Paretic propulsion as a measure of walking performance and functional motor recovery post-stroke: A review

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ARTICLE INFO

ABSTRACT

Background: Although walking speed is the most common measure of gait performance post-stroke, improved walking speed following rehabilitation does not always indicate the recovery of paretic limb function. Over the last decade, the measure paretic propulsion (Pp, defined as the propulsive impulse generated by the paretic leg divided by the sum of the propulsive impulses of both legs) has been established as a measure of paretic limb output and recently targeted in post-stroke rehabilitation paradigms. However, the literature lacks a detailed synthesis of how paretic propulsion, walking speed, and other biomechanical and neuromuscular measures collectively relate to post-stroke walking performance and motor recovery.
Objective: The aim of this review was to assess factors associated with the ability to generate Pp and identify rehabilitation targets aimed at improving Pp and paretic limb function.
Methods: Relevant literature was collected in which paretic propulsion was used to quantify and assess propulsion symmetry and function in hemiparetic gait.
Results: Paretic leg extension during terminal stance is strongly associated with Pp. Both paretic leg extension and propulsion are related to step length asymmetry, revealing an interaction between spatiotemporal, kinematic and kinetic metrics that underlies hemiparetic walking performance. The importance of plantarflexor function in producing propulsion is highlighted by the association of an independent plantarflexor excitation module with increased Pp. Furthermore, the literature suggests that although current rehabilitation techniques can improve Pp, these improvements depend on the patient's baseline plantarflexor function.
Significance: Pp provides a quantitative measure of propulsion symmetry and should be a primary target of post-stroke gait rehabilitation. The current literature suggests rehabilitation techniques that target both plantarflexor function and leg extension may restore paretic limb function and improve gait asymmetries in individuals post stroke.

1. Introduction

Stroke is a leading cause of long-term disability in the United States [1], with 50% of stroke survivors over age 65 suffering from hemiparesis [2]. Individuals with post-stroke hemiparesis typically exhibit impaired walking performance, characterized by slow walking speeds and asymmetrical spatiotemporal, kinematic and kinetic patterns [3]. Since the ability to walk is important for independent living and maintaining a high quality of life, improving gait performance is the primary rehabilitation goal in the first year post-stroke [4]. Walking speed is the most common measure of gait performance post-stroke [5,6] due to its strong correlation with an individual's stage of motor recovery [5,7]. However, improved walking speed following rehabilitation is not always indicative of recovery of paretic leg function as speed improvements may also be the result of compensations provided by the non-paretic limb. For example, asymmetric kinetic patterns characterized by greater force production by the non-paretic limb can be observed even when hemiparetic individuals achieve speeds associated with community ambulatory walking ability ($\geq$ 0.8 m/s) [6]. This kinetic asymmetry highlights that underlying neurological impairments can persist despite restoration of near normal walking speeds.

Generating the forces required to propel the body forward during walking has been defined as an essential requirement of gait [8], and

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greater propulsive ground reaction forces (GRFs) are required to achieve faster walking speeds [9]. These propulsive forces are predominantly produced by the ankle plantarflexors [10], which play a critical role in gait as primary providers of both vertical support and forward propulsion during the second half of stance [10]. Individually, the biarticular gastrocnemius provides energy to the leg to initiate swing while the uniarticular soleus delivers energy to the trunk, and thus to the center of mass (COM), to provide body support against gravity and forward propulsion [10]. Plantarflexor weakness is a common impairment in hemiparetic individuals and has been shown to limit gait speed [11]. In addition to impaired foot dorsiflexion and eversion, post-stroke hemiparesis often impairs plantarflexion on the paretic side, which leads to a reduction in the propulsive force produced by the paretic limb relative to the non-paretic limb. Thus, improvement in propulsion symmetry following rehabilitation would suggest recovery of paretic limb neuromuscular function. However, since walking speed alone does not measure paretic limb function, additional metrics are needed to more completely quantify neurological recovery of the paretic limb.

One such metric is paretic propulsion (Pp), which was developed to assess propulsion symmetry as a quantitative measure of the coordinated output from the paretic leg [6]. Pp describes the contribution of the paretic leg in propelling the COM forward during walking and is defined as the percentage of propulsion performed by the paretic leg [6]. The percentage of propulsion generated by the paretic leg is calculated by dividing the total propulsive impulse (i.e., the positive time integral of the anterior/posterior force curve) performed by the paretic leg by the sum of the propulsive impulses performed by both legs, such that a Pp of 50% (Pp = 0.5) indicates perfect symmetry [6]. A Pp value less than 0.5 indicates the paretic limb is producing less propulsion compared to the non-paretic limb. Pp has been shown to strongly correlate with hemiparetic severity based on the Brunnstrom motor recovery stages [12] (Fig. 1, [6]), suggesting improvements in propulsion symmetry may lead to improved motor function in the paretic limb. Consequently, Pp has emerged as a common mechanics-based measure of hemiparetic walking performance [13–16] and recently therapies have targeted propulsion as a primary outcome [17] and predictor of gait outcomes [18].

Despite the considerable research investigating propulsion symmetry and its relationship with walking speed and other biomechanical and neuromuscular measures in post-stroke gait [6,17,19–25], the literature lacks a detailed analysis of how these metrics are collectively related to post-stroke walking performance and motor recovery. Thus, the goal of this review is to summarize factors associated with an individual's ability to generate paretic propulsion and assess how this information can be used to inform therapies designed to improve recovery of paretic limb function. Section 2 describes the associations between paretic propulsion and other measures of walking performance. Section 3 reviews the factors that contribute to the ability to produce paretic propulsion. Finally, Section 4 assesses the effectiveness of current rehabilitation techniques and suggests future directions for rehabilitation to improve both paretic propulsion and walking speed.

