data during static standing. Lower limb inertial properties were estimated based on the subject model and anthropometric measurements of limb positions and joint centers. Marker data were filtered using a low-pass, 2nd order Butterworth filter (10 Hz). Sagittal joint angles were calculated from the transformations between model segments. Sagittal joint moments were calculated from inverse dynamics using low-pass filtered ground reaction force data (2nd order Butterworth at 20 Hz) and joint angle measurements. Sagittal joint powers were calculated as the product of joint moments and angular velocities. Kinetic data were normalized to body weight, and stance was identified as the period when vertical forces >10 N. Kinematic and kinetic data were further normalized to percentage gait cycle (%GC) and average step cycle profiles were created for all complete steps.

Specific kinematic and kinetic variables of interest included specific spatiotemporal measures that describe the walking patterns, and those that may estimate patterns of recovery vs compensation. Spatiotemporal measures of interest included cadence and stride length, as well as paretic single limb stance (%SLS) and step length asymmetry (SLA; paretic /non-paretic step length). Joint kinematic variables included sagittal hip, knee and ankle angles, including peak flexion and extension, total range of motion (ROM), and hip-knee joint coordination, defined by hip-knee phase plots and quantified in terms of stride-to-stride consistency across multiple gait cycles using the average coefficient of correspondence (ACC). The ACC ranges between 0 to 1, with higher values indicating a greater consistency in hip-knee joint coordination during walking. In general, we suggest that greater changes in ROM and hip-knee ACCs in the non-paretic limb may be estimates of neurological recovery, whereas patterns of greater change in the non-paretic limb would be characteristic of compensation.

Kinetic metrics of interest included average positive sagittal ankle, knee and hip powers, which were calculated by integrating joint power within the stride only when values were positive.

\[
\frac{\int_{HS}^{HS} \text{Power}_{\text{joint}}}{\text{stride time}}
\]

Equation 1:
Powers were averaged over specific portions of the gait cycle (GC) consistent with peak power generation (i.e., concentric or positive power) profiles observed during walking. Figure 1 depicts a single-subject example of paretic-limb sagittal joint powers, with both positive and negative powers identified by others previously. As the present study used averaged vs peak joint powers, portions of the gait cycle were identified that encompassed the peaks identified, and powers were averaged over this range. The specific portions of the GC chosen were slightly larger (greater range of %GC) than would be expected in individuals without neurological injury given the variability in neuromuscular coordination of patients post-stroke demonstrated previously. Importantly, only positive powers were averaged during those portions (negative values were not averaged with positive values). Specific phases included positive ankle powers during 40–80% of the GC (A2), associated with concentric plantar flexor activity for propulsion. For knee joints, powers were calculated from 10–50% GC, which is typically associated with knee extension during the primary loading throughout most of single limb stance (K2). Positive hip joint powers were calculated in two separate bins including 0–30% of the GC associated with hip extensor activity following initial contact (H1) and during 50–100% of the GC associated with hip flexor activity prior to and during swing (H3). Other power absorption (negative) phases (i.e., A1, K1, K3-K4, H2, Fig 1) are not included in this analysis. In this analyses, greater gains in joint powers in the paretic vs non-paretic limbs would be indicative of patterns of recovery vs compensation.

**Statistical analysis**

Data are presented as mean ± standard deviation (SD) throughout the text and tables. All variables were checked for normality using the Shapiro–Wilk test, presented as mean (SD) with analyses conducted using SPSS (v22, IBM, Armonk, NY). One-way analyses of variance (ANOVA) and chi-squared analyses were utilized to evaluate BSL differences in demographics, clinical characteristics, and gait biomechanics between groups. Two separate one-way ANOVA analyses were then conducted to compare training-induced changes between groups, including differences in changes at peak TM at BSL and POST and differences in changes at POST with speeds matched to BSL peak speeds (MATCH). Significance was set at $\alpha=0.05$, with post-hoc Bonferroni comparisons.

Stepwise multiple linear regression analyses were used to investigate relationships between changes ($\Delta$) in SSV and peak TM speed from BSL to POST and changes in specific variables of joint kinematics and kinetics across all participants combined as opposed to independent training groups given their smaller sample sizes. Spatiotemporal variables were omitted due to their known contributions to changes in speed and correlation with other kinematic/kinetic parameters.

