COMMENTARY

Post-Stroke Walking Behaviors Consistent with Altered Ground Reaction Force Direction Control Advise New Approaches to Research and Therapy

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Abstract Recovery of walking after stroke requires an understanding of how motor control deficits lead to gait impairment. Traditional therapy focuses on removing specific observable gait behaviors that deviate from unimpaired walking; however, those behaviors may be effective compensations for underlying problematic motor control deficits rather than direct effects of the stroke. Neurological deficits caused by stroke are not well understood, and thus, efficient interventions for gait rehabilitation likely remain unrealized. Our laboratory has previously characterized a post-stroke control deficit that yields a specific difference in direction of the ground reaction force (F, limb endpoint force) exerted with the hemiplegic limb of study participants pushing on both stationary and moving pedals while seated. That task was not dependent on F to retain upright posture, and thus, the task did not constrain F direction. Rather, the F direction was the product of neural preference. It is not known if this specific muscle coordination deficit causes the observed walking deviations, but if present during walking, the deficit would prevent upright posture unless counteracted by compensatory behaviors. Compensations are presented that mechanically counteract the F misdirection to allow upright posture. Those compensations are similar to behaviors observed in stroke patients. Based on that alignment between predictions of this theory and clinical observations, we theorize that post-stroke gait results from the attempt to compensate for the underlying F misdirection deficit. Limb endpoint force direction has been shown to be trainable in the paretic upper limb, making it a feasible goal in the lower limb. If this F misdirection theory is valid, these ideas have tremendous promise for advancing the field of post-stroke gait rehabilitation.

Introduction

For humans with post-stroke hemiparesis, the ability to walk promotes independent living and is held as an important goal [1–4]. The neurological consequences of a stroke, however, lead to walking difficulty [1, 5–7]. Fortunately, recent advances in understanding neuroplasticity have established the potential for restorative therapy as an improvement over traditional therapy techniques which consist primarily of attempts to alter compensatory behaviors [8]. The mechanisms by which the neurological insult disrupts gait are poorly understood [9, 10], however, as evidenced by the inconclusive pool of studies surrounding theoretically promising therapeutic approaches [11–18]. Such lack of understanding is not surprising given that even non-disabled locomotion is poorly understood at the neural control level [6, 19–21]. Development of more effective rehabilitation must be facilitated by understanding the mechanisms by which stroke disrupts gait.

This paper aims to inform new research and clinical directions by investigating the theoretical implications of how a particular motor control deficit observed in post-stroke individuals contributes to walking deviations. The preferred control of ground reaction force (F) direction has been shown to be altered post-stroke [22]. In that study, participants performed a seated pushing task that involved isometric legs pushing on a fixed pedal while in a fixed seat. The pedal measured F magnitude and direction. Because F direction

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was unspecified, the F direction produced reflected central nervous system priority. Non-disabled participants and the non-paretic leg of post-stroke participants preferred to direct F near the center-of-mass (CM), independent of leg posture (Fig. 1a) [22, 23]. Post-stroke participants, however, showed a preference for directing F anterior to the CM with their paretic limb. Both ischemic and hemorrhagic stroke participants exhibited this behavioral preference in chronic and acute phases. That miscoordination would disrupt upright posture by pitching the person backward if used in a situation where F is the only force available to control whole-body angular momentum (WBAM), such as during hand-free walking (Fig. 1b).

Study of non-impaired walking suggests that this typically CM-directed control of F is a component of walking [24]. The anteriorly misdirected F has not been directly observed during walking, because it is impossible to remain upright when using that deviation in isolation. With F control altered in the manner described post-stroke, successful walking would require behaviors that compensate for the mechanical effects of the disrupted motor control. Those compensations would obscure the underlying F misdirection deficit, preventing direct measurement of a misdirected force during walking. Thus, to assess the plausibility that this disrupted motor control affects walking, this work proposes behaviors that are mechanically sufficient to counteract a misdirected F and compares them with behaviors observed in post-stroke gait. Similarity between the proposed compensatory behaviors and the behaviors observed in post-stroke gait supports this theory of misdirected F direction as a major deficit causing walking problems. Therapy that addresses the motor deficit of F misdirection could be highly effective by eliminating the need for compensatory behaviors. The expectation is that rehabilitative strategies developed to target this specific motor control deficit rather than target behaviors that compensate for that deficit should result in more effective gait restoration.

Significance of the Ground Reaction Force (F) in Motor Control of Gait

Walking has specific mechanical requirements. The primary mechanical requirement is to retain upright posture so as to prevent a fall. Anteriorly misdirected F after stroke would disrupt gait by interfering with the way people typically meet that requirement. From this, compensatory behaviors can be predicted.

In mechanical terms, upright posture requires supporting the body against gravity and controlling rotation of the whole body. This means WBAM (Fig. 1b) must be controlled by an external force on the body. The ground reaction force (F) is the only means available to provide that support by producing a torque about the CM to alter WBAM. F results from the net
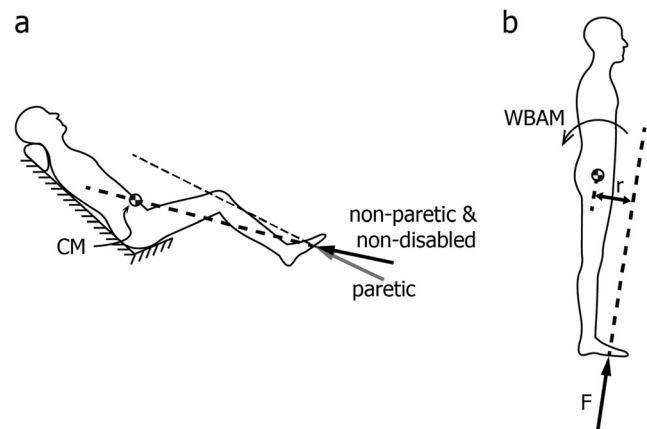

Fig. 1 Seated pushing efforts show F directed toward the CM except when affected by stroke, where F is biased anteriorly (**a**) [22]. Anteriorly biased F would produce a posterior pitching acceleration (counter-clockwise change in WBAM) disrupting upright posture (**b**)

common output of numerous neural motor signals, and because stroke alters those signals, the effect of the neurological insult is embedded in F. Therefore, this work will focus on F to provide insight into both the stroke-induced neural deficit and the resulting impact on walking.

