Central Drive to the Paretic Ankle Plantarflexors Affects the Relationship Between Propulsion and Walking Speed After Stroke

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Background and Purpose: The ankle plantarflexor muscles are the primary generators of propulsion during walking. Impaired paretic plantarflexion is a key contributor to interlimb propulsion asymmetry after stroke. Poststroke muscle weakness may be the result of a reduced force-generating capacity, reduced central drive, or a combination of these impairments. This study sought to elucidate the relationship between the neuromuscular function of the paretic plantarflexor muscles and propulsion deficits across individuals with different walking speeds.

Methods: For 40 individuals poststroke, we used instrumented gait analysis and dynamometry coupled with supramaximal electrostimulation to study the interplay between limb kinematics, the neuromuscular function of the paretic plantarflexors (ie, strength capacity and central drive), propulsion, and walking speed.

Results: The strength capacity of the paretic plantarflexors was not independently related to paretic propulsion. Reduced central drive to the paretic plantarflexors independently contributed to paretic propulsion deficits. An interaction between walking speed and plantarflexor central drive was observed. Individuals with slower speeds and lower paretic plantarflexor central drive presented with the largest propulsion impairments. Some study participants with low paretic plantarflexor central drive presented with similarly fast speeds as those with near-normal central drive by leveraging a compensatory reliance on nonparetic propulsion. The final model accounted for 86% of the variance in paretic propulsion ($R^2 = 0.86$, $F = 33.10$, $P < 0.001$).

Discussion and Conclusions: Individuals poststroke have latent paretic plantarflexion strength that they are not able to voluntarily access. The magnitude of central drive deficit is a strong indicator of propulsion impairment in both slow and fast walkers.

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INTRODUCTION

Symmetrical propulsive force generation from the individual limbs is a hallmark feature of healthy bipedal locomotion that contributes to an economical and stable gait.[1] In contrast, asymmetrical propulsion is characteristic of poststroke hemiparesis[2] and is associated with impaired walking function.[3,4] Poststroke neuromotor impairments are heterogeneous and the propulsive force generated by each limb depends on both kinematics and kinetics.[5,6] Prior work has shown that the ankle plantarflexor muscles are the primary generators of propulsive power during walking[7] and that the trailing limb angle plays an important role in translating ankle plantarflexion torque into propulsion (Figure 1A).[5,8] However, little is known about how the nature of plantarflexor muscle weakness affects propulsion in persons with stroke.

During clinical testing, the maximum force that a patient produces voluntarily is often used to measure their strength; however, poststroke weakness may be the result of different underlying deficits.[9-12] For example, a muscle that produces low voluntary forces may have reduced strength capacity (eg, reduced physiologic cross-sectional area due to muscle atrophy), reduced central drive (ie, latent capacity to produce higher forces), or a combination of these deficits. More specifically, strength capacity refers to the force produced by a fully activated muscle, and central drive is a measure of strength that relates a muscle's maximum voluntary force to its strength capacity and therefore reflects the percentage of the strength capacity that can be voluntarily generated by an individual. By combining dynamometry with neuromuscular electrostimulation, the extent and nature of poststroke muscle weakness can be assessed (Figure 1B).[11,13]

Personalizing propulsion-targeting interventions[14-16] to the needs of individual patients is necessary for the advance of poststroke gait rehabilitaiton[17] and likely requires identifying the nature of their ankle plantarflexion weakness. The objective of this study was to evaluate the interplay

