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Evaluation of lower limb cross planar kinetic connectivity signatures post-stroke ☆

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ABSTRACT

Following stroke, aberrant three dimensional multijoint gait impairments emerge that present in kinematic asymmetries such as circumduction. A precise pattern of cross-planar coordination may underlie abnormal hemiparetic gait as several studies have underscored distinctive neural couplings between medio-lateral control and sagittal plane progression during walking. Here we investigate potential neuromechanical constraints governing abnormal multijoint coordination post-stroke. 15 chronic monohemispheric stroke patients and 10 healthy subjects were recruited. Coupled torque production patterns were assessed using a volitional isometric torque generation task where subjects matched torque targets for a primary joint in 4 directions while receiving visual feedback of the magnitude and direction of the torque. Secondary torques at other lower limb joints were recorded without subject feedback. We find that common features of cross-planar connectivity in stroke subjects include statistically significant frontal to sagittal plane kinetic coupling that overlay a common sagittal plane coupling in healthy subjects. Such coupling is independent of proximal or distal joint control and limb biomechanics. Principal component analysis of the stroke aggregate kinetic signature reveals unique abnormal frontal plane coupling features that explain a larger percentage of the total torque coupling variance. This study supports the idea that coupled cross-planar kinetic outflow between the lower limb joints uniquely emerges during pathological control of frontal plane degrees of freedom resulting in a generalized extension of the limb. It remains to be seen if a pattern of lower limb motor outflow that is centrally mediated contributes to abnormal hemiparetic gait.

1. Introduction

Following stroke, the emergence of aberrant stereotypical three dimensional (3D) gait impairments becomes evident. Yet previous research has traditionally limited the study of contributions to asymmetric gait to isolated single joint deficiencies. For example putative mechanisms to compensate for paretic ankle plantarflexion weakness (Dietz et al., 1981; Higginson et al., 2006), impaired knee flexion weakness and velocity (Goldberg and Anderson, 2004), and over activity of hip flexors (Sung and Bang, 2000; Piazza and Delp, 1996) have been extensively investigated. Similar studies have focused their investigation to exclusively sagittal plane mechanics for specific gait rehabilitation outcome measures (Jonkers et al., 2009; Daly et al., 2004). However the unique 3D observations of post-stroke gait abnormalities include impaired dynamic coupling between multiple lower limb joints across sagittal and frontal planes such as circumduction (Kerrigan et al., 2001). It is not known if a basic pattern of lower limb motor outflow that is centrally mediated underlies an impaired coordination pattern.

The ability to meet the biomechanical demands of gait following stroke may be neurally constrained to produce a precise pattern of cross-planar multijoint coordination. More specifically, sensitivity to frontal plane control may potentially be especially salient as several lines of investigation have underscored distinctive cross-planar couplings between medio-lateral (ML) control and sagittal plane progression during normal walking (Mackinnon and Winter, 1993). Both experimental (Cruz and Dhaher, 2008; Rogers et al., 2004) and simulation data (Allen et al., 2013; Finley et al., 2008) have implicated distinctive cross-planar neural couplings as potential contributors to stroke gait pathologies. Evidence for reciprocal heteronymous reflex meditated connectivity between hip adductors

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and knee extensors are observed when single joint angular perturbations were separately applied to the hip and knee (Finely et al., 2008). Frontal plane hip abduction in stroke gait is persistently observed even after applying assistive knee flexion torque (Sulzer et al., 2009). This suggests that paretic frontal plane movements are a potential reflection of altered neural constraints rather than the result of voluntary kinematic compensations. Key experimental evidence from healthy subjects also show that lower limb sagittal plane muscles RF and VL make contributions to frontal plane hip acceleration (Hunter et al., 2009). Coupling of cross-planar kinetic outflow is further supported by modeling studies that demonstrate how muscles that primarily contribute to anterior–posterior (AP) COM acceleration also contribute to the ML acceleration (Pandy et al., 2010). It remains to be seen whether the pathological engagement of control of frontal plane mechanics leads to the emergence of unique neural couplings.

Accordingly we hypothesize that previously highlighted evidence of cross-planar couplings is reflective of a more generalized kinetic constraint across multiple joints in the lower limb. This work seeks to explicitly quantify post-stroke lower limb kinetic outflow by identifying preferences in a volitional torque production during a graded target matching task. 3D modeling has shown that while stability control of the sagittal plane in normal walking can be largely accounted for by the passive mechanics of the limb, active control is required to maintain stability in the frontal plane (Bauby and Kuo, 2000; O'Conner and Kuo, 2009). We further explore this by testing the hypothesis that engaging biomechanical demands in the frontal plane leads to the emergence of aberrant kinetic constraints across multiple lower limb joints that are independent of limb biomechanics. Characterizing the differential effects of abnormal torque patterns and strength impairments may improve clinical treatment of gait dysfunction. Interventions targeting multisegmental abnormalities may facilitate functional improvements in post-stroke gait dysfunction.