The contributions of F to walking can be understood by expressing F as a magnitude and direction. F magnitude is largely responsible for supporting body weight. F direction is largely responsible for variations in torque about the CM, which determines body orientation. Both F magnitude and direction deficits are potential mechanisms of stroke gait disruption.

Sufficient F magnitude is necessary to support the body against gravity. F magnitude capacity is often reduced in the paretic limb after stroke [10, 25–29]. Insufficient F magnitude would adversely affect gait, because without producing sufficient F magnitude to oppose gravity, a person will fall down. F magnitude capacity could be improved by strengthening exercises, in which case those exercises would be expected to improve gait. Such strengthening exercises, however, do not provide the consistent and functionally significant improvements to gait that should result if F magnitude were the primary deficit [30–34].

F magnitude deficiency is not the only potential problem disrupting post-stroke gait; F direction also needs to be considered. Due to the large effect of small changes in F direction on WBAM, the consequences of a deficit in F direction are expected to cause a person to fall over and not down as was the case for F magnitude deficit.

The direction of F during walking has been studied in various populations with and without walking difficulties [35–42]. The F direction produced by the paretic limb during walking following a stroke has been identified as atypical [38, 43] and has been used as a target metric for therapy [37]. That therapeutic intervention had promising results showing that the direction of F

correlated with functional ability, which supports the targeting of F direction deviations [37]. Those studies of F direction during walking, as well as others examining atypical muscle activation synergies and timing [44, 45], support the theory of miscoordination as a neural mechanism for impairment following stroke. They do not, however, precisely characterize that miscoordination. It remains unclear which of the atypical behaviors previously described were due to neuromotor dysfunction and which were compensatory strategies [46]. Our objective is to examine the plausibility of a specific miscoordination in the hemiparetic limb during walking that has not been addressed by these previous studies to inform understanding of stroke and develop more effective therapy.

Post-Stroke Gait Behavior

Post-stroke gait behavior consists of direct effects of the neural injury and compensations for those direct effects [46]. Stroke alters nervous system motor control, producing direct effects such as reduced muscle activation overall or differentially across muscles. If the direct effects have significant impact on attaining behavioral goals, they are likely to elicit compensations. Compensations may also cause other disruptions to gait, inducing further compensations [47]. Compensations that reduce the effect of F on WBAM will be called *primary compensations*. Those induced by primary compensations will be called *secondary compensations*.

Primary compensations can reduce the undesired effects of F on WBAM in four ways. F alters WBAM via magnitude and direction of torque about the CM, as torque is the product of force magnitude and the moment arm (r) (Fig. 1b). The four ways to reduce the effect of F on WBAM are to (1) decrease F magnitude in the paretic limb (and increase F magnitude in the non-paretic limb to maintain sufficient average vertical force), (2) decrease the duration of time that paretic leg F acts, (3) reduce the moment arm of F by redirecting F, or (4) reduce the moment arm of F by repositioning the body. Primary compensations for F misdirection must address one or more of these as presented in separate sections below.

Primary Compensation: 1) Decrease F Magnitude

Reducing the magnitude of the misdirected F decreases the torque about the CM and thus reduces the postural disruption. However, that magnitude reduction interferes with the walking requirement of average net force of the ground acting on the feet having a sufficient vertical component to counter the force of gravity. Thus, decreasing the magnitude of the paretic leg F necessitates the
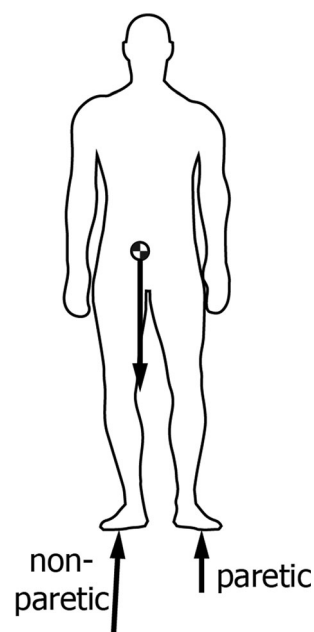

Fig. 2 Positioning the CM laterally closer to the non-paretic foot reduces the F magnitude required of the paretic foot

secondary compensation of increased non-paretic leg F magnitude (Fig. 2).

A common feature of hemiparetic gait is decreased magnitude of paretic leg F, often referred to as lateral weight-bearing asymmetry [10, 25–28, 48]. The common rationale is that weak paretic leg muscles prevent the generation of sufficient force magnitude. Or, if able to generate sufficient force, the weakened muscles operate closer to their peak force capacity which increases fatigue and motivates the person to produce less paretic leg force. An alternate explanation for reduced paretic leg F magnitude is that it reduces the postural imbalance induced by the misdirected F. The magnitude of the paretic leg contribution to the F required for body support can be reduced by positioning the CM laterally off the midline and biased toward the non-paretic leg (Fig. 2). The weakness explanation is discredited by the failure of strength increases to reduce the asymmetry as discussed above [30–34].

