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RESEARCH ARTICLE | *Modularity and Compositionality in Motor Control: Acknowledging Emilio Bizzi*

Motor module generalization across balance and walking is impaired after stroke

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Allen JL, Kesar TM, Ting LH. Motor module generalization across balance and walking is impaired after stroke. *J Neurophysiol* 122: 277–289, 2019. First published May 8, 2019; doi:10.1152/jn.00561.2018.—Muscle coordination is often impaired after stroke, leading to deficits in the control of walking and balance. In this study, we examined features of muscle coordination associated with reduced walking performance in chronic stroke survivors using motor module (a.k.a. muscle synergy) analysis. We identified differences between stroke survivors and age-similar neurotypical controls in the modular control of both overground walking and standing reactive balance. In contrast to previous studies that demonstrated reduced motor module number poststroke, our cohort of stroke survivors did not exhibit a reduction in motor module number compared with controls during either walking or reactive balance. Instead, the pool of motor modules common to walking and reactive balance was smaller, suggesting reduced generalizability of motor module function across behaviors. The motor modules common to walking and reactive balance tended to be less variable and more distinct, suggesting more reliable output compared with motor modules specific to either behavior. Greater motor module generalization in stroke survivors was associated with faster walking speed, more normal step length asymmetry, and narrower step widths. Our work is the first to show that motor module generalization across walking and balance may help to distinguish important and clinically relevant differences in walking performance across stroke survivors that would have been overlooked by examining only a single behavior. Finally, because similar relationships between motor module generalization and walking performance have been demonstrated in healthy young adults and individuals with Parkinson's disease, this suggests that motor module generalization across walking and balance may be important for well-coordinated walking.

NEW & NOTEWORTHY This is the first work to simultaneously examine neuromuscular control of walking and standing reactive balance in stroke survivors. We show that motor module generalization across these behaviors (i.e., recruiting common motor modules) is reduced compared with controls and is associated with slower walking speeds, asymmetric step lengths, and larger step widths. This is true despite no between-group differences in module number, suggesting that motor module generalization across walking and balance is important for well-coordinated walking.

electromyography; gait; muscle coordination; muscle synergy; postural control

INTRODUCTION

More than 50% of stroke survivors have persistent mobility impairments that limit their quality of life (Mayo et al. 1999; Miller et al. 2010), and within the first year following a stroke, up to 70% of community-dwelling stroke survivors experience a fall due to loss of balance (Weerdesteyn et al. 2008). Appropriate muscle coordination is required for well-coordinated walking and maintaining balance, particularly in response to external perturbations such as slips and trips. However, muscle coordination is often impaired after stroke for both gait (Clark et al. 2010; Knutsson and Richards 1979; Shiavi et al. 1987) and balance control (de Kam et al. 2018; Di Fabio et al. 1986; Kirker et al. 2000; Marigold and Eng 2006).

Motor module (a.k.a. muscle synergy) analysis has been used to provide valuable insights into changes in muscle coordination associated with reduced walking performance in neurological populations such as stroke (Ivanenko et al. 2013; Seamon et al. 2018; Ting et al. 2015). Motor modules are groups of coactive muscles that are flexibly recruited over time to transform movement goals into biomechanical outputs (Ting et al. 2015). To date, motor module analysis for lower limb muscle coordination poststroke has primarily focused on the number of motor modules recruited during gait behaviors. Our recent work utilized novel metrics of motor module consistency, distinctness, and generalization to examine other features of muscle coordination, and identified differences related to gait and balance performance in both healthy adults (Sawers et al. 2015) and individuals with Parkinson's disease (Allen et al. 2017). However, it remains unclear how these modular features of muscle coordination are affected after stroke. Identifying features of muscle coordination associated with impaired gait and balance performance poststroke through motor module analysis may provide important insights into neuro-

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muscular mechanisms underlying impaired motor performance.

Compared with neurotypical controls, stroke survivors often recruit a smaller number of motor modules during walking, i.e., reduced neuromuscular complexity (Clark et al. 2010). This reduction in motor module number is due to merging of different healthy modules, assumed to reflect a lack of independent drive to motor modules that perform different functions. Motor module merging poststroke is consistent with the clinical definition of muscle synergies, in which abnormal coupling of muscles across the limb are observed (Knutsson and Richards 1979; Shiavi et al. 1987). Furthermore, the reduction in motor module number poststroke is associated with impaired gait and balance function (Barroso et al. 2017; Bowden et al. 2010; Clark et al. 2010) and limits the ability to perform more complex locomotor tasks (e.g., changing speed, cadence, step length, and step height; Routson et al. 2014). Motor module number is also better correlated with gait and balance function than are lower limb Fugl-Meyer assessments typically used to measure the severity of motor impairment (Bowden et al. 2010). Previous studies have also shown that increases in motor module number after rehabilitation are accompanied by improved walking performance (Ferrante et al. 2016; Routson et al. 2013).

However, the ability to recruit a given number of motor modules does not directly translate to a specific level of motor performance. Although recruiting fewer motor modules is associated with slower walking speeds poststroke, differences in speed still remain in individuals who recruit an identical number of modules (Clark et al. 2010). Similarly, improved walking performance after poststroke rehabilitation can occur without increasing motor module number (Hashiguchi et al. 2016; Routson et al. 2013).

To aid in distinguishing important and clinically relevant impairments in motor performance, we recently introduced novel motor module metrics of consistency and distinctness (Allen et al. 2017; Sawers et al. 2015). These novel metrics reflect variations in muscle coordination across different repetitions of the same behavior (e.g., across multiple gait cycles for walking). We posit that greater motor module consistency and distinctness reflects greater stability of motor output across repetitions of a behavior (consistency) organized around production of more well-defined biomechanical output (distinctness), leading to higher levels of motor performance. Indeed, we recently observed greater motor module consistency and distinctness during a balance-challenging walking behavior among expert professional ballet dancers compared with novice nondancers (Sawers et al. 2015). Similarly, we found that improved gait and balance performance after rehabilitation in individuals with Parkinson's disease was associated not with increased module number but increased consistency and distinctness (Allen et al. 2017). Although it is well established that stroke survivors walk with increased step-to-step variability (e.g., spatiotemporal variability; Balasubramanian et al. 2009), whether reduced motor performance in stroke survivors is accompanied by reduction in motor module consistency and distinctness is unknown.

Although maintaining balance is critical for walking, especially in the presence of external disturbances, little is known about motor modules recruited for standing balance poststroke (de Kam et al. 2018) or how they compare with motor modules recruited during walking. Recent evidence suggests that generalization of motor modules across walking and balance behaviors, i.e., recruiting a common set of motor modules, may be an important feature of muscle coordination underlying differences in walking performance. In healthy, young adults, many of the motor modules recruited during walking are also recruited to control both standing and walking balance in response to external perturbations (Chvatal and Ting 2012, 2013; Oliveira et al. 2012). Furthermore, higher levels of motor module generalization across walking and balance behaviors are associated with better motor performance. Long-term training over many years in professional ballet dancers leads to better motor performance on a balance-challenging beam-walking behavior compared with nondancers and is associated with recruiting more common motor modules across gait and balance movement behaviors (Sawers et al. 2015). The generalization of motor modules across gait and balance can also be affected by neurological disorders; individuals with Parkinson's disease exhibit lower levels of motor module generalization across gait and balance behaviors compared with healthy adults, and improvements in motor performance after rehabilitation are associated with increases in motor module generalization (Allen et al. 2017). Whether motor module generalization is reduced in stroke survivors whose ability to selectively recruit appropriate patterns of muscle coordination is impaired (e.g., Clark et al. 2010; Knutsson and Richards 1979; Shiavi et al. 1987) remains unknown.

In the present study, we analyzed electromyography (EMG) data from muscles spanning the hip, knee, and ankle during overground walking and multidirectional perturbations to standing to examine how the modular control of walking and balance is affected in stroke survivors. This is the first study to compare motor modules recruited for walking and standing reactive balance in stroke survivors. We hypothesized that the ability to selectively and consistently recruit patterns of neuromuscular control appropriate for a given movement behavior is impaired after stroke. On the basis of this hypothesis, we predicted that stroke survivors *1*) recruit fewer motor modules in walking and in balance that *2*) exhibit less consistency and distinctness such that *3*) fewer common motor modules are recruited across walking and balance behaviors.