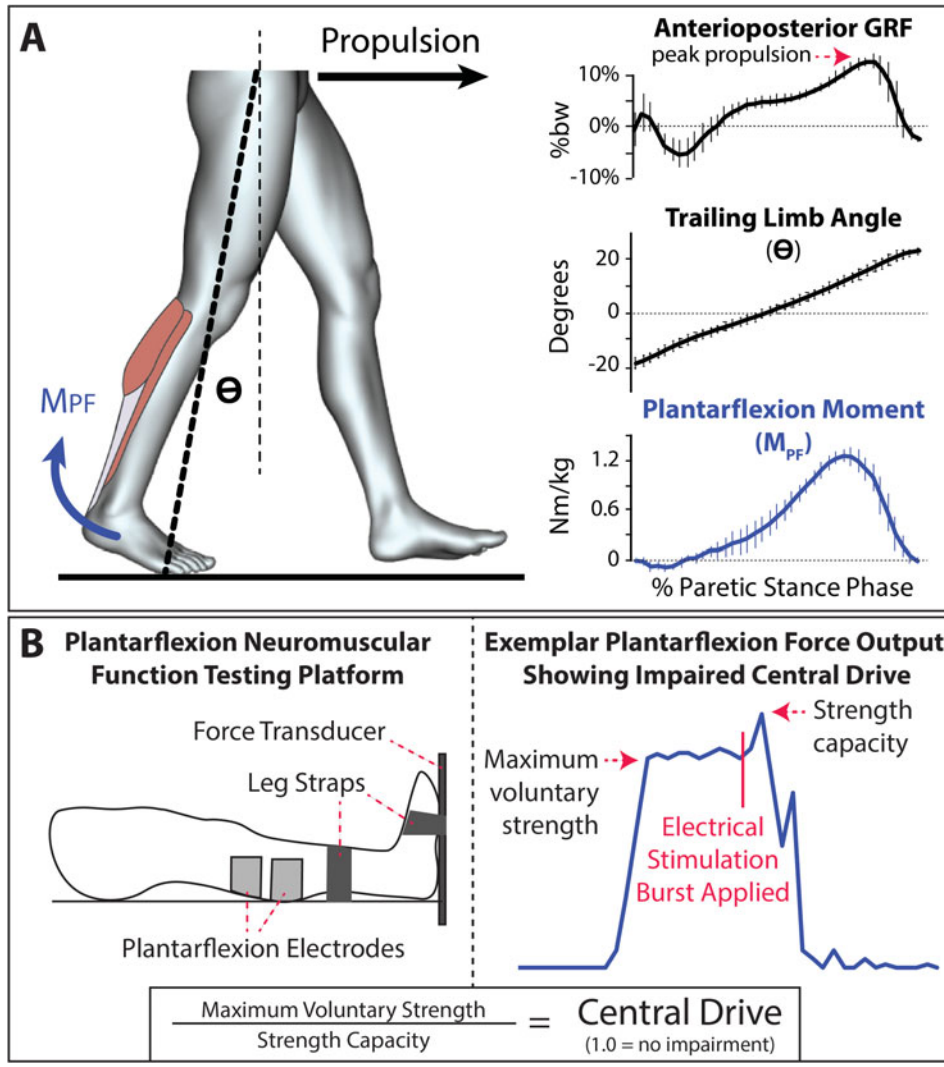

Figure 1. (A) Propulsion is produced when an ankle plantarflexion moment (M_{PF}) is generated and when the limb is oriented behind the body (θ). (B) Combining isometric strength testing with muscle electrostimulation allows assessment of both a muscle's maximum voluntary strength and strength capacity. The ratio of these force measurements is used to compute central drive.

between the paretic trailing limb angle, the neuromuscular function of the ankle plantarflexor muscles, and paretic limb propulsion across speed-based subsets of community-dwelling people poststroke. We hypothesized that, beyond deficits in trailing limb angle and plantarflexion strength capacity, an impaired central drive to the paretic plantarflexors would primarily underlie paretic limb propulsion deficits after stroke.

METHODS

Participants

Forty individuals with chronic stroke (>6 months after stroke) participated in this study. Inclusion criteria were a single cortical or subcortical stroke, sufficient cognitive function to follow instructions and communicate with the investigators, the ability to walk for 4 minutes without orthotic support or the assistance of another individual, passive ankle range of motion to position the ankle in a neutral position with the knee fully extended, and passive hip extension range of motion to extend the hip at least 10°. Individuals were excluded from participating if they had a history of multiple strokes, cerebellar stroke, lower extremity joint replacement, bone or joint problems that limited their ability to walk, neglect or hemianopia, unexplained dizziness during the past 6 months, or chest pain or shortness of breath without exertion. All participants provided written informed consent to participate in this study. All study procedures were approved by the University of Delaware's Institutional Review Board.