Primary Compensation: 2) Decrease F Duration

Reducing the duration of the misdirected F decreases the alteration of WBAM (Fig. 1b). With shortened paretic F duration, the requirement to counter the force of gravity requires an increase in non-paretic leg F magnitude and/or duration. Thus, asymmetric stance/swing timing is predicted from F misdirection.

Post-stroke gait is characterized by specific asymmetries. As the non-paretic leg swing requires the paretic limb to solely support body weight, a shortened

non-paretic swing time facilitates decreased duration of the paretic limb F. This makes it more difficult for the non-paretic foot to advance forward a normal distance, resulting in short non-paretic step length. By decreasing the amount of time supported by the paretic limb, the non-paretic stance phase is relatively prolonged. Walking after stroke commonly exhibits those features [48–57].

Primary Compensation: 3) Redirect F

F may be generated in a more favorable direction by utilizing passive joint torques instead of those produced by the post-stroke neural activation. The direction of F with respect to the leg, for a given leg posture, is a function of the relative hip, knee, and ankle torques [58]. Modulating the relative magnitudes of those joint torques alters F direction. Hip, knee, and ankle torques can be generated by neural activation of muscle or by passive mechanisms such as the ligament and bone forces that engage near the end of a joint range of motion [53]. Stroke appears to reduce the ability of the neural system to select among various coordination strategies [59, 60]. Thus, passive joint torques may become an attractive option with which to control F following stroke.

To support the body against the force of gravity, knee buckling (uncontrolled flexion) must be prevented. Hyperextending the knee joint (extension beyond hip-knee-ankle co-linearity) engages ligament and bone forces that produce a knee flexion torque that resists further knee extension (Fig. 3a, b). Thus, knee hyperextension allows the leg to produce F without the use of muscles crossing the knee. No hip torque is required, so F will have a line of action that passes near the hip joint (neglecting leg weight and limb acceleration). That orientation for the F line of action is likely to be closer to the CM than the misdirected F produced by the stroke-induced preferred muscle coordination [22]. Thus, upright posture would be more readily maintained by adopting a preference for a hyperextended knee angle to passively produce the knee torque necessary to support body weight.

Knee hyperextension is a common post-stroke behavior [25, 53, 61]. Other investigators have proposed that knee hyperextension is caused by excessive ankle plantar-flexor torque (plantar-flexor spasticity [53, 62]), impaired knee proprioception, spastic quadriceps, or weak knee extensors [63]. While those certainly can contribute to knee hyperextension, the present theory that knee hyperextension is an effective compensation for F misdirection also predicts secondary compensations of excessive plantar-flexion torque and spastic quadriceps to maintain knee hyperextension (see "Secondary Compensations: Preservation of Knee Hyperextension"
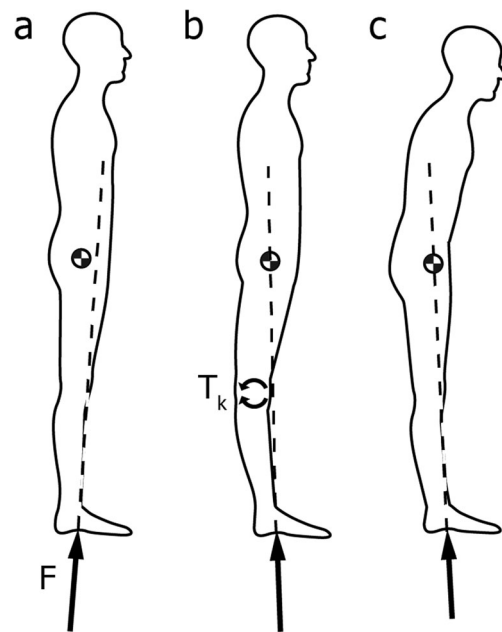

Fig. 3 F direction can be reoriented from the neurally miscoordinated direction which is anterior to CM (**a**) to the appropriately directed orientation near the CM (**b**) by relying on passive knee joint torque generated with knee hyperextension. Alternatively, leg rotation posteriorly (**c**) redirects F, and rotation of the torso anteriorly keeps the CM over the feet, resulting in a hip flexed posture that prevents the posterior pitching torque (**a**) because the F line of action now passes through the CM

section). These primary and secondary compensations are likely to develop because of the stability benefits of directing F closer to the CM.

Primary Compensation: 4) Align CM with Force

Instead of redirecting F to align with the CM, a body posture adjustment can position the CM closer to the F line of action. The direction of F depends on the relative torques at the hip, knee, and ankle. Thus, for a given set of joint torques, the force line of action has a specific direction relative to the leg, regardless of how the leg is oriented in space. Flexing the body at the hip joint shifts the whole-body CM anteriorly relative to the leg and simultaneously reorients the leg posteriorly, pivoting about the ankle. This maneuver brings the F line of action closer to the CM, thus reducing the posterior pitching torque (Fig. 3a, c).

Humans with hemiplegia frequently have a body posture that is characterized by an excessive hip flexion angle during walking [63–65]. That body posture can effectively eliminate or reduce the postural imbalance caused by the abnormal force control by aligning the CM with the force line of action (Fig. 3c). The compensation is only necessary on the paretic side, so in many individuals the flexion of the hip is present only on the paretic side (sometimes designated "pelvic retraction") [28, 50, 66].