2. Paretic propulsion and walking performance measures

Achieving walking speeds associated with community ambulation post-stroke is an important rehabilitation goal since self-selected walking speed is strongly associated with community involvement, quality of life, and mortality across healthy and pathological populations [26–28]. Although walking speed is a powerful measure of overall walking ability, it does not provide insight into specific neurologic impairments as evidenced by the relatively weak relationship between walking speed and hemiparetic severity (as rated by Brunnstrom stages) [6,19]. However, measures of gait asymmetry, including Pp and step length asymmetry metrics, strongly correlate to hemiparetic severity and therefore may be more appropriate for characterizing neurologic recovery than walking speed alone [6,19]. This section will review the findings of the several studies [6,19,22,29–31] that have investigated the relationships between propulsion symmetry, step length symmetry, and walking speed in hemiparetic gait to gain insight into how these walking performance measures relate to motor recovery post-stroke.

2.1. Propulsion symmetry and walking speed

The propulsive forces generated by each limb contribute to the overall acceleration of the body during gait and are therefore critical to walking speed [9]. In individuals with hemiparesis it is common for a disproportionate amount of the propulsive forces to be produced by the non-paretic limb during walking, particularly in individuals with moderate or severe impairment [6]. Patients with the most severe motor impairment post-stroke also exhibit greater reliance on the non-paretic limb for weight-bearing and balance control [32,33], which
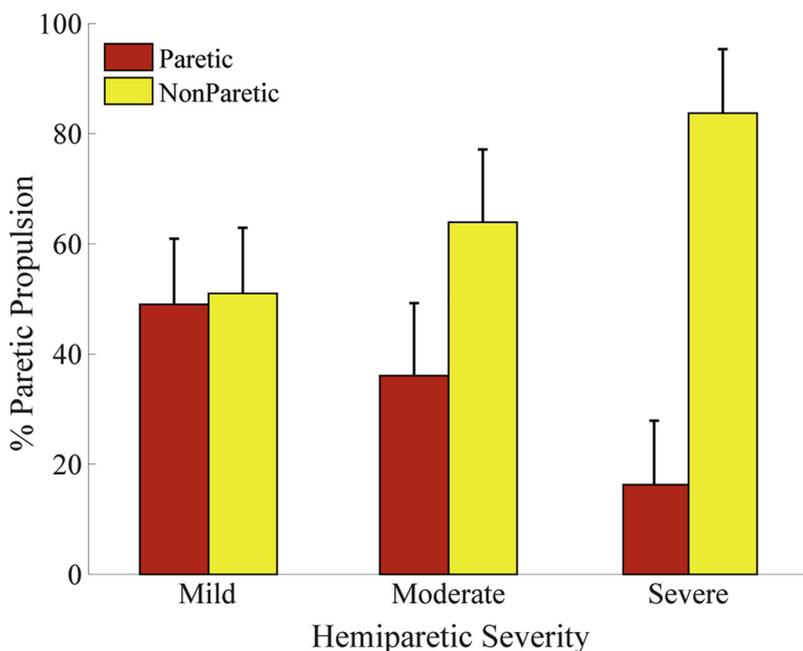

Fig. 1. Comparison of the average propulsion (expressed as a percentage of the total propulsion) generated by the paretic (red bars; Pp) and nonparetic legs (yellow bars) of subjects of differing hemiparetic severity. There are substantial differences in the percent of Pp in those with severe and moderately severe hemiparesis when compared with those with mild severity. Compensation by the nonparetic leg is noticeable in the asymmetry shown by the moderate and severe groups. Error bars indicate SD for each variable. Adapted from [6]. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
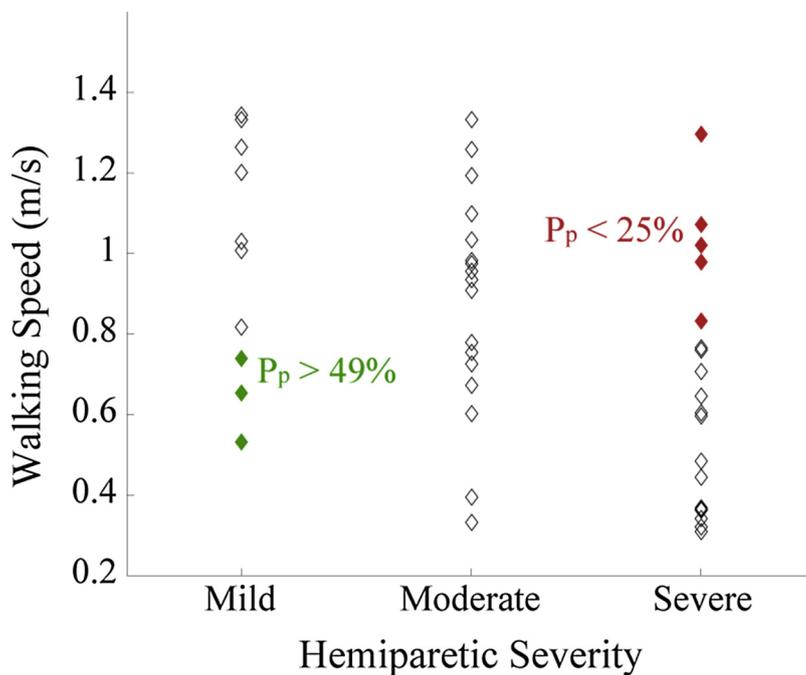
Fig. 2. Individual walking speed data for subjects in each of the hemiparetic severity groups. Although there is a weak overall trend for walking speed to decrease with severity, the variability is substantial and the speed ranges in each of the groups overlap substantially. Five participants with severe hemiparesis are still able to achieve normal walking speeds (> 0.8 m/s), but all demonstrate substantial impaired Pp (filled red markers). Conversely, 3 participants with mild hemiparesis walked < 0.8 m/s but have normal Pp (> 49%; filled green markers). Adapted from [6]. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