Considering the inherent interdependency between gait variables, principal component (PC) analyses were applied to 16 specific gait variables (joint kinematics and positive powers) as a method to extract the primary features (i.e., reduce the dimensions) of the training-induced changes with smaller sample sizes. PC analyses were performed separately for each training paradigm to discern potential contributions of biomechanical variables accounting for changes observed with each training paradigm. For each LT group, participants and
variables were arranged in a matrix with gait variables as columns and participants (observations) as rows. The PCs were retained according to the scree plot which delineates individual contribution of each PC to explain the overall variance. The goal was that all the retained PCs together explained ≥80% of the overall variance. To examine the validity and robustness of the calculated PCs, a bootstrapping procedure was conducted of each LT group with 1000 iterations on a random subset of 80% of participants in that group. The factor loadings (the correlation between original gait variables and each PC) and component score coefficients (the contribution or weight of original variables to form each PC) were then cross validated using Pearson’s correlations (r) and significance values. To limit the redundancy of contributing gait variables across the orthogonal PCs and in light of bootstrapping technique to reassure robustness of the executed PCs, a significant threshold of p<0.01 was used to determine significance (vs r-values\textsuperscript{24}) i.e., variables with significant (p<0.01) contributions to the first three PCs were reported.

**Results**

Fifty participants (17 LV, 15 HF, 18 HV) with valid kinematic data were included (Table 1) with no between-group differences in BSL demographics or clinical characteristics. Data from 40 of 90 participants were lost due to loss of marker placement during TM testing (n=22), termination of study participation without POST biomechanical assessments (n=2), inability to walk at least 0.1 m/s at BSL (n=5), or transition of laboratory location (n=11). Eight participants were unable to accurately place each limb on separate TM belts, resulting in 42 participants with valid kinetic data (15 LV, 14 HF and 13 HV). Baseline peak TM speeds within each group were nearly identical to speeds in the full clinical trial\textsuperscript{9}.

Comparison of other demographic and clinical characteristics indicate minimal differences between groups, except for body weight (p=0.02) with differences in gender (p=0.06) and duration post-stroke (p=0.15) approaching significance.

**Changes in gait kinematics and kinetics**

Changes in both SSV [HV:0.14±0.14 m/s; HF:0.18±0.10 m/s; LV:0.02±0.06 m/s] and peak TM speed [HV:0.41±0.10 m/s; HF:0.44±0.17 m/s; LV:0.11±0.13 m/s] were significantly different between groups favoring HF and HV over LV (both p<0.01). Speed-related differences were reflected by consistent differences in cadence and stride lengths (Table 2, both p<0.01). All groups demonstrated improvements in temporal symmetry (i.e., % paretic single-limb stance) or step length asymmetry (SLA), with no significant differences between groups. For MATCH comparisons, there were no differences in spatiotemporal parameters (Supplemental Table).

Evaluation of joint kinematic and kinetic variables at BSL peak TM speeds revealed no differences between groups. Training-induced changes in kinematics indicated limited differences except for changes in intralimb hip-knee consistency (i.e., ACC) in paretic and non-paretic limbs (both p<0.05; Table 2), with differences in non-paretic hip-knee ACC between HF vs LV (Δ=0.08, p<0.05) and paretic hip-knee ACC between HV vs LV approaching significance (Δ=0.05, p=0.05). For MATCH comparison, few differences were
observed (Supplementary Table) except for nonparetic hip-knee ACC (HF > HV; p<0.05),
and non-paretic hip ROM (HV and LV > HF; p<0.05).

For kinetic variables, several training-induced changes were different between groups (Table
2). Results from ANOVAs and post-hoc comparisons revealed significant differences in
bilateral hip power at initial contact (H1) and terminal stance/pre-swing (H3; p<0.01), and
nonparetic ankle power (A2; p<0.001) following HF vs LV paradigms. Nonparetic ankle
propulsive powers were also increased following HV vs LV training (A2; p<0.05), with
additional differences in paretic hip power (H3; p<0.05). Substantial differences in paretic
A2 powers observed following HV and LV training were not significantly different (p=0.06).
For MATCH comparisons, only differences in paretic knee power were significant following
HV vs LV (K2; p<0.05; Supplementary Table).

