Gait & Posture 62 (2018) 395–404

Contents lists available at ScienceDirect

Gait & Posture

journal homepage: www.elsevier.com/locate/gaitpost

Full length article

Pelvic excursion during walking post-stroke: A novel classification system

Virginia L. Little[a,b], Theresa E. McGuirk[a,b], Lindsay A. Perry[b,1], Carolynn Patten[a,b,c,*]

[a] Neural Control of Movement Lab, Malcom Randall VA Medical Center, Gainesville, FL 32608, USA
[b] Brain Rehabilitation R&D Center (151A), Malcom Randall VA Medical Center, 1601 SW Archer Rd., Gainesville, FL 32608-1197, USA
[c] Department of Physical Therapy, College of Public Health & Health Professions, University of Florida, Gainesville, FL 32610-0154, USA

ARTICLE INFO

Keywords:
Stroke
Gait
Pelvis
Biomechanics
Hemiparesis
Classification

ABSTRACT

Background: Researchers and clinicians often use gait speed to classify hemiparetic gait dysfunction because it offers clinical predictive capacity. However, gait speed fails to distinguish unique biomechanical characteristics that differentiate aspects of gait dysfunction.

Research question: Here we describe a novel classification of hemiparetic gait dysfunction based on biomechanical traits of pelvic excursion. We hypothesize that individuals with greater deviation of pelvic excursion, relative to controls, demonstrate greater impairment in key gait characteristics.

Methods: We compared 41 participants (61.0 ± 11.2yrs) with chronic post-stroke hemiparesis to 21 non-disabled controls (55.8 ± 9.0yrs). Participants walked on an instrumented split-belt treadmill at self-selected walking speed. *Pelvic excursion* was quantified as the peak-to-peak magnitude of pelvic motion in three orthogonal planes (i.e., tilt, rotation, and obliquity). Raw values of pelvic excursion were compared against the distribution of control data to establish deviation scores which were assigned bilaterally for the three planes producing six values per individual. Deviation scores were then summed to produce a *composite pelvic deviation score*. Based on composite scores, participants were allocated to one of three categories of hemiparetic gait dysfunction with progressively increasing pelvic excursion deviation relative to controls: Type I (n = 15) – minimal pelvic excursion deviation; Type II (n = 20) – moderate pelvic excursion deviation; and Type III (n = 6) – marked pelvic excursion deviation. We assessed resulting groups for asymmetry in key gait parameters including: kinematics, joint powers temporally linked to the stance-to-swing transition, and timing of lower extremity muscle activity.

Results: All groups post-stroke walked at similar self-selected speeds; however, classification based on pelvic excursion deviation revealed progressive asymmetry in gait kinematics, kinetics and temporal patterns of muscle activity.

Significance: The progressive asymmetry revealed in multiple gait characteristics suggests exaggerated pelvic motion contributes to gait dysfunction post-stroke.

1. Introduction

Gait speed, step asymmetry, metabolic cost, and muscle activity patterns during walking have all been used to classify hemiparetic gait dysfunction [1–5]. Often, researchers and clinicians use gait speed changes to assess treatment-related improvements without regard to the neuromechanical walking pattern. On this background, hemiparetic gait dysfunction is only moderately responsive to treatment [6,7].

Gait speed offers myriad advantages as a classification variable [8,9]. However, speed-based classifications of gait dysfunction are inherently limited, most notably by lack of specificity. In contrast, a gait classification system based on walking mechanics is likely to reveal underlying impairments of gait dysfunction post-stroke.

Biomechanical investigations of gait dysfunction typically focus on deviations in kinematics and kinetics of lower extremity joints with little attention to motion at the pelvis.[2] Given the critical role of the pelvis as a structural link between the trunk and the lower extremities, pelvic excursion[3] is a logical biomechanical focus for investigating hemiparetic gait dysfunction [10]. Indeed, biomechanical deviations of pelvis motion, including exaggerated frontal (i.e., lateral tilt and

* Corresponding author at: Brain Rehabilitation Research Center (151A), Malcom Randall VA Medical Center, 1601 SW Archer Rd., Gainesville, FL 32608, USA.
E-mail addresses: ginnylittle@phhp.ufl.edu (V.L. Little), tmcguirk@phhp.ufl.edu (T.E. McGuirk), lperry@usa.edu (L.A. Perry), patten@phhp.ufl.edu (C. Patten).
[1] Present address: Department of Physical Therapy, University of St. Augustine, 1 University Blvd., St. Augustine, FL 32086, USA.
[2] Pelvis, as used throughout the text, refers to the bony structure that consists of bilateral ilia, ischia, pubi, the sacrum, and the coccyx.
[3] Pelvic excursion refers to motion of the bony pelvis.

https://doi.org/10.1016/j.gaitpost.2018.03.052
Received 1 December 2017; Received in revised form 9 March 2018; Accepted 30 March 2018
0966-6362/ Published by Elsevier B.V.