Secondary Compensations: Preservation of Knee Hyperextension

The preservation of the hyperextended knee posture (primary compensation 3 above) may be enhanced by activation of selected muscles. Depending on the laxity of the knee joint, the knee may be able to hyperextend far enough that it will remain stable in the hyperextended position. However, tight knee ligaments may limit hyperextension to only a few degrees, such that minor disturbances could begin to flex the knee. A flexed knee will begin to collapse, possibly triggering the preferred muscle coordination that produces the misdirected *F*. To avoid that, hip extension torque and/or ankle plantar-flexion torque can assist in maintaining knee extension [62]. Those specific joint torques have been observed post-stroke [67–69]. Of the various joint torque patterns that can serve to keep the knee extended [70–72], individuals may choose among those patterns. That latitude may result in the variability in joint torque patterns observed [26, 73]. Co-contraction of knee muscles stiffens the joint and can also be used to stabilize the hyperextended knee posture as is observed post-stroke [29, 53, 72, 74].

The improvement in balance provided by a hyperextended knee posture during stance could motivate other behaviors to sustain that knee posture. Keeping the knee hyperextended during swing phase could serve to ensure that the knee can passively bear weight during initial heel contact. An abnormally extended knee joint during swing phase is commonly observed after stroke [53, 61, 73, 75, 76]. Knee extended posture during swing could be attained by abnormal activation of knee extensor muscles around the time of swing initiation, as is observed after stroke [76].

During stance phase, knee extension can be facilitated by an ankle plantar-flexor torque [68, 69]. Plantar-flexor muscles generate a torque at the ankle joint that attempts to increase the distance between the toes and the knee. With plantar-flexor muscles active, the floor prevents the toes from moving down. Thus, the knee is forced backward, helping to keep the knee extended. The plantar-flexor torque shifts the center of pressure forward under the foot and, if excessive, may even lift the heel off the ground. The center of pressure under the hemiparetic foot is often anterior compared to that of non-disabled subjects [25, 77], and frequently the heel does not touch the ground (pes equinus) [25, 63]. An anterior center of pressure also rotates *F* direction posteriorly in mechanical linked segment interactions [78] which also aids in aligning the *F* line of action with the CM.

Initial foot-ground contact under the forefoot, rather than the heel, can also be used to promote knee extension. In non-disabled adult walking, ankle dorsi-flexor muscles are active at initial foot-ground contact to keep the force of the ground on the heel from causing the foot to approach the floor too rapidly [69]. However, that dorsi-flexor muscle torque also tends to flex the knee [68, 69]. Normally, knee extensor muscles counteract knee flexion, limiting it to 10–20 degrees [79]. If a person with hemiparesis is avoiding the use of muscles to prevent knee collapse (see above), ankle dorsi-flexion torque should be avoided because of the knee flexing effect. The simplest way to avoid ankle dorsi-flexion torque is to not activate those muscles. If the foot is in a standard posture at initial contact (heel touchdown first) and the pretibial muscles are not activated, the foot will quickly plantar-flex until it is arrested by the floor. This is called drop-foot and often is accompanied by a slapping sound as the foot sole hits the floor [79–81]. An alternate approach is to hold the foot in a plantar-flexed posture so that initial ground contact is made with the forefoot. No dorsi-flexion torque is needed and the foot will not slap onto the ground. A plantar-flexor torque can be employed and will assist in keeping the knee extended. To summarize, the commonly observed plantar-flexor actions (torque and posture) support the primary compensation of a knee extended posture.

Additional Secondary Compensations

Additional compensations may be required to relieve negative consequences of the behaviors that support knee hyperextension (presented above). Both the plantar-flexor torque and the plantar-flexed foot posture, in combination with the extended knee, may be carried over to the swing phase to ensure that the advantages are present during stance phase without having to repeatedly activate and deactivate those features on every cycle. Without some knee flexing and ankle dorsi-flexing, the leg will be too long for the foot to clear the ground when advancing the foot. A readily available solution is to raise the hip joint during swing phase ("hip-hiking") and/or swing the leg laterally ("circumduction"). These are common behaviors observed in post-stroke gait [25, 51, 53, 82].

A continuous demand for plantar-flexion action could lead to spasticity and contracture of the posterior tibial muscles and flaccidity of the pretibial muscles. The ability of the triceps surae muscles to supply the required plantar-flexion action can be enhanced by increasing the gain of the stretch reflex of those muscles. The knee has inherent passive stability when forced into hyperextension. Thus, there is little risk of excessive plantar-flexor torque causing too much knee hyperextension. Thus, strong and possibly even excessive reflex gain in the triceps surae to support the plantar-flexion actions could be expected. If excessive, this could manifest as spasticity. The plantar-flexed posture leads to chronic use of the posterior tibial muscles at a shortened length, leading to contracture. Disuse and possible antagonist inhibition could lead to flaccidity of the pretibial muscles. These are observed post-stroke [25, 63].

The sense of instability induced by the misdirected force may also induce co-contraction of multiple leg muscles.

Co-contraction is a common means to control an unstable load [83–85]. The critical joint that enables the leg to bear weight is the knee, thus co-contraction is primarily expected in muscles that cross the knee. This is observed post-stroke [29, 53, 72, 74].

Discussion

Rehabilitation of post-stroke gait should be guided by an understanding of the mechanisms by which stroke disrupts gait. It is theorized here that a contributing mechanism to post-stoke gait disruption is a specific miscoordination of leg muscles that is characterized by F that is anteriorly misdirected. This theory predicts specific gait behaviors that are well suited to compensate for the postural instability caused by that miscoordination. Those specific behaviors are commonly observed in post-stroke gait, supporting this theory. The availability of various compensations allows individual patients to present with differing behaviors [44], all emerging as solutions to a common underlying miscoordination.