METHODS

Subjects

Nine individuals poststroke (3 men, 57.2 ± 12.7 yr, 85.5 ± 24.4 kg, 6 right-sided hemiparesis, 46.3 ± 23.1 mo poststroke, Fugl-Meyer lower extremity score 23.7 ± 3.7; all values means ± SD) and eight sex-, age-, and weight-similar neurotypical controls (3 men, 62.0 ± 6.6 yr, 76.4 ± 19.1 kg) participated in the current study (Table 1). All participants provided written informed consent before participating according to protocols approved by the institutional review boards at both Emory University and Georgia Institute of Technology.

Inclusion criteria for individuals poststroke were *1*) chronic stroke (>6 mo poststroke), *2*) first (single) lesion, *3*) Fugl-Meyer lower extremity score >12 and <34, *4*) ambulatory with or without an assistive device, and *5*) ability to stand unassisted for at least 15 min. Exclusion criteria were *1*) inability to communicate with investigators, *2*) lower extremity joint pain, contractures, major sensory deficits, or cardiovascular or respiratory symptoms contraindicative of walking, *3*) any other significant non-stroke-related impairment affecting balance, walking, or cognition, and *4*) cerebellar signs.

Table 1. *Subject demographics*

Subject	Sex	Age, yr	Mass, kg	Affected Side	Months Poststroke	Stroke Type	LE-FM	Walking Speed, m/s
				Stroke group				
S1	F	61	44.0	R	73	H	23	0.54
S2	M	70	74.0	R	60	H	22	0.30
S3	M	53	98.6	L	35	I	19	1.58
S4	F	65	97.8	L	47	I	20	0.77
S5	F	63	85.9	R	48	I	23	1.01
S6	F	43	133.5	L	11	I	26	0.88
S7	F	67	72.7	L	50	I	30	1.36
S8	F	31	88.7	R	15	H	27	0.71
S9	M	62	74.2	R	78	NR	NR	0.80
				Control group				
C1	F	63	49.8					1.76
C2	F	68	62.4					1.27
C3	F	60	86.0					1.23
C4	M	56	113.1					1.24
C5	F	67	81.4					1.28
C6	M	57	81.0					1.06
C7	M	72	73.6					1.49
C8	F	53	64.3					1.53

F, female; H, hemorrhagic; I, ischemic; LE-FM, lower extremity Fugl-Meyer score; M, male; NR, not reported.

Inclusion criteria for neurotypical controls included *1*) ambulatory with or without an assistive device, *2*) ability to stand unassisted for at least 15 min, and *3*) age $\geq$ 18 yr. Exclusion criteria were *1*) inability to communicate with researchers, *2*) lower extremity joint pain, contractures, major sensory deficits, or cardiovascular or respiratory symptoms contraindicative of walking, *3*) history or evidence of orthopedic, muscular, or physical disability, *4*) taking current medications that may affect balance, *5*) history or evidence of vestibular, auditory, or proprioceptive impairment, *6*) history or indication of orthostatic hypotension, and *7*) history of any neurological disease or insult.

Experimental Protocol

Surface EMG was recorded from 12 muscles per leg: gluteus maximus (GMAX), gluteus medius (GMED), tensor fascia lata (TFL), adductor magnus (ADMG), biceps femoris long head (BFLH), rectus femoris (RFEM), vastus lateralis (VLAT), medial gastrocnemius (MGAS), lateral gastrocnemius (LGAS), soleus (SOL), peroneus longus (PERO), and tibialis anterior (TA). All EMG data were collected at 1,200 Hz except for *subjects S1–S4*, for whom EMG was collected at 1,080 Hz. Three-dimensional (3-D) kinematics were measured at 120 Hz with an 8-camera (*subjects S1–S4*) or 10-camera (all other subjects) Vicon motion capture system and a custom 25-marker set that included head-arms-trunk, pelvis, thigh, shank, and foot segments.

All subjects completed two experimental conditions: standing reactive balance and walking at self-selected speed.

Standing reactive balance. Reactive balance performance was assessed through postural responses to ramp-and-hold translations of the support surface during standing while subjects stood on an instrumented platform that translated in 12 equally spaced directions in the horizontal plane (see Fig. 1*B*). Subjects were instructed to cross their arms and maintain their balance without stepping or using their arms. All subjects were exposed to the same level of perturbation (displacement 7.5 cm, velocity 15 cm/s, acceleration 0.1 *g*). This perturbation level was such that all subjects could maintain balance on a majority of trials such that few corrective steps were observed. Perturbations across directions were presented in random order, and we continued collecting data until three good trials were collected in each direction (for a total of 36 trials without stepping or arm movement). Stance width was self-selected and enforced to be the same across all trials.

Self-selected walking speed. Each subject also walked overground at self-selected walking speed over a ~25-ft distance. At least three trials were collected per subject. Subjects were instructed to walk as they would normally while keeping their head up and looking straight ahead.

EMG Data Processing

EMG data were high-pass filtered at 35 Hz, demeaned, rectified, and low-pass filtered at 40 Hz with custom MATLAB routines. Subject-specific EMG data matrices for each leg and condition (i.e., walking and reactive balance) were assembled as described below. The assembled EMG data matrices for each condition were then normalized to the maximum activation observed during walking at self-selected speed.

For standing reactive balance, EMG data were analyzed during four different time bins: one before the perturbation and three during the automatic postural response (APR; Fig. 1*B*) (Chvatal and Ting 2013). Specifically, mean muscle activity was calculated during a 120-ms background period that ended 170 ms before the perturbation and during each of three 75-ms bins beginning 150 ms after perturbation onset. Mean muscle activity values for each muscle during each bin for each trial were assembled to form an $m \times t$ data matrix, where m is the number of muscles (12) and t is the number of data points (3 trials $\times$ 12 directions $\times$ 4 time bins = 144).

For walking, at least 10 gait cycles were analyzed per subject to ensure adequate capture of step-to-step variability in muscle recruitment (Oliveira et al. 2014). For consistency with reactive balance processing, EMG data for walking were averaged over 75-ms bins. Only data from the middle 20 ft of the 25-ft walkway were analyzed to avoid the effects of gait initiation and termination (Fig. 1*A*). Trials were concatenated end to end to form an $m \times t$ data matrix. The number of conditions, t (trials $\times$ time bins), varied across subjects. The minimum size of t was 149, and there was no significant difference in the size of t between groups [388.1 $\pm$ 139.8 for stroke subjects, 341.6 $\pm$ 81.8 for controls; $t(15) = 0.822$, $P = 0.424$].

Motor Module Analysis

Four sets of motor modules were identified for each subject [i.e., 2 legs $\times$ 2 conditions (walking and standing reactive balance)]. Motor modules were identified by applying a nonnegative matrix factorization algorithm on the EMG data matrices (NNMF; Lee and Seung

