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Descending neural drives to ankle muscles during gait and their relationships with clinical functions in patients after stroke

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HIGHLIGHTS

- Descending neural drive to each muscle was reduced on the paretic side during gait.
- Neural drive to antagonist muscles was increased during gait in stroke patients.
- Increased drive to antagonist muscles was related to paretic ankle muscle weakness.

ABSTRACT

Objective: The objective of this study was to investigate the descending neural drive to ankle muscles during gait in stroke patients using a coherence analysis of surface electromyographic (EMG) recordings and the relationships of the drive with clinical functions.
Methods: EMG recordings of the paired tibialis anterior (TA), medial and lateral gastrocnemius (MG and LG), and TA–LG muscles were used to calculate intramuscular, synergistic, and agonist–antagonist muscle coherence, respectively, in 11 stroke patients and 9 healthy controls. Paretic motor function, sensory function, spasticity, ankle muscle strength, and gait performance were evaluated.
Results: Paretic TA–TA and MG–LG beta band (15–30 Hz) coherences were significantly lower compared with the non-paretic side and controls. TA–LG beta band coherence was significantly higher on both sides compared with controls. Paretic TA–TA beta band coherence positively correlated with gait speed, and paretic TA–LG beta band coherence negatively correlated with paretic ankle plantar flexor muscle strength.
Conclusions: The intramuscular and synergistic muscle neural drives were reduced during gait on the paretic side in stroke patients. The agonist–antagonist muscle neural drive was increased to compensate for paretic ankle muscle weakness.
Significance: Descending neural drive reorganization to agonist–antagonist muscles is important for patients with paretic ankle muscle weakness.

1. Introduction

Gait is a fundamental component of human daily life. Patients who have suffered central nervous system (CNS) lesions that impair descending motor pathways have difficulty walking independently (Dietz et al., 1995; Jørgensen et al., 1995; Rossignol, 2000). Thus, walking in humans depends on the integrated action of hierarchical levels of supraspinal and spinal neural control (Nielsen, 2003; Yang and Gorassini, 2006), within which the contributions of the primary motor cortex and corticospinal tract are particularly important (Barthélemy et al., 2011; Petersen et al., 2012). Significant activation has been observed in the primary sensorimotor cortex (SMC) during gait in healthy human subjects using neuroimaging techniques (Fukuyama et al., 1997; Miyai

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et al., 2001; la Fougere et al., 2010). Previous transcranial magnetic stimulation studies have demonstrated that the corticospinal tract is rhythmically excited throughout the gait cycle (Schubert et al., 1997; Capaday et al., 1999; Petersen et al., 2001).

After stroke, patients exhibit asymmetrical SMC activation during gait due to the reduced SMC activation in the affected hemisphere (Miyai et al., 2002). More symmetric SMC activation and increased corticospinal excitability correlate with improvements in gait parameters after stroke rehabilitation (Miyai et al., 2003; Yen et al., 2008). These results suggest that increased SMC activation and corticospinal excitability in the affected hemisphere may play an important role in locomotor recovery after stroke. Therefore, a new clinically convenient technique that can assess the functional contribution of the SMC and that can be used as a surrogate marker of corticospinal control during gait in patients after stroke is needed.

Recent studies have suggested that coherence analyses of paired surface electromyographic (EMG) recordings can quantitatively evaluate the descending neural drive from the SMC during gait (Halliday et al., 2003; Hansen et al., 2005; Norton and Gorassini, 2006; Nielsen et al., 2008; Barthélemy et al., 2010, 2015; Petersen et al., 2010, 2013; Willerslev-Olsen et al., 2015). Coherence analyses measure the linear correlation between a pair of signals in the frequency domain (Halliday et al., 1995), and the beta to gamma frequency bands are strongly related to the corticospinal drive (Conway et al., 1995; Mima and Hallett, 1999; Grosse et al., 2002; Petersen et al., 2012). EMG–EMG intramuscular and synergistic muscle coherences are observed in these frequency bands in healthy subjects (Farmer et al., 1993; Halliday et al., 2003), and they are reduced in patients with CNS disorders during static muscle contraction (Farmer et al., 1993; Fisher et al., 2012) and gait (Hansen et al., 2005; Nielsen et al., 2008; Barthélemy et al., 2010, 2015; Petersen et al., 2013). Furthermore, EMG–EMG beta band coherence can detect cortical excitability changes following transcranial direct current stimulation over the SMC (Power et al., 2006). Therefore, an EMG–EMG coherence analysis might be a promising candidate marker of the corticospinal control of walking.