**Association between gait biomechanics and locomotor function**

Stepwise, multiple linear regression analyses estimated the relative contributions of training-
induced changes in kinematic and kinetic variables to improvements in walking speeds
(ΔSSV and Δpeak TM speed). For all participants, peak TM speeds were positively
associated with nonparetic kinetic and kinematic variables, explaining up to 64% of the
variance. Conversely, ΔSSV were positively related to paretic ankle power with
contributions of non-paretic hip power, also accounting for 47% of the variance.

\[
\Delta \text{peak TM} = 0.014 \times (\Delta \text{nonparetic } A_2) + 1.094 \times (\Delta \text{nonparetic } H_3) + 0.873 \times (\Delta \text{nonparetic ACC}) + 0.13
\]

Equation 2:

\[
\Delta \text{SSV} = 0.009 \times (\Delta \text{paretic } A_2) + 0.006 \times (\Delta \text{nonparetic } H_3) + 0.037
\]

Equation 3:

Subsequently, PC analyses was utilized to extract the primary features of the training-
induced changes in biomechanical variables (joint kinematics and kinetics) within each
group. Analyses of the 16 joint kinematic and kinetic variables revealed 5 robustly
reproducible PCs that accounted for >80% of the variance in each training group. The first
three PCs explained >50% of the variance and are discussed further (Figure 2). For LV
training, PC1 was characterized by paretic ankle ROM and hip power (H1 and H3), as well
as non-paretic H3, PC2 was defined by nonparetic hip-knee ACC and paretic ankle power
(A2), and the third PC was defined by nonparetic hip power (H1). For HF, the first three PCs
were organized differently; PC1 was characterized by nonparetic ACC and A2, and paretic
knee (K2) and hip (H3) power, PC2 was defined by bilateral H1, and PC3 was characterized
by nonparetic hip ROM and H3. For HV, all PCs were from the paretic limb; the first PC
focused on paretic H1 and H3 as well as ankle ROM and PC2 was defined by paretic A2,
with no specific gait variables with significant correlations to PC3.

**Discussion**

The present study detailed changes in locomotor kinematics and kinetics following 3
different LT paradigms in participants post-stroke, revealing consistent differences in
treadmill speed, stride length and cadence between high vs low intensity training. Between
group comparisons of hip-knee ACCs and joint powers indicated more consistent differences between HF and LV in the non-paretic limb, whereas differences between HV and LV appear to favor more paretic limb variables. Subsequent PC analyses revealed PCs loaded on paretic-limb kinetic variables and hip-knee kinematic consistency with HV training. Conversely, PCs in HF and LV were characterized by changes in both non-paretic and paretic limb hip-knee ACCs and specific kinetic variables. The combined findings could provide some insight into patterns of compensation and recovery in patients post-stroke with specific locomotor interventions.

Previous studies detailing differential changes in joint kinematic and kinetics following various training paradigms provided to individuals post-stroke are scarce. While a primary limitation of this study is the small sample size, particularly within each training group, significant between-group differences were nonetheless observed for many variables. Differences in spatiotemporal patterns and kinematic consistency between high- vs low-intensity groups are consistent with previous and preliminary studies and reinforce the notion that high-intensity training does not entrain abnormal kinematic patterns. Rather, such training appears to facilitate more “normal” movement strategies. Analyses of joint powers further suggest the specific methods for delivering high-intensity training may influence neuromuscular strategies. The PC analyses indicate greater loading of paretic-limb changes with HV, which may be indicative improve neurological recovery underlying gains in walking function. Further, greater loading of bilateral limb changes in HF suggest patterns of recovery and compensation underlying locomotor improvements.

The potential significance of these findings may be two-fold. First, strategies that focus on providing large amounts of stepping practice during forward walking at high intensity may result in gains in locomotor function, although improvements may be more dependent on both “normal” and compensatory strategies. A separate conclusion is that HV training that focused on paretic-limb deficits resulted in greater recovery of neuromuscular strategies used prior to injury indicative of neurological recovery. Importantly, compensatory patterns are also observed in all groups (Table 2) consistent with previous results. In specific patients with substantial distal impairments that require AFO, such compensations will be necessary. Nonetheless, the combined findings suggest attention to specific biomechanical locomotor deficits may influence patterns of neurological and functional recovery in patients post-stroke. Namely, attention to specific locomotor deficits that contribute to forward progression (i.e., propulsion, limb swing) appears to elicit gains in those biomechanical deficits with HV training. Further, greater gains in dynamic stability were observed following HV, where very little changes in balance are observed with HF training. The combined findings emphasize the potential significance of performing stepping training in variable context, and further highlight the importance of specificity of training to elicit desired motor outcomes post-stroke.