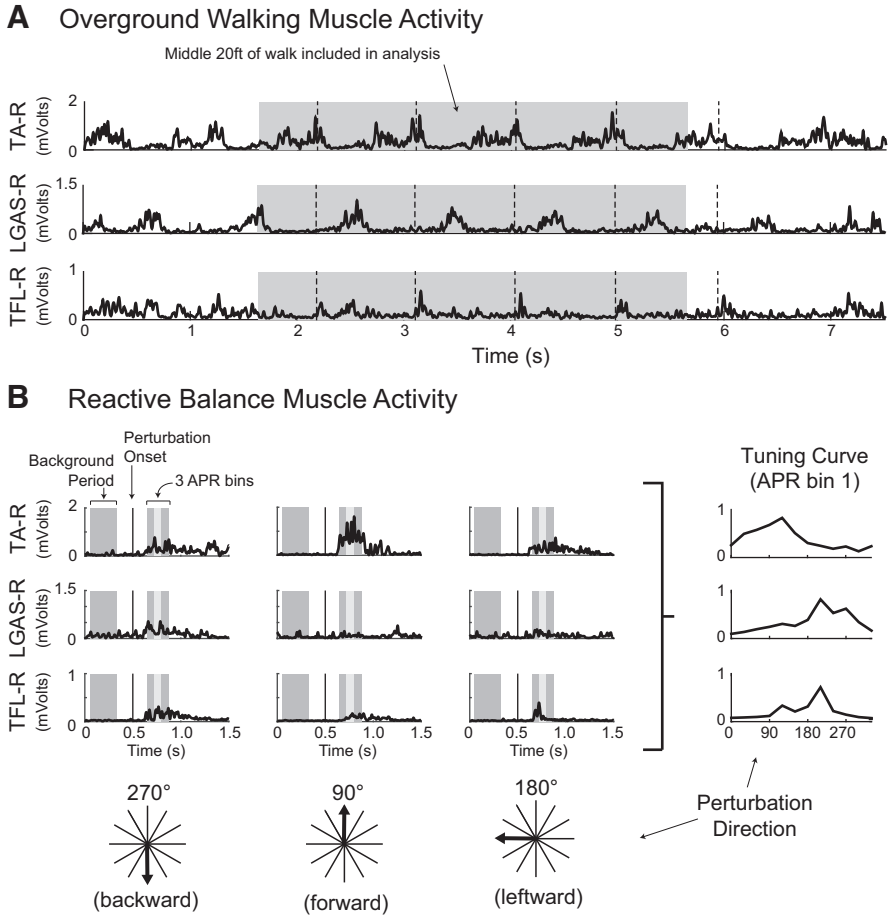

Fig. 1. Example processed electromyography (EMG) from select muscles during overground walking and reactive balance. *A*: muscle activity for walking was recorded while participants walked overground at their self-selected speed for at least 3 trials over a 25-ft walkway. Dashed lines represent right heel strikes. For each trial, only data from the middle 20 ft of the 25-ft walkway were analyzed (represented by shaded region) to avoid the effects of gait initiation and termination. Data from all trials for a subject were concatenated before motor module extraction to form an $m \times t$ data matrix, where m is the number of muscles and t is the number of time points across all trials. *B*: muscle activity for reactive balance was assessed through ramp-and-hold perturbations in 12 evenly spaced directions. *Left*: responses to backward, forward, and leftward perturbations are illustrated. EMG responses occurred ~150 ms after perturbation onset (denoted by vertical lines). Mean EMG activity was calculated during a background period before the perturbation and during three 75-ms time bins during the automatic postural response (APR; shaded regions). *Right*: tuning curves of mean muscle activity from perturbation responses as a function of perturbation directions for the first APR bin. Before motor module extraction, the tuning curves were assembled to form an $m \times t$ data matrix (3 trials $\times$ 12 directions $\times$ 4 time bins = 144). TFL-R, tensor fascia lata; LGAS-R, lateral gastrocnemius; TA-R, tibialis anterior.

1999) such that EMG = W $\times$ C, where W is an $m \times n$ matrix with n motor modules and C is an $n \times t$ matrix of motor module activation coefficients. To ensure equal weighting of each muscle during the extraction process, each row in the EMG data matrices (i.e., each muscle) was scaled to unit variance before motor module extraction and rescaled to original units afterward (Allen et al. 2017; Chvatal and Ting 2013; Torres-Oviedo and Ting 2007).

The number of motor modules, n, per condition was chosen as described in Allen et al. (2017). Briefly, 1–12 motor modules (W) were extracted from each EMG data matrix. The goodness of fit between actual and reconstructed EMG was evaluated with variability accounted for (VAF), defined as 100 $\times$ squared uncentered Pearson's correlation coefficient (Zar 1999). The number of motor modules was chosen such that the lower bound of the 95% confidence interval on VAF exceeded 90% (Allen et al. 2017; Cheung et al. 2009; Hayes et al. 2014). Confidence intervals were calculated using a bootstrapping procedure (250 samples with replacement).

Motor module spatial structure (i.e., W's) was analyzed using the following primary outcome metrics: motor module variability, motor module distinctness, and motor module generalizability.

Motor module number. Motor module number (n) was defined as the number of motor modules independently extracted from the EMG data matrices for walking and reactive balance. To test our prediction that individuals poststroke would recruit fewer motor modules, we compared the number of motor modules independently extracted in each leg (control, nonparetic, paretic) using separate Kruskal-Wallis tests for walking and reactive balance.

Motor module variability. Motor module variability (W_{var}) was defined as the variability of motor module structure across different movement observations. This analysis quantifies the variability of motor module spatial structure (W) across different subsets of the EMG data set with a multistep process (similar to Allen et al. 2017). First, each EMG matrix was resampled 100 times in which 80% of the data was randomly sampled without replacement. From each resampled matrix, a new set of motor modules was extracted, where the number of motor modules, n, was identical to the number previously identified from the entire data set. Because each extraction does not extract modules in the same order, a k-means algorithm was used to cluster similar modules across the 100 resampled extractions. The initial seed for the k-means algorithm was the motor modules extracted using all the data. The variability of each motor module was then quantified as the radius of the n-sphere (in 12-D space) that encompassed the all cluster points (e.g., the 100 different resampled motor modules) in that module to 95% confidence, which was then averaged across all modules within a task. For an example in 2-D (i.e., 2 muscles), see Fig. 3*A*. To test our prediction that individuals poststroke would exhibit increased motor module variability on the paretic leg, we compared motor module variability in each leg (control, nonparetic, paretic) using separate one-way ANOVAs for walking and reactive balance.

Motor module distinctness. Motor module distinctness (W_{dis}) was defined as the mean distance between the n-spheres of each module in 12-D space, where the more distinct the motor modules are for a task, the greater the distance. For a 2-D representative example, see Fig. 3*A*. To test our prediction that individuals poststroke would exhibit decreased motor module distinctness on the paretic leg, we compared the number of motor modules independently extracted in each leg (control, nonparetic, paretic) using separate one-way ANOVAs for walking and reactive balance.

Motor module generalizability. Motor module generalizability ($\%n_{shared}$) was defined as the percentage of motor modules that were similar between reactive balance and walking. First, the number of

shared motor modules across walking and reactive balance (n_{shared}) was identified with Pearson's correlation coefficients (Allen et al. 2017; Chvatal and Ting 2013). A pair of motor modules were considered "shared" if $r \geq 0.708$, which corresponds to the critical value of r^2 for 12 muscles at $P = 0.01$. The amount of shared motor modules was expressed as a percentage to account for the fact that each subject recruited a different number of total motor modules. The percentage of shared motor modules was calculated as $100 \times [n_{shared}/(n_{walk} + n_{balance} - n_{shared})]$. To test our prediction that individuals poststroke would recruit fewer common motor modules across walking and balance behaviors, we compared the percentage of shared motor modules across walking and reactive balance in each leg (control, nonparetic, paretic) using a one-way ANOVA.

Motor module temporal recruitment (i.e., C's) was analyzed using the primary outcome metric of motor module recruitment variability (C_{var}).

Motor module recruitment variability. Step-to-step (for walking) and trial-to-trial (for reactive balance) variability of motor module recruitment coefficients was quantified using the average root mean square error (RMSE) of the C's (see Fig. 4*A*). To test our prediction that individuals poststroke would exhibit increased variability of motor module recruitment, we compared motor module recruitment variability in each leg (control, nonparetic, paretic) using separate one-way ANOVAs for walking and reactive balance. All statistics were performed in SPSS (version 25; IBM SPSS, Chicago, IL) with $\alpha = 0.05$.

Finally, the following secondary analyses were also performed. We examined whether W_{var}, W_{dis}, and C_{var} differed between shared vs. nonshared modules using a separate two-way ANOVA with "leg" and "shared" as factors. The leg factor had three levels (control, paretic, nonparetic), and the shared factor had two levels (shared, nonshared). In this analysis, metrics for each individual module were assessed (instead of averaged within a subject) and split into two groups (shared and nonshared). Leg was included as a factor to check that a similar leg effect was identified as in the one-way ANOVA examining the effect of leg as reported above.