suggests functional asymmetries between limbs may reflect a compensation strategy. However, although a moderate positive correlation between Pp and walking speed has been established in group data, some individuals achieve community walking speeds despite generating greatly reduced propulsion on their paretic limb compared to their non-paretic limb (Fig. 2) [6]. Therefore, Pp could be used in conjunction with walking speed to identify what, if any, compensatory strategy is being used [6]. For example, an individual walking at a speed associated with community ambulatory ability but with low Pp must be using some compensatory mechanism to achieve their walking speed. On the other hand, a few individuals from the same study had symmetric Pp and mild severity but walked at limited community walking speeds (0.4-0.8 m/s), which suggests an alternative impairment, rather than reduced paretic limb propulsion, limits walking speed in these individuals [6]. A subsequent study reported that Pp is capable of differentiating between limited community and community walkers, but does not distinguish between household (< 0.4 m/s) and limited community speed classifications [34]. These studies suggest that Pp captures a different aspect of post-stroke walking performance than walking speed.

In addition to evaluating post-stroke walking at self-selected speeds, studies have investigated walking performance at speeds faster than the subject's preferred pace [29,31]. One study found no systematic effect of increased walking speed on changes in Pp in their cohort of 46 hemiparetic subjects [29], supporting the idea that walking speed and Pp provide independent measures of functional recovery post-stroke. Others have quantified the peak propulsive forces generated by the paretic and non-paretic limb at self-selected and fast walking speeds and found that subjects increased propulsive forces on both limbs to achieve the faster pace [31]. However, linear regression analysis revealed only changes in non-paretic propulsive forces independently contributed to increased walking speeds [31]. This suggests subjects often rely on their non-paretic limb to achieve walking speeds faster than their preferred pace. Similarly, standing balance has been shown to improve in individuals post-stroke without improvements in paretic muscle function and with continued reliance on the non-paretic limb [33,35]. Thus, targeted training of the paretic limb may be necessary to improve paretic limb function and achieve symmetric propulsion with improvements in walking speed.

2.2. Paretic propulsion and step length asymmetry

In addition to propulsion asymmetry, step length asymmetry between the paretic and non-paretic limb is often observed in hemiparetic gait [19,22,29,30,36–39]. Step length asymmetry can be quantified using either the ratio of the paretic step length to the non-paretic step length (i.e., step length ratio or SLR) [19] or the ratio of the paretic step length to the sum of the paretic and non-paretic step lengths (i.e., paretic step ratio or PSR) [22]. The limitation of SLR is that it can become indeterminate or negative for individuals with severe impairment who exhibit a zero or negative non-paretic step length, respectively. In these cases, PSR will remain positive, allowing for easier interpretation and comparison between individuals. Large variability in step length asymmetry is observed among hemiparetic subjects: some individuals walk with longer paretic steps, some take longer non-paretic steps, and still others walk with symmetric step lengths [19]. Like Pp, there is only a weak relationship between step length asymmetry and walking speed in post-stroke subjects as a group [19,36–38], suggesting measures of step length asymmetry may also provide different information about post-stroke walking performance than walking speed alone.

There is evidence for a moderate to strong relationship between measures of step length asymmetry and propulsion asymmetry [19,29]. Balasubramanian and colleagues reported a strong negative correlation between SLR and Pp ($r = -0.785$; Fig. 3), suggesting that individuals who produce greater propulsion in their paretic limb take longer non-paretic steps than paretic steps [19]. Indeed, in the same study three out of the four subjects who walked with longer non-paretic steps than paretic generated over half of their propulsive forces on their paretic limb [19]. The study concluded that a high SLR is due in large part to relatively greater propulsion on the non-paretic limb [19], suggesting SLR and Pp may be mechanically related. This strong correlation between step length asymmetry and Pp has been observed at both self-selected and fastest-comfortable walking speeds [29], indicating the relationship between step length and propulsion is independent of gait speed.

Studies have leveraged this relationship between step length asymmetry and propulsion to identify mechanisms underlying walking performance in post-stroke gait [22,30,39]. Peterson et al. found that an individual's step length asymmetry group (either high, symmetric or
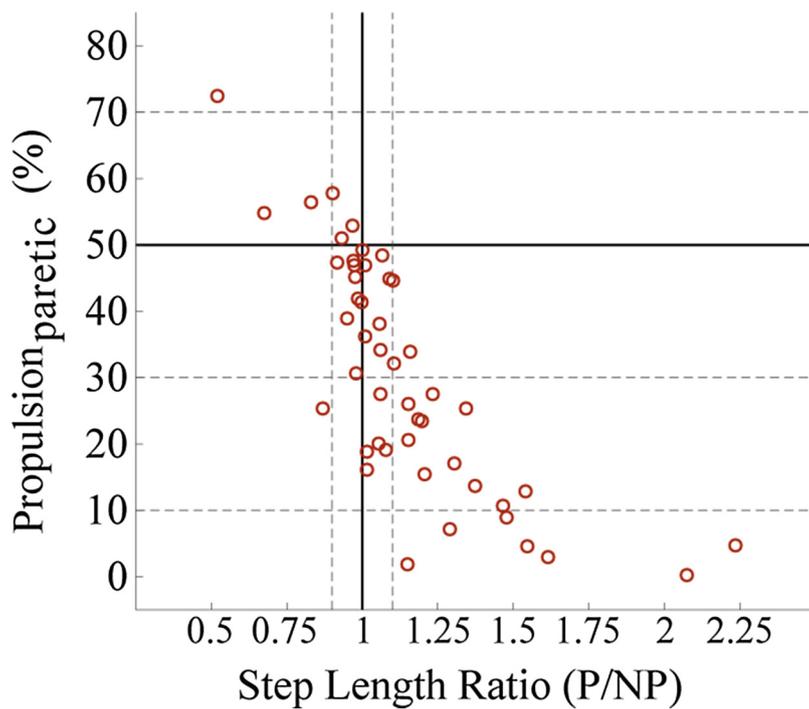
Fig. 3. Relationship between step length ratio and Pp. The solid vertical line indicates symmetric steps (SLR = 1), vertical dashed lines indicate the SLR subdivisions at SLR equal to 0.9 and SLR = 1.1. Solid horizontal line indicates symmetric propulsive force generation by the paretic leg (Pp = 50%), horizontal dashed lines indicate differing levels of paretic leg propulsion (10%, 30%, and 70% PP). Step length ratio quantifies step length asymmetry and Pp quantifies the contribution of the paretic leg to the task of propulsion. Note the decreasing Pp as SLR increases. Abbreviations: Pp, paretic leg propulsion (in percent); NP, non-paretic leg; P, paretic leg. Adapted from [19].