Measuring Limb Propulsion During Walking

Kinetic and kinematic data were collected via an 8-camera motion analysis system (Motion Analysis Corp, Santa Rosa, California) as participants walked at their usual self-selected walking speed on a split-belt treadmill (Bertec

Corp, Columbus, Ohio) instrumented with 2 independent 6 degree of freedom force plates capturing at 1080 Hz. Usual walking speed was determined over ground using a 10-m walk test. Before data collection, participants completed a 1-minute treadmill acclimation bout. Treadmill walking speed was reduced if the participants felt that their overground usual walking speed was not a comfortable treadmill walking speed. Previous work has described in detail the gait analysis setup.[15,18] Participants wore an overhead support harness with no body weight support and used a handrail if needed for safety. Kinematic and kinetic data were filtered using a bidirectional fourth-order Butterworth low-pass filter at 6 and 30 Hz, respectively. Propulsive force was determined as the maximum anterior ground reaction force during stance phase,[3,19] and propulsion asymmetry was computed as previously described.[14] The trailing limb angle was computed using kinematic data as previously described.[3] All motion analysis data were processed using commercial software (Visual 3D version 5.0, C-Motion Inc, Germantown, Maryland). Variables were averaged across strides within the 30-second trial.

Muscle Performance Testing

The burst superimposition test was used to assess the maximum voluntary strength, strength capacity, and central drive of the paretic ankle plantarflexor muscles (Figure 1B).[11,13] Participants lay supine on a dynamometer (KIN-COM III, Chattecx Corp, Chattanooga, Tennessee) with the paretic knee fully extended and the ankle in a neutral position. The foot and shank were secured in place by Velcro straps and shoulder restraints were used to ensure that all generated force was captured by the transducer and not reduced due to body displacement. Electrical pulses were delivered using a neuromuscular electrical stimulator (Grass S8800, Grass Technologies, Warwick, Rhode Island) with an SIU8T stimulus isolation unit. Two self-adhesive surface electrodes (Versa-Stim $3'' \times 5''$, CONMED Corp, New York) were placed over the ankle plantarflexors, one proximally and the other on the distal portion of the muscle belly of the calf. The cathode was placed over the widest portion of the muscle belly, covering both the medial and lateral heads of the gastrocnemius. The anode was placed over the distal portion of the gastrocnemius muscle belly. Custom software (LabView 5.1, National Instruments, Austin, Texas) was used to control electrical pulse delivery.

An initial maximal single pulse (600 μs, 135 V) was delivered to the resting muscle. Two seconds after the initial pulse, participants were encouraged to maximally contract their ankle plantarflexors to allow measurement of their maximum voluntary strength. A maximal electrical stimulation burst (600-μs pulse duration, 100-ms train duration, 135 V, 100-Hz train) was delivered 5 seconds following the first pulse when the participants achieved their volitional maximum. The following equation was used to calculate each person's strength capacity, or the force produced with full muscle activation:

$$Strength\ Capacity = Maximum\ Voluntary\ Force + Fstim \quad [1]$$

where *Fstim* is the additional force produced by the muscle after the applied burst of electrical stimulation. A cubic adjustment was then applied to the strength capacity calculation to improve the accuracy and reliability for low levels of volitional force production.[11,13] Central drive to the plantarflexors was calculated as the ratio of these strength measures:

$$Central\ Drive = Maximum\ Voluntary\ Strength\ /\ Strength\ Capacity \quad [2]$$

A central drive value less than 1.0 indicates a deficit in voluntary access to the full force-generating ability of the muscle, and thus a deficit in the central drive to the muscle.

Statistical Analyses

All analyses were conducted using commercial statistical software (SPSS version 24, IBM Corp, Armonk, New York). Alpha was set to 0.05. Means ± standard error are reported for all variables. Between-limb differences in ankle plantarflexor strength capacity, maximum voluntary strength, and central drive were assessed using independent *t* tests. Moderated regression was then used to assess the relationship between paretic propulsion, walking speed, trailing limb angle, and each of the ankle plantarflexor measurements. Maximum voluntary strength was found to be highly correlated with both strength capacity ($r = 0.777$) and central drive ($r = 0.833$), whereas central drive and strength capacity were not correlated ($r = 0.359$). Similarly, walking speed and trailing limb angle were found to be highly correlated ($r = 0.860$).