To investigate the relationship between generalizability and motor performance in individuals poststroke, we first calculated several spatiotemporal parameters of walking to serve as measures of walking performance: walking speed, step width, step length asymmetry, and stride time variability. Walking speed for each trial was defined as the average velocity of the C7 marker in the middle 20 ft of the walkway and averaged across trials. Stride time was calculated as the difference in the timing of foot contact between subsequent steps of the same leg. Timing of foot contact was determined on the basis of visual inspection of foot marker position. Stride time variability was calculated using the coefficient of variation (SD/mean $\times$ 100). Step length and width were calculated as absolute difference in ankle marker anterior-posterior and mediolateral position, respectively, between subsequent initial foot contact events. Step length asymmetry was defined as paretic step length/(paretic step length + nonparetic step length) for the stroke group. With this definition, a value of 0.5 indicates perfect step length symmetry, >0.5 indicates longer paretic than nonparetic step length, and <0.5 indicates shorter paretic than nonparetic step length. For the control group, step length asymmetry was calculated as right step length/(right step length + left step length). Group differences in walking speed, step width, step length asymmetry, and stride variability were evaluated using unpaired *t*-tests for each measure. Pearson's correlation analyses were then performed in the stroke group between each measure and motor module generalization in the paretic leg.

We then examined the structure of the motor modules shared between walking and reactive balance. To facilitate comparison of modules between legs, motor modules from walking that were also recruited in reactive balance were pooled across legs and grouped with a hierarchical cluster analysis [MATLAB statistics toolbox functions pdist (Minkowski option; $P = 3$), linkage (Ward option), and cluster]. The number of unique shared modules across legs was determined by identifying the minimum number of clusters that partitioned motor modules such that no cluster contained more than one module per leg (Cheung et al. 2005; Sawers et al. 2017). Next, we examined whether the recruitment of any of these shared modules in the paretic leg of stroke survivors was associated with walking speed, step width, step length asymmetry, or stride time variability. For each shared module (e.g., each module in Fig. 5), we first identified in each subject the walking module with maximum similarity to the shared module using Pearson's correlations. Across all subjects, we then correlated these similarity values to walking speed, step width, step length asymmetry, and stride time variability. This analysis was repeated for each of the shared modules.

RESULTS

Primary Results

Motor module number, *n* (Fig. 2*B*), was not significantly different between control, paretic, and nonparetic legs for either walking ($P = 0.801$) or reactive balance ($P = 0.486$). The median number of motor modules recruited for walking was 4 in controls (range 2–5), 4 in the nonparetic leg (range 3–5), and 4 in the paretic leg (range 3–5). The median number of motor modules recruited for reactive balance was 3 in controls (range 2–4), 3 in the nonparetic leg (range 3–4), and 4 in the paretic leg (range 2–5).

We found that W_{var} (Fig. 3*B*) did not differ between control, paretic, or nonparetic legs in either walking [$F(2,31) = 0.054$, $P = 0.948$] or reactive balance [$F(2,31) = 0.116$, $P = 0.891$]. Variability for walking across control legs was 0.31 ± 0.20 (range 0.09–0.66), across paretic legs was 0.30 ± 0.23 (range 0.06–0.77), and across nonparetic legs was 0.28 ± 0.20 (range 0.06–0.66). Variability for reactive balance across control legs was 0.33 ± 0.1 (range 0.12–0.60), across paretic legs was 0.29 ± 0.19 (range 0.08–0.63), and across nonparetic legs was 0.32 ± 0.16 (range 0.15–0.63).

Similarly, W_{dis} (Fig. 3*C*) did not differ between control, paretic, or nonparetic legs in either walking [$F(2,31) = 0.039$, $P = 0.962$] or reactive balance [$F(2,31) = 0.115$, $P = 0.892$]. Distinctness for walking across control legs was 1.24 ± 0.45 (range 0.35–1.83), across paretic legs was 1.28 ± 0.63 (range 0.04–1.95), and across nonparetic legs was 1.30 ± 0.56 (range 0.40–1.95). Distinctness for reactive balance across control legs was 0.94 ± 0.39 (range 0.06–1.50), across paretic legs was 1.03 ± 0.57 (range 0.03–1.56), and across nonparetic legs was 0.95 ± 0.52 (range 0.00–1.46).

In contrast, we found significant differences in motor module generalizability (e.g., the percentage of motor modules that were shared between reactive balance and walking, $\%n_{shared}$) between groups [$F(2,31) = 3.689$, $P = 0.037$; Fig. 2*C*]. Between-leg comparisons revealed a trend for reduced motor module generalizability compared with controls in both the paretic ($P = 0.066$) and nonparetic legs ($P = 0.098$), but not between the paretic and nonparetic legs ($P = 0.984$). Motor module generalizability was $37.0 \pm 17.0\%$ across control legs (range 12.5–75%), $20.3 \pm 19.7\%$ across paretic legs (range 0–50%), and $21.7 \pm 14.8\%$ across nonparetic legs (range 0–50%).

We also found significant differences in C_{var} (Fig. 4*B*) between groups, but only for walking [$F(2,29) = 9.028$, $P < 0.001$]. Between-leg comparisons revealed that recruitment variability for walking was greater in the nonparetic leg com-

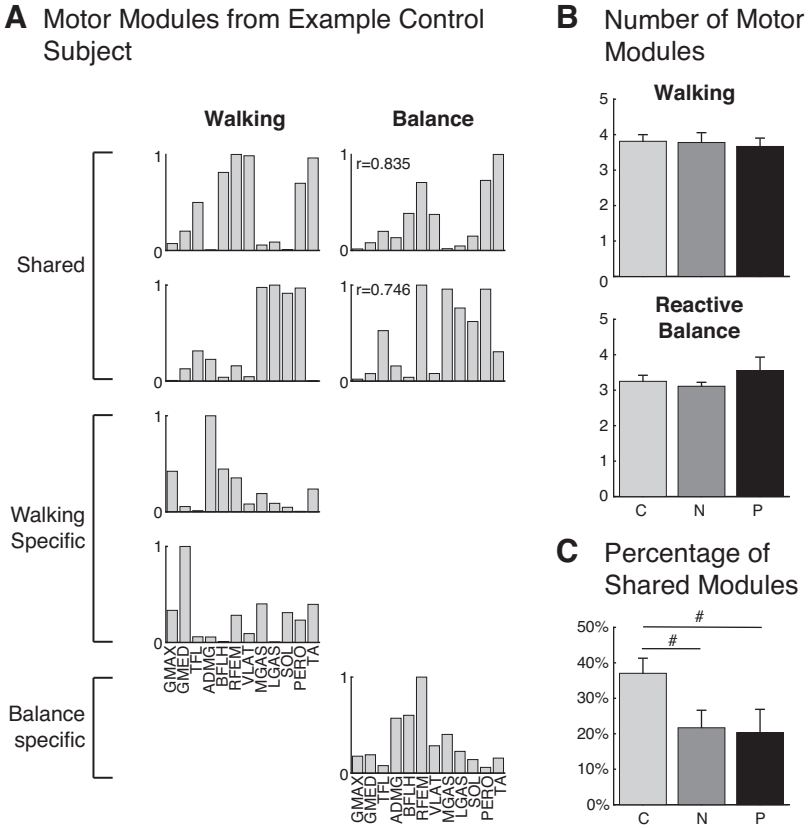

Fig. 2. Motor module number and generalization across walking and reactive balance. *A*: representative motor modules from a control subject during walking (*left*) and reactive balance (*right*). Motor modules were extracted from each behavior independently and identified as shared across behaviors if $r > 0.708$. *B*: the number of motor modules recruited during overground walking (*top*) and reactive balance (*bottom*) did not differ between control (C; $n = 16$), nonparetic (N; $n = 9$), and paretic legs (P; $n = 9$). *C*: the percentage of shared motor modules was decreased in both the nonparetic and paretic legs compared with control legs. Sharing across behaviors was quantified as a percentage of total number of unique motor modules (i.e., 40% of the motor modules, or 2 of 5, were shared across behaviors in the representative subject in *A*). #$P < 0.1$. GMAX, gluteus maximus; GMED, gluteus medius; TFL, tensor fascia lata; ADMG, adductor magnus; BFLH, biceps femoris long head; RFEM, rectus femoris; VLAT, vastus lateralis; MGAS, medial gastrocnemius; LGAS, lateral gastrocnemius; SOL, soleus; PERO, peroneus longus; TA, tibialis anterior.

pared with both the control legs ($P = 0.001$) and the paretic leg ($P = 0.024$), with no difference between paretic and control legs ($P = 0.587$). No significant differences were identified for reactive balance [$F(2,31) = 0.368$, $P = 0.695$]. Recruitment variability for walking across control legs was 0.078 ± 0.008 (range 0.061–0.092), across paretic legs was 0.083 ± 0.007 (range 0.068–0.093), and across nonparetic legs was 0.097 ± 0.015 (range 0.080–0.119). Recruitment variability for reactive balance across control legs was 0.077 ± 0.044 (range 0.039–0.210), across paretic legs was 0.063 ± 0.057 (range 0.008–0.202), and across nonparetic legs was 0.065 ± 0.028 (range 0.016–0.105). Note that due to missing gait events, data from *subject S3* was not included in the analysis for walking.

