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Intrinsic and functional stiffness vs. antagonist co-activation: a correlational analysis in stroke patients

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ABSTRACT

Tonus has been considered the basis of central nervous system (CNS) functional adaptation to fulfill the dual purpose of movement and postural control [1]. Among the multiple pathologies of CNS, stroke has been the most investigated in the context of tonus dysfunction, typically through measures involving the evaluation of muscle resistance against stretching at rest [2]. However, the recent evidence demonstrating that the increased muscle resistance obtained under these conditions is more related to intrinsic components, being not associated to movement dysfunction [3], turn relevant the assessment of biomechanical variables that could reflect tonus dysfunction in a highly demanding postural control functional context, like upright standing [4] and postural phases of gait initiation and stand-to-sit [5, 6]. By representing muscle synergies organization within postural tone to increase joint stiffness and postural stability, antagonist co-activation at the ankle level would reflect tonus dysfunction in poststroke subjects [7]. Furthermore, the evaluation of ankle stiffness through the analysis of stabilometric data obtained by a force platform during a functional task such as upright standing [8], here called functional stiffness, may also be used as a functional alternative to intrinsic stiffness assessment. Based on the previously exposed, it can be hypothesized that functional stiffness, rather than intrinsic stiffness, would best reflect the muscle synergies involved in the maintenance of postural stability during functional tasks.

The main aim of the present study was to evaluate the level of correlation between intrinsic stiffness at rest, functional stiffness in upright standing and antagonist co-activation in upright standing and postural phases of gait initiation and stand-to-sit, at the ankle joint of post-stroke subjects.

Key words: Tonus; Motor Control; Stroke; Stiffness; Antagonist co-activation.

METHODS

The contralesional (CONTRA) and ipsilesional (IPSI) limbs of eight participants with a history of ischemic stroke in the subcortical territory of middle cerebral artery for at least 6 months were evaluated (Table 1). Ankle intrinsic stiffness evaluation at neutral position (NP) was performed in seated position with knee extension by an isokinetic dynamometer programmed in passive mode at 5º/sec, according to Sousa [9]. Functional stiffness values in the anteroposterior direction were obtained during 15sec of upright standing on a force platform, according to the method proposed in Winter [8]. The antagonist co-activation of the tibialis anterior (TA)/soleus (SOL), TA/gastrocnemius medialis (GM) and Ventral/Dorsal pairs of each limb was calculated for upright standing and postural phases of the transition to gait and sitting, which were defined by the analysis of ground reaction forces and center of pressure (CoP) data obtained by two force platforms. The averaged values of three valid trials of intrinsic stiffness, functional stiffness and antagonist co-activation were used for analysis.

Table 1. Characterization of the participants according to gender, age (years), weight (kg), height (cm), lesion location (confirmed by computed tomography), injured hemisphere and evolution time (months).

ID	Gender	Age	Weight	Height	Lesion Location	Injured Hemisphere	Evolution Time
1	Female	39	65	170	Cortico-subcortical	Left	27
2	Male	47	95	176	Lenticulo-capsular	Right	30
3	Male	34	86	185	Lenticulo-capsular	Right	18
4	Female	32	60	173	Cortico-subcortical	Right	30
5	Female	38	68	163	Lenticulo-capsular	Right	24
6	Male	61	89	176	Lenticulo-capsular	Right	6
7	Female	57	65	160	Striato-capsular	Left	12
8	Female	54	75	158	Fronto-parieto-insular	Right	90

RESULTS

A significant and high correlation was found between antagonist co-activation of IPSI TA/SOL pair in upright standing and functional stiffness (r=0.810; p=0.015) (Table 2). There were no statistically significant correlations between intrinsic stiffness and antagonist co-activation and functional stiffness variables in none of the limbs.

Table 2. Correlation between intrinsic stiffness, functional stiffness and antagonist co-activation variables (with the respective correlation coefficient (r) and test value (p)).

Co-activation variables			Stiffness variables	
Functional task	Muscle pair	Limb	"NP" IPSI	Functional stiffness
Upright standing	TA/SOL	IPSI	0.038 (0.939)	0.810 (0.015)
	TA/GM		0.143 (0.760)	0.690 (0.058)
	Ventral/Dorsal		0.288 (0.535)	0.687 (0.071)
Gait initiation	SOL/TA		0.486 (0.329)	-0.214 (0.645)
	GM/TA		0.314 (0.544)	-0.179 (0.702)
	Dorsal/Ventral		0.314 (0.544)	-0.179 (0.702)
Stand-to-sit initiation	SOL/TA		0.071 (0.879)	-0.190 (0.651)
	GM/TA		0.464 (0.294)	-0.333 (0.420)
	Dorsal/Ventral		0.321 (0.482)	-0.476 (0.233)
			-0.357 (0.432)	
Functional task	Muscle pair	Limb	"NP" CONTRA	Functional stiffness
Upright standing	TA/SOL	CONTRA	-0.250 (0.589)	0.357 (0.385)
	TA/GM		0.250 (0.589)	0.452 (0.260)
	Ventral/Dorsal		-0.357 (0.432)	0.288 (0.493)
Gait initiation	SOL/TA		0.600 (0.208)	-0.357 (0.432)
	GM/TA		0.543 (0.268)	-0.250 (0.589)
	Dorsal/Ventral		0.771 (0.072)	-0.214 (0.645)
Stand-to-sit initiation	SOL/TA		0.107 (0.819)	0.143 (0.738)
	GM/TA		0.714 (0.071)	0.405 (0.320)
	Dorsal/Ventral		0.464 (0.294)	0.048 (0.911)
			-0.214 (0.645)	
			r (p value)	

CONCLUSIONS

The global tendency for significant correlations between antagonist co-activation in upright standing and functional stiffness, coupled with the absence of significant correlations between intrinsic stiffness and antagonist co-activation and functional stiffness variables, seems to demonstrate, as hypothesized, the relation between motor control variables evaluated in dynamic conditions and that muscle resistance to passive stretching does not seem to reflect the behavior of these muscles involved in muscle synergies during a functional task.

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