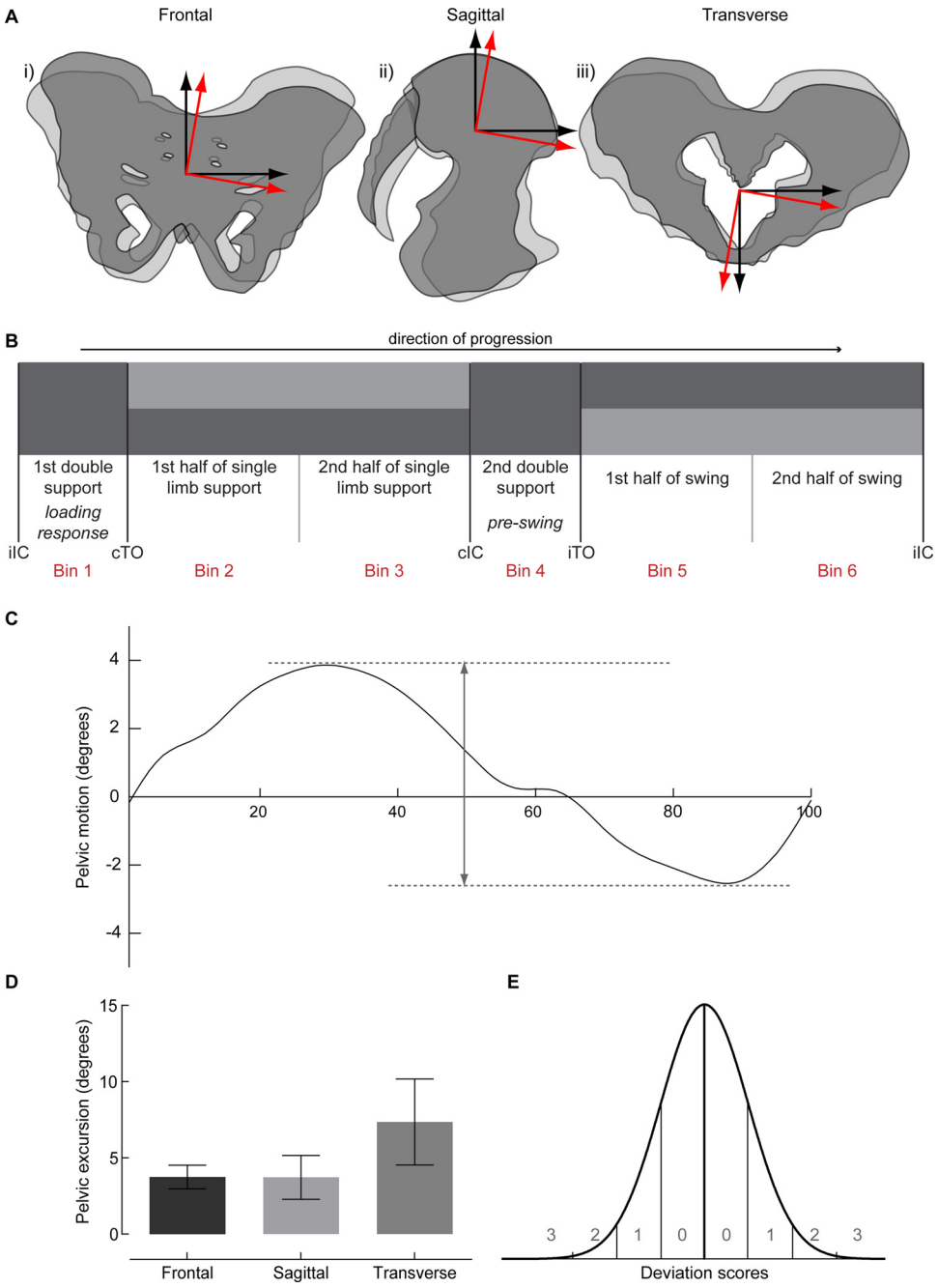

Fig. 1. Quantification of pelvic excursion. A) *Pelvic motion*. Darker structures and red arrows illustrate pelvic rotation with respect to anatomic orientation (lighter structures) within i) frontal, ii) sagittal, and iii) transverse planes with respect to the lab reference frame (black arrows). Pelvic motion was quantified across the gait cycle for each individual, in each plane. B) *Gait cycle*. We parse the gait cycle in 6 biomechanically relevant phases using the following gait events: ipsilateral initial contact (iIC), contralateral toe off (cTO), contralateral initial contact (cIC), and ipsilateral toe off (iTO). The first and second halves of single limb support and swing are delineated at the mid-point between the two adjacent gait events. Darker shading represents periods of foot-floor contact; lighter shading represents periods of reference limb swing. C) *Pelvic excursion*. Single-subject data from the transverse plane illustrated here. To establish normative data, we first measured pelvic excursion bilaterally in healthy controls and quantified the peak-to-peak magnitude of pelvic motion (degrees) throughout the gait cycle in each of the three orthogonal planes. D) *Reference data from controls*. Subsequently, raw values of pelvic excursion for healthy controls and participants post-stroke were compared against the distribution of these reference control data. E) *Deviation scores*. Pelvic deviation was defined in each plane by determining the number of standard deviations between an individual raw value and the reference control mean for a specific plane of motion, effectively computing a z-score truncated at the whole number. Thus, raw values > 1 but < 2 sd from the reference control mean produced a deviation value = 1. It follows that raw values ≥ 2 but < 3 sd yield a deviation value of 2, and raw values ≥ 3 sd a deviation value of 3.

displacement) and transverse plane (i.e., rotation) motion occur following stroke [11,12].

Despite recognition of pelvic motion impairments, our understanding of how they relate to other biomechanical gait impairments following stroke remains limited. In health, we recognize 3-dimensional motion of the pelvis decreases energy cost by minimizing vertical center of mass excursion during walking [10,13]. Excessive pelvic motion in any direction could increase the metabolic cost of walking, a well-recognized sequela following stroke [13–15]

Evidence suggests regaining control of pelvic motion may lead to better gait outcomes. For instance, individuals post-stroke who improve overground walking speed following intervention also reveal kinematic changes suggestive of reduced anterior pelvic tilt [7]. However, whether control of pelvic motion during walking is a prerequisite to or by-product of improved walking function remains unknown.

Here we report results of an exploratory analysis aimed at developing a classification scheme for hemiparetic gait dysfunction based on the magnitude of pelvic excursion deviation. We investigated differences between controls and pelvic excursion deviation categories derived from the classification. We examined neuromechanical differences by leg and quantified the magnitude of asymmetry between legs to determine if asymmetries were larger for groups with more pelvic excursion deviation.

2. Methods

2.1. Participants

Data presented here represent a secondary analysis from a cross-sectional study investigating the immediate effects of locomotor training parameters. Twenty-one healthy controls (age: 55.8 ± 9.0yrs) and 41 individuals post-stroke (age: 61.0 ± 11.2yrs; chronicity:

70.5 ± 64.0mo) participated. We recruited independent ambulators with hemiparesis due to a single, unilateral hemispheric stroke (confirmed with neuroimaging). All procedures described herein were approved by the local Institutional Review Board and conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent before participation.

2.2. Instrumentation and data processing

Participants walked on an instrumented split-belt treadmill (Bertec, Columbus, OH), that measured 3-dimensional ground reaction forces (sampling frequency: 2000 Hz). We recorded marker data using 12 infrared cameras (Vicon MX, Vicon Motion Systems Ltd., Oxford, UK; sampling frequency: 200 Hz), a modified Helen Hayes marker set (41 single reflective markers and 11 rigid clusters), and surface electromyograms (EMG) using active, pre-amplified electrodes (MA-311, Motion Lab Systems, Baton Rouge, LA; sampling frequency: 2000 Hz) placed bilaterally over the motor point of eight lower extremity muscles (medial gastrocnemius (MG), soleus (SO), tibialis anterior (TA), gluteus medius (GM), biceps femoris (BF), semimembranosus (SM), rectus femoris (RF), and vastus medialis (VM)). Participants wore a safety harness (Robertson Mountaineering, Henderson, NV) for fall arrest while walking without upper extremity support.

We collected, labeled, and reduced marker data using Vicon Nexus (Vicon Motion Systems Ltd., Version 1.6.1, Oxford, UK), modeled using Visual 3D (C-Motion, Version 4.82.0, Germantown, MD), and processed with custom Matlab (The MathWorks, Version 7.7.0 R2008b, Natick, MA) scripts to calculate joint kinematics. We calculated joint kinetics using inverse dynamics. Kinematic and kinetic data were lowpass filtered (Butterworth, 4th order, 6 Hz and 10 Hz cutoff, respectively). EMG data were demeaned and bandpass filtered (Butterworth, 4th order, 20 Hz–450 Hz) with a 60 Hz notch filter applied to remove electrical noise (~roll-off: −49.315 dB/Hz). Filtered EMG signals were rectified and smoothed with a bi-directional lowpass filter (Butterworth, 4th order, 20 Hz cutoff). All motion data were time-normalized to the gait cycle. On average, 30 ± 8 step cycles per leg were used to calculate gait variables for each participant.

2.3. Clinical data

We assessed the motor component of the Lower Extremity Fugl-Meyer (LE FM) [16,17] and measured self-selected (SSWS) and fastest-comfortable (FCWS) overground walking speeds with an instrumented walkway (GaitRite, Version 3.9, Havertown, PA). Participants post-stroke walked without customarily worn ankle foot orthoses. If necessary, we provided an Aircast (DJO, Vista, CA) to prevent excessive ankle motion (n = 6) and allowed assistive device use for safety (n = 4).

2.4. Key gait parameters

We quantified pelvic excursion as the peak-to peak magnitude of pelvic motion in three orthogonal planes (i.e., tilt, rotation, and obliquity; Fig. 1) to build a classification system based on control of pelvic motion (described below). Once participants were classified, we investigated the pattern of asymmetry in the following key gait parameters across groups.

2.4.1. Kinematics

We analyzed six kinematic characteristics: peak hip extension (magnitude and timing), peak knee flexion (magnitude and timing), knee angle at initial contact (i.e., 0%GC), and ankle angle at terminal swing (i.e., 99%GC).

2.4.2. Kinetics

We analyzed the magnitude and timing (%GC) of the three joint powers (i.e., product of net joint moment and angular velocity) contributing to forward limb progression at the stance-to-swing transition: concentric ipsilateral ankle plantarflexor power in pre-swing (iA2), concentric contralateral hip extensor power in early stance (cH1), and concentric ipsilateral hip flexor power occurring throughout pre-swing and initial swing (iH3) [7,18].