Secondary Results

Our secondary analyses on W_{var} and W_{dis} revealed that motor modules common to walking and reactive balance tended to be less variable and more distinct than those that were not common across behaviors. For W_{var} (Fig. 3*B*, *bottom*), we found a significant main effect of shared [$F(1,235) = 12.433$, $P = 0.001$], no effect of leg [$F(2,235) = 0.728$, $P = 0.484$], and no interaction effect [$F(2,235) = 0.414$, $P = 0.662$]. Motor module variability was lower in shared vs. nonshared modules with a medium effect size (ES = 0.45). A lower value of variability means that motor module structure was more consistently recruited from step to step. For W_{dis} (Fig. 3*C*, *bottom*), there was a trend for a main effect of shared [$F(1,235) = 3.740$ $P = 0.054$] and no effect of leg [$F(2,235) = 0.070$, $P = 0.783$] or shared × leg interaction [$F(2,235) = 0.451$, $P = 0.638$]. Motor module distinctness was higher in shared vs. nonshared modules, although the effect size was small (ES = 0.22). In contrast, no differences in C_{var} (Fig. 4*B*, *right*) were identified; there was no effect of shared [$F(1,229) = 1.04$, $P = 0.309$], leg [$F(2,229) = 0.869$, $P = 0.421$], or shared × leg interaction [$F(2,229) = 0.219$, $P = 0.803$].

Our secondary analyses on motor module generalizability revealed several associations with walking performance. Stroke survivors walked at slower speeds ($P = 0.008$; Table 2), with increased step width ($P = 0.003$) and increased step length asymmetry ($P = 0.020$).We identified a moderate positive relationship between paretic leg motor module generalization and walking speed ($r = 0.46$; see Fig. 6*A*) such that higher levels of generalization across walking and reactive balance were associated with walking at faster speeds. We also identified a large negative relationship between paretic leg motor module generalization and step width ($r = -0.55$) such that higher levels of generalization across walking and reactive balance were associated with narrower step widths. Similarly, we identified a moderate negative correlation between paretic leg motor module generalization and step length asymmetry ($r = -0.30$) such that higher levels of generalization were associated with more symmetric step lengths. We found no relationship between paretic leg motor module generalization and stride time variability. Note that due to missing gait events, data from *subject S3* was not included in the analysis of step width, step length asymmetry, or stride time variability.

A total of three unique shared motor modules were identified across participants (Fig. 5). Shared *module 1* primarily consisted of the ankle plantarflexors and was recruited in 13 of 16 control legs, 8 of 9 nonparetic legs, and 4 of 9 paretic legs.

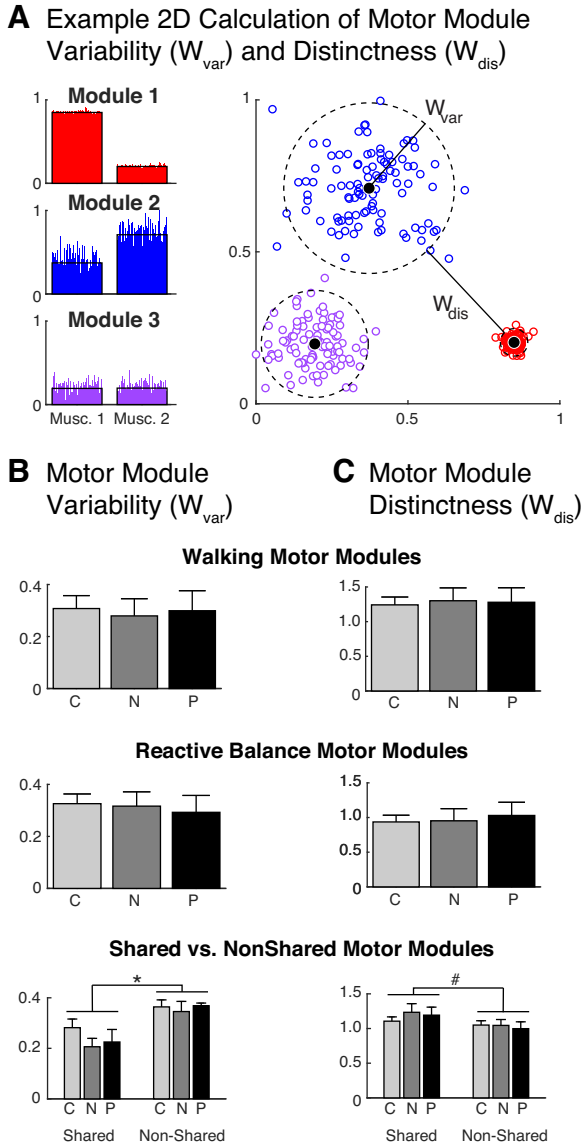

Fig. 3. Motor module variability and distinctness. *A*: a 2-dimensional (i.e., 2 muscles) example of motor module variability and distinctness calculation. *Left*: colored bars for each muscle weighting represent the contribution of each muscle within a module over each of the 100 different resampled module extractions. Black bars indicate the mean across all resampled extractions. *Right*: each point in a cluster represents 1 of the 100 resampled motor modules as depicted on *left*. Motor module variability (W_{var}) is defined as the radius encompassing 95% of the resampled points (dashed circle), and motor module distinctness (W_{dis}) is the average distance between each cluster (solid line). *B* and *C*: motor module variability and distinctness did not differ between control (C; $n = 16$), nonparetic (N; $n = 9$), and paretic legs (P; $n = 9$) in either walking (*top*) or reactive balance (*middle*). However, in all legs, the motor modules that were shared across the two behaviors exhibited less variability and were more distinct than those that were recruited in only one of the behaviors (*bottom*). $*P < 0.05$; $\#P < 0.1$. Musc., muscle.

Across all stroke subjects, the similarity of paretic leg modules during walking to this plantarflexor synergy exhibited a large positive correlation with walking speed ($r = 0.57$; Fig. 6*B*) and moderate negative correlation with step width ($r = -0.45$) and step length asymmetry ($r = -0.40$). Shared *module 2* consisted of hip, knee, and ankle muscles and was recruited in 7 of 16 control legs, 2 of 9 nonparetic legs, and 2 of 9 paretic legs. Across all stroke subjects, the similarity of paretic leg modules during walking to this proximal synergy exhibited a moderate negative correlation with step width ($r = -0.31$; Fig. 6*C*), large negative correlation with step length asymmetry ($r = -0.65$), and moderate negative correlation with stride time variability ($r = -0.46$). Shared *module 3* consisted primarily of the ankle dorsiflexors with low level recruitment of more proximal knee and hip muscles. This module was recruited in 8 of 16 control legs, 0 of 9 nonparetic legs, and 4 of 9 paretic legs. Across all stroke subjects, the similarity of paretic leg modules during walking to this TA module exhibited a moderate negative correlation with walking speed ($r = -0.32$, Fig. 6*D*), moderate positive correlation with step length asymmetry ($r = 0.46$), and large positive correlation with stride time variability ($r = 0.69$).

DISCUSSION

In the present study, we have shown that examining muscle recruitment across movement behaviors with different biomechanics and neural control may reveal important insights into changes in neuromuscular control underlying motor performance that might otherwise be overlooked during studies focused on a single behavior. Our study is the first to compare the modular control of walking and standing reactive balance in stroke survivors. Our results provide evidence that motor module generalization across walking and standing reactive balance is reduced poststroke and associated with lower walking performance. Our results demonstrate that even when stroke survivors exhibit similar number and consistency of motor modules compared with neurotypical individuals in a single behavior, as shown in prior results (Allen et al. 2017; Sawers et al. 2015), comparing motor modules across behaviors can reveal important differences related to motor ability.