Post-stroke gait therapy should be based on the most proximal cause for which there exists adequate understanding and potential means to induce positive change. The theory here aims to extend understanding to a more proximal factor in the causal chain between the ischemic event and gait difficulties. It is proposed that the motor control deficit characterized by F misdirection is responsible for a range of behaviors and may be a more proximal cause than those presented in the literature. The ability to therapeutically correct the F misdirection has not yet been established in the post-stroke lower limb but has been in the upper limb. Practice reaching in a robot-induced force field induces specific changes in post-stroke reaching kinematics that require the limb end point to be producing force against the handle in a new direction [86–88]. This success in retraining end point force direction in the paretic upper limb suggests that post-stroke individuals are capable of learning appropriate force direction in the lower limb [89, 90] if provided with the necessary training environment.

Therapies that are able to correct the F misdirecting motor control deficit are expected to also eliminate the gait deviations that are compensations for that deficit. On the contrary, therapies that attempt to eliminate those compensations without first retraining F direction are expected to be less effective in restoring functional gait. Without retraining, F direction compensations are still needed. Therapy that eliminates some compensations will just necessitate the adoption of other compensations that are likely to be less optimal though may be less apparent to therapists or researchers.

Relationships among the existing literature explanations for post-stroke gait deviations and the F misdirection theory need to be explored. This theorized mechanism is consistent with and extends existing explanations for post-stroke gait behaviors. For example, stiff-legged gait has been attributed to inappropriate joint torques that result in a leg posture with excessive knee extension [25, 53, 61]. Those torques vary across individuals and include excess knee extension and ankle plantar-flexion [53, 63]. While it is possible that stroke causes a variety of joint torque deficits, an alternate hypothesis is that knee hyperextension is an effective compensation for F misdirection. Individuals then adopt diverse joint torque strategies to achieve that limb posture [25].

Another example of how the force misdirection theory explains current therapeutic experiences is its predictions of apparent progress during therapy [91–94] as well as minimal transfer to overground walking [12–14, 95–97] for body weight supported treadmill training (BWSTT). The BWSTT environment places external forces on the walker via the harness that can counteract the postural disturbance caused by F misdirection (posterior pitching). The harness can apply forces to the torso that do not pass through the CM and thus can exert a torque to oppose the torque produced when F is misdirected. In this way the BWSTT environment acts similar to the behavioral compensations discussed above, muting the effect of the F misdirection. By reducing or eliminating the need for the walker to use the compensatory behaviors that were adopted to stabilize upright posture during overground walking, the BWSTT environment allows the walker to utilize the misdirected F without experiencing postural disturbance. Thus, no compensatory behaviors are employed and the walking kinematics appear closer to normal [91–94]. However, the BWSTT environment is unlikely to induce change in the motor control deficit producing the F misdirection, and thus, the same compensatory behaviors are necessary once overground walking is resumed [12–14, 95–97].

Limitations

There is an inherent limitation in the ability to observe direct empirical evidence that post-stroke F misdirection disrupts gait. Physics constrain F direction in order to remain upright while walking in a steady state. Namely, the average torque about the CM must be zero. The presence of an F in some other direction would preclude walking by producing a non-zero average torque. Thus, F misdirection cannot be directly observed during walking. Rather, a task other than walking must be used to observe this motor control deficit. That task must not be dependent on F for balance. Seated pushing provided that environment and allowed the observation that post-stroke individuals have an F misdirecting motor control deficit [22]. The use of a non-walking task to discover the F misdirection is not unlike the use of non-walking strength tests to postulate that post-stroke walking is degraded by reduced F magnitude capacity [10].

It is also recognized that the explanation for gait deviations discussed here is not the only plausible one. Strong supporting evidence for the force misdirection theory, however, is that the single motor deficit of F misdirection can specifically predict a broad range of observed behaviors and the response of patients to therapy (e.g., BWSTT).

The evidence on compensatory behaviors presented here provides indirect support for the theory. Direct evidence of measuring F misdirection during post-stroke gait may be possible in a mechanical environment that specifically counteracts the postural disturbance of the F misdirection, allowing the person to produce the misdirected F without compensatory behaviors. Current clinical body weight support harness systems may provide that mechanical environment.

Conclusion

The foot force misdirection observed post-stroke [22] is evidence of a motor control deviation that should be considered as a possible explanation for the gait abnormalities typically present in that population. Anteriorly biased F direction in post-stroke individuals would disrupt posture and prevent upright locomotion if used during walking with no other changes in balance strategy. Compensatory behaviors, however, could preserve upright posture during walking despite the presence of that F direction bias. Compensations are proposed here for their ability to accommodate an anteriorly biased F direction during walking. Those compensations are similar to observed post-stroke behavior. This similarity supports the theory that deviation in F direction may be an underlying explanation for gait abnormalities in stroke patients and is therefore an appropriate target for therapy.

References

1. Lord SE, McPherson K, McNaughton HK, Rochester L, Weatherall M. Community ambulation after stroke: how important and obtainable is it and what measures appear predictive? Arch Phys Med Rehabil. 2004;85:234–9.

2. Bohannon RW, Andrews AW, Smith MB. Rehabilitation goals of patients with hemiplegia. Int J Rehabil Res. 1988;11:181–3.

3. Bohannon RW, Horton MG, Wikholm JB. Importance of four variables of walking to patients with stroke. Int J Rehabil Res. 1991;14:246–50.

4. Desrosiers J, Malouin F, Bourbonnais D, Richards CL, Rochette A, Bravo G. Arm and leg impairments and disabilities after stroke rehabilitation: relation to handicap. Clin Rehabil. 2003;17:666–73.

5. Perry J, Garrett M, Gronley JK, Mulroy SJ. Classification of walking handicap in the stroke population. Stroke. 1995;26:982–9.

6. Loeb GE. Neural control of locomotion: how do all the data fit together? Bioscience. 1989;39:800–4.

7. Danielsson A, Willen C, Sunnerhagen KS. Physical activity, ambulation, and motor impairment late after stroke. Stroke Res Treat. 2012;2012:818513.

