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Does muscle coactivation influence joint excursions during gait in children with and without hemiplegic cerebral palsy? Relationship between muscle coactivation and joint kinematics☆

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ABSTRACT

Background: The theoretical role of muscle coactivation is to stiffen joints. The aim of this study was to assess the relationship between muscle coactivation and joint excursions during gait in children with and without hemiplegic cerebral palsy.
Methods: Twelve children with hemiplegic cerebral palsy and twelve typically developing children underwent gait analysis at three different gait speeds. Sagittal hip, knee, and ankle kinematics were divided into their main components corresponding to joint excursions. A coactivation index was calculated for each excursion from the electromyographic envelopes of the rectus femoris/semitendinosus, vastus medialis/semitendinosus, or tibialis anterior/soleus muscles. Mixed linear analyses of covariance modeled joint excursions as a function of the coactivation index and limb.
Findings: In typically developing children, increased coactivation was associated with reduced joint excursion for 8 of the 14 linear models (hip flexion, knee loading, knee extension in stance, knee flexion in swing, ankle plantarflexion from initial contact to foot-flat, ankle dorsiflexion in stance and in swing). Conversely, ankle plantarflexion excursion at push-off increased with increasing tibialis anterior/soleus coactivation. In the involved limbs of the children with cerebral palsy, knee loading, ankle plantarflexion at push off, and ankle dorsiflexion in swing decreased, while hip extension increased, with increasing muscle coactivation.
Interpretation: The relationships between muscle coactivation and joint excursion were not equally distributed in both groups, and predominant in typically developing children. The results suggest that excessive muscle coactivation is not a cause of stiff-knee gait in children with hemiplegic cerebral palsy, but appears to be related to spastic drop foot.

1. Introduction

In patients with spastic paresis, the relationships between the different aspects of the upper motor neuron syndrome (weakness, spasticity, coactivation, loss of selectivity), and gait parameters or functional performance remain controversial. Whether these impairments are equally or independently distributed in a child with cerebral palsy (CP), and which impairments are behind the abnormal gait pattern and limitation of function, are a matter of debate (Gage et al., 2009). These pathological gait patterns of children with CP are a mixture of primary, secondary, and tertiary abnormalities. It is important to distinguish between these deviations, as the primary (disordered motor control) and secondary (musculo-skeletal deformities) abnormalities are impairments and must be corrected as far as possible, while tertiary abnormalities should resolve following treatment of the impairments (Gage et al., 2009). One of the primary impairments which has been considered as detrimental to functional performance in children with CP during gait is muscle coactivation (Unnithan et al., 1996; Gross et al., 2013), defined as the simultaneous activation of two antagonist muscles. It is often believed to be the direct result of the upper motor neuron lesion (Gracies, 2005), however increased coactivation has also been found in the non-paretic limbs of children with hemiplegic cerebral palsy (Gross et al.,

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2013), and of adults with stroke (Lamontagne et al., 2000). This suggests that it could also be a compensatory mechanism (i.e. to increase joint stability to cope with poor motor control). It is classically believed that the coactivation of the antagonist muscle opposes the moment of force created by the agonist muscle, thus restraining movement (Winter, 2009). However, some studies have questioned the role of coactivation to restrict joint motion or force/moment production. While a recent study found a negative correlation between coactivation of the tibialis anterior/soleus muscle pair and dorsiflexion torque during isometric contractions in adult subjects with hemiparesis (Vinti et al., 2013), other studies have found no association between the level of coactivation and force production in stroke or cerebral palsy patients (Horstman et al., 2009; Moreau et al., 2009). It is thus necessary to quantify coactivation during functional tasks such as gait and to relate it to kinematic impairments, in order to give insights into its potential deleterious role on joint excursions.

The aim of the present study was therefore to investigate the relationship between coactivation of agonist-antagonist muscle pairs in the lower limb and the excursion of the corresponding joints during gait in typically developing (TD) children and children with hemiplegic cerebral palsy (CP). We hypothesized that in both TD and children with CP, there would be a negative association between coactivation and joint excursion, i.e. muscle coactivation would reduce active joint motion.