low PSR) determined the relationship between the paretic ankle moment impulse and propulsion impulse during pre-swing [22]. Individuals with high PSR who exhibit relatively longer paretic steps and relatively reduced paretic propulsion achieve smaller paretic leg extension, which is associated with a smaller ankle moment contribution to the anterior-posterior GRF [22]. In addition, Allen et al. found that while all hemiparetic subjects in their sample exhibited some level of plantarflexor impairment, the degree of plantarflexor impairment and the compensatory mechanism used to overcome the reduced paretic plantarflexor function were related to the level of step length asymmetry [30]. The high PSR group was the least able to generate paretic propulsion and compensated by producing greater plantarflexor and knee extensor moment impulses on their non-paretic limb [30]. The symmetric PSR group relied on the paretic and non-paretic hip flexors to accelerate the leg forward during pre-swing and swing to increase step length. However, the consequence of greater paretic hip flexor impulse was reduced Pp, as unlike the non-paretic plantarflexors, the paretic plantarflexors were unable to compensate for the loss of propulsion [30]. Individuals with a low PSR were unable to generate sufficient energy to the paretic leg during pre-swing due to impaired paretic plantarflexors and hip flexor output, leading to a short paretic swing time and decreased propulsion on the non-paretic limb. Finally, Roerdink et al. demonstrated that the combined contributions of asymmetries in trunk progression (indicative of propulsion) and forward foot placement (indicative of swing capacity) influence overall step length asymmetry [39]. For example, one subject had an 18% asymmetry in trunk progression (greater during the paretic step), a −10% asymmetry in forward foot placement (greater during the non-paretic step) but just a 4% asymmetry in step length, which fell within the step length asymmetry range of control subjects [39]. Therefore, a small step length asymmetry can be achieved by trunk progression and foot placement asymmetries that are of similar magnitude but in opposite directions, which suggests step length symmetry alone does not indicate gait symmetry [39]. Thus, both a measure of propulsion symmetry, such as Pp, and measures of step length asymmetry are needed to gain insight into patient-specific paretic muscle impairments and the compensatory mechanisms contributing to hemiparetic walking performance.

3. Factors influencing paretic propulsion

Several biomechanical and neuromuscular variables, including joint kinematics, kinetics and muscle activity, contribute to propulsion generation. Several studies have evaluated the relationship between paretic propulsion and one or more of these variables to provide insight into the mechanisms of paretic propulsion generation. Understanding the relative contribution of these variables to paretic propulsion may help identify specific rehabilitation targets to improve paretic limb function during post-stroke gait.

3.1. Kinematic variables

A critical kinematic variable associated with propulsion generation is leg extension during terminal stance [17,22,40–42]. Leg extension has been quantified using two similar, yet slightly different, metrics: 1) the angle between the vertical axis and a line from the pelvis COM to the foot COM [22], and 2) the angle between the vertical axis and a line from the center of pressure to the greater trochanter (trailing limb angle) [17,40]. Regardless of metric, leg extension was positively correlated with propulsion in both healthy [40] and hemiparetic populations [17,22,41]. Increased leg extension during terminal stance is associated with greater anteriorly directed forces, and thus greater propulsion (Fig. 4). A proposed mechanism to improve Pp through increased paretic leg extension is to target the recruitment of the paretic soleus, vasti, and gluteus maximus [22], which are primary contributors to hip and knee extension during gait [43,44]. Since leg extension angle is sensitive to PSR [22], targeting leg extension to improve Pp may be most effective in individuals with a high PSR, who achieve less paretic leg extension [22] and produce lower Pp [19,22] compared to individuals with a low or symmetric PSR.