8. Behrman AL, Bowden MG, Nair PM. Neuroplasticity after spinal cord injury and training: an emerging paradigm shift in rehabilitation and walking recovery. Phys Ther. 2006;86:1406–25.

9. Hollands KL, Pelton TA, Tyson SF, Hollands MA, van Vliet PM. Interventions for coordination of walking following stroke: systematic review. Gait Posture. 2011;35:349–59.

10. Hsu AL, Tang PF, Jan MH. Analysis of impairments influencing gait velocity and asymmetry of hemiplegic patients after mild to moderate stroke. Arch Phys Med Rehabil. 2003;84:1185–93.

11. Dickstein R. Rehabilitation of gait speed after stroke: a critical review of intervention approaches. Neurorehabil Neural Repair. 2008;22:649–60.

12. Dobkin BH, Duncan PW. Should body weight-supported treadmill training and robotic-assistive steppers for locomotor training trot back to the starting gate? Neurorehabil Neural Repair. 2012;26:308–17.

13. Dobkin BH. Strategies for stroke rehabilitation. Lancet Neurol. 2004;3:528–36.

14. Moseley AM, Stark A, Cameron ID, Pollock A. Treadmill training and body weight support for walking after stroke. Cochrane Database Syst Rev 2003;CD002840.

15. Pollock A, Baer G, Pomeroy V, Langhorne P. Physiotherapy treatment approaches for the recovery of postural control and lower limb function following stroke. Cochrane Database Syst Rev. 2003;2.

16. Teasell RW, Bhogal SK, Foley NC, Speechley MR. Gait retraining post stroke. Top Stroke Rehabil. 2003;10:34–65.

17. Hornby TG, Campbell DD, Kahn JH, Demott T, Moore JL, Roth HR. Enhanced gait-related improvements after therapist- versus robotic-assisted locomotor training in subjects with chronic stroke: a randomized controlled study. Stroke. 2008;39:1786–92.

18. Mehrholz J, Werner C, Kugler J, Pohl M. Electromechanical-assisted training for walking after stroke. Cochrane Database Syst Rev 2007;4.

19. Farley CT, Ferris DP. Biomechanics of walking and running: center of mass movements to muscle action. Exerc Sport Sci Rev. 1998;26:253–85.

20. Craik RL, Oatis CA. Gait analysis: theory and application. St Louis: Mosby; 1995.

21. Full RJ, Koditschek DE. Templates and anchors: neuromechanical hypotheses of legged locomotion on land. J Exp Biol. 1999;202(Pt 23):3325–32.

22. Rogers LM, Brown DA, Gruben KG. Foot force direction control during leg pushes against fixed and moving pedals in persons post-stroke. Gait Posture. 2004;19:58–68.

23. Gruben KG, Rogers LM, Schmidt MW, Tan L. Direction of foot force for pushes against a fixed pedal: variation with pedal position. Mot Control. 2003;7:362–77.

24. Gruben KG, Boehm WL. Ankle torque control that shifts the center of pressure from heel to toe contributes non-zero sagittal plane angular momentum during human walking. J Biomech 2014.

25. Kim CM, Eng JJ. Magnitude and pattern of 3D kinematic and kinetic gait profiles in persons with stroke: relationship to walking speed. Gait Posture. 2004;20:140–6.

26. Kim CM, Eng JJ. Symmetry in vertical ground reaction force is accompanied by symmetry in temporal but not distance variables of gait in persons with stroke. Gait Posture. 2003;18:23–8.

27. Chen CY, Hong PW, Chen CL, Chou SW, Wu CY, Cheng PT, et al. Ground reaction force patterns in stroke patients with various

degrees of motor recovery determined by plantar dynamic analysis. Chang Gung Med J. 2007;30:62–72.

28. De Quervain IA, Simon SR, Leurgans S, Pease WS, McAllister D. Gait pattern in the early recovery period after stroke. J Bone Joint Surg Am. 1996;78:1506–14.

29. Hidler JM, Carroll M, Federovich EH. Strength and coordination in the paretic leg of individuals following acute stroke. IEEE Trans Neural Syst Rehabil Eng. 2007;15:526–34.

30. Morris SL, Dodd KJ, Morris ME. Outcomes of progressive resistance strength training following stroke: a systematic review. Clin Rehabil. 2004;18:27–39.

31. Mudge S, Barber PA, Stott NS. Circuit-based rehabilitation improves gait endurance but not usual walking activity in chronic stroke: a randomized controlled trial. Arch Phys Med Rehabil. 2009;90:1989–96.

32. Sullivan KJ, Brown DA, Klassen T, Mulroy S, Ge T, Azen SP, et al. Effects of task-specific locomotor and strength training in adults who were ambulatory after stroke: results of the STEPS Randomized Clinical Trial. Phys Ther. 2007;87:1580–602.

33. van de Port IG, Wood-Dauphinee S, Lindeman E, Kwakkel G. Effects of exercise training programs on walking competency after stroke: a systematic review. Am J Phys Med Rehabil. 2007;86:935–51.

34. van de Port IG, Wevers LE, Lindeman E, Kwakkel G. Effects of circuit training as alternative to usual physiotherapy after stroke: randomised controlled trial. BMJ. 2012;344:e2672.

35. Franz JR, Maletis M, Kram R. Real time feedback encourages old adults to increase their propulsion during walking. Clin Biomech. 2014; 29.1:68–74.

36. Oh K, Baek J, Park S. Gait strategy changes with acceleration to accommodate the biomechanical constraint on push-off propulsion. J Biomech. 2012;45:2920–6.