Several previous studies have investigated intramuscular coherence during gait in patients with CNS disorders, such as spinal cord injury (SCI) (Hansen et al., 2005; Barthélemy et al., 2010, 2015), stroke (Nielsen et al., 2008), and cerebral palsy (Petersen et al., 2013; Willerslev-Olsen et al., 2015). Intramuscular coherence recorded at the tibialis anterior (TA) muscle during gait correlated with the gait parameters in patients after SCI (Barthélemy et al., 2010, 2015) and children with cerebral palsy (Petersen et al., 2013; Willerslev-Olsen et al., 2015). Although several physical functions such as lower limb muscle strength are strongly correlated with gait function in patients after stroke (Nadeau et al., 1999b), it is unclear whether these clinical functions also correlate with EMG–EMG intramuscular and synergistic muscle coherence during gait in patients after stroke.

EMG–EMG coherence between agonist and antagonist muscles has not generally been observed during gait in healthy subjects (Halliday et al., 2003; Norton and Gorassini, 2006). However, the beta to low-gamma (24–40 Hz) band coherence between agonist–antagonist muscles during gait is only present in patients who responded to treadmill training compared with non-responding patients after SCI (Norton and Gorassini, 2006). These results imply that increases in the descending neural drive to agonist–antagonist muscles might contribute to the generation of functional, but not necessarily normal, walking (Norton and Gorassini, 2006). Although increased agonist–antagonist muscle coactivation has frequently been observed during gait in patients after stroke (Rosa et al., 2014), no previous studies have investigated the EMG–EMG coherence between agonist–antagonist muscles during gait in patients after stroke.

Clarifying the relationship between clinical functions and EMG–EMG coherence during gait would be helpful in understanding the mechanisms by which corticospinal control of walking contributes to functional recovery in patients after stroke. The aim of the present study was to investigate the EMG–EMG intramuscular, synergistic muscle, and agonist–antagonist muscle coherences during gait and their relationships with clinical functions in patients after stroke.

2. Methods

2.1. Subjects

Eleven patients who suffered from a stroke and age- and sex-matched nine healthy control subjects participated in the present study (Table 1). The inclusion criteria of the patients were the following: (1) a single stroke that occurred more than 6 months prior to the present study, (2) no history of other neurological diseases (e.g., parkinsonism and ataxia) or rheumatic or orthopedic conditions that could interfere with gait, (3) the ability to walk independently without an ankle–foot orthosis for 10 m several times (Functional Ambulation Category score of at least 4) (Holden et al., 1984), and (4) no difficulty understanding the experimental tasks because of cognitive problems. None of the healthy controls had a neurological or orthopedic disorder or apparent gait abnormality. All subjects provided informed consents prior to beginning the study. All procedures were approved by the ethics committee of Kyoto University Graduate School and Faculty of Medicine, and were consistent with the Declaration of Helsinki.

2.2. Measurements of clinical functions

The Brunnstrom recovery stage descriptions (Brunnstrom, 1966) were used to evaluate the motor functions of the paretic

Table 1
Characteristics of the subjects.

	Patients $n = 11$	Controls $n = 9$	p
Age (years)	59.27 ± 11.58	55.78 ± 3.87	0.365
Height (cm)	163.36 ± 10.22	165.00 ± 8.50	0.706
Weight (kg)	63.95 ± 11.48	60.39 ± 10.53	0.483
Sex (n): male/female	7/4	6/3	0.999
Affected side (n): right/left	4/7		
Time post-stroke (years)	5.85 ± 2.09		
Brunnstrom recovery stage (n): I/II/III/IV/V/VI	0/0/4/3/4/0		
Cutaneous sensation (points)	5.09 ± 4.09		
Position sense (number of correct answers)	7.36 ± 3.35		
Modified Ashworth scale (n): 0/1/1+/2/3/4	0/4/5/1/1/0		
Ankle muscle strength (Nm/kg)			
DF on the paretic side	0.23 ± 0.13		
PF on the paretic side	0.38 ± 0.15		
Functional Ambulation Category (n): 1/2/3/4/5	0/0/0/3/8		
Gait speed (m/s)	0.58 ± 0.21	0.55 ± 0.04	0.637
Swing time symmetry ratio	1.53 ± 0.34		
Stride time coefficient of variation	4.80 ± 1.18		

The data are reported as mean ± standard deviation or n. A Functional Ambulation Category score of 4 indicates that the patient can ambulate independently on level surfaces, but requires supervision or physical assistance to negotiate stairs, inclines, or non-level surfaces, and the score of 5 indicates that the patient can ambulate independently on non-level and level surfaces, stairs, and inclines. DF: dorsiflexor, PF: plantar flexor.