3.2. Kinetic variables

In addition to leg extension angle, the ankle moment has been identified as a significant predictor of the propulsive ground reaction force in both healthy [40] and hemiparetic [41] populations. Hemiparetic individuals produce lower internal plantarflexor moment and propulsive force impulses (i.e., the time integral of the moment and
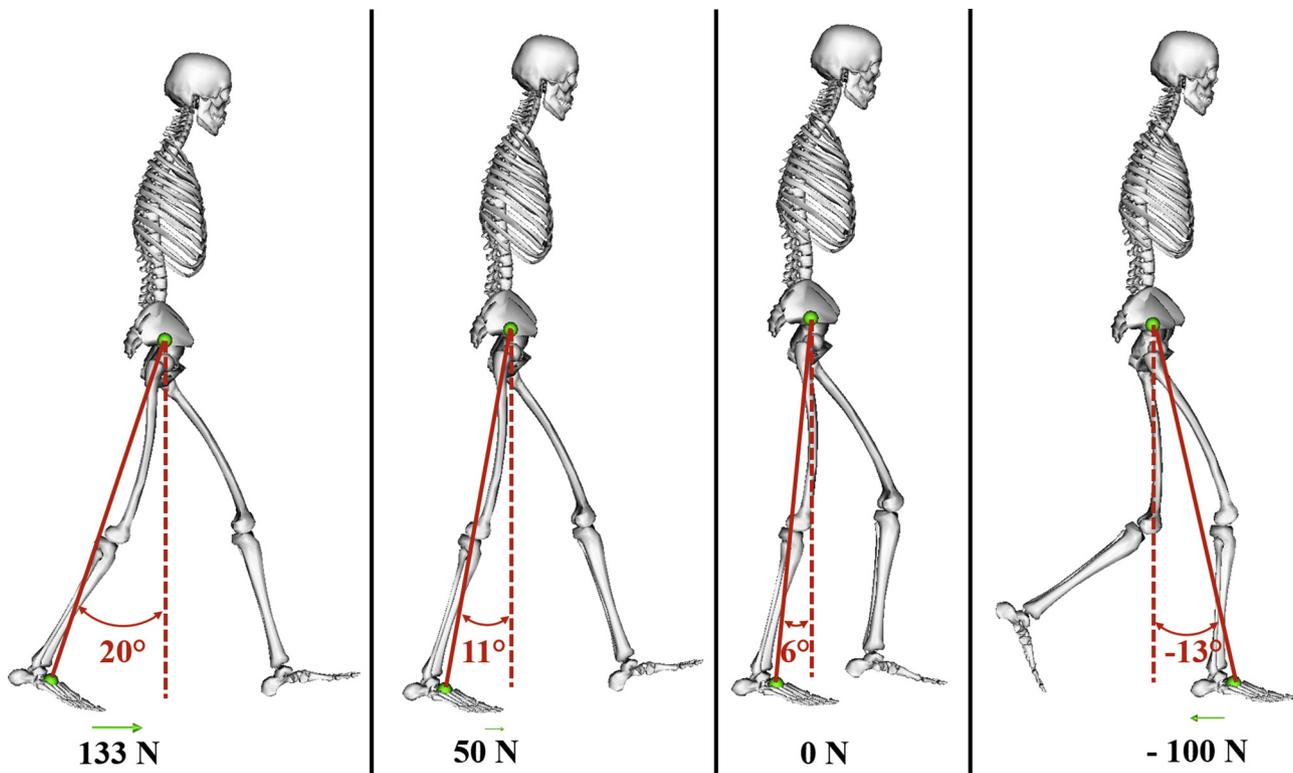
Fig. 4. Greater leg extension angles are associated with greater anterior ground reaction forces. Leg extension angle was calculated as the angle between the vertical axis and a line from the pelvis COM to the foot COM [22].

force, respectively) on their paretic limb compared to controls, particularly during pre-swing [30]. The contribution of the plantarflexor moment impulse to the propulsion impulse [22] and the degree of plantarflexor impairment [30] are associated with an individual's step length asymmetry. Individuals who walk with a high PSR exhibit the most severely impaired plantarflexor moment [30] and Pp [19,22,30]. Together, these findings highlight the key role of the plantarflexors in generating propulsion and contributing to overall walking performance.

The relationships between Pp and knee and hip joint kinetics are also influenced by kinematics. During pre-swing, the knee extensors generate an internal extension moment that is associated with the propulsion impulse. However, the knee extensor moment and propulsion impulses were positively related in non-paretic and control limbs but negatively related in paretic limbs [22]. The position of the foot relative to the pelvis (i.e., leg extension angle) affects the knee extensors' ability to propel the COM forward. Therefore, leg extension angle and PSR likely alter the relationship between knee extension moment and propulsion [22]. Additionally, hemiparetic individuals with a symmetric PSR generate increased hip flexion moments to overcome paretic plantarflexor weakness, yet still exhibit a reduced propulsion impulse by the paretic limb compared to control limbs [30]. The Pp deficits in individuals with a symmetric PSR may be due to excessive internal hip flexion moments, which have been associated with reduced propulsion impulse [22]. Finally, the paretic hip abduction moment is related to lateral foot placement and the capacity to transfer weight to the paretic limb [45]. The positive relationship between the peak vertical and anterior paretic GRFs during stance suggests weight bearing on the paretic limb may be associated with Pp [45]. Therefore, both sagittal and frontal plane kinetics influence the ability to produce propulsion on the paretic limb.

3.3. Muscle activity

The ability to produce propulsion during the paretic stance phase is strongly related to the net activity of the paretic and non-paretic muscles during paretic pre-swing. This net activity occurs because the propulsion of the COM produced by the paretic muscles can be negated by simultaneous negative acceleration (braking) of the COM produced by the non-paretic muscles during the non-paretic limb's weight acceptance phase. Paretic plantarflexor muscle activity is strongly associated with Pp, with increased gastrocnemius and soleus activity relating to increased propulsion of the paretic limb [20,46]. Furthermore, increased activity in the paretic soleus, gastrocnemius, and gluteus medius is associated with improved functional walking status [46,47]. In contrast, individuals with poorer functional walking ability exhibit increased activity in the non-paretic hamstrings during paretic pre-swing (non-paretic weight acceptance), which causes a net negative anterior-posterior acceleration of the COM due to the increased braking contribution from the non-paretic hamstrings [46,47]. The net negative anterior-posterior acceleration during paretic pre-swing reduces the overall paretic propulsive GRF. Early activation of the paretic tibialis anterior and the rectus femoris during pre-swing is inversely related to propulsion [20], implying that coordination between the paretic flexors and extensors is needed to maximize propulsion mechanics.