2.4.3. Muscle activity

We calculated the proportion of integrated EMG (iEMG) in each phase of gait (Fig. 1) relative to the total iEMG over the cycle. Thus, iEMG identifies temporal differences in EMG activation across the cycle, but does not inform regarding EMG amplitude. For each muscle, we investigated iEMG within the bin revealing peak proportional activity in controls, specifically: loading response for the VM, RF, TA, and GM; the second half of single limb support for the plantarflexors; and the second half of swing for the hamstrings [10]. In addition, we investigated pre-swing for the TA given the prevalent claim that 'so-called' foot drop is a primary concern after stroke [19].

2.5. Pelvic classification

To establish normative data, we measured pelvic excursion bilaterally in healthy controls. Subsequently, pelvic excursion values for healthy controls and participants post-stroke were compared against the distribution of these normative data to establish deviation scores. Deviation scores were assigned bilaterally for the three planes producing six values per individual, which were summed to produce a composite pelvic deviation score (Fig. 2). The maximum composite score observed in controls was used as the initial delimiter then doubled to establish a second criterion, creating three categories of progressively increasing pelvic excursion deviation (PED) in hemiparetic individuals relative to controls as follows: Type I – minimal pelvic excursion deviation; Type II – moderate pelvic excursion deviation; and Type III – marked pelvic excursion deviation.

Following classification, we evaluated limb asymmetry for key gait parameters described above. Finally, we investigated the presence of a systematic relationship between the extent of pelvic deviation and limb asymmetry using a two-pronged approach: 1) including controls to determine the overall pattern of asymmetry and 2) excluding controls to identify patterns specific to the stroke PED groups.

2.6. Statistical analysis

Limb asymmetry (i.e., difference between legs), was the primary outcome for the key gait parameters. PED group was defined as an ordinal variable (*i.e.*, 0 = control, 1 = Type I, 2 = Type II, 3 = Type III). To test for the presence of either a linear or quadratic increase in asymmetry, we used a least squares model (covariates: age, sex, treadmill speed) to test for differences in the mean limb asymmetry across PED groups. We refer to increased magnitude of difference between legs across PED groups, from Controls (if included) to Type III, as 'progressive asymmetry'. We investigated differences in clinical data between groups with either a Kruskal-Wallis Test or a 1-way ANOVA and used descriptive statistics to evaluate demographics. To control for Type I error, statistical significance was established at $p < 0.05$ for all variables. We analyzed clinical data with JMP Pro software (Version 11.0.0, SAS Institute Inc., Cary, NC) and all other data with SAS software (Version 9.3, SAS Institute Inc., Cary, NC).

3. Results

3.1. Overview

Self-selected gait speed and age were similar between stroke PED groups. FM scores revealed greater motor impairment for participants with the most pelvic excursion deviation. Also, we noted progressive

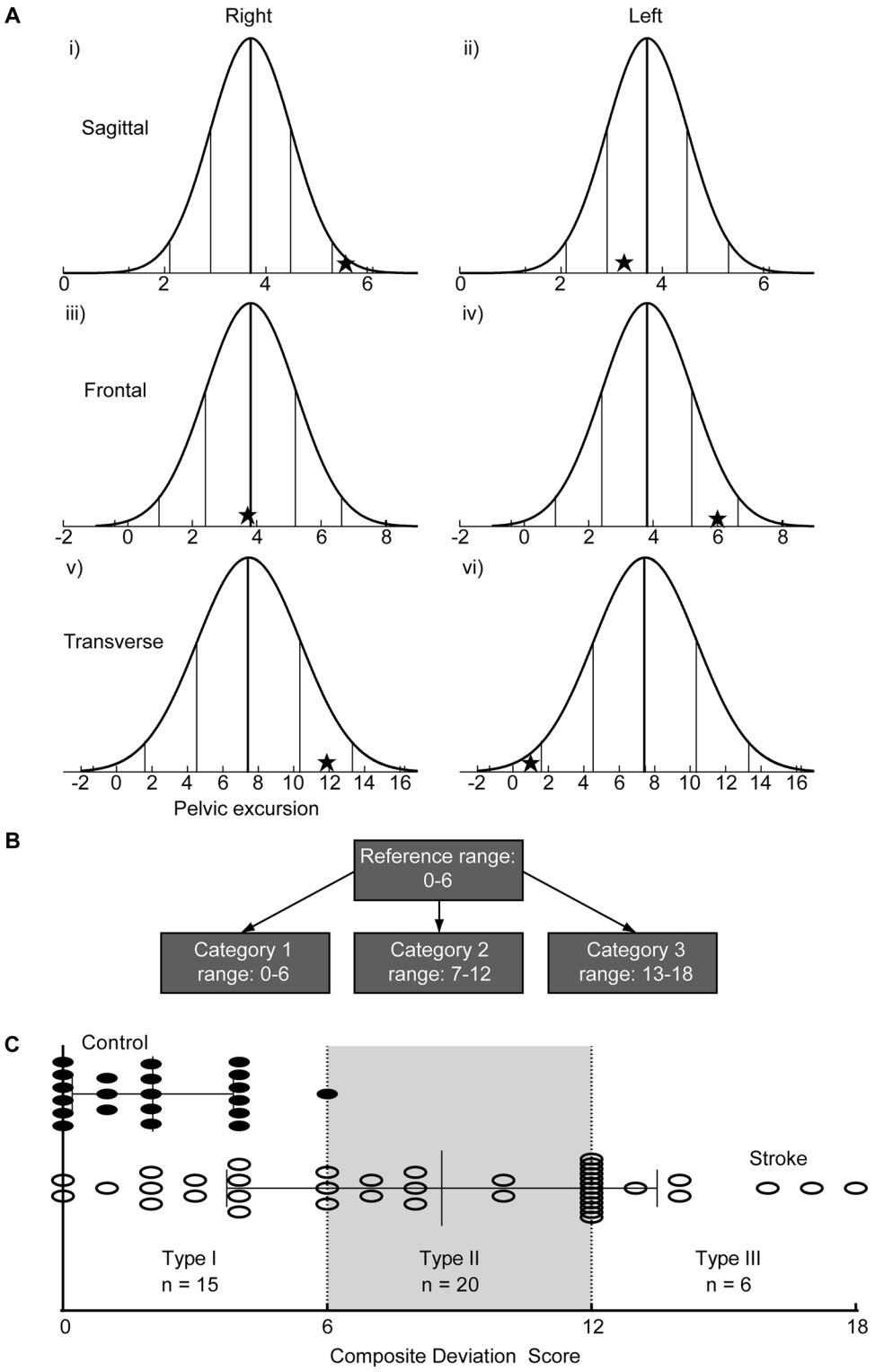

Fig. 2. Pelvic excursion deviation score. A) *Comparison between individual pelvic excursion values and the control mean to determine pelvic excursion deviation*. The distribution of composite pelvic deviation scores from healthy controls was used to establish criteria for classification of pelvic deviation in persons post-stroke. Control distributions of pelvic excursion are illustrated for both legs and the respective planes of motion (i–vi). Individual values of pelvic excursion (x-axis) for each leg and plane of motion were assigned a truncated z-score (i.e., rounded to whole number) corresponding to 0–1, 1–2, or 2–3 standard deviations (noted by vertical lines) from controls. Deviation values were assigned for each leg in all three planes producing six values per individual, which were summed to produce a *composite pelvic deviation score*. We illustrate pelvic excursion deviation values (★) for a hypothetical case: i) R, sagittal = 2, ii) L, sagittal = 0, iii) R, frontal = 0, iv) L, frontal = 1, v) R, transverse = 1, vi) L, transverse = 2, summing to produce a composite deviation score of 6. B) *Schematic illustrating use of the control range to determine the criterion composite deviation values to distinguish between categories*. The maximum composite score observed in controls was used as the initial delimiter. The maximum control score was doubled to establish a second criterion. C) *Distribution of composite deviation scores in our sample*. Combined, this convention established three categories of hemiparetic gait dysfunction with progressively increasing pelvic excursion deviation relative to controls: Type I – considered to have minimal pelvic excursion deviation, produced a composite deviation score less than or equal to controls; Type II – moderate pelvic excursion deviation, produced composite deviation scores greater than the first, but less than the second, delimiter; and Type III – considered to have marked pelvic excursion deviation, produced composite deviation scores exceeding twice the maximal pelvic excursion observed in controls. Closed and open circles denote controls and participants post-stroke, respectively. The dotted lines represent the primary (composite score = 6) and secondary (composite score = 12) criteria used to distinguish pelvic deviation categories.

asymmetry across multiple biomechanical variables, including: kinetics, kinematic magnitude and timing, and iEMG. Progressive asymmetry was most prominent in the magnitude and timing of kinematics.