Additional limitations to the present study include the inability to include additional participants from the training study due to foot placement during testing, or those unable to walk at least 0.1 m/s on the treadmill at BSL. These constraints are related to those with significant impairments in locomotor function and whether these findings can be applied to non-ambulatory patients early post-stroke are not clear. Further, regression analyses on
training subgroups were not possible because of sample sizes, and the use of PC analyses partly overcomes this issue\textsuperscript{28}, although alternative research suggests otherwise\textsuperscript{32}. Additional work is needed to confirm these findings across larger, more diverse patient populations.

**Conclusions**

Providing stepping training at higher intensities resulted in significant gains in spatiotemporal parameters, kinematic consistency and power generation as compared to lower intensity activities. Post-hoc comparisons and PC analyses suggest greater paretic-limb coordination and joint powers were observed following high-intensity training in variable contexts, whereas changes following high-intensity training targeting only walking forward resulted in greater trend of bilateral improvements. The data support the hypotheses that focused attention to paretic limb deficits during variable stepping tasks at high-intensities can result in improved paretic kinematics and kinetics indicative of recovery vs compensation.

**Supplementary Material**

Refer to Web version on PubMed Central for supplementary material.

**Acknowledgements/Sources of Funding**

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**References**


Figure 1.
Single subject data for paretic hip, knee and ankle powers throughout the gait cycle at both baseline (BSL; gray) and post-training (POST; black). The designated portions of the gait cycle (A1–2, K1–4, H1–3) are identified to indicate approximate gait cycle phases when peak joint powers are observed.
Figure 2.
Principle component analyses for each training subgroup, delineating which biomechanical variables contribute to each principle component in A) LV, B) HF, and C) HV. For each training group, the first 3 principle components and the variables that contribute to them are indicated.
Table 1.

Demographic and baseline characteristics.

<table>
<thead>
<tr>
<th></th>
<th>LV (n=17)</th>
<th>HF (n=15)</th>
<th>HV(n=18)</th>
<th>p-values</th>
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</thead>
<tbody>
<tr>
<td>gender (M/F)</td>
<td>10/7</td>
<td>8/7</td>
<td>16/2</td>
<td>0.06</td>
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<td>side of paresis (R/L)</td>
<td>6/11</td>
<td>8/7</td>
<td>4/12</td>
<td>0.32</td>
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<tr>
<td>AFO (yes/no)</td>
<td>13/4</td>
<td>11/4</td>
<td>10/8</td>
<td>0.36</td>
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<tr>
<td>age (years)</td>
<td>54±13</td>
<td>57±11</td>
<td>58±9.6</td>
<td>0.50</td>
</tr>
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<td>weight (kg)</td>
<td>74±17</td>
<td>89±16</td>
<td>92±18</td>
<td>0.02</td>
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<tr>
<td>height (m)</td>
<td>1.7±1.2</td>
<td>1.7±1.1</td>
<td>1.8±1.1</td>
<td>0.78</td>
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<tr>
<td>post-injury duration (mo)</td>
<td>19±18</td>
<td>23±12</td>
<td>42±63</td>
<td>0.15</td>
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<tr>
<td>Fugl-Meyer assessment</td>
<td>23±4.9</td>
<td>23±4.6</td>
<td>23±5.8</td>
<td>0.97</td>
</tr>
<tr>
<td>baseline SSV</td>
<td>0.58±0.23</td>
<td>0.56±0.30</td>
<td>0.51±0.28</td>
<td>0.94</td>
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<td>baseline TM speed</td>
<td>0.85±0.40</td>
<td>0.75±0.39</td>
<td>0.73±0.44</td>
<td>0.62</td>
</tr>
</tbody>
</table>
Table 2.
Comparison of kinematic and kinetic variables at BSL and POST as mean (SD). Changes were compared using one-way ANOVA. P: paretic; NP: non-paretic.