The ability to produce Pp is also associated with the complexity of an individual's motor strategy, with fewer independent motor modules (or groups of co-excited muscles) associated with lower Pp [48]. Specifically, the presence of an independent plantarflexor module has been shown to be associated with greater Pp [48]. When plantarflexor activity is merged with the early stance extensor module, premature plantarflexor activation contributes to increased early stance braking while prolonged activation of the rectus femoris and vasti increases late stance braking (Fig. 5). These impaired motor strategies ultimately reduce the net propulsion produced on the paretic limb [48,49]. However, even if the plantarflexor module is independent, individuals often
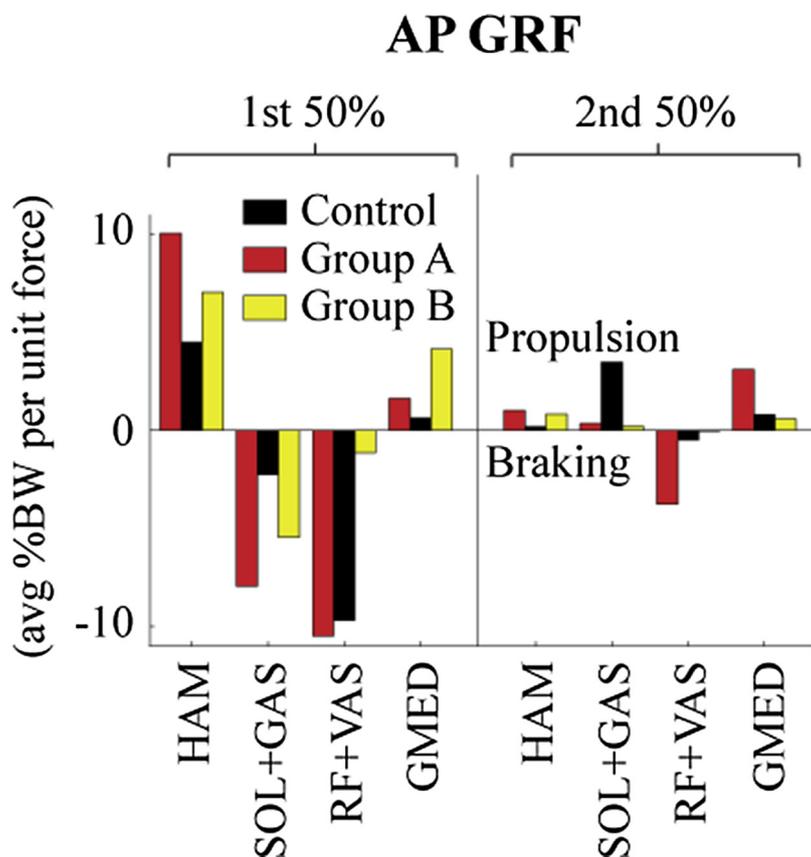
AP GRF

Fig. 5. Average contributions to the normalized GRF (GRF normalized by body weight (BW); contribution computed per unit muscle force) during the 1st and 2nd half of stance in the AP direction for the non-impaired control (black), Group A (red, merged plantarflexor module) and Group B (yellow, independent plantarflexor module) simulations. Hemiparetic values in Groups A and B correspond to muscle groups on the paretic leg. Muscle groups: hamstrings (HAM), soleus (SOL), gastrocnemius (GAS), rectus femoris (RF), vasti (VAS), and gluteus medius (GMED). During the 2nd half of stance, Group A's increased braking from RF + VAS coupled with reduced propulsion from SOL + GAS compared to controls leads to the reduced net propulsion observed when the plantarflexor module is merged with the early stance extensor module (RF + VAS). Adapted from [49]. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

have impaired forward propulsion generation due to premature plantarflexor activity [49], emphasizing the importance of both appropriate plantarflexor activation magnitude and timing in producing propulsion.

4. Current rehabilitation efforts and future directions

Several rehabilitation approaches, including exercise therapy, gait training techniques, functional electrical stimulation, and orthotic devices, have been designed to improve walking capacity in individuals post-stroke. A review of these treatment methods revealed that outcomes are comparable across treatments, which primarily resulted in patients achieving a limited community walking level [50]. The primary outcome measure of most studies evaluating these rehabilitation methods is walking speed. However, more recently the effects of rehabilitation methods on paretic limb function (i.e., paretic propulsion) have also been reported. This section will review the efficacy of these treatments from studies that report a measure of paretic limb function as a treatment outcome.

Gait training typically involves treadmill based training with or without body weight support to promote desired kinematics and spatiotemporal patterns, which is then followed by transference of newly trained skills to overground walking [51]. The overground walking portion of the training focuses on endurance and dynamic balance while walking over various terrains and negotiating obstacles. The majority of participants gain moderate improvements in walking speed (0.12 to 0.30 m/s), yet do not achieve functional community walking speeds [50]. Even when community walking speeds were achieved following body weight supported treadmill training, Pp did not improve [52]. A recent study identified the underlying mechanisms that distinguish individuals who achieve a clinically meaningful change in self-selected walking speed (≥ 0.16 m/s, i.e., responders) with gait training from those who do not (non-responders) by evaluating changes in measures of motor control, balance, functional walking ability, and endurance due to gait training [23]. Responders demonstrated

significant improvements in all measures except step length asymmetry and daily step activity. Although responders significantly improved Pp as a result of training, there was no association between the change in Pp and the change in self-selected walking speed due to training for responders, suggesting some responders used alternative compensatory mechanisms to achieve faster walking speeds. However, the non-responders' change in self-selected walking speed was negatively correlated with the change in the deviation of Pp from symmetry. Non-responders with smaller changes in self-selected walking speed exhibited increased deviations from symmetric propulsion with training, which demonstrates the importance of Pp to assess walking capacity. A subsequent study evaluated the effect of gait training on module quality and walking performance in individuals post-stroke. The study analyzed healthy controls and those who through the course of therapy improved their muscle activation patterns such that they went from having one or two merged modules to four independent modules post-therapy [53]. Although the hemiparetic subjects achieved a statistically significant improvement in Pp from pre- to post-training, their post-training Pp values were significantly lower than that of the controls, indicating the presence of lingering impairments in the hemiparetic subjects' ability to produce propulsion from their paretic limb. The same study revealed a trend of greater Pp post-therapy when more independent modules were present pre-therapy, suggesting that gait training may most effectively improve paretic limb function in individuals who initially have more independent modules.