3.2. Biomechanically based gait classification identifies three stroke groups

Mean composite pelvic excursion score was 2.1 (range 0–6) for controls and 8.6 (range 0–18) for participants post-stroke (Fig. 2C). Based on the range of composite pelvic excursion scores in controls, the primary and secondary delimiters used to classify pelvic deviation were 6 and 12, respectively. Thus, we categorized participants post-stroke into three groups: Type I (n = 15), Type II (n = 20) and Type III (n = 6).

3.3. Clinical data: progressive deficit in motor impairment but not gait speed

3.3.1. Demographics

All groups were of similar age (Control: 55.8 ± 9.0yrs, Type I:

64.9 ± 8.2yrs, Type II: 59.4 ± 9.7yrs, Type III: 56.2 ± 19.5yrs; $F_{(3,58)} = 2.47$, $p = 0.07$). Our sample was predominantly male (n = 42); this relationship carried over to each of the groups: Type I: 10/15, Type II: 16/20, Type III: 5/6, Control: 11/21. Stroke chronicity was similar across stroke groups (Type I: 58.3 ± 65.6mo, Type II: 83.0 ± 61.7mo, Type III 59.7 ± 71.1mo; $p = 0.49$). Hemiparetic side was relatively evenly distributed within each stroke group (R/L – Type I: 7/8, Type II: 11/9, Type II: 4/2).

3.3.2. Gait speed

All stroke groups walked slower than controls (SSWS: 1.3 ± 0.2m/s, $F_{(3,58)} = 70.52$, $p < 0.0001$) but no differences in self-selected overground gait speed (Type I: 0.7 ± 0.2m/s, Type II: 0.6 ± 0.2m/s, Type III: 0.5 ± 0.2m/s; p's > 0.05) were revealed between stroke PED groups. Interestingly, we detected differences among participant categories in fastest-comfortable walking speed. Again, all stroke participant groups revealed slower FCWS than controls (FCWS: 1.9 ± 0.2m/s, $F_{(3,58)} = 52.28$, $p < 0.0001$). Type II (0.8 ± 0.2m/s) walked slower ($p < 0.05$) than Type I (1.1 ± 0.4m/s). However, FCWS in Type III (0.9 ± 0.6m/s) was not different from either Type I or Type II (p's > 0.05).

3.3.3. Lower extremity motor impairment

Type II (22.5/34 ± 7) and Type III (20/34 ± 10.5) revealed lower FM scores than Type I (27/34 ± 8; $X^2_{(2)} = 8.67$, $p = 0.01$), indicating greater motor impairment. However, Type II and Type III did not differ.

3.4. Gait asymmetry corresponds with pelvic excursion deviation

3.4.1. Kinematics

Peak hip extension and peak knee flexion revealed linear increases in the magnitude of asymmetry across groups, such that Type III revealed the greatest asymmetries (Fig. 3). Similarly, we found a progressive asymmetry for peak hip extension timing and peak knee flexion timing (Fig. 4). The knee angle at initial contact did not reveal a progressive asymmetry (p's > 0.05; not illustrated). However, the ankle angle at terminal swing revealed a linear progressive asymmetry among groups post-stroke ($p = 0.04$), with a quadratic increase noted when compared against controls ($p = 0.04$; Fig. 3F).

3.4.2. Kinetics

Progressive asymmetry was detected across all groups for the concentric hip extension power (H1; Fig. 5A) produced in loading. Neither the concentric ankle plantarflexor power in pre-swing (A2; Fig. 5C) nor the concentric hip flexor power in early swing (H3; Fig. 5E) (all p's > 0.05) revealed a statistically significant progressive asymmetry. Fig. 5C (A2) and 5E (H3) illustrate increases in the calculated asymmetries from Type I to Type III that fail to reach statistical significance, likely due to high variability among the Type II individuals.

No progressive asymmetry was detected for the timing of peak power production for ankle plantarflexion (A2), hip extension (H1) or hip flexion (H3; all p's > 0.05).

3.4.3. Temporal patterns of muscle activity

Progressive asymmetry between legs was detected in loading response for both TA and RF (Fig. 6). While pre-swing revealed no progressive asymmetry in the tibialis anterior (p's > 0.05), all groups post-stroke revealed exaggerated paretic TA activity.

Progressive asymmetry was not detected for all other muscles whether controls were included in the analysis or not (all p's > 0.05). However, we note marked disorganization in the timing of bilateral plantarflexors (Supplement 1) and more subtle changes to bilateral hamstrings (Supplement 2) activity patterns.

4. Discussion

Here we developed a classification scheme for hemiparetic gait dysfunction based on pelvic excursion deviation in individuals post-stroke. Importantly, SSWS was similar between stroke groups exhibiting increasing magnitude of pelvic excursion deviation, but the relevant biomechanical and physiologic measures revealed progressive asymmetry. Our results emphasize the ability of biomechanical measures to elucidate mechanisms underlying hemiparetic gait dysfunction. Future work that investigates remediating these impaired mechanisms could lead to targeted treatment strategies.

4.1. Clinical gait assessments provide limited insight

Researchers predominantly use speed-based measures of gait to classify and predict walking function following stroke [2,20]. However, many factors contribute to gait speed, so on its own it is a non-specific outcome variable [2,20,21]. Speed-based classifications may confirm walking function is compromised, but fail to inform how or why. Gait speed similarities across stroke groups strengthen our argument that classification based on pelvis excursion deviation provides additional insight unavailable through speed-based classifications.

Such limitations of speed-based classification schemes have prompted others to classify the hemiparetic gait pattern itself [22]. Cluster analysis revealed distinct gait patterns characterized primarily by gait speed and paretic knee kinematics [22]. Other work revealed a disruptive influence by the nonparetic leg on normal phasing of paretic muscle activity during locomotion [23]. Therefore both paretic and nonparetic limb data are pertinent to incorporate in analyses of hemiparetic gait dysfunction [24]. Furthermore, the kinetic variables used in the current study were pre-selected based on their known roles in forward progression, a functional phase requiring unloading of the trailing limb with subsequent loading of the leading limb. The data investigated here provide additional insight regarding biomechanical impairments during this critical phase of the gait cycle as body weight transitions between support limbs in the presence of excessive pelvic excursion. While deficits in A2 and H3 power production are observed bilaterally, asymmetrical power production is most evident in H1, the stabilizing hip extensor power during loading response. Exaggerated pelvic excursion may contribute directly to the inability of the hip extensors to produce sufficient power during paretic loading response.

4.2. Insights gained from biomechanical analyses

Quantification of pelvic excursion incorporates tri-axial pelvic rotation based on the rationale that pelvic rotations decrease energy cost by minimizing the vertical excursion of the center of mass in normal gait [10,13]. Excessive motion about any of the three axes could significantly increase the metabolic cost of walking, a well-recognized sequela following stroke [13–15]. Exaggerated motion of the pelvis is documented following stroke [11,12], yet the relationship between impairments at the pelvis and other biomechanical impairments distal to the pelvis remain unclear.

