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The influence of gait speed on co-activation in unilateral spastic cerebral palsy children $\overset{\scriptscriptstyle \bigwedge}{\overset{\scriptscriptstyle \leftarrow}{}}$

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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

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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

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			

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 interelectrode 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



Fig. 1. Illustration of the co-activation index computation (co-activation area) for a typically developing child limb in the rectus femoris (rf)/semitendinosus (ST) muscle couple.

(random variable). The random variable allows linking all measurements obtained for the same subject and thus correlation between them. It also distinguishes within-individuals variance from betweenindividuals variance. The first mixed model tested the effect of the type of lower limb (fixed independent variables): IL, UL, and TDL for a muscle couple. The second mixed model evaluated the behavior of the three muscle couples (fixed independent variables): RF/ST, VM/ST and SO/TA within the same lower limb type. If there were significant interactions with gait speed, pairwise contrasts were used to statistically identify differences in the CA slopes.

All statistical analyses were carried out using R 2.13. (R Development Core Team, 2011) for Windows XP. The "nlme" package was used to implement a linear mixed model. The alpha-risk was set at 0.05. Bonferroni adjustment was used for pairwise comparisons involving an alpha-risk at 0.016 (0.05/3) for the comparison between two groups.

4. Results

No significant differences between CP and TD children were found for age (P=0.78), height (P=0.57) or body mass (P=0.91).

No significant differences were found between the three different types of limbs (IL, UL, and TDL) for the effect of speed condition on the non-dimensional gait speed values (*slow*: F = 0.08, *P*-value 0.92, *spontaneous*: F = 0.17, *P*-value 0.84, *fast*: F = 0.23, *P*-value 0.79).

Fig. 2 shows changes in the CA index as a function of the nondimensional gait speed for all children. Gait speed had a significant effect on the CA index for all the muscle couples and the lower limb types (Table 2). CA increased with gait speed in each case.

Interactions (Table 2) showed the effect of the limb type on the regression between the CA index and the non-dimensional gait speed. Comparison of the two groups of children showed significant differences between the IL and the TDL for all muscle couples but no differences between the UL and the TDL. In the CP group, the effect of gait speed was significantly greater in the IL compared with the UL.

The effect of gait speed on the CA index for the three muscle couples within each limb was analyzed using the second mixed model. A significant interaction was found in the TDL (Table 3). Pairwise contrasts revealed a different behavior of the RF/HA muscle couple, with a smaller increase in the CA index with gait speed than the other two muscles couples (Fig. 3c). There were no differences between the slopes of the VM/HA and SO/TA couples. In contrast, in the CP group no interactions were found (Table 3) for either the IL



Fig. 2. Changes in the CA index as a function of the non-dimensional gait speed for all children and effect of the limb type on the regression between CA and non-dimensional gait speed. Gait speed had a significant effect on the CA index for all the muscle couples and the lower limb types. The linear regressions show no differences between the UL and the TDL. Contrary, the effect of gait speed on the CA index in the IL was higher than its effect in the UL and TDL for all the muscle couples. UL: uninvolved limbs of CP children. IL: involved limbs of CP children.

or UL. This indicates that the impact of gait speed on the CA index was similar for the three muscle couples (Fig. 3a and b) in CP children.

5. Discussion

The aim of this study was to determine the respective effects of gait speed and impairments caused by an upper motor neuron syndrome on CA in the lower limbs during gait. The effect of gait speed on muscle CA values in the lower limbs of hemiplegic CP and TD children was characterized. As hypothesized, the CA index was speed-dependent in both populations and this effect was most pronounced in the IL. Speed-dependent changes in CA occurred in both thigh and shank muscles. However, the level of speeddependent CA differed between segments for the TD children (the

Table 2

Mixed model results indicating the effect of the type of lower limb (TDL: typically developing children limbs; IL: involved limbs; UL: uninvolved limbs) on the regression between the non-dimensional gait speed and the CA index for the three different muscle couples. The CA index increases significantly with gait speed in all muscle couples and lower limb types. Significant interactions show that the increase of the CA index with gait speed is higher in the IL than in the UL or in the TDL, whatever the muscle couple. No difference is present between the UL and the TDL.