<table>
<thead>
<tr>
<th>Spatiotemporal</th>
<th>LV</th>
<th>HF</th>
<th>HV</th>
<th>p-values (group)</th>
</tr>
</thead>
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<tr>
<td>speed (m/sec)</td>
<td>0.85±0.40</td>
<td>0.96±0.42</td>
<td>1.20±0.40</td>
<td>&lt;0.01; HV/HF&gt;LV</td>
</tr>
<tr>
<td>stride length (m)</td>
<td>0.54±0.26</td>
<td>0.54±0.19</td>
<td>0.60±0.16</td>
<td>&lt;0.05; HV/HF&gt;LV</td>
</tr>
<tr>
<td>cadence (step/min)</td>
<td>97±23</td>
<td>102±21</td>
<td>117±18</td>
<td>&lt;0.05; HV/HF&gt;LV</td>
</tr>
<tr>
<td>SLS (%)</td>
<td>28±5.74</td>
<td>30±5.5</td>
<td>29±8.43</td>
<td>0.63</td>
</tr>
<tr>
<td>SLA (PNP)</td>
<td>1.1±0.42</td>
<td>1.0±0.15</td>
<td>0.97±0.11</td>
<td>0.95</td>
</tr>
</tbody>
</table>

| Kinematics non-paretic | ankle ROM | 26±5.4 | 27±5.7 | 23±4.7 | 26±5.6 | 25±6.1 | 28±5.7 | 0.30 |
| knee ROM | 57±9.3 | 57±8.9 | 51±12 | 55±12 | 57±6.8 | 59±6.5 | 0.07 |
| hip ROM | 43±6.1 | 45±7.2 | 40±8.1 | 44±8.9 | 43±6.3 | 47±6.7 | 0.30 |
| ACC | 0.89±0.07 | 0.90±0.08 | 0.87±0.09 | 0.95±0.06 | 0.90±0.08 | 0.94±0.04 | <0.05; HF>LV |
| Kinematics paretic | ankle ROM | 19±9.4 | 19±7.3 | 18±5.4 | 19±6.7 | 18±4.8 | 19±4.0 | 0.38 |
| knee ROM | 42±9.4 | 40±8.4 | 42±9.5 | 43±14 | 37±12 | 34±11 | 0.36 |
| hip ROM | 37±5.8 | 38±5.5 | 38±6.2 | 38±8.2 | 36±7.5 | 37±6.3 | 0.72 |
| ACC | 0.87±0.06 | 0.88±0.07 | 0.86±0.08 | 0.90±0.07 | 0.84±0.10 | 0.88±0.10 | <0.05; HV>LV |

| Kinetics non-paretic | NP A2 | 15±10 | 16±9.4 | 8.8±7.2 | 19±10 | 10±8.0 | 21±11 | <0.01; HV/HF>LV |
| NP K2 | 3.1±2.9 | 3.9±2.9 | 3.9±4.1 | 3.0±3.6 | 2.9±3.1 | 3.2±2.5 | 0.13 |
| NP H1 | 16±14 | 16±13 | 6.5±5.8 | 20±14 | 8.3±7.6 | 16±9.0 | <0.01; HF>LV |
| NP H3 | 11±8.6 | 13±8.2 | 9.8±7.6 | 21±11 | 8.0±5.8 | 16±11 | 0.01; HF>LV |
| Kinetics paretic | P A2 | 4.3±4.7 | 4.8±4.5 | 4.1±4.6 | 6.8±4.8 | 4.6±6.7 | 10±11 | 0.06 |
| P K2 | 3.8±3.4 | 6.3±5.4 * | 2.8±2.5 | 6.4±4.0 | 4.1±3.3 | 10±8.7 | 0.17 |
| P H1 | 5.9±6.8 | 5.3±4.4 | 4.6±3.9 | 12±10 | 3.3±3.8 | 7.1±7.5 | <0.01; HF>LV |
| P H3 | 9.9±8.5 | 10±9.1 | 6.8±6.0 | 16±10 | 6.4±7.5 | 13±14 | <0.01; HV/HF>LV |