



The influence of gait speed on co-activation in unilateral spastic cerebral palsy children[☆]



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ABSTRACT

Background: Physiological co-activation of antagonistic muscles during gait allows stability of loaded joints. Excessive co-activation restrains motion and increases energy expenditure. Co-activation is increased by gait speed and in the case of upper motor neuron lesions. This study aimed to assess the pathological component of co-activation in children with unilateral cerebral palsy.

Methods: 10 children with unilateral cerebral palsy and 10 typically developing children walked at spontaneous, slow and fast speeds. The spatio-temporal parameters and electromyographic activity of the rectus femoris, vastus medialis, semi-tendinosus, tibialis anterior and soleus of both lower limbs were recorded. A co-activation index was computed from the EMG envelopes. A mixed linear model was used to assess the effect of walking speed on the index of the antagonistic muscle couples (rectus femoris/semi-tendinosus, vastus medialis/semi-tendinosus and tibialis anterior/soleus) in the different limbs.

Findings: A greater effect of walking speed on co-activation was found in the involved limbs of children with cerebral palsy for all muscle couples, compared with their uninvolved limbs and the limbs of typically developing children. In typically developing children, but not in children with cerebral palsy, the effect of gait speed on the co-activation index was lower in the rectus femoris/semi-tendinosus than in the other agonist/antagonist muscle couples.

Interpretations: In children with cerebral palsy, a pathological component of muscle activation might be responsible for the greater increase in co-activation with gait speed in the involved limb. Altered motor control could explain why the co-activation in the rectus femoris/semi-tendinosus couple becomes more sensitive to speed.

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1. Introduction

Children with cerebral palsy (CP) present with pathological gait patterns which are a mixture of primary, secondary, and tertiary abnormalities (Gage and Schwartz, 2009). The primary abnormalities are directly related to the lesions of the brain structures involved in motor control. Musculo-skeletal deformities are the consequences of the abnormal forces due to the brain injuries and are referred to as the secondary abnormalities. The tertiary effects of the brain injury are the coping mechanisms used by the child. It is important to distinguish between these three levels using comprehensive gait analysis since the primary and secondary abnormalities are impairments and must be corrected as much as possible, while the tertiary ones should

resolve following the treatment of the impairments (Gage and Schwartz, 2009). Primary impairments include loss of selective control of muscles (synergies and co-activations), motor weakness, balance impairment, and abnormal muscle tone (usually spasticity). Co-activation (CA) is defined as the simultaneous electrical activity of agonist and antagonist muscle groups (Ikeda et al., 1998). CA induces co-contraction (CC), the mechanical action of two antagonist muscles which cross the same joint and act in the same plane (Olney, 1985). CA is a normal, physiological aspect of motor behavior, but inappropriate CA is one of the main abnormal findings in patients with upper motor neuron lesions. The positive effects of CC have been reported in many studies (Bowsher et al., 1993; Damiano, 1993; Detrembleur et al., 1997; Hubley-Kozey et al., 2008). Indeed, CC increases joint stiffness and therefore improves joint stability, pressure distribution over joint surfaces, as well as motor precision (Humphrey and Reed, 1983; Johansson and Westling, 1988; Valero-Cuevas, 2005). Inappropriate CC has been considered as detrimental to functional performance in children with CP during gait (Leonard et al., 1991; Unnithan et al., 1996a). The simultaneous activity

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of antagonist muscles creates opposing joint moments (Unnithan et al., 1996b) thus decreasing agonist force production and restraining movement. Walking speed is therefore reduced and energy expenditure increased (Winter, 2009). The paradox of normal, useful CA versus pathological CA raises a problem in the interpretation of dynamic EMG deviations in children with CP (Table 1).

It is important to consider walking speed when investigating CA. In typically developing (TD) children, speed significantly increases the relative duration of myoelectrical activity (EMG-time pattern) in the shank muscles (triceps surae, tibialis anterior, and peroneus longus), which means there is a greater overlap in the duration of agonist/antagonist activation with gait speed (Detrembleur et al., 1997). Unnithan et al. (1996a) showed that CA increases in both TD and CP children in the lower limb muscles when gait speed is increased. Thus, CA appears to be influenced by two main factors in children with CP: walking speed, similarly to TD children, and the upper motor neuron syndrome. This could lead to confusion when interpreting EMG data during gait analysis and for decision-making processes. It is therefore important to be able to differentiate between EMG changes related to variations in gait speed and changes related to the pathology. Because of the heterogeneous nature of children with CP and the use of varied methodologies, this question has been little studied. Moreover, the relative influences of the pathology and gait speed on the distal and proximal segments of the same limb have not been studied. This could provide some insights into the motor control of the lower limb during gait.

The aim of this study was therefore to analyse the pathological component of CA in children with unilateral CP. To this end, we investigated the impact of gait speed on CA, in muscles of the thigh and shank in children with unilateral CP and matched TD children. We hypothesized that the effect of gait speed would be different depending on the healthy or pathological nature of the limb, with a greater impact on the involved spastic limbs (IL) than in the uninvolved limbs (UL) or in the normal limbs of TD children (TDL). We also looked for differences of the effect of speed between segments (thigh vs. shank).