2. Methods

2.1. Participants

This study was granted approval by the institutional ethical committee and all children and parents provided informed consent. Twelve children with hemiplegic CP (mean age = 9.9 years, SD = 3.9) and 12 age-matched TD children (mean age = 9.7 years, SD = 2.4) were recruited for the study. Children with CP were included if they had strictly unilateral involvement with a predominance of spasticity (Baxter et al., 2007), a GMFCS level of I (Palisano et al., 1997), and habitually walked without an ankle-foot orthosis.

2.2. Data Acquisition

The children were asked to walk barefoot and unassisted down a 12-m walkway in the gait lab in three speed conditions. Orthoses were not permitted in order to record natural gait. Previous studies have shown that the level of coactivation in lower limb muscles increases with gait speed in both TD and children with CP (Unnithan et al., 1996; Damiano et al., 2000; Gross et al., 2013). Gait speed is also known to influence joint excursions (Stansfield et al., 2006; Schwartz et al., 2008). Therefore, the children were asked to walk at different speeds so that a range of muscle coactivation levels and of joint excursions could be analyzed. It was not necessary to precisely control gait speed, as our objective was to obtain a range of speeds. The children were first asked to walk at their self-selected comfortable speed. Next, they were instructed to walk markedly faster, without running. Finally, they were instructed to walk slower than during their spontaneous gait.

For each speed, three practice trials were performed so that the child could accommodate his/her gait. The data of the following four successive gait trials were retained for analysis. Six optoelectronic Vicon MX-F40 cameras (Oxford Metrics, Oxford, UK) recorded the displacement of reflective markers which were positioned on the subjects according to the Plug-in Gait conventions (Davis et al., 1991). Electromyographic (EMG) data were simultaneously recorded at 1000 Hz using a wireless surface EMG system (ZeroWire EMG, Aurion S.r.l., Milano, Italy). The activity of 5 muscles was recorded in each lower limb: rectus femoris (RF), vastus medialis (VM), semitendinosus (ST), tibialis anterior (TA) and soleus (SO). The electrodes were positioned according to the SENIAM recommendations (Hermens et al., 2000).

2.3. Extraction of the Kinematic Parameters of Interest

The three markers on each foot were used for the automatic detection of gait events (Desailly et al., 2009) which was then refined using synchronized high-speed videos.

The data reduction method for kinematics described by Benedetti et al. (1998) was used to define ten phases for analysis, based on sagittal kinematic data, for each joint: two for the hip, four for the knee and four for the ankle (Fig. 1). Joint excursion was calculated as the difference between the two peak values constituting the boundaries of each phase. The peak values were H_2, H_3, H_5, for the hip, K_1, K_2, K_3, K_5, and K_{13} for the knee, and A_1, A_2, A_3, A_5, and $_{A10}$ for the ankle. All these values were defined as in Benedetti et al. (1998), except the K_{13} parameter, defined as minimal knee flexion in the swing phase, and the A_{10} parameter, corresponding to maximal ankle dorsiflexion in the swing phase, which were added in this study. For the hip joint, *extension* ($= H_2–H_3$) and *flexion* ($= H_5–H_3$), constituting the biphasic kinematics of the hip, were analyzed. For the knee joint, *knee loading* ($= K_2–K_1$), *extension in stance* ($= K_2–K_3$), *flexion from late stance to initial swing* ($= K_5–K_3$), and *extension in swing* ($K_5–K_{13}$) were retained as parameters of interest. For the ankle joint, *plantarflexion from initial contact to foot-flat* ($A_1–A_2$), *dorsiflexion in stance* ($= A_3–A_2$), *plantarflexion during push-off* ($= A_3–A_5$), the key-parameters of the ankle/foot kinematics described by Perry (1992) as the first, second, and third foot rockers were analyzed, as well as *dorsiflexion in swing* ($= A_{10}–A_5$), which is crucial to foot clearance in swing.

These phases were identified for all joints in 99 to 100% of the gait cycles except for the knee loading phase ($K_1–K_2$, Fig. 1), which could be defined in 196 cycles (66.7%).