In addition to the importance of Pp as an important outcome measure for evaluating the effect of rehabilitation on paretic limb function, changes in Pp following gait training have been shown to be indicative of underlying changes in muscle function [24]. A comparison of the simulated muscle function of two subjects who walked at the same self-selected speeds pre- and post-rehabilitation but had opposite changes in Pp (one increased while the other decreased) revealed that the subject who increased Pp also had increased contributions from the paretic soleus, gastrocnemius, and hamstrings to the paretic anterior-posterior
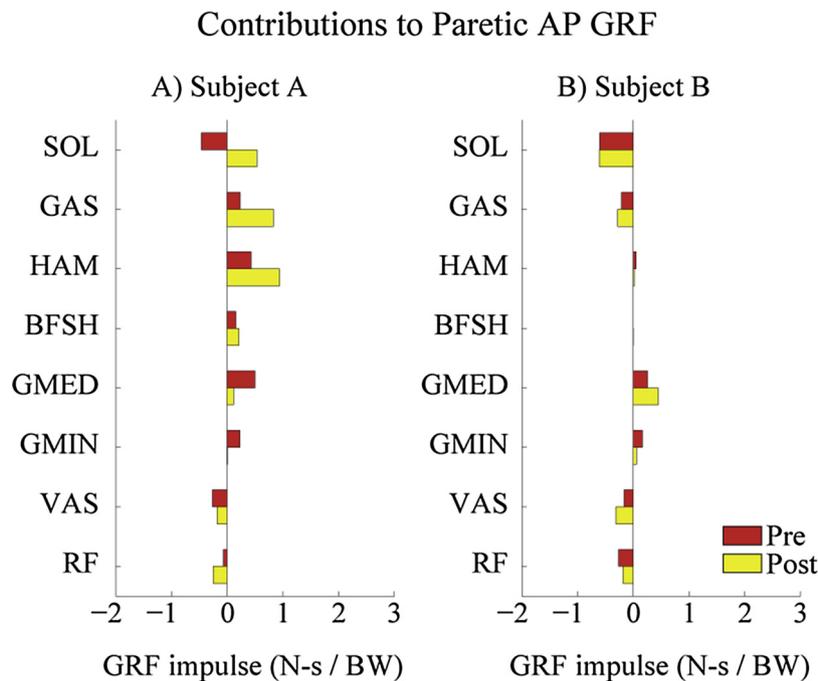
Contributions to Paretic AP GRF

Fig. 6. Muscle contributions from the paretic leg to the anterior posterior ground reaction forces during the paretic propulsive phase for A) Subject A who increased Pp with rehabilitation and B) Subject B who decreased Pp with rehabilitation. Adapted from [24].

GRF (propulsion) following training (Fig. 6). In contrast, the subject who decreased Pp with training had greater negative contributions from soleus and gastrocnemius to the anterior-posterior paretic GRF (braking) prior to training and this contribution did not change as a result of training. Moreover, the subject with increased Pp had reduced contributions from the non-paretic soleus to non-paretic propulsion while the subject with decreased Pp increased their contributions from the non-paretic plantarflexors to propulsion following training. These findings further emphasize the importance of the paretic plantarflexors in producing paretic propulsion and how Pp can be used to identify compensatory strategies acquired as a result of training.

Recently, a post-stroke rehabilitation approach has been designed to promote appropriate plantarflexor function through functional electrical stimulation (FES) of the paretic ankle dorsiflexors during paretic swing (for foot clearance) and plantarflexors during paretic terminal stance (for propulsion) [54]. By combining fast treadmill training, which has been shown to increase paretic hip extension [55,56], with FES (Fast-FES) over a 12-week training program, hemiparetic patients exhibited significant improvements in self-selected walking speed, peak propulsion, propulsion symmetry, and peak trailing limb angle [57]. The absolute change, but not the relative change, in self-selected walking speed was significantly associated with all baseline metrics except peak trailing limb angle, with better baseline function associated with greater changes in self-selected walking speed [57]. Additional analyses revealed that the ability to increase peak paretic propulsive force from self-selected to fast walking speeds at baseline is predictive of greater gains in peak paretic propulsive force from Fast-FES training [58]. A follow-up study with a larger group of hemiparetic subjects determined that slower hemiparetic walkers with greater Pp at baseline achieved the greatest improvements in self-selected and fastest walking speeds with Fast-FES training compared to walkers with smaller Pp and even faster walkers with high Pp [18]. Furthermore, their findings demonstrated that baseline Pp was important for predicting changes in self-selected and fastest walking speed due to training and suggested that walking speed alone was insufficient for identifying candidates for targeted gait training because, as shown above, walking speed alone does not distinguish between underlying hemiparetic motor impairments [18].

To identify the underlying mechanisms contributing to improved function with Fast-FES training, a subsequent randomized control trial compared the effects of 12 weeks of gait training at either self-selected walking speed, fastest possible speed, or with the Fast-FES paradigm on self-selected walking speed and paretic and non-paretic propulsive forces in hemiparetic patients [17,31]. Across all training groups, paretic propulsion force increased 23% with the change in paretic propulsion force explaining 52% of the variance in the change in self-selected speed from pre- to post-training [17]. Moreover, there was a stronger relationship between the change in paretic propulsive force and the change in self-selected walking speed ($R^2 = 0.54$) as a result of training than between the change in non-paretic propulsive force and the change in self-selected walking speed ($R^2 = 0.43$) [31]. The change in trailing limb angle was a greater independent contributor than paretic ankle moment to the observed increase in paretic propulsive force across all training groups, with only the Fast-FES group demonstrating a significant increase in paretic ankle moment due to training [17]. The authors suggested trailing limb angle is more modifiable than ankle moment and therefore restoration of paretic propulsion is accomplished primarily by the hip and knee extensors, which govern the trailing limb angle [17]. However, the plantarflexors are the primary muscles that contribute to propulsion [43]. Therefore, their role in restoring paretic propulsion cannot be neglected, particularly given that the soleus has also been shown to be a key contributor to knee extension during late stance [43,44]. Furthermore, the large inter-subject variation in treatment outcomes [17,57] suggests a need to understand the mechanisms underlying the relationship between increased leg extension and improvements in Pp.