To avoid model multicollinearity and overfit, maximum voluntary strength and trailing limb angle were excluded from the final model, which evaluated the relationship between paretic propulsion and walking speed, central drive, and strength capacity. All regression assumptions were assessed, and centered variables were used to further minimize multicollinearity. Significant moderation (indicated by a significant interaction) was plotted within ±1 standard deviation of the moderator variables[20,21] and further examined for measurements of paretic and nonparetic limb propulsion and propulsion symmetry across subgroups identified by stratifying study participants based on the moderators.[22,23]

RESULTS

Paretic limb data were available for all 40 participants. Due to technical issues during data collection, nonparetic limb data were available for only 39 participants. Study participants walked, on average, 0.69 ± 0.05 m/s and generated 12.74 ± 0.86% body weight (%bw) of nonparetic limb propulsion and 8.54 ± 0.82%bw of paretic limb propulsion during walking—a 33% difference in propulsion across limbs ($P = 0.001$). Study participants' maximum voluntary plantarflexion strength was, on average, 384 ± 27 Newtons (N) of plantarflexion force produced with their nonparetic limbs and 231 ± 25 N of plantarflexion force produced with their paretic limbs—a 40% difference in plantarflexion force across limbs ($P < 0.001$). The strength capacity of the nonparetic plantarflexors was, on average, 538 ± 23 N, whereas the strength capacity of the paretic plantarflexors was, on average, 433 ± 25 N—a 19% difference in strength capacity across limbs ($P = 0.003$) (Figure 2A). Central drive to the nonparetic ankle plantarflexors was measured to be 70 ± 3%. In contrast, central drive to the paretic

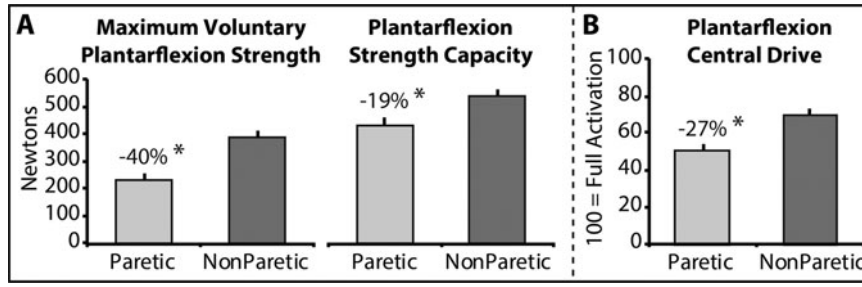

Figure 2. (A) Maximum plantarflexion force produced voluntarily and with a superimposed burst of electrical stimulation for the paretic and nonparetic plantarflexors of 40 people poststroke. (B) Deficits in central drive to the paretic and nonparetic plantarflexors, computed as the ratio of maximum voluntary strength and strength capacity. Error bars are the standard error. *$P < 0.05$.

ankle plantarflexors was measured to be only 51 ± 4%. Between limbs, the paretic plantarflexors had a 27% lower central drive (Figure 2B).

A regression model including only walking speed accounted for 71% of the variance in paretic limb propulsion observed across participants. The addition of ankle plantarflexor muscle function measurements to the model increased the variance explained to 86% (Table 1). Independent of the effect of a slower walking speed ($\beta = -2.123$, $P < 0.001$), deficits in paretic plantarflexor central drive ($\beta = 0.035$, $P < 0.001$) independently contributed to deficits in paretic limb propulsion. In contrast, deficits in paretic plantarflexor strength capacity were not independently related to deficits in paretic propulsion ($\beta = -0.004$, $P = 0.051$). An interaction between walking speed and paretic plantarflexor central drive was observed ($\beta = 0.187$, $P = 0.007$). Examination of the interaction revealed that, in study participants walking at speeds faster than 0.40 m/s, those with a higher central drive to the paretic ankle plantarflexor muscles presented with markedly more paretic limb propulsion (~15%bw) than individuals walking at the same speeds but with less central drive to the paretic plantarflexor muscles (~10%bw). Regardless of central drive impairment, slow walking speed was associated with the lowest levels of paretic propulsion (~5%bw) (Figure 3).