Motor Module Generalization Across Walking and Standing Reactive Balance Is Reduced in Stroke Survivors

Although prior studies have demonstrated that differences in motor module number can explain differences in walking performance (Clark et al. 2010; Fox et al. 2013; Rodriguez et al. 2013; Steele et al. 2015), we did not identify reduced motor module number in our sample of stroke survivors in either walking or standing reactive balance. This negative finding can be explained by the relatively high motor function of the stroke survivors in the current study. Reduced motor module number during walking, attributed to merging of two or more motor modules, is common among stroke survivors (Clark et al. 2010). Although we identified a couple cases of motor module merging in the current study (e.g., Fig. 7), this group of stroke survivors on average did not exhibit a reduction in motor module number. However, motor module merging, and thus reduced motor module number, was previously found to be associated with slower walking speeds (Bowden et al. 2010; Clark et al. 2010) and poorer motor function (i.e., Fugl-Meyer scores; Cheung et al. 2012). The functional characteristics of the stroke survivors in the current study were on the higher end of those previously examined, with faster walking speeds (0.31–1.58 m/s in the current study vs. 0.14–1.16 m/s in Clark et al. 2010) and better motor function (Fugl-Meyer lower extremity scores 19–30 in the current study vs. 8–34 in Clark et al. 2010). Similarly, a recent study examining motor modules during reactive balance found no difference in motor

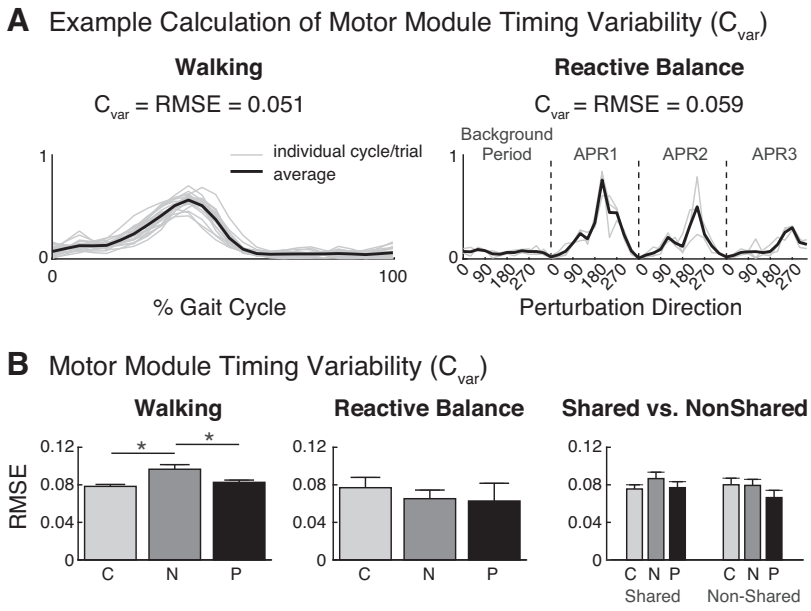

Fig. 4. Motor module timing variability. *A*: example motor module recruitment timing and calculation of module timing variability (C_{var}). For walking (*left*), C_{var} was calculated as the root mean square error (RMSE) of the timing curve across gait cycles. For reactive balance (*right*), C_{var} was calculated as the RMSE of the timing curve across perturbation directions (12 directions) and time bins [4 time bins: background and automatic postural response (APR) 1, APR2, and APR3]. Gray lines represent individual gait cycles for walking or trials for reactive balance. *B*: C_{var} during walking (*left*) was increased in the nonparetic leg (N; $n = 8$) compared with both the paretic (P; $n = 8$) and control legs (C; $n = 16$). No differences were identified between legs during reactive balance (*middle*). No differences were identified between modules that were shared across the two behaviors compared with those that were recruited in only one of the behaviors (*right*). $*P < 0.05$.

module number between controls and mild-to-moderately impaired stroke survivors (de Kam et al. 2018). Thus the similar number of motor modules in stroke survivors vs. controls found in the current study is consistent with the high-functioning stroke survivors of these prior studies.

Accumulating evidence suggests that motor module generalization may help to distinguish important and clinically relevant differences in walking performance across individuals, especially among high-functioning stroke survivors. Motor module generalization, defined as recruiting a common set of motor modules across different movement behaviors, has previously been examined across different locomotor behaviors to understand limitations in the control of walking poststroke (Routson et al. 2014). Although maintenance of balance is critical for walking, no study has examined generalization of motor modules across walking and balance in stroke survivors with walking impairments. In two recent studies, we provided evidence that the amount of motor module generalization across walking and reactive balance is associated with walking performance. In particular, improved walking performance is accompanied by increased motor module generalization after long-term training in healthy young adults (Sawers et al. 2015) and rehabilitation in individuals with Parkinson's disease (Allen et al. 2017). In the present study, we show that higher levels of motor module generalization across walking and balance are also associated with better walking performance in stroke survivors, such as walking speed, step length asymmetry, and step width. Taken together, these studies add to our understanding of how walking is controlled, providing compelling evidence that motor module generalization across walking and balance underlies well-coordinated walking.

Generalization of motor modules across walking and standing reactive balance may reflect automatic control of walking. Rapid changes in the coordination of muscles is required to recover from discrete perturbations, such as those experienced by participants in the current study during the standing reactive balance paradigm, and are thought to be governed by brain stem circuits (Stapley and Drew 2009). We previously demonstrated in healthy young adults that a common set of motor modules are recruited across walking and reactive balance (Chvatal and Ting 2012, 2013), suggesting a convergence of control on the automatic brain stem-mediated recruitment of spinal motor modules. A stroke, depending on the location

Table 2. *Spatiotemporal parameters of walking*

	Controls	Stroke	*P* Value
Step width, cm	19.84 (2.23)	28.16 (6.16)	0.003
Step length asymmetry	0.50 (0.01)	0.54 (0.05)	0.020
Stride time variability, %	3.4 (0.8)	4.1 (1.4)	0.250
Walking speed, m/s	1.36 (0.22)	0.88 (0.39)	0.008

Values for control and stroke groups are means (SD).

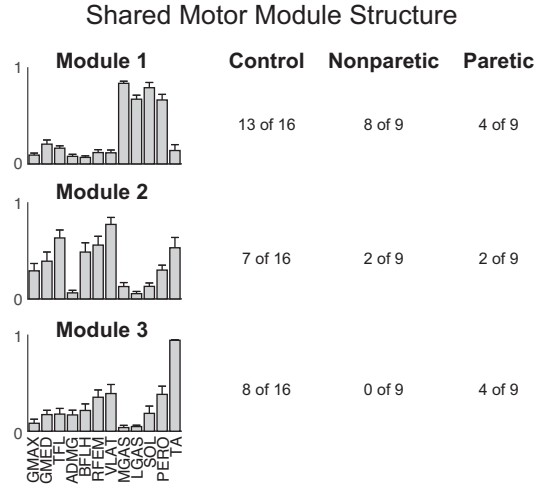

Fig. 5. Structure of motor modules shared across walking and reactive balance. Three unique motor modules were identified across all legs as shared across behaviors. The numbers at *right* indicate in how many legs per group (control, nonparetic, paretic) each of these motor modules were present. GMAX, gluteus maximus; GMED, gluteus medius; TFL, tensor fascia lata; ADMG, adductor magnus; BFLH, biceps femoris long head; RFEM, rectus femoris; VLAT, vastus lateralis; MGAS, medial gastrocnemius; LGAS, lateral gastrocnemius; SOL, soleus; PERO, peroneus longus; TA, tibialis anterior.