2. Methods

Approval was obtained from the institutional ethical committee and all children and parents provided informed consent. Ten hemiplegic CP children (mean age = 10.2 years, SD = 3.6) and 10 age-matched TD children (mean age = 10.2 years, SD = 2.04) were recruited for the study. The number of children was chosen for this observational study based upon previous works (Unnithan et al., 1996a). Inclusion criteria for hemiplegic CP children were: i. unilateral, spastic CP as defined by Bax et al. (2005); ii. ability to walk independently at least 10 m, and iii. age between 6 and 18 years. This age range was chosen because Beck et al. (1981) showed that a mature gait pattern is present by the age of 5 years in the typical development of human walking. Exclusion criteria were: i. a previous orthopedic or neurosurgical intervention, ii. botulinum toxin injections within the last twelve months. iii. inability to understand the instructions given during the gait analysis. Exclusion criteria for TD children were: i. any previous neurological lesion or orthopedic surgery, ii. lower limb length difference greater than

0.5 cm, iii. scoliosis, and iv. any injury of the lower limbs within the last 12 months.

A wireless surface EMG system (ZeroWire EMG, Aurion S.r.l., Milano, Italy) was used to acquire the electromyographic activity of 5 muscles on each lower limb: rectus femoris (RF), Vastus Medialis (VM), Semitendinosus (ST), Tibialis Anterior (TA) and Soleus (SO). After skin preparation (shaving if necessary, alcohol rubbing, cleaning with water, drying), surface-EMG electrodes were placed according to the SENIAM recommendations (Hermens et al., 2000). The inter-electrode distance was 20 mm in order to minimize cross-talk.

Children were asked to walk barefoot and unassisted down a 12-meter walkway in the gait lab in three different speed conditions. First, they were asked to walk at their self-selected speed, next, they were instructed to walk as fast as possible without running and finally, they were instructed to walk markedly slower than their spontaneous gait, without stopping. It has been shown that the spontaneous walking speed of TD and hemiplegic CP children is comparable, as well as their speeds in slow or fast gait (Fonseca et al., 2001). Therefore we used this simple speed instruction protocol to obtain a range of speed in each population. For each speed condition, three practice trials were performed so that the child could accommodate his/her gait. Four successive gait trials were then recorded. Six optoelectronic Vicon MX-F40 cameras (Oxford Metrics, Oxford, UK) recorded the displacement of reflective markers which were positioned on the feet (second metatarsal, heel and lateral malleolus) according to the Plug-in Gait conventions (Davis et al., 1991). These three markers were used for the automatic detection of gait events (Desailly et al., 2009) which was then refined using synchronized high-speed videos. EMG data were simultaneously recorded at 1000 Hz.

A customized Matlab program (the Mathworks, Natick, MA, USA) written using the Biomechanical ToolKit (Btk Development Core Team, 2011), was used for post-processing of the data files (c3d). Spatio-temporal parameters were calculated from the gait events and were scaled to the body size, as proposed by Hof (Hof, 1996), in order to improve comparisons between subjects despite variations in height and weight. Thus, gait speed (v) was scaled to the leg length (l_0 : from greater trochanter to ground) and the acceleration of gravity (g) in order to obtain a non-dimensional gait speed (v^*)

$$v^* = v / \sqrt{g \cdot l_0}.$$

EMG measurements were full wave rectified and filtered using a fourth-order Butterworth 8.9 Hz low pass filter (Shiavi et al., 1987) with phase correction to create the linear envelope for each recorded gait cycle. The CA index was calculated following the method described in Unnithan et al. (1996a) for three agonist–antagonist muscle couples (RF/ST, VM/ST, SO/TA) for both legs in the CP children and the TD children. For each muscle, the maximal value provided from all gait trials was used to normalize the range of the linear envelope. This normalization method was preferred to the use of a maximal voluntary contraction performed on a dynamometer because of the difficulty for children, especially with CP, to produce a sufficient and reproducible level of voluntary muscle activation (Damiano et al., 2000). Integration of the overlapping area between the two normalized linear envelopes defined the CA index (Fig. 1).

3. Statistical analysis

Between group differences for age, height, and body mass were examined using a Wilcoxon test.

A repeated measures Anova was used to test the effect of each speed condition (slow, spontaneous, fast) on the measured non-dimensional gait speed values (dependent variables) in the UL, IL, and TDL.

A linear mixed ANCOVA modeled the CA index (dependent variable) as a function of the non-dimensional gait speed and type of lower limb (covariates) with regard to subject repeatability

Table 1
Subjects' characteristics and between-group comparisons (CP: children with cerebral palsy; TD: typically developing children).

Variable	CP children (n = 10) Mean (Standard deviation)	TD children (n = 10) Mean (Standard deviation)	Wilcoxon test (W/p-value)
Subject characteristics			
Age (years)	10.1 (2.4)	10.2 (4.1)	59.5/0.78
Height (mm)	1410 (183)	1384 (145)	46.5/0.57
Body mass (kg)	33.8 (12.1)	33.3 (9.8)	53.0/0.91

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