To further explore the interaction between walking speed and paretic plantarflexor central drive impairment, we examined differences in limb propulsion ability across subsets of individuals stratified based on these 2 variables. In brief, we identified the 20 slowest versus 20 fastest participants, then further dichotomized the groups based on paretic plantarflexor central drive impairment. This 2-step subgrouping resulted in four (n = 10) subgroups (see Table 2) and revealed that fast participants (ie, 0.99 ± 0.04 m/s) with a high central drive to the paretic ankle plantarflexor muscles (ie, 77 ± 2%) presented with substantially higher paretic limb propulsion and low propulsion asymmetry than each of the other subgroups (Figure 4A). In contrast, slow participants (ie, 0.37 ± 0.06 m/s) with a low central drive to the paretic ankle plantarflexor muscles (ie, 26 ± 3%) presented with the highest propulsion asymmetry (Figure 4B).

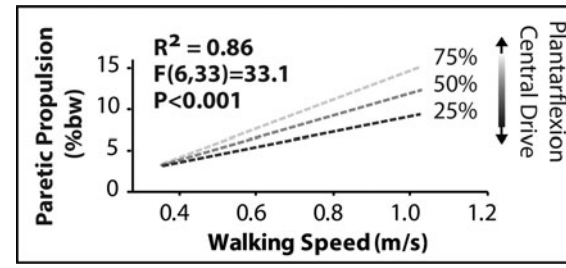

Figure 3. Relationship between paretic limb propulsion and walking speed is moderated by the central drive to the paretic plantarflexors. Simple slopes are calculated using ±1 standard deviation of the moderator variables—walking speed and central drive to the paretic plantarflexors.

Table 1. Models of Paretic Limb Propulsive Force During Walking After Stroke

Model Statistics				Predictor Statistics		
Model	R^2	F	P	**Predictors**	β	P
Speed (includes only speed)	0.71	93.51	<0.001	Constant	−0.471	<0.001
				Walking speed	13.007	<0.001
Speed+ (includes plantarflexion function variables)	0.86	33.10	<0.001	Constant	3.418	<0.001
				Walking speed	−2.123	<0.001
				Central drive[a]	0.035	<0.001
				Strength capacity[b]	−0.004	0.051
				Central drive × walking speed	0.187	0.007
				Central drive × strength capacity	0.000	0.114
				Walking speed × strength capacity	0.014	0.073

[a]Assessed as the ratio of maximum voluntary strength to strength capacity.
[b]Maximum voluntary strength of the ankle plantarflexor muscles, assessed using dynamometry and supramaximal neuromuscular electrical stimulation.

Table 2. Differences in Propulsive Ability for Walking Speed and Plantarflexor Central Drive Subgroups[a]

Subgroup	Walking Speed, m/s	Central Drive, %	Limb Propulsion, %bw		Propulsion Asymmetry, %
			P	NP	
Fast-high	0.99 ± 0.04	77 ± 2	14.58 ± 1.07	15.79 ± 1.74	−4 ± 14
Fast-low	0.93 ± 0.08	40 ± 3	9.41 ± 1.11	15.08 ± 1.97	32 ± 7
Slow-high	0.48 ± 0.07	60 ± 5	6.13 ± 1.21	9.93 ± 1.14	35 ± 13
Slow-low	0.37 ± 0.06	26 ± 3	4.05 ± 0.85	10.17 ± 1.14	60 ± 7

Abbreviations: NP, nonparetic; P, paretic; %bw, % body weight.
[a]Mean ± standard error are reported for each variable.