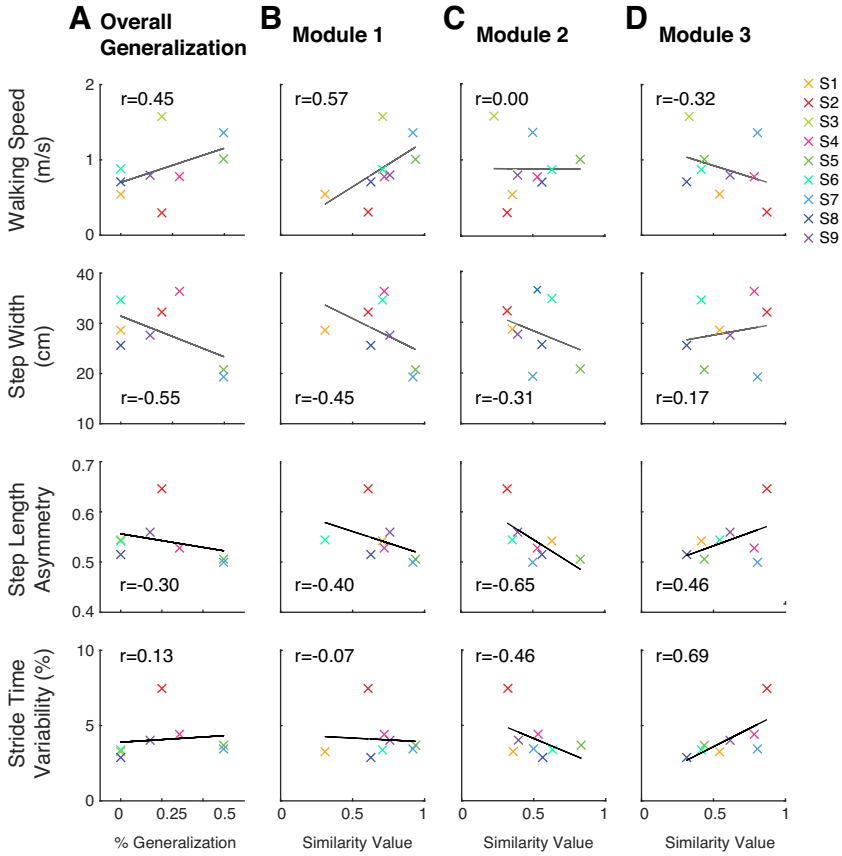

Fig. 6. Association between measures of walking performance and overall motor module generalization and recruitment of shared *modules 1–3* in the paretic leg of individuals poststroke (S1–S9). Module numbers correspond to the shared modules identified across all subjects illustrated in Fig. 5. *A*: motor module generalization was positively correlated with walking speed and negatively correlated with step width and step length asymmetry. *B*: recruitment of a module that resembled shared *module 1* was associated with increased walking speed, reduced step width, and reduced step length asymmetry (e.g., values becoming closer to 0.5, which represents perfect symmetry of step lengths). *C*: recruitment of a module that resembled shared *module 2* was associated with reduced step width, step length asymmetry, and stride time variability. *D*: recruitment of a module that resembled shared *module 3* was associated with decreased walking speed, increased step length asymmetry, and increased stride time variability. Symmetric step lengths occur at values of 0.5, and values above symmetry occur due to longer paretic than nonparetic steps.

of the lesion and the affected brain areas, may disrupt the neural pathways governing the control of walking such that they no longer converge on the same motor modules. Loss of the ability to recruit the automatic reactive balance motor modules during walking poststroke is consistent with reduced gait automaticity in this population (e.g., using dual-task paradigms; Dennis et al. 2009; Hyndman et al. 2006; Liu-Ambrose et al. 2007; Plummer-D'Amato et al. 2008). Consistent with the idea that generalization of motor modules across walking and reactive balance reflects automaticity, post hoc analyses revealed that the shared walking/balance modules in the paretic leg more closely resembled the motor modules independently recruited in reactive balance ($r = 0.68 \pm 0.19$) vs. walking ($r = 0.58 \pm 0.21$), with a medium effect size (Cohen's $d = 0.50$; Cohen 1992). These results are also consistent with our recent study in individuals with Parkinson's disease in whom improvements in gait and balance function after rehabilitation were accompanied by increased motor module generalization due to walking motor modules becoming more similar to the reactive balance motor modules (Allen et al. 2017). Future studies are needed to directly test the relationship between motor module generalization and gait automaticity.

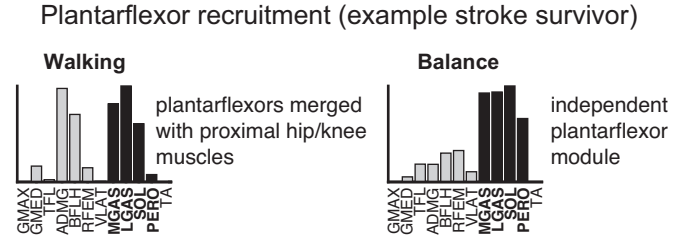

Fig. 7. Example stroke survivor data illustrating merged plantarflexor module during walking but independent recruitment of the plantarflexors during reactive balance. GMAX, gluteus maximus; GMED, gluteus medius; TFL, tensor fascia lata; ADMG, adductor magnus; BFLH, biceps femoris long head; RFEM, rectus femoris; VLAT, vastus lateralis; MGAS, medial gastrocnemius; LGAS, lateral gastrocnemius; SOL, soleus; PERO, peroneus longus; TA, tibialis anterior.

Motor Modules Generalized Across Walking and Standing Reactive Balance Are More Distinct and Consistent in Their Structure

Generalized motor modules may reflect consistent patterns of muscle coordination to produce well-defined motor output that is required by multiple movement behaviors. We found that motor modules common to both walking and reactive balance are more distinct and consistent in their structure (i.e., the W's) than behavior-specific motor modules in both stroke survivors and controls (Fig. 3*C*). These results are consistent with our prior studies where we found higher levels of motor module generalization were accompanied by higher levels of distinctness and consistency in both healthy young adults (Sawers et al. 2015) and individuals with Parkinson's disease (Allen et al. 2017). That similar results have been found across different population groups suggest that this phenomenon reflects a general muscle recruitment strategy for well-coordinated movement. We posit that the distinctness of generalized motor modules reflects patterns of muscle coordination that have been fine-tuned to produce a unique motor output that may be required across multiple movement behaviors, and furthermore, that their structure is consistent across repetitions

of each behavior may reflect a stable neural solution to produce that motor output. We speculate that the lack of group level differences (i.e., stroke compared with controls) reflects the heterogeneity of brain lesion location and lesion load affected in our cohort of stroke survivors. This is in contrast to our prior study in Parkinson's disease in which the affected brain areas across subjects are more localized (i.e., basal ganglia and related structures; Allen et al. 2017). Future studies with a larger sample of stroke survivors could take advantage of the heterogeneity of stroke locations to investigate which neural structures are important for producing motor modules with distinct and consistent structure.

Our results also suggest that stroke survivors with mild-to-moderate impairment may rely on their nonparetic leg to make step-to-step adjustments in muscle recruitment during walking. We found that motor module recruitment during walking was more variable in stroke survivors than in controls (i.e., the C's, Fig. 4), which is consistent with our prior study in which increased variability was associated with lower skill level in healthy young adults (Sawers et al. 2015). However, only the nonparetic leg exhibited increased recruitment variability, with no differences between generalized or behavior-specific modules. Increased recruitment variability in the nonparetic leg may reflect greater flexibility in muscle recruitment that accommodates or compensates for paretic leg deficits.

The Motor Modules Generalized Across Walking and Standing Reactive Balance Varied Across Subjects

We identified three motor modules that were frequently generalized across walking and reactive balance (Fig. 5) that resemble motor modules previously identified during walking in healthy adults (Clark et al. 2010): an independent plantarflexor module, a proximal hip and knee module, and an ankle dorsiflexor module. Surprisingly, generalization across walking and reactive balance was altered not only in the paretic leg of stroke survivors but also in their nonparetic leg (Figs. 2 and 5). Such changes in nonparetic leg generalization likely represent a compensatory strategy to overcome deficits in the paretic leg and are consistent with prior studies demonstrating changes in both paretic and nonparetic leg muscle recruitment during walking and reactive balance (e.g., de Kam et al. 2018; Di Fabio et al. 1986; Raja et al. 2012). We discuss below how the generalization of each of these modules may be related to walking performance after stroke. However, studies with larger sample sizes will be needed to specifically test these speculations and to evaluate differences in movement strategies.