37. Awad LN, Reisman DS, Kesar TM, Binder-Macleod SA. Targeting paretic propulsion to improve poststroke walking function: a preliminary study. Arch Phys Med Rehabil. 2014;95:840–8.

38. Awad LN, Binder-Macleod SA, Pohlig RT, Reisman DS. Paretic propulsion and trailing limb angle are key determinants of long-distance walking function after stroke. Neurorehabil Neural Repair. 2015;29:499–508.

39. Bowden MG, Balasubramanian CK, Neptune RR, Kautz SA. Anterior-posterior ground reaction forces as a measure of paretic leg contribution in hemiparetic walking. Stroke. 2006;37:872–6.

40. Bullimore SR, Burn JF. Consequences of forward translation of the point of force application for the mechanics of running. J Theor Biol. 2006;238:211–9.

41. Gruben KG, Boehm WL. Force direction pattern stabilizes sagittal plane mechanics of human walking. Hum Mov Sci. 2012;31:649–59.

42. Gruben KG, Boehm WL. Ankle torque control that shifts the center of pressure from heel to toe contributes non-zero sagittal plane angular momentum during human walking. J Biomech. 2014;47: 1389–94.

43. Sharma S, McMorland AJ, Stinear JW. Stance limb ground reaction forces in high functioning stroke and healthy subjects during gait initiation. Clin Biomech (Bristol, Avon) 2015.

44. Mulroy S, Gronley J, Weiss W, Newsam C, Perry J. Use of cluster analysis for gait pattern classification of patients in the early and late recovery phases following stroke. Gait Posture. 2003;18:114–25.

45. Shiavi R, Bugle HJ, Limbird T. Electromyographic gait assessment, part 2: preliminary assessment of hemiparetic synergy patterns. J Rehabil Res Dev. 1987;24:24–30.

46. Bowden MG, Behrman AL, Woodbury M, Gregory CM, Velozo CA, Kautz SA. Advancing measurement of locomotor rehabilitation outcomes to optimize interventions and differentiate between recovery versus compensation. J Neurol Phys Ther. 2012;36:38–44.

47. Goodman MJ, Menown JL, West Jr JM, Barr KM, Vander Linden DW, McMulkin ML. Secondary gait compensations in individuals without neuromuscular involvement following a unilateral imposed equinus constraint. Gait Posture. 2004;20:238–44.

48. Titianova EB, Tarkka IM. Asymmetry in walking performance and postural sway in patients with chronic unilateral cerebral infarction. J Rehabil Res Dev. 1995;32:236–44.

49. Bogataj U, Gros N, Malezic M, Kelih B, Kljajic M, Acimovic R. Restoration of gait during two to three weeks of therapy with multichannel electrical stimulation. Phys Ther. 1989;69:319–27.

50. Carlsoo S, Dahllof A, Holm J. Kinetic analysis of gait in patients with hemiparesis and in patients with intermittant claudication. Scand J Rehabil Med. 1974;6:166–79.

51. Chen G, Patten C, Kothari DH, Zajac FE. Gait differences between individuals with post-stroke hemiparesis and non-disabled controls at matched speeds. Gait Posture. 2005;22:51–6.

52. Dettmann MA, Linder MT, Sepic SB. Relationships among walking performance, postural stability, and functional assessments of the hemiplegic patient. Am J Phys Med Rehab. 1987;66:77–90.

53. Olney SJ, Richards C. Hemiparetic gait following stroke. Part I: characteristics. Gait Posture. 1996;4:136–48.

54. Peat M, Dubo HI, Winter DA, Quanbury AO, Steinke T, Grahame R. Electromyographic temporal analysis of gait: hemiplegic locomotion. Arch Phys Med Rehabil. 1976;57:421–5.

55. Wall JC, Ashburn A. Assessment of gait disability in hemiplegics. Hemiplegic gait Scand J Rehabil Med. 1979;11:95–103.

56. Wall JC, Turnbull GI. Gait asymmetries in residual hemiplegia. Arch Phys Med Rehabil. 1986;67:550–3.

57. Allen JL, Kautz SA, Neptune RR. Step length asymmetry is representative of compensatory mechanisms used in post-stroke hemiparetic walking. Gait Posture. 2011;33:538–43.

58. Hof AL. The force resulting from the action of mono- and biarticular muscles in a limb. J Biomech. 2001;34:1085–9.

59. Bobath B. Adult hemiplegia: evaluation and treatment. Elsevier Health Sciences, 1990.

60. Neckel ND, Blonien N, Nichols D, Hidler J. Abnormal joint torque patterns exhibited by chronic stroke subjects while walking with a prescribed physiological gait pattern. J Neuroeng Rehab. 2008;5:19.

61. Barela JA, Whitall J, Black P, Clark JE. An examination of constraints affecting the intralimb coordination of hemiparetic gait. Hum Mov Sci. 2000;19:251–73.

62. Silver-Thorn B, Herrmann A, Current T, McGuire J. Effect of ankle orientation on heel loading and knee stability for post-stroke individuals wearing ankle-foot orthoses. Prosthet Orthot Int. 2011;35: 150–62.

63. O'Sullivan SB. Stroke. In: O'Sullivan SB, Schmitz TJ, editors. Physical rehabilitation: assessment and treatment. Philadelphia: F.A. Davis Company; 1994. p. 78–553.

64. Lehmann JF, Condon SM, Price R, DeLateur BJ. Gait abnormalities in hemiplegia - their correction by ankle-foot orthoses. Arch Phys Med Rehabil. 1987;68:763–71.

65. Turani N, Kemiksizoglu A, Karatas M, Ozker R. Assessment of hemiplegic gait using the Wisconsin Gait Scale. Scand J Caring Sci. 2004;18:103–8.