	Slope of the regression	Dimensionless gait speed effect	Interaction effects Lower limb type/dimensionless gait speed	
Muscle couples			Inter-subject comparisons (with TDL as reference)	Inter-CP children comparisons (with UL as reference)
		F-value _(dof) (P-value)	F-value _(dof) (P-value)	F-value _(dof) (P-value)
Rectus femoris/semi-	tendinosus			
TDL	0.11	131.19 _(1.1250) (< 0.001 *)		
UL	0.26	44.85 _(1,1250) (<0.001*)	$0.24_{(1,1250)}(0.62)$	
IL	0.47	44.85 _(1,1250) (< 0.001*)	42.66 _(1,1250) (< 0.001*)	21.55 _(1,533) (<0.001*)
Vastus medialis/semi	-tendinosus			
TDLs	0.17	316.22 _(1.1250) (< 0.001*)		
UL	0.28	152.54 _(1.1250) (< 0.001 *)	$0.42_{(1.1250)}(0.5125)$	
IL	0.40	152.53 _(1,1250) (< 0.001*)	35.09 _(1,1250) (< 0.001*)	8.72 _(1,533) (0.003*)
Soleus/tibialis anterio	or			
TDLs	0.19	204.79 _(1,1250) (< 0.001*)		
UL	0.23	78.01 _(1,1250) (< 0.001 *)	$2.95_{(1,1250)}(0.08)$	
IL	0.36	78.01 _(1,1250) (< 0.001*)	9.78 _(1,1250) (0.002*)	12.52 _(1,533) (0.001*)

Bold p-values associated with * indicate statistical significance (p<0.05).

RF/ST muscle couple was less affected by walking speed than the shank muscle couples) but not for the children with CP.

5.1. Computation of the co-activation index

In the literature, CC and CA have been measured during different motor tasks, whether analytical evaluations (dynamometric exertions) or functional tasks (stepping, jumping, walking, running). Various methods of EMG data processing have been used in order to quantify CA using indices (Damiano et al., 2000; Falconer, 1985; Ikeda et al., 1998; Unnithan et al., 1996a). These studies all used different CA index computing processes. We chose to use a CA index computation method which was very similar to the "co-contraction index" described by Unnithan et al. (1996a), based only on EMG data. This choice was made because to date, that study is the only one which investigated CA in TD and CP children in both thigh and shank muscles during gait, similarly to the present study. The intensity of electrical muscular coactivation (CA index) provides information about coactivation of agonist/antagonist muscle couples and therefore insights into motor control, as EMG is the primary signal to describe the input to the muscular system (Winter, 2009).

Table 3

Results of the mixed linear model comparing the impact of dimensionless gait speed on the CA index in the three muscle couples (rectus femoris (RF)/semi-tendinosus (ST), vastus medialis (VM)/semi-tendinosus and soleus (SO)/tibialis anterior (TA)) for the different limb types: typically developing children limbs (TDL) and involved limbs (IL), uninvolved limbs (UL) of CP children. The interaction effect (muscles/non dimensional gait speed) indicates a significant effect of the muscle couple, i.e. the regressions between CA and speed present significantly different slopes for the different couples. Consequently, pairwise comparisons were used to identify differences. The results show that the CA index as a function of gait speed is significantly different (lower) for the RF/ST than for VM/ST and SO/TA, in the TDL only.

	Interaction effect Muscles/dimensionless gait speed F-value(dep. (P-value)	Pairwise comparisons of slopes		
	r varae(ab) (r varae)	Muscles	F-value _(dof) (P-value)	
IL III	$2.14_{(1,791)} (0.11)$ $2.35_{(1,791)} (0.10)$			
TDL	24.61 _(1,2176) (<0.001*)	RF/ST — VM/ST RF/ST — SO/TA	23.69 _(1,2176) (< 0.001 *) 25.54 _(1,2176) (< 0.001 *)	
		VM/ST – SO/TA	$0.32_{(1,2176)}(0.57)$	

Cross-talk between muscles is known to be an important source of errors when using surface EMG electrodes. Surface EMG studies of agonist/ antagonist behavior can be susceptible to cross-talk where the activity of distant but more strongly activated antagonist muscles contaminates the agonist EMG recording (De Luca and Merletti, 1988). Cross-talk from an antagonistic muscle could render the CA index inaccurate. In this study, the CA index was compared between the two legs of CP subjects, and between CP subjects and TD children who were age-matched. Therefore any cross-talk which occurred should be almost the same between groups although an interaction cannot be excluded. Although fine wire electrodes are known to reduce cross-talk (Nene et al., 2004), we chose not to use such an invasive technique.