Perry described two functional units of the body during gait [10]. As part of the passenger unit, the pelvis stabilizes the head, arms and trunk over the support leg. Alternatively, as part of the locomotor unit, the pelvis is paramount to controlling external forces imposed by foot-floor contact. These external forces create moments about each lower extremity joint. As the proximal component of the hip joint, particular demand is placed on the pelvis to counteract the moment of inertia produced by the mass of the trunk, while simultaneously controlling forces arising from foot-floor contact. Assessment of pelvic excursion therefore encompasses potential stability limitations from forces originating both superior and inferior to the pelvis. In addition to impairments of pelvic rotation, individuals post-stroke frequently demonstrate increased lateral pelvic displacement [12,25,26]. Lateral pelvic

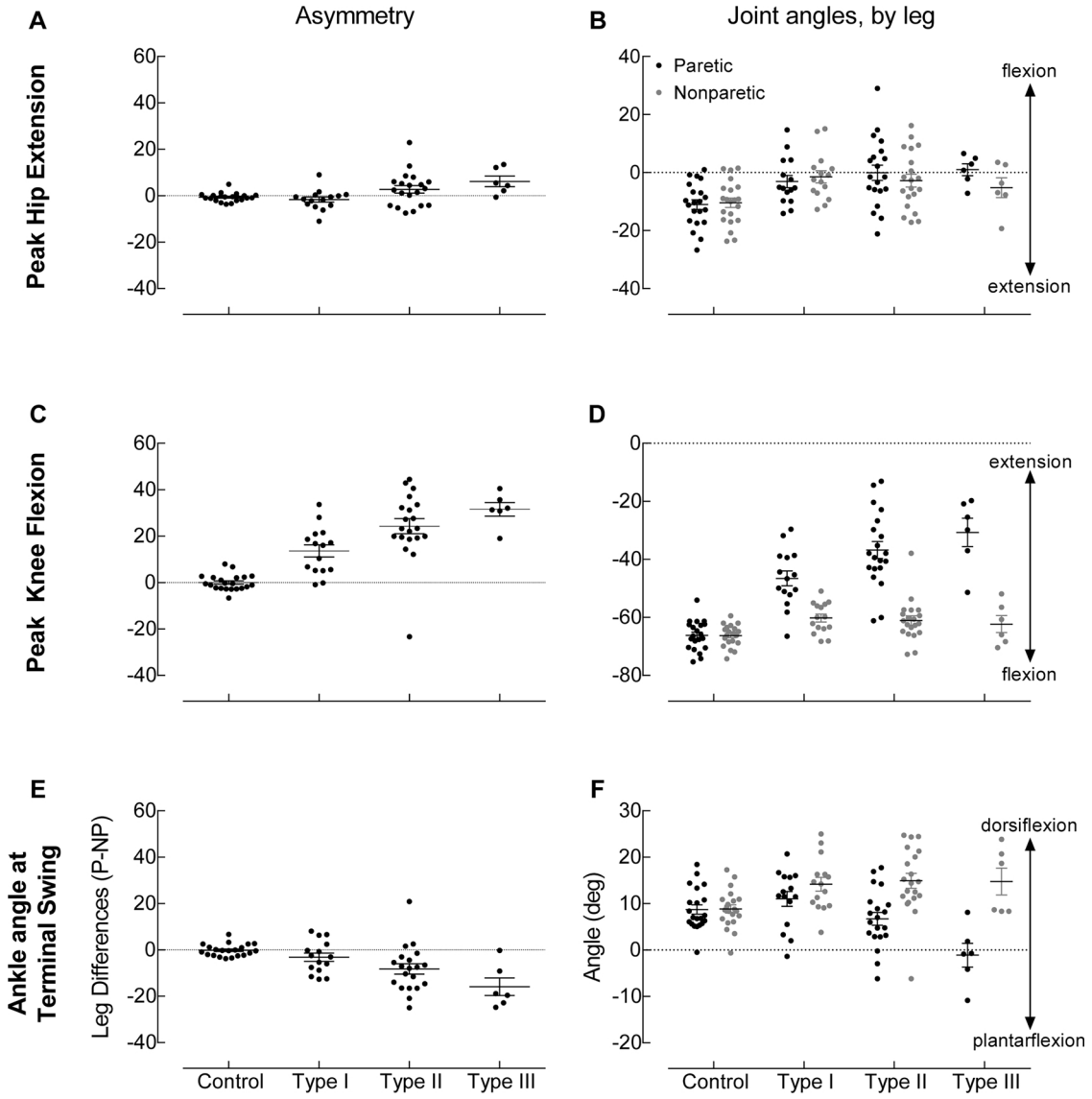

Fig. 3. Progressive asymmetries detected in kinematic *magnitude.* The left column illustrates calculated kinematic asymmetry between paretic and nonparetic legs (P-NP) for (A) peak hip extension, (C) peak knee flexion, and (E) ankle angle at terminal swing. The dotted line at zero indicates symmetry. The right column illustrates raw kinematic values by leg for (B) peak hip extension, (D) peak knee flexion, and (F) ankle angle at terminal swing. Paretic and nonparetic legs depicted by black and grey circles, respectively. The left leg was experimentally assigned as the paretic leg in controls. A dotted reference line is provided at neutral (0° flexion/extension) for ease of interpretation. Notably, the linear increases in asymmetry detected for peak hip extension and peak knee flexion were present whether controls were included (p's < 0.01) in the analysis or not (p's $= 0.02$). While the factor driving differences in peak hip extension is unclear, the differences in peak knee flexion appear to be driven by paretic leg impairment (D). Similarly, the differences contributing to ankle angle asymmetry at terminal swing appear to be driven by the paretic leg following stroke (F). Data are mean ± standard error of the mean with each subject represented individually to illustrate group distributions.

displacement was not included in the current classification, though it is likely linked to pelvic rotation in the frontal plane (e.g., pelvic drop or Trendelenburg) [10].

More recently, reductions in anterior pelvic tilt during walking were associated with better gait-related outcomes following intervention for hemiparetic gait dysfunction [7]. While it is not possible to infer causation from these data, they suggest the importance of reestablishing control of pelvic motion during walking as a prerequisite to positive response to walking-related interventions following stroke. Given the progressive asymmetries we revealed in several kinematic, kinetic and physiologic variables concurrent with exaggerated pelvic excursion, we hypothesize interventions that reestablish control of pelvic motion during walking will also be likely to improve the asymmetries noted here.

4.3. Interventions to remediate underlying impairments of gait dysfunction

We hypothesized that classifying walking mechanics would reveal underlying impairments of hemiparetic gait that could be used to design specifically targeted rehabilitation interventions. Indeed, our Type III PED group, who revealed the most pelvic excursion, also revealed the greatest asymmetry in several gait characteristics, including: timing and magnitude of peak hip extension and peak knee flexion, ankle angle at terminal swing, and concentric hip extension power (H1). The Type III PED group would likely benefit most from impairment level training targeting power production of the paretic hip extensors and ankle plantarflexors to improve H1 and knee flexion during pre-swing. Consistent with this premise, recent work shows a mediolateral corrective force directed toward the pelvis on the paretic side early in the stance phase can improve paretic leg muscle activation and improve pelvic displacement symmetry [27].