2. Methods

2.1. Subject

A total of 25 participants (Table 1) were recruited for this study including 15 with a single unilateral stroke and 10 unimpaired age-matched control subjects. All subjects gave written informed consent. The study was approved by the Northwestern University Review Board. Impaired subjects presented with right side hemiparesis. A licensed physical therapist scored each subject for a variety of clinical tests (Table 1). All subjects were able to walk 5 m without assistance and had no history of orthopedic injury or surgery to their lower limbs.

2.2. Experimental set-up

Subjects were secured in a motorized, instrumented exoskeleton (Lokomat; Hocoma, Zurich, Switzerland) and isometrically locked in 2 different gait specific postures. The dimensions of the orthosis were adjusted for each participant to align the orthosis joint centers with those of the subject (Fig. 1). The subject's lower extremities were secured to the orthosis via cuffs instrumented with 4 total six-degree-of freedom (DOF) load cells (JR3, Woodland, CA) to measure the interaction forces and moments of the paretic test limb. The test limb was completely unloaded by the Lokomat such that subjects did not have to actively support the limb during torque production.

2.3. Protocol

Subjects produced voluntary isometric torques in 4 directions at a primary lower limb joint corresponding to both sagittal plane and frontal plane targets at the hip and ankle. The hip targets are flexion, extension, abduction, and adduction. The ankle targets are dorsiflexion, plantarflexion, eversion, and inversion. Subjects received instantaneous visual feedback of the primary joint torque produced (Fig. 2). The torques generated at each joint were calculated from thigh, shank, and ankle load cell signals using static equilibrium equations. While the subject matched target torques at the primary joint, secondary torques produced at the other joints across planes were recorded simultaneously. Specific instruction regarding the kinetic outflow at the secondary joints would have confounded the ability to investigate the intrinsic across-joint coupling. Feedback of secondary joint torque output was not provided.

The experimental protocol consisted of two parts: in part 1, maximum voluntary torques (MVT) produced at the hip (MVHT), knee (MVKT), and ankle (MVAT) were recorded along the 4 directions in the sagittal and frontal planes. Only knee sagittal plane torques were recorded. In part 2 isometric subjects performed torque target matching at either the hip or the ankle joints for a normalized percentage of MVT. Subjects were presented with targets in randomized order of the 4 directions and were instructed to match primary joint target torques within $\pm 5\%$ of the torque magnitude and hold for a minimum of 200 ms for a successful trial. To investigate the influence of supraspinal drive, two levels of randomized target torque magnitude, 20% and 40% of MVT for the primary joint were tested. To investigate the influence of biomechanics, 2 different gait specific postures were examined. In the toeoff posture (TO) the lower limb was rigidly locked at 15° hip extension, 45° knee flexion, and 90° ankle dorsiflexion (Winter, 1984). For the midswing posture (MS), the lower limb was rigidly stabilized at 10° hip extension, 65° knee flexion, and 90°ankle dorsiflexion (Winter, 1984). MVTs were recorded for each target direction for each posture.

Table 1
Stroke and control subject information. The clinical assessments were performed by a licensed physical therapist. Abbreviations: H – Hemorrhagic; I – Ischemic; TUG – Time up and go test; LMFM – Lower Motor Fugl–Meyer. Modified Ashworth scores tabulated for knee flexion and ankle dorsiflexion. All subjects had no cognitive deficits and passed a mini-mental examination. Subjects were not excluded due to sensory or proprioceptive impairments. Subjects were excluded if unable to meet the following range of motion requirements: ankle dorsiflexion to 90°, knee flexion to 90°, knee extension to 0°, hip flexion to 90° and hip extension to 10°.

Subject	Stroke	Gender	Age	Post (y)	Ashworth Knee	Ashworth Ankle	TUG (s)	Berg	LMFM
S1	H	F	52	3	0	0	11.4	46/56	18/34
S2	I	F	58	25	1/5	0	11.5	53/56	21/34
S3	I	M	49	3	0	3/5	12	43/56	25/34
S4	I	M	56	8	1/5	1/5	12	55/56	26/34
S5	I	F	31	9 m	0	0	11.7	51/56	18/34
S6	I	M	46	7 m	0	0	8.5	54/56	30/34
S7	H	F	50	2	0	0	13.5	54/56	26/34
S8	H	M	53	3	0	0	9.8	55/56	25/34
S9	I	M	39	2	0	0	13.4	46/56	27/34
S10	H	F	51	2	0	1/5	17	49/56	24/34
S11	I	M	46	2	0	0	8.2	51/56	30/34
S12	I	M	46	3	0	0	20.2	52/56	25/34
S13	I	F	63	8	0	1/5	15.3	55/56	20/34
S14	I	M	60	16	0	0			30/34
S15	I	F	34	2	0	0			32/34
Stroke mean (SD)		M=8	49 (9)	5(7)					25(4)
Control mean (SD)		M=8	45(13)						