Another possible confounding factor in the study of CA in CP and TD children is the different walking speeds in these two populations (Gage and Schwartz, 2009). Using the scaled data, the gait speed in the slow, spontaneous, and fast walking conditions was similar between the TD and CP children, therefore CA was assessed on a comparable range of walking speeds. Although the use of a treadmill would have offered a more accurate control of gait speed, it has been shown that during treadmill walking, hip flexion is altered and other gait disturbances occur (Alton et al., 1998; Lee and Hidler, 2008). In this study, the inter-subject variability was mastered by the use of a non-dimensional gait speed, and the intra-subject variability (from cycle to cycle) was mastered by the mixed linear model.

5.2. Effect of gait speed on CA

Our results are in accordance with previous studies that showed an increase in muscle CA with gait speed in children with CP (Damiano et al., 2000; Unnithan et al., 1996a) and in TD children (Detrembleur et al., 1997; Frost et al., 1997; Unnithan et al., 1996a). Similar results have also been found in healthy adults (Peterson and Martin, 2010). It has been suggested that in TD children, the increase in CA with gait speed is a strategy to increase joint stability, which is challenged by higher mechanical constraints during faster gait (Detrembleur et al., 1997).

For all muscle couples (RF/ST, VM/ST, and TA/SO), there was a greater effect of gait speed on the CA index in the IL of the CP children compared with their UL or with the TDL. This greater effect of speed on CA values on the affected side of CP children could be related to the altered velocity threshold of the stretch reflexes due to the



Fig. 3. Effect of the muscle couple: rectus femoris (rf)/semitendinosus (ST), vastus medialis (vm)/semitendinosus (st) and soleus (so)/tibialis anterior (TA) on the co-activation index (CA) evolution with gait speed for the three lower limb types: a) involved limbs (IL); b) uninvolved limbs (UL) and c) typically developing children limbs (TDL). Linear regressions only revealed a significant difference for the TDL (Fig. c): the impact of gait speed in the CA index was lower in the RF/ST than in the other muscle couples.

cerebral lesions. It might also be related to increased short-latency reflex bursts following foot-contact in leg muscles (Leonard et al., 1991). Our results suggest that a pathological component of muscle activation due to the upper motor neuron lesion and consequent lack of reciprocal inhibition might be responsible for the increase in CA with gait speed in the IL. These results are in contrast with data in adult hemiplegic patients, who present more co-activation on their non-paretic side, probably by compensatory, stabilization mechanisms (Lamontagne et al., 2000). A more precise characterization of the nature and origin of the pathological component in this excessive co-activation might require timing analysis of the EMG, to test for spasticity. Timing analysis is performed to detect muscle activation (Hortobágyi et al., 2009). We are not aware of studies using timing quantitative analysis to characterize the time delay of initiation of muscle firing, for instance to test for spasticity. Investigations using a musculo-skeletal model could also be used to evaluate the correlation between CA and muscle stretch to test this hypothesis.

In the TDL, the effect of walking speed on the CA index was significantly lower for the RF/ST muscle couple than for VM/ST and SO/TA. In contrast, in the CP children, no differences in the degree of CA between the different muscles were found in either the UL or the IL. These results indicate that physiologically, CA in the RF/HA couple is less sensitive to gait speed that in the other muscle couples. This specific aspect of motor control in this couple appears to be altered in CP children. In healthy subjects, the RF has been shown to contract only during the stance-to-swing transition, to allow limb advancement, especially at higher gait speeds (Nene et al., 2004). The hamstrings contract from terminal swing until the end of the loading response (LR) to decelerate knee extension and stabilize the knee during the LR (Perry, 2010). Since the RF and HA contract at opposite times of the gait cycle, there is little CA in this couple, even during fast gait. This probably explains why speed has little effect on the CA index in this muscle couple in the TD children. However, in the IL of CP children the CA in the RF/ST could be more affected by speed because of inappropriate activity related to spasticity. Larger, more controlled studies are needed to confirm that the upper motor neuron lesions in CP children reduce the ability to enhance selective muscle contraction. Two hypotheses could be put forward to explain why this difference was also found in the UL. Firstly, spasticity could be present in the uninvolved side. Even if the children were classified as having unilateral forms of CP according to the current taxonomy, it is possible that an upper motor neuron syndrome may also affect the uninvolved limb (Bax et al., 2005). Secondly, the increased CA of the RF/HA couple in the UL could attempt to compensate for the impairments of the IL. If this increased CA in the unaffected side occurs during stance phase, particularly during double-support, it may improve stability at faster gait speeds. The decomposition of the gait cycle into phases and computation of a CA index for each phase could answer this question in future studies.