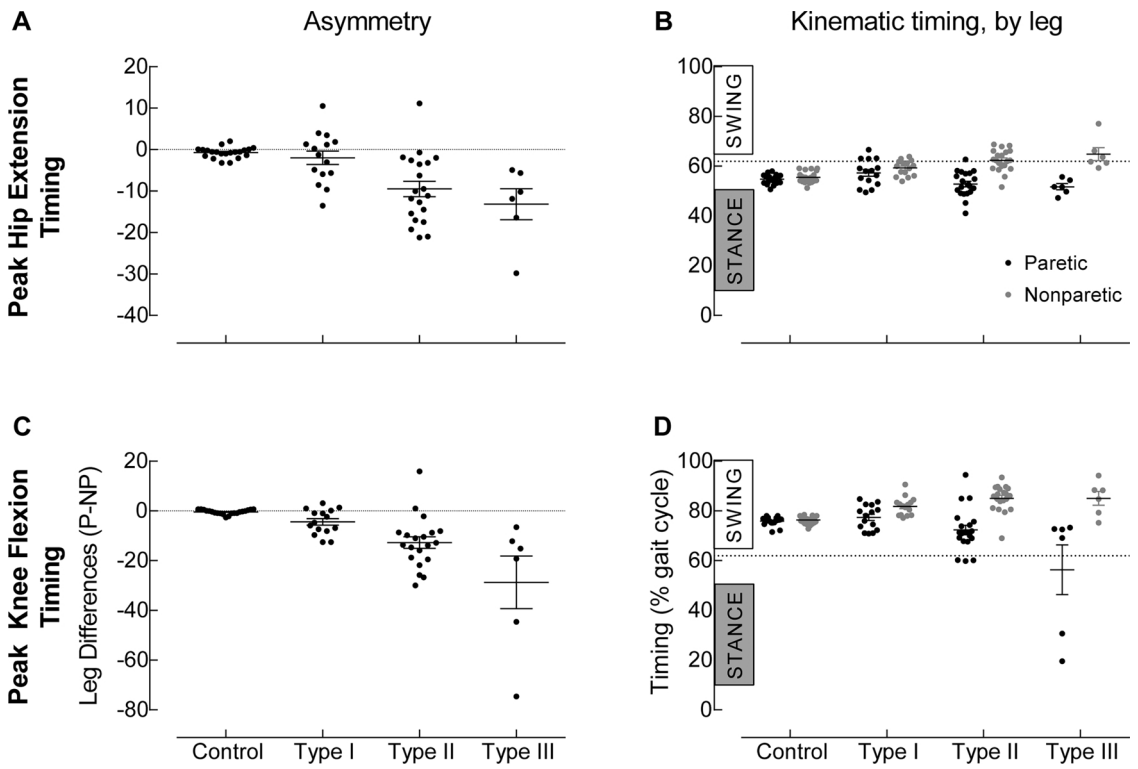

Fig. 4. Progressive asymmetries detected in kinematic *timing*. Here, we illustrate asymmetries in kinematic timing. Paretic and nonparetic legs depicted by black and grey circles, respectively. For comparison, the left leg was experimentally assigned as the paretic leg in controls. A standard reference for toe-off (dotted horizontal line) is provided at 62% of the gait cycle [10]. The left column illustrates calculated asymmetry between paretic and nonparetic legs (P-NP) for (A) peak hip extension timing, and (C) peak knee flexion timing. The dotted line at zero indicates symmetry. The right column illustrates raw kinematic values by leg for (B) peak hip extension timing, and (D) peak knee flexion timing. For the timing of peak hip extension, the increase in asymmetry was linear assessed both with ($p < 0.01$) and without controls ($p = 0.04$); this progressive asymmetry was driven by paretic leg impairment (B). However, for the timing of peak knee flexion, the increase in asymmetry was quadratic when controls were included ($p = 0.04$) and linear among groups post-stroke ($p < 0.01$). Asymmetry in peak knee flexion timing also appears to be driven by paretic leg impairments (D). Data are mean ± standard error of the mean with each subject represented individually to illustrate group distributions.

4.4. Limitations

Our investigation was deliberately limited in scope, to gain understanding of how pelvis motion interacts with other characteristics of hemiparetic gait. As a starting point we categorized hemiparetic gait dysfunction on the magnitude of pelvic excursion deviation, although more elaborate classification schemes could be developed. Furthermore, the cutoffs for the classification scheme at one and two times the maximal control mean (Fig. 2C) may seem arbitrary. However, our sample post-stroke revealed individuals who produced pelvic motion within the bounds of the control distribution. To categorize these individuals while simultaneously acknowledging their pelvic motion did not deviate from controls, we capped the first category at the maximal value of the control distribution (i.e., Type I). Since the composite deviation scores were the sum of six individual deviation scores that each could not exceed a value of three, the maximum possible composite deviation score was 18. Thus, including a second cutoff at twice the maximum of the control distribution provided equally spaced boundaries for the classification scheme.

Use of a composite score proved useful to establish guidelines for categorization based on general control of pelvic motion; however, there are also inherent limitations to this approach. Use of a composite score of pelvic excursion deviation does not afford the opportunity to detect explicit deviations or impairments in any given plane of pelvic motion. Furthermore, identifying specific impairments in controlling pelvic motion (e.g., tilt or rotation) during walking, may help to elucidate which components should be addressed in treatment interventions targeting improved control of the bony pelvis during walking.

An important limitation is the use of data obtained during treadmill walking, which reveals reduced frontal and transverse pelvic excursions compared to overground walking in healthy controls [28–30]. To our knowledge, the relationship of pelvic kinematics between treadmill and overground walking following stroke remains unknown. Thus, we cannot assume similar reductions in frontal and transverse plane excursion on the treadmill post-stroke. Therefore we recommend caution when comparing our results with data collected from persons post-stroke during overground walking.

We acknowledge use of the biomechanically-based classification method presented here is not presently feasible in the clinical setting given the expense and time needed to quantify pelvic excursion. However, speed-based metrics failed to distinguish our groups. Indeed, our goal was to determine if our classification system would be informative and we found that it was. Furthermore, clinicians with strong observational skills might be able to detect pelvic deviations and with the increased availability of wearable technologies, quantitative assessment of pelvic motion during gait may soon be clinically accessible.

5. Conclusions

We performed a first look at the role of pelvic motion control in hemiparetic walking. Our biomechanically-based classification system identified progressive asymmetry in several key biomechanical and physiologic measures between three groups of individuals post-stroke with increasing deviation in pelvic excursion. Similarity in gait speed between stroke groups suggests classification based on pelvic excursion deviation provides greater insight, unavailable from speed-based