Gait training incorporating real-time biofeedback of paretic anterior-posterior GRFs has recently been shown to improve paretic propulsion during a single training session [59]. Significant improvements in paretic propulsion were maintained following a 30 min washout period [59]. Additionally, significant improvements in paretic trailing limb angle, peak paretic plantarflexor moment, and step length symmetry were retained following a short (2 min) washout period, but not following longer (15 and 30 min) washout periods [59]. These findings suggest biofeedback may be a promising technique to improve paretic propulsion. However, further investigations are necessary to assess the

long-term efficacy of multi-session biofeedback gait training.

In addition to gait training techniques, efforts have been made to develop external devices that assist with propulsion in hemiparetic subjects. These devices include wearable suits and passive-dynamic ankle foot orthoses (PD-AFOs) that overcome the limitations of traditional solid AFOs, which have been shown to impede propulsive force and ankle plantarflexor moment generation during gait in healthy adults [60]. A soft robotic exosuit was developed to improve paretic limb function and reduce energy expenditure during hemiparetic gait by augmenting the function of the paretic plantarflexors during mid-to-late stance and the paretic dorsiflexors during swing [61]. On average, participants' peak paretic propulsive force improved by 11% and 13% and peak propulsion asymmetry improved by 20% and 16% during treadmill and overground walking, respectively, with slower hemiparetic walkers demonstrating the greatest relative improvements in propulsion symmetry [61]. A significant improvement in ankle power symmetry during terminal stance was also observed with the powered exosuit [62]. Additionally, the optimal timing of powered plantarflexor assistance was subject-dependent and mistimed plantarflexor force assistance was shown to impair paretic propulsion in some subjects [61], demonstrating the heterogeneity of plantarflexor dysfunction in the post-stroke population and the need for rehabilitation techniques that target an individual's specific impairments. While the soft exosuit demonstrates promising potential for improving paretic propulsion, the long-term effect of exosuit use on hemiparetic neuromotor function and walking performance is currently unknown and requires further investigation.

PD-AFO devices have been designed to supplement push-off in patients with impaired plantarflexor function by acting like a spring and storing energy at mid-stance and returning energy to the leg during terminal stance. While the effects of PD-AFO stiffness on gait mechanics and energetics have been tested in simple models [63] and in healthy subjects [64,65] and those with musculoskeletal injuries [66,67], further research is needed to assess the effect of PD-AFOs on walking performance in post-stroke subjects.

While the interventions described here can lead to increased paretic limb output, not all persons with hemiparesis improve Pp with therapy [23,24] and even in those who do increase Pp, propulsion deficits on the paretic limb can persist [53]. Moreover, the individuals with the most severe propulsion asymmetries have been shown to benefit least from current rehabilitation techniques [53,58], which suggests there exists a baseline threshold of plantarflexor function to benefit from current locomotor rehabilitation techniques [18]. However, such a threshold has not been defined and likely depends on the type of locomotor rehabilitation being utilized. In addition, it is unclear if individuals with sub-threshold plantarflexor function at baseline are incapable of improving paretic plantarflexor function due to impaired corticomotor function [68] or if reliance on a compensation strategy that hinders propulsion is interfering with their ability to learn a propulsion-based walking strategy [18]. Future work is needed to determine how to identify the cause of subthreshold plantarflexor function at the onset of therapy as well as to develop interventions specific to the underlying cause that are capable of improving Pp.

Several variables critical to the production of Pp have been identified, including appropriate magnitude and timing of plantarflexor output [20,24,46,48,49,53,61], appropriate timing of leg flexor activation [20], leg extension angle during terminal stance [17,22,40], and ankle moment generation [19,22,30]. Previous research has demonstrated that hemiparetic individuals are capable of developing an independent plantarflexor module through locomotor training [53]. Therefore, the identification of an independent plantarflexor module at rehabilitation baseline may be important to determine if developing an independent plantarflexor module should be a rehabilitation goal. However, more research may be needed to identify the underlying mechanisms of locomotor training that facilitate the development of an independent plantarflexor module in order to design therapies that target increased modular complexity in hemiparetic individuals with merged modular control. In addition, appropriately timed paretic hip and knee extensor and flexor function during gait is required to facilitate greater paretic leg extension and Pp. Impaired leg extension may be due to weak extensor muscles or excessive hip and knee flexor activity. Therefore, improving terminal stance leg extension in hemiparetic individuals may require hip and knee extensor strengthening as well as functional training to promote appropriately timed muscle activation during terminal stance. Furthermore, the positive correlation between leg extension and Pp may be due to greater contributions to propulsion from the hip and knee extensors resulting from increased force produced by these muscles or due to a more advantageous kinematic position for the plantarflexors to produce greater contributions to propulsion. The ability of an individual to leverage a particular mechanism to increase leg extension and improve Pp is likely dependent on an individual's specific impairments. Therefore, identifying the mechanisms underlying the relationship between leg extension and Pp may inform patient-specific rehabilitation designed to increase Pp in hemiparetic gait.

In conclusion, Pp should be a primary target and outcome measure of post-stroke gait rehabilitation. The current literature suggests rehabilitation techniques that target both plantarflexor function and leg extension may restore paretic limb function and correct gait asymmetries in hemiparetic individuals. However, due to the heterogeneity of individuals post-stroke, not all individuals benefit from the same type of treatment. Thus, there is a critical need for methods to predict which patients will benefit from current treatments. Given that baseline Pp has been associated with functional outcomes of gait training [18], Pp is a promising measure to help predict patient outcomes. Future work is needed to develop new rehabilitation techniques to improve gait symmetry in individuals whose function does not improve with existing therapy, particularly those with very minimal Pp.

Funding

Conflict of interest

The authors have no conflicts of interest to declare.

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