5.3. Limitations of the study

The clinical applications of the results of this study might be restrained by the small sample size. Larger studies into the CA of children with hemiplegic CP must be carried out in order to confirm our results even if, like Unnithan et al. (1996a), we obtained significant results. As aforementioned, the assessment of muscular activation by the use of a CA index does not allow interpretation of the nature of the excessive activation. A markedly different methodology is necessary for that purpose, for example timing analysis. Moreover, we calculated a CA index for the entire gait cycle, while the computation of indexes for the different sub-phases of the cycle might be necessary for a better identification of the pathological and adaptive components of CA in the involved and uninvolved limbs. The precise nature of the link between gait speed and co-activation is unattainable by our results: a different testing protocol, using a treadmill and multiple speeds separated by small intervals would be necessary to precise the effect of speed on CA in the different limbs (linear or exponential).

5.4. Potential clinical applications

Excessive muscular co-activation is present in the involved limbs of unilateral CP children when they increase walking speed. As the ability to increase walking speed in this population is decreased (Fonseca et al., 2001), a hypothesis can be made about the causal link between increased co-activation and decreased ability to accelerate in these children. In this way, our results bring arguments for the use of treatments of muscular hyperactivity (for instance botulinum toxin) to improve walking speed in this population of unilateral CP children. The effects of such treatments on muscular co-activation should be evaluated. Moreover, our findings should help the clinicians performing dynamic EMG analysis of these children to understand the changes with time, or treatment, for a single individual, in the light of walking speed.

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References

- Alton, F., Baldey, L., Caplan, S., Morrissey, M., 1998. A kinematic comparison of overground and treadmill walking. Clin. Biomech. 13, 434–440.
- Bax, M., Goldstein, M., Rosenbaum, P., Leviton, A., Paneth, N., Dan, B., et al., 2005. Proposed definition and classification of cerebral palsy, April 2005. Dev. Med. Child Neurol. 47, 571–576.
- Beck, R.J., Andriacchi, T.P., Kuo, K.N., Fermier, R.W., Galante, J.O., 1981. Changes in the gait patterns of growing children. J. Bone Joint Surg. 63, 1452–1457.
- Bowsher, K.A., Damiano, D., Vaughan, C.L., 1993. Joint torques and co-contraction during gait for normal and cerebral palsy children. J. Biomech. 26, 326.
- Btk Development Core Team, 2011. Btk: a Biomechanical ToolKit.
- Damiano, D., 1993. Reviewing muscle cocontraction: is it developmental, pathological, or motor control issue? Phys. Occup. Ther. Pediatr. 12, 3–20.
- Damiano, D., Martellotta, T., Sullivan, D., Granata, K., Abel, M., 2000. Muscle force production and functional performance in spastic cerebral palsy: relationship of cocontraction. Arch. Phys. Med. Rehabil. 81, 895–900.
- Davis, R., Õunpuu, S., Tyburski, D., Gage, J., 1991. A gait analysis data collection and reduction technique. Hum. Mov. Sci. 10, 578–587.
- De Luca, C.J., Merletti, R., 1988. Surface myoelectric signal cross-talk among muscles of the leg. Electroencephalogr. Clin. Neurophysiol. 69, 568–575.
- Desailly, E., Yepremian, D., Sardain, P., Lacouture, P., 2009. Foot contact event detection using kinematic data in cerebral palsy children and normal adults gait. Gait Posture 29, 76–80.
- Detrembleur, C., Willems, P., Plaghki, L., 1997. Does walking speed influence the time pattern of muscle activation in normal children? Dev. Med. Child Neurol. 39, 803–807.
- Falconer, K., 1985. Quantitative assessment of co-contraction at the ankle joint in walking. Electromyogr. Clin. Neurophysiol. 25, 135–149.
- Fonseca, S.T., Holt, K.G., Saltzman, E., Fetters, L., 2001. A dynamical model of locomotion in spastic hemiplegic cerebral palsy: influence of walking speed. Clin. Biomech. 16, 793–805.
- Frost, G., Dowling, J., Dyson, K., Bar-Or, O., 1997. Cocontraction in three age groups of children during treadmill locomotion. J. Electromyogr. Kinesiol. 7, 179–186.
- Gage, J.R., Schwartz, M.H., 2009. Consequences of brain injury on musculoskeletal development. In: Gage, J.R., Schwartz, M.H., Koop, S.E., Novacheck, T.F. (Eds.), The Identification and Treatment of Gait problems in Cerebral Palsy. Mac Keith Press, London, pp. 107–129.