Generalization of the proximal hip and knee module (*module 2*; Fig. 5) across walking and reactive balance, which was notably absent in most stroke subjects, may be associated with the automatic control of mediolateral stability during walking. *Module 2* has large contributions from muscles with actions in the mediolateral plane (i.e., GMED and TFL). A module with similar structure to *module 2* was previously identified in healthy adults to be recruited for both standing reactive balance (Chvatal and Ting 2013; Torres-Oviedo and Ting 2007) and walking (Chvatal and Ting 2013; Clark et al. 2010;). This module is a major contributor to mediolateral ground reaction forces during walking (Allen and Neptune 2012), which act to prevent the center-of-mass from moving too far beyond the base of support during early stance. As such, not recruiting this module during walking may be a potential source of mediolateral instability. A common strategy to compensate for poor mediolateral balance is to increase step width, which acts to increase lateral base of support and provide greater margins of stability (McAndrew Young and Dingwell 2012; Vistamehr et al. 2016). Indeed, stroke subjects walked with greater step widths than older adult controls (Table 2), and recruitment of the proximal hip and knee extensor module was negatively correlated with step width in the paretic leg of stroke survivors (Fig. 6).

Increased automatic control of mediolateral stability due to recruitment of the reactive balance proximal knee and hip module during walking may also enable better control of stepping. Recruitment of this module was associated with more symmetric step lengths and less stride time variability (Fig. 6*C*). These relationships are consistent with the role of the proximal hip/knee module for both body support and mediolateral ground reaction forces (Allen and Neptune 2012). All subjects in the stroke group who walked with asymmetric step lengths had longer paretic than nonparetic steps (step length asymmetry >0.5). Longer paretic steps have been associated with less percentage body weight supported by the paretic leg and spending less time in paretic single-leg stance (Balasubramanian et al. 2007). As such, the nonparetic leg has less time to progress forward during swing, resulting in a shorter nonparetic step. An absence of this shared proximal hip/knee module likely contributes to an inability to support weight on the paretic leg and thus the observed asymmetric step lengths. Similarly, an inability to maintain weight on the paretic leg and medially direct the center-of-mass during single-leg stance may also lead to less control over stride timing. In addition, the hip muscles contained in this module are important for pelvic list and as such enable transfer of power to the contralateral limb (e.g., in this case the nonparetic limb) during swing (Allen and Neptune 2012; Anderson et al. 2004). This interpretation is consistent with the increased stride time variability associated with absence of this shared module as well as a recent study which found the hip abductors (muscles contained in this shared module) to be important for stride time variability (Arvin et al. 2015).

Recruitment of an independent plantarflexor motor module (*module 1*; Fig. 5) in the paretic leg across both walking and reactive balance may facilitate faster walking with more symmetric steps lengths and automatic control of mediolateral stability. This module was generalized across behaviors in a majority of control (13 of 16) and nonparetic (8 of 9) legs, but in less than half of paretic legs (4 of 9). That this module is positively correlated with walking speed is consistent with the role of the plantarflexors in generating forward propulsion (Liu et al. 2008; Neptune et al. 2008) and for successful walking performance poststroke (Routson et al. 2013). Similarly, that this module is negatively correlated with step width is consistent with the role of the plantarflexors, in addition to the proximal hip/knee module, in generating mediolateral ground reaction forces (Allen and Neptune 2012; Pandy et al. 2010). Similar to the proximal hip/knee module, that the plantarflexor module was associated with more symmetric stepping is consistent with the role of the ankle plantarflexors in providing body support during stance (Allen and Neptune 2012; Liu et al. 2008; McGowan et al. 2010).

Generalization of the independent TA module (*module 3*; Fig. 5) was also notably absent in many stroke subjects. The TA is important for the control of foot and leg kinematics during the swing phase (Allen and Neptune 2012) and the transition to stance phase (Anderson and Pandy 2003; Perry 1992), and reduced TA strength has been associated with slower walking speeds in stroke survivors (Lin et al. 2006). As such, we expected the absence of this shared module in stroke survivors to also negatively impact walking performance. However, our results suggest the opposite such that recruitment of the independent TA module is associated with slower walking speeds, increased step length asymmetry, and increased stride time variability (Fig. 6*D*). As a post hoc analysis, we examined whether a similar relationship between TA module recruitment and walking performance existed in the control group. We found that recruitment of this module in controls was not associated with any of our measures of walking performance (walking speed: $r = -0.09$; step length asymmetry: $r = 0.01$; stride time variability: $r = -0.05$). A potential explanation is that those stroke subjects who had better levels of walking performance did so through compensatory multimuscle coactivation patterns in which the TA was coactivated with other muscle groups. Indeed, changes in TA recruitment timing and coactivation with muscles crossing the ankle, knee, and hip are common after stroke (Den Otter et al. 2007; Lamontagne et al. 2000; Tan and Dhaher 2014). Furthermore, improved walking performance in stroke survivors can occur without a return to normal muscle coordination patterns (Buurke et al. 2008; Den Otter et al. 2006). This relationship warrants investigation in a larger sample of stroke survivors in which we can also examine whether particular patterns of compensatory multimuscle coordination are associated with better walking performance.

Examining Multiple Movement Behaviors Provides Unique Insights into Neuromuscular Control

Examining muscle recruitment across movement behaviors with different biomechanics and neural control may reveal important insights into changes in neuromuscular control underlying motor performance that might otherwise be overlooked. Several of our stroke survivors exhibited classic patterns of motor module merging during walking, such as the merging of the ankle plantarflexors with more proximal hip and knee extensors (Clark et al. 2010). Recruitment of this merged module is associated with slow walking speeds and asymmetric gait patterns (Clark et al. 2010), and regaining independent recruitment of the ankle plantarflexors during walking is associated with improved walking performance after rehabilitation (Routson et al. 2013). Despite recruiting a merged plantarflexor module during walking, some individuals in the current study retained independent recruitment of the ankle plantarflexors during reactive balance (e.g., Fig. 7). One explanation for this phenomenon is that it is simply an artifact of the methodology used to select the number of motor modules (4 in balance, 3 in walking). However, we can rule this out because the plantarflexors were still merged with the proximal hip and knee extensors when four motor modules were extracted from walking and the independent plantarflexor module during reactive balance remained even when the number of motor modules extracted was reduced to three. Whether the merged plantarflexor control during walking was due to a choice (i.e., developed compensatory strategy) or a constraint (i.e., altered neural pathway integrity during walking) is unclear. Nevertheless, these results suggest that this poststroke individual retains the capability to independently recruit the plantarflexors in some capacity and might be more likely to regain independent plantarflexor control during walking through targeted rehabilitation. The specific muscle groups to target depend on whether the merged pattern represents a compensatory strategy or neural constraint.

Conclusions

Our work is the first to provide evidence that motor module generalization across walking and standing reactive balance may distinguish important and clinically relevant differences in muscle coordination affecting walking performance in stroke survivors that would have been overlooked by examining walking alone. The relationship between walking performance and motor module generalization across walking and balance identified in the present study in stroke survivors is consistent with our prior studies examining individuals with Parkinson's disease (Allen et al. 2017) and expert ballet dancers (Sawers et al. 2015). Although the sample sizes in each study were small (between 6 and 11 per group), taken together, these studies provide strong evidence for generalization of neural control across walking and reactive balance as important for well-coordinated walking. Because of the small sample size of the current study, we chose not to investigate how the generalization of specific motor modules was associated with measures of walking performance beyond walking speed, step width, and stride time variability or differences in movement strategies (i.e., joint kinematics). Furthermore, because we have to date only identified correlational associations between motor module generalization and walking performance, future studies are needed to further investigate whether decreased motor module generalization is an important causal factor limiting walking performance.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

J.L.A. and L.H.T. conceived and designed research; J.L.A. performed experiments; J.L.A. analyzed data; J.L.A., T.M.K., and L.H.T. interpreted results of experiments; J.L.A. prepared figures; J.L.A. and L.H.T. drafted manuscript; J.L.A., T.M.K., and L.H.T. edited and revised manuscript; J.L.A., T.M.K., and L.H.T. approved final version of manuscript.

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