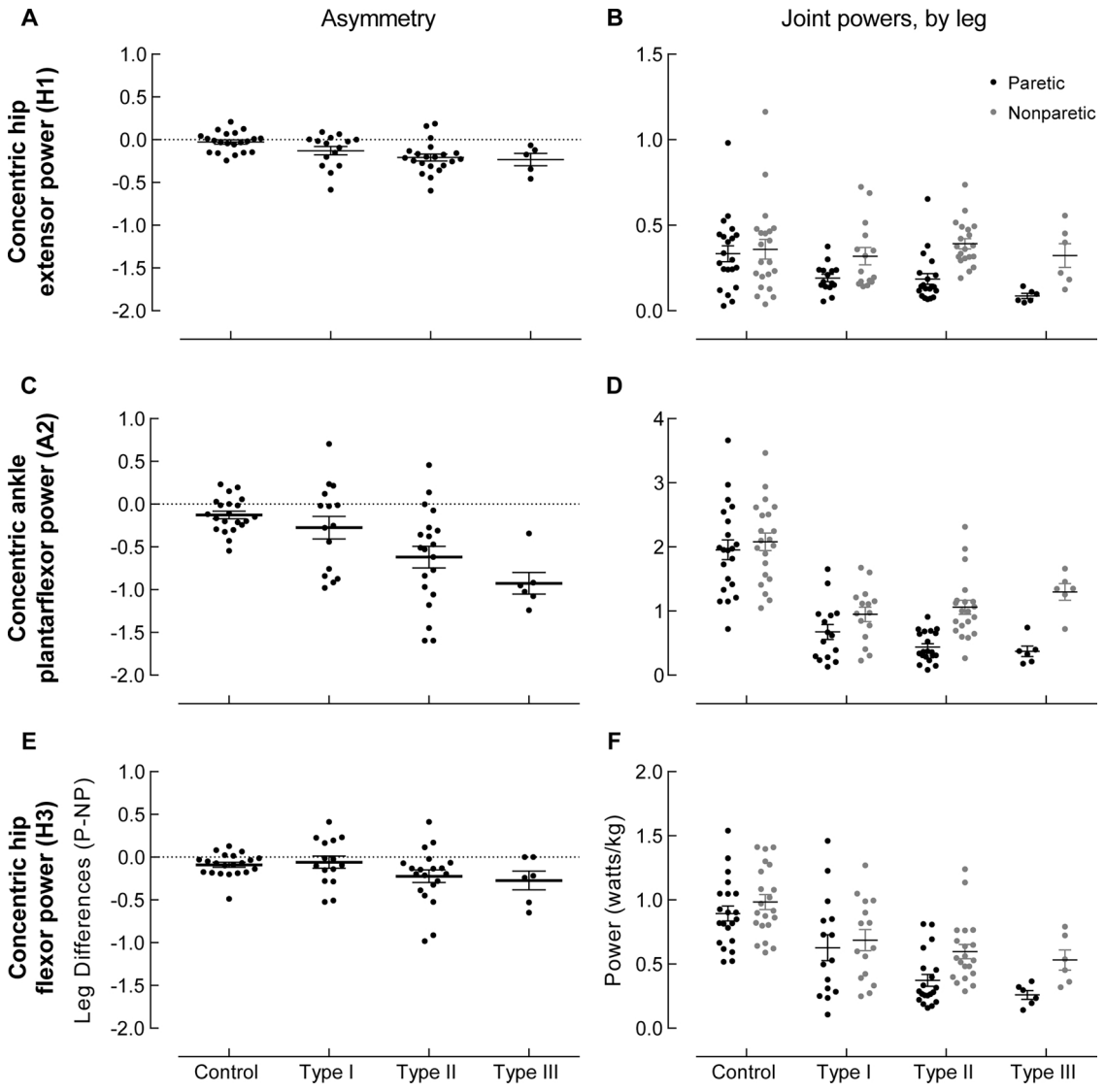

Fig. 5. Magnitude of joint powers. *Top row*: concentric hip extensor power (**H1**; A, B); *middle row*: concentric ankle plantarflexion power (**A2**; C, D); *bottom row*: concentric hip flexor power (**H3**; E, F). The left column illustrates calculated kinetic asymmetry between paretic and nonparetic legs (P-NP) with a dotted line at zero to indicate symmetry. The right column illustrates raw kinetic values by leg. Black and grey circles depict paretic and nonparetic legs, respectively. The left leg was experimentally assigned as the paretic leg in controls. The increase in H1 asymmetry was linear ($p = 0.02$) and appears to be driven by an impairment of power production in the paretic leg following stroke (A–B). Interestingly, once controls were excluded from the analysis, no progressive asymmetry was detected among the groups post-stroke ($p > 0.05$). The asymmetries noted in (C) A2 and (E) H3 fail to reach statistical significance likely due to the high variability in Type II. Data are mean ± standard error of the mean with each subject represented individually to illustrate group distributions.

classifications.

Our data suggest restoring control of pelvic motion during walking may be an appropriate intervention target following stroke. Further biomechanical investigations are needed to determine whether control of pelvis motion is a requisite precursor to, or a byproduct of, improvements in walking function.

Conflict of interest statement

The authors have no conflicts of interest to declare.

Acknowledgements

This research was supported by the Department of Veterans Affairs, Rehabilitation Research & Development Service, Project #A6365B (CP) and Research Career Scientist Award #F7823S (CP) and the VA Brain Rehabilitation Research Center of Excellence (B6793C). Dr. Little received support from the Foundation for Physical Therapy and NIH T32 Neuromuscular Plasticity Training Grant (No. 5 T32 HD043730-08, K Vandenborne, PI).

This material is the result of work supported with resources and the use of facilities at the NF/SG Veterans Administration Health Care System, Gainesville, FL, USA. The contents do not represent the views of the Department of Veterans Affairs or the United States Government. The funding source played no role in either writing this manuscript or the decision to submit for publication. The corresponding author retains full access to all data in the study and assumes final responsibility for the decision to submit for publication.

We thank: Helen Emery, PT for her assistance with data collection and Kelly Rooney, MS for her assistance with data processing.

A portion of this work was presented in abstract form at the Combined Sections Meeting (APTA) and an annual meeting for the American Society for Biomechanics. Virginia L. Little conducted this work in partial fulfillment of the requirements for the Ph.D. degree.

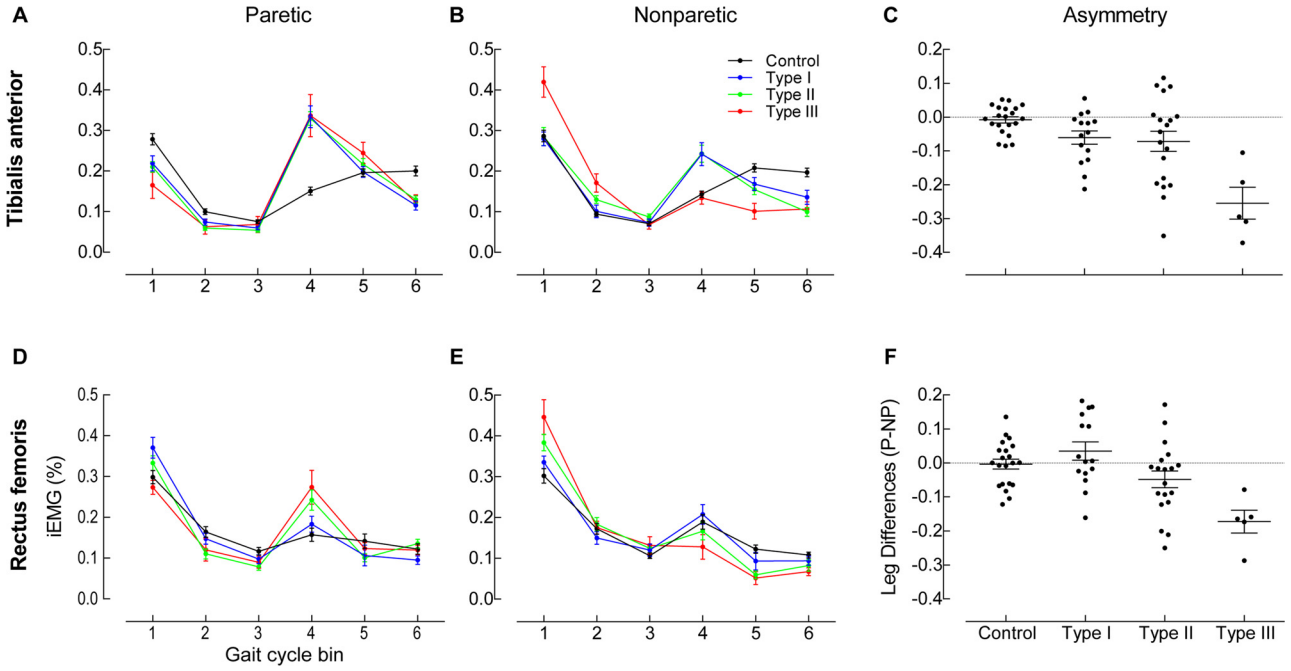

Fig. 6. Temporal patterns of muscle activity. We quantified integrated EMG (iEMG) during biomechanically relevant phases of gait which was parsed into 6 bins: 1) loading response, 2) first half of single limb support, 3) second half of single limb support, 4) pre-swing, 5) first half of swing, and 6) second half of swing. Paretic (left column) and nonparetic (middle column) iEMG is illustrated for the tibialis anterior (A, B) and rectus femoris (D, E) muscles. Error bars depict one standard error. Progressive asymmetry of iEMG was detected only in the tibialis anterior and the rectus femoris during loading response, although the details of these asymmetries differ. For the tibialis anterior, the increase in the asymmetry is linear across all groups ($p < 0.01$), and once the controls are excluded, the asymmetry increase is quadratic ($p = 0.03$; C). However, for the rectus femoris the opposite is true. A quadratic increase in asymmetry is noted with controls included in the model ($p = 0.02$), yet a linear progression is present among only the groups post-stroke ($p < 0.01$; F). While all groups post-stroke reveal proportionally greater tibialis anterior activity in paretic pre-swing (bin 4) compared to the nonparetic leg, a progressive asymmetry was not detected statistically (asymmetry not illustrated). Since iEMG represents temporal information regarding muscle activity rather than magnitude of activity, higher values of paretic tibialis anterior iEMG may result from our sample post-stroke using a lift- or pull-off strategy rather than the typical push-off noted in pre-swing of normal gait pointing to their retained capacity to activate the tibialis anterior following stroke. Asymmetry data are mean ± standard error of the mean with each subject represented individually to illustrate group distributions. The dotted line at zero indicates symmetry.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.gaitpost.2018.03.052.