- Hermens, H.J., Freriks, B., Merletti, R., Rau, G., Disselhorst-Klug, C., Stegeman, D.F., et al., 2000. European Recommendations for Surface Electromyography: Results of the SENIAM Project.
- Hof, A., 1996. Scaling gait data to body size. Gait Posture 4, 222–223.
- Hortobágyi, T., Solnik, S., Gruber, A., Rider, P., Steinweg, K., Helseth, J., et al., 2009. Interaction between age and gait velocity in the amplitude and timing of antagonist muscle coactivation. Gait Posture 29, 558–564.
- Hubley-Kozey, C., Deluzio, K., Dunbar, M., 2008. Muscle co-activation patterns during walking in those with severe knee osteoarthritis. Clin. Biomech. 23, 71–80.
- Humphrey, D.R., Reed, D.J., 1983. Separate cortical systems for control of joint movement and joint stiffness: reciprocal activation and coactivation of antagonist muscles. In: Mackel, R. (Ed.), Motor Control Mechanisms in Health and Disease. Raven Press, New York, pp. 347–372.
- Ikeda, A.J., Abel, M.F., Granata, K.P., Damiano, D.L., 1998. Quantification of cocontraction in spastic cerebral palsy. Electromyogr. Clin. Neurophysiol. 38, 497–504.
- Johansson, R.S., Westling, G., 1988. Coordinated isometric muscle commands adequately and erroneously programmed for the weight during lifting task with precision grip. Exp. Brain Res. 71, 59–71.
- Lamontagne, A., Richards, C.L., Malouin, F., 2000. Coactivation during gait as an adaptive behavior after stroke. J. Electromyogr. Kinesiol. 10, 407–415.
- Lee, S.J., Hidler, J., 2008. Biomechanics of overground vs. treadmill walking in healthy individuals. J. Appl. Physiol. 104, 747–755.
- Leonard, C.T., Hirschfeld, H., Forssberg, H., 1991. The development of independent walking in children with cerebral palsy. Dev. Med. Child Neurol. 33, 567–577.
- Nene, A., Byrne, C., Hermens, H., 2004. Is rectus femoris really a part of quadriceps? Assessment of rectus femoris function during gait in able-bodied adults. Gait Posture 20, 1–13.
- Olney, S., 1985. Quantitative evaluation of cocontraction of knee and ankle muscles in normal walking. In: Winter, D.A., Norman, R.W., Wells, R.P., Hayes, K.C., Patla, A.E. (Eds.), Biomechanics IX-A, Human Kinetics, Champaign (IL), pp. 114–117.
- Perry, J., 2010. Gait Analysis: Normal and Pathological Function, 2nd edition. Slack Incorporated. Thorofare.
- Peterson, D.S., Martin, P.E., 2010. Effects of age and walking speed on coactivation and cost of walking in healthy adults. Gait Posture 31, 355–359.
- R Development Core Team, 2011. R: A Language and Environment for Statistical Computing (Vienna, Austria).
- Shiavi, R., Bugle, H.J., Limbird, T., 1987. Electromyographic gait assessment, part 2: preliminary assessment of hemiparetic synergy patterns. J. Rehabil. Res. Dev. 24, 24–30.
- Unnithan, V.B., Dowling, J.J., Frost, G., Volpe Ayub, B., Bar-Or, O., 1996a. Cocontraction and phasic activity during gait in children with cerebral palsy. Electromyogr. Clin. Neurophysiol. 36, 487–494.
- Unnithan, V.B., Dowling, J.J., Frost, G., Bar-Or, O., 1996b. Role of cocontraction in the O2 cost of walking in children with cerebral palsy. Med. Sci. Sports Exerc. 28, 1498–1504.
- Valero-Cuevas, F.J., 2005. An integrative approach to the biomechanical function and neuromuscular control of the fingers. J. Biomech. 38, 673–684.
- Winter, D.A., 2009. Biomechanics and Motor Control of Human Movement, 4th edition. John Wiley & Sons Inc., New York.