DISCUSSION

A compensatory reliance on the nonparetic limb to generate forward propulsion is a hallmark characteristic of poststroke hemiparesis that is associated with slow walking speeds[2,4,24] and a high energy cost of walking.[14,20,25] An increase in propulsion symmetry driven by an increase in propulsion output from the paretic limb is an essential rehabilitation goal for gait restoration after stroke,[15,24] yet an elusive objective due to our limited understanding of the neuromuscular deficits underlying propulsion deficits across the heterogeneous poststroke population. This study elucidates the relationship between the neuromuscular function of the paretic ankle plantarflexor muscles—the primary generators of positive power during walking[7]—and deficits in paretic propulsion across study participants with different walking speeds.

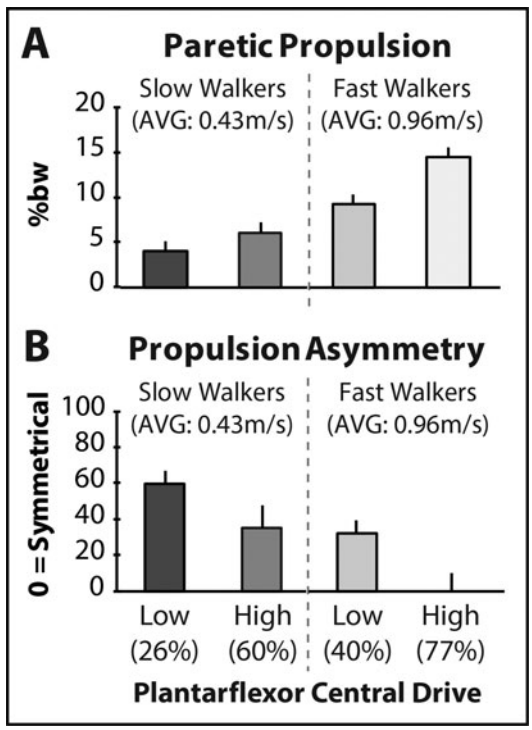

Figure 4. (A) Paretic limb propulsion and (B) interlimb propulsion asymmetry ratio for four (n = 10) subgroups created by stratifying study participants based on their walking speed and central drive to the paretic plantarflexors. Error bars are the standard error.

Beyond deficits in plantarflexion strength capacity, a reduced central drive to the paretic plantarflexors explains (i) deficits in paretic propulsion during hemiparetic walking and (ii) the compensatory reliance on the nonparetic limb for propulsion, especially in fast walkers. Individuals in the chronic phase of stroke recovery appear to have untapped potential for plantarflexion force generation that may be exploitable during gait training to retrain more physiological walking patterns. Indeed, our previous work shows that 12 weeks of gait training with the addition of functional electrical stimulation (FES) to the paretic plantarflexor muscles can facilitate therapeutic increases in paretic plantarflexion moments during walking, whereas gait training without FES does not result in an increase in paretic plantarflexion moments.[6] Further development and study of interventions that can help people poststroke fully access the force-generating potential of their ankle plantarflexor muscles is warranted. Moreover, the development and clinical translation of diagnostic tools that can assess the extent and nature of ankle plantarflexor dysfunction, as well as monitor changes resulting from intervention, is needed.

Independent of differences in walking speed across study participants, a reduced central drive to the paretic ankle plantarflexors was found to be an independent contributor to paretic propulsion deficits, whereas plantarflexor strength capacity was not. Our observation of deficits in paretic plantarflexor central drive being associated with increased propulsion asymmetry in both slow and fast walkers further highlights the importance of considering central drive when prescribing interventions for the heterogeneous poststroke population. That is, being able to more fully activate the paretic ankle plantarflexor muscles during isometric strength testing is indicative of being able to generate more symmetrical propulsive forces from the lower limbs during walking. Taken together, these findings suggest that the combination of dynamometry with neuromuscular electrical stimulation has the potential to play an important role in examining the nature of clinically assessed plantarflexor muscle weakness. Given that central drive deficits are present in other muscle groups that play crucial roles during different functional movements (eg, the knee extensors[26] and elbow flexors[27]), examining the relationship between the neuromuscular function (ie, strength capacity and central drive) of these muscles and their respective functional movements could provide important diagnostic information for the prescription of targeted interventions.