References

[1] E. Knutsson, C. Richards, Different types of disturbed motor control in gait of hemiparetic patients, Brain 102 (1979) 405–430.
[2] J. Perry, M. Garrett, J.K. Gronley, S.J. Mulroy, Classification of walking handicap in the stroke population, Stroke 26 (1995) 982–989.
[3] S.J. Olney, C. Richards, Hemiparetic gait following stroke. Part I: characteristics, Gait Posture 4 (1996) 136–148.
[4] C.K. Balasubramanian, M.G. Bowden, R.R. Neptune, S.A. Kautz, Relationship between step length asymmetry and walking performance in subjects with chronic hemiparesis, Arch. Phys. Med. Rehabil. 88 (2007) 43–49.
[5] B. Brouwer, K. Parvataneni, S.J. Olney, A comparison of gait biomechanics and metabolic requirements of overground and treadmill walking in people with stroke, Clin. Biomech. 24 (2009) 729–734.
[6] P.W. Duncan, K.J. Sullivan, A.L. Behrman, S.P. Azen, S.S. Wu, S.E. Nadeau, et al., Body-weight-supported treadmill rehabilitation after stroke, N. Engl. J. Med. 364 (2011) 2026–2036.
[7] S.J. Mulroy, T. Klassen, J.K. Gronley, V.J. Eberly, D.A. Brown, K.J. Sullivan, Gait parameters associated with responsiveness to treadmill training with body-weight support after stroke: an exploratory study, Phys. Ther. 90 (2010) 209–223.
[8] B. Bilney, M. Morris, K. Webster, Concurrent related validity of the GAITRite walkway system for quantification of the spatial and temporal parameters of gait, Gait Posture 17 (2003) 68–74.
[9] U.B. Flansbjer, A.M. Holmback, D. Downham, C. Patten, J. Lexell, Reliability of gait performance tests in men and women with hemiparesis after stroke, J. Rehabil. Med. 37 (2005) 75–82.
[10] J. Perry, Gait Analysis: Normal and Pathological Function, SLACK Incorporated, Thorofare, NJ, 1992.
[11] D.C. Kerrigan, E.P. Frates, S. Rogan, P.O. Riley, Hip hiking and circumduction: quantitative definitions, Am. J. Phys. Med. Rehabil./Assoc. Acad. Physiatr. 79 (2000) 247–252.
[12] K.J. Dodd, M.E. Morris, Lateral pelvic displacement during gait: abnormalities after stroke and changes during the first month of rehabilitation, Arch. Phys. Med. Rehabil. 84 (2003) 1200–1205.
[13] J.B. Saunders, V.T. Inman, H.D. Eberhart, The major determinants in normal and pathological gait, J. Bone Joint Surg. Am. 35-A (1953) 543–558.
[14] D.J. Farris, A. Hampton, M.D. Lewek, G.S. Sawicki, Revisiting the mechanics and energetics of walking in individuals with chronic hemiparesis following stroke: from individual limbs to lower limb joints, J. Neuroeng. Rehabil. 12 (2015) 24.
[15] M. Kafri, M.J. Myslinski, V.K. Gade, J.E. Deutsch, High metabolic cost and low energy expenditure for typical motor activities among individuals in the chronic phase after stroke, J. Neurol. Phys. Ther. 38 (2014) 226–232.
[16] A.R. Fugl-Meyer, L. Jaasko, I. Leyman, S. Olsson, S. Steglind, The post-stroke hemiplegic patient. 1. a method for evaluation of physical performance, Scand. J. Rehabil. Med. 7 (1975) 13–31.
[17] P.W. Duncan, M. Propst, S.G. Nelson, Reliability of the Fugl-Meyer assessment of sensorimotor recovery following cerebrovascular accident, Phys. Ther. 63 (1983) 1606–1610.
[18] D.A. Winter, P. Eng, Kinetics: our window into the goals and strategies of the central nervous system, Behav. Brain Res. 67 (1995) 111–120.
[19] M. Kafri, E. Sasson, Y. Assaf, Y. Balash, O. Aizenstein, J.M. Hausdorff, et al., High-level gait disorder: associations with specific white matter changes observed on advanced diffusion imaging, J. Neuroimaging 23 (2013) 39–46.
[20] S. Masiero, R. Avesani, M. Armani, P. Verena, M. Ermani, Predictive factors for ambulation in stroke patients in the rehabilitation setting: a multivariate analysis, Clin. Neurol. Neurosurg. 109 (2007) 763–769.
[21] M.G. Bowden, C.K. Balasubramanian, A.L. Behrman, S.A. Kautz, Validation of a speed-based classification system using quantitative measures of walking performance poststroke, Neurorehabil. Neural Repair 22 (2008) 672–675.
[22] S. Mulroy, J. Gronley, W. Weiss, C. Newsam, J. Perry, Use of cluster analysis for gait pattern classification of patients in the early and late recovery phases following stroke, Gait Posture 18 (2003) 114–125.
[23] S.A. Kautz, C. Patten, Interlimb influences on paretic leg function in poststroke hemiparesis, J. Neurophysiol. 93 (2005) 2460–2473.
[24] I. Jonkers, S. Delp, C. Patten, Capacity to increase walking speed is limited by impaired hip and ankle power generation in lower functioning persons post-stroke, Gait Posture 29 (2009) 129–137.
[25] S.F. Tyson, Trunk kinematics in hemiplegic gait and the effect of walking aids, Clin. Rehabil. 13 (1999) 295–300.

[26] E. De Bujanda, S. Nadeau, D. Bourbonnais, Pelvic and shoulder movements in the frontal plane during treadmill walking in adults with stroke, J. Stroke Cerebrovasc. Dis. 13 (2004) 58–69.
[27] C.J. Hsu, J. Kim, R. Tang, E.J. Roth, W.Z. Rymer, M. Wu, Applying a pelvic corrective force induces forced use of the paretic leg and improves paretic leg EMG activities of individuals post-stroke during treadmill walking, Clin. Neurophysiol. 128 (2017) 1915–1922.
[28] L. Vogt, K. Pfeifer, W. Banzer, Comparison of angular lumbar spine and pelvis kinematics during treadmill and overground locomotion, Clin. Biomech. 17 (2002) 162–165.
[29] R. Staszkiewicz, W. Chwala, W. Forczek, J. Laska, Three-dimensional analysis of the pelvic and hip mobility during gait on a treadmill and on the ground, Acta Bioeng. Biomech./Wroclaw Univ. Technol. 14 (2012) 83–89.
[30] N. Chockalingam, F. Chatterley, A.C. Healy, A. Greenhalgh, H.R. Branthwaite, Comparison of pelvic complex kinematics during treadmill and overground walking, Arch. Phys. Med. Rehabil. 93 (2012) 2302–2308.