66. Bogardh E, Richards CL. Gait analysis and relearning of gait control in hemiplegic patients. Physiother Can. 1981;33:223–30.

67. Siegel KL, Kepple TM, Stanhope SJ. Using induced accelerations to understand knee stability during gait of individuals with muscle weakness. Gait Posture. 2005;23:435–40.

68. Higginson JS, Zajac FE, Neptune RR, Kautz SA, Burgar CG, Delp SL. Effect of equinus foot placement and intrinsic muscle response on knee extension during stance. Gait Posture. 2006;23:32–6.

69. Matjacic Z, Olensek A, Bajd T. Biomechanical characterization and clinical implications of artificially induced toe-walking: differences between pure soleus, pure gastrocnemius and combination of soleus and gastrocnemius contractures. J Biomech. 2006;39:255–66.

70. Kepple TM, Siegel KL, Stanhope SJ. Relative contributions of the lower extremity joint moments to forward progression and support during gait. Gait Posture. 1997;6:1–8.

71. Winter DA. Overall principle of lower limb support during stance phase of gait. J Biomech. 1980;13:923–7.

72. Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiparetic patients. Brain. 1979;102:405–30.

73. Kerrigan DC, Frates EP, Rogan S, Riley PO. Spastic paretic stiff-legged gait—biomechanics of the unaffected limb. Am J Phys Med Rehab. 1999;78:354–60.

74. Lamontagne A, Richards CL, Malouin F. Coactivation during gait as an adaptive behavior after stroke. J Electromyogr Kinesiol. 2000;10:407–15.

75. Riley PO, Kerrigan D. Kinetics of stiff-legged gait: induced acceleration analysis. IEEE Trans Rehab Eng. 1999;7:420–6.

76. Kerrigan DC, Gronley J, Perry J. Stiff-legged gait in spastic paresis. a study of quadriceps and hamstrings muscle activity. Am J Phys Med Rehabil. 1991;70:294–300.

77. Kinsella S, Moran K. Gait pattern categorization of stroke participants with equinus deformity of the foot. Gait Posture. 2008;27:144–51.

78. Gruben KG, Boehm WL. Mechanical interaction of center of pressure and force direction in the upright human. J Biomech. 2012;45:1661–5.

79. Perry J. Gait analysis: normal and pathological function. Thorofare: Slack Incorporated; 1992.

80. Takebe K, Basmajian JV. Gait analysis in stroke patients to assess treatments of foot-drop. Arch Phys Med Rehabil. 1976;57:305–10.

81. Blaya JA, Herr H. Adaptive control of a variable-impedance ankle-foot orthosis to assist drop-foot gait. IEEE Trans Neural Syst Rehab Eng. 2004;12:24–31.

82. Kerrigan DC, Frates EP, Rogan S, Riley PO. Hip hiking and circumduction: quantitative definitions. Am J Phys Med Rehabil. 2000;79:247–52.

83. Deserres SJ, Milner TE. Wrist muscle activation patterns and stiffness associated with stable and unstable mechanical loads. Exp Brain Res. 1991;86:451–8.

84. Bourbonnais D, Vanden Noven S, Pelletier R. Incoordination in patients with hemiparesis. Can J Public Health. 1992;83 Suppl 2: S58–63.

85. Milner TE. Adaptation to destabilizing dynamics by means of muscle cocontraction. Exp Brain Res. 2002;143:406–16.

86. Patton JL, Kovic M, Mussa-Ivaldi FA. Custom-designed haptic training for restoring reaching ability to individuals with poststroke hemiparesis. J Rehabil Res Dev. 2006;43:643–56.

87. Patton JL, Mussa-Ivaldi FA. Robot-assisted adaptive training: custom force fields for teaching movement patterns. IEEE Trans Biomed Eng. 2004;51:636–46.

88. Patton JL, Stoykov ME, Kovic M, Mussa-Ivaldi FA. Evaluation of robotic training forces that either enhance or reduce error in chronic hemiparetic stroke survivors. Exp Brain Res. 2006;168:368–83.

89. Bourbonnais D, Bilodeau S, Lepage Y, Beaudoin N, Gravel D, Forget R. Effect of force-feedback treatments in patients with chronic motor deficits after a stroke. Am J Phys Med Rehab. 2002;81:890–7.

90. Pang MY, Yang JF. The initiation of the swing phase in human infant stepping: importance of hip position and leg loading. J Physiol. 2000;528(2):389–404.

91. Hassid E, Rose D, Dobkin B. Improved gait symmetry in hemiparetic stroke patients during body weight-supported treadmill stepping. J Neurol Rehab. 1997;11:21–6.

92. Visintin M, Barbeau H. The effects of body weight support on the locomotor pattern of spastic paretic patients. Can J Neurol Sci. 1989;16:315–25.

93. Hesse S, Konrad M, Uhlenbrock D. Treadmill walking with partial body weight support versus floor walking in hemiparetic subjects. Arch Phys Med Rehabil. 1999;80:421–7.

94. Burgess JK, Weibel GC, Brown DA. Overground walking speed changes when subjected to body weight support conditions for nonimpaired and post stroke individuals. J Neuroeng Rehab. 2010;7:6.

95. Duncan PW, Sullivan KJ, Behrman AL, Azen SP, Wu SS, Nadeau SE, et al. Body-weight-supported treadmill rehabilitation after stroke. N Engl J Med. 2011;364:2026–36.

96. Suputtitada A, Yooktanan P, Rarerng-Ying T. Effect of partial body weight support treadmill training in chronic stroke patients. J Med Assoc Thai. 2004;87 Suppl 2:S107–11.

97. Helbostad JL. Treadmill training and/or body weight support may not improve walking ability following stroke. Aust J Physiother. 2003;49:278.