Interestingly, although deficits in paretic plantarflexor central drive were associated with reduced paretic propulsion

during walking, some individuals with large central drive deficits still achieved similarly fast walking speeds as those with near-normal paretic plantarflexor central drive. Indeed, the subset of fast participants who presented with low paretic plantarflexor central drive appear to have been able to leverage a compensatory reliance on the nonparetic limb to meet the propulsive demands of walking at a fast speed. That is, although reduced central drive to the paretic plantarflexor muscles may be a primary impairment underlying paretic propulsion deficits, it does not necessarily result in reduced walking speed because fast walking speeds may be achieved through nonparetic limb propulsion.[2] However, as previously noted, increasing walking speed through asymmetric locomotor strategies is associated with a high energy cost of walking.[20]

Clinically, muscle weakness is measured as the maximum voluntary force; however, individuals with the same maximum voluntary force may have a very different balance in the underlying deficits (ie, reduced strength capacity vs reduced central drive). With potentially different underlying deficits, well-matched and targeted treatments are required. Indeed, beyond more traditional muscle-strengthening approaches,[28] low voluntary plantarflexion force production can be increased during task-specific gait training by facilitating access to latent plantarflexion strength or supplementing low plantarflexion strength capacity. For example, as noted previously, FES to the ankle plantarflexor muscles during poststroke gait training has been shown to improve paretic ankle plantarflexor moment generation[6] and paretic propulsion.[15]

Limitations

Although muscle strength is the product of both muscle activation and the volume and contractile properties of the muscle fibers, this investigation did not include measurements of the latter. Our finding that paretic plantarflexor strength capacity was not an independent explanatory factor in our analyses when accounting for central drive deficits suggests that poststroke changes in these properties are not primary determinants of impaired paretic propulsion; however, this study cannot rule out these changes as potential contributing factors. Moreover, isometric muscle strength likely does not fully reflect the dynamic muscle function required during walking. Measurements of muscle power may have proven to be more related to propulsion function. Another limitation of this study is that our method for measuring strength capacity assumes that *Fstim* maximally activates the muscle's residual force-generating capacity. It is possible that, for some individuals, higher levels of $Fstim$ may have been required to measure their true maximum force-generating capacity.

A comprehensive report of differences in locomotor biomechanics (eg, hip, knee, and ankle kinetics and kinematics) across study participant subsets walking at different speeds and with different plantarflexion impairments was beyond the scope of this study. Such a detailed analysis would build on our finding of a compensatory reliance on the nonparetic limb for forward propulsion in specific subsets by revealing differences in inter- and intralimb gait strategies. Moreover, although this study accounts for differences in walking speed across participants, it cannot speak to the ability of individuals from different subsets to modulate their walking speeds. A follow-up study could investigate how different plantarflexion impairments influence the ability to modulate walking speed, as well as other clinically meaningful locomotor variables (eg, the energy cost of walking and gait stability).

CONCLUSIONS

The present study highlights the importance of central drive to the paretic plantarflexor muscles to paretic limb propulsion. However, it is possible that some individuals poststroke may require a combination of interventions that increase both strength capacity and central drive. Beyond suggesting that clinicians should consider the nature of their patient's muscle weakness when prescribing gait training interventions, this research motivates the clinical translation of interventions that can target central drive (eg, neuromodulatory interventions, including FES) and innovative intervention studies to determine whether the differential targeting of central drive versus strength capacity produces different outcomes.

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Nominating Committee Member (3-year term)

Interested members are encouraged to speak with former and current position holders to learn more about these opportunities to serve the Academy. Contact information for the board positions can be found at www.neuropt.org/about-us/leadership and for the SIG position under each SIG listing on the Special Interest Group tab.

More information about each position, eligibility requirements and the nomination form is available at www.neuropt.org/members/ nomination-2020. You must log into the site to see this information.

If you have questions about the open positions or the process please contact a member of the Nominating Committee:
Nora Fritz at nora.fritz@wayne.edu
Tara McIsaac at tmcisaac@atsu.edu
Alicia Flach at adflach@mailbox.sc.edu