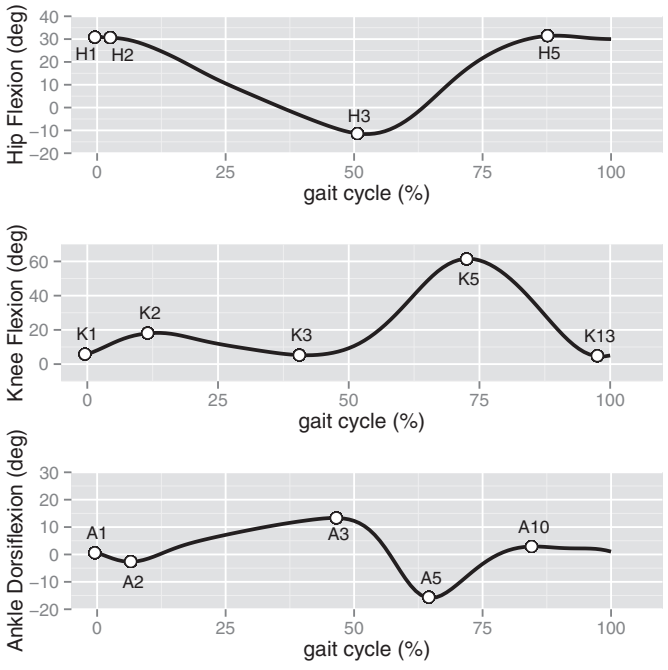

Fig. 1. Illustration of the processing of the kinematic data to obtain the joint excursions: hip flexion/extension (top); knee flexion/extension (middle); and ankle dorsi/plantarflexion (bottom) during the gait cycle. H_1, H_2, H_3, H_5, K_1, K_2, K_3, K_5, A_1, A_2, A_3 and A_5 were defined as in Benedetti et al. (1998). The K_{13} parameter, defined as minimal knee flexion in the swing phase, and the A_{10} parameter, corresponding to maximal ankle dorsiflexion in the swing phase, were added in this study. For the hip joint, *extension* ($= H_2–H_3$) and *flexion* ($= H_5–H_3$) were analyzed. For the knee joint, *knee loading was defined as* $K_2–K_1$, *extension in stance* as $K_2–K_3$, *flexion from late stance to initial swing* as $K_5–K_3$, and *extension in swing* as $K_5–K_{13}$. For the ankle joint, *plantarflexion* from initial contact to foot-flat ($= A_1–A_2$), *dorsiflexion in stance* ($= A_3–A_2$), *plantarflexion during push-off* ($= A_3–A_5$), as well as *dorsiflexion in swing* ($= A_{10}–A_5$) were analyzed.

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2.4. EMG Processing and Calculation of the Coactivation Index

EMG recordings were full wave rectified and filtered using a fourth-order Butterworth 8.9 Hz low pass filter (Shiavi et al., 1998) with phase correction, to create a linear envelope for each gait cycle (see Fig. 2). For each muscle and each child, the maximal EMG value obtained from all the gait trials was used to normalize the range of the linear envelope (Damiano et al., 2000; Gross et al., 2013). The EMG envelopes of two antagonist muscles were integrated within each identified phase (Fig. 2) to obtain a coactivation index corresponding to the joint excursion:

$$\text{Coactivation Index} = \frac{\int \min(\text{EMG}_1(t),\ \text{EMG}_2(t))dt}{\int \text{EMG}_1\ (t)\ dt + \int \text{EMG}_2\ (t)\ dt} \quad (1)$$

where $\text{EMG}_1\ (t)$ and $\text{EMG}_2\ (t)$ are the EMG signals of the two antagonist muscles (see Fig. 2). The coactivation index was 0 if one or both muscles were not active during the phase (Falconer and Winter, 1985).

In this way, several indexes of coactivation were obtained for each stride. The following muscle pairs were analyzed: for hip excursions: RF/ST, for knee excursions, both RF/ST and VM/ST and for ankle excursions TA/SO. All the computations were performed using MATLAB R2008b (Mathworks, Natick, MA, USA) and the open-source Biomechanical ToolKit package for MATLAB (Barre and Armand, 2014).

2.5. Statistical Analysis

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

Mixed linear models (analyses of covariance [ANCOVAs]) with repeated measures were used to assess the relationship between the coactivation index and joint excursion in the groups of TD and CP children. The lower limbs were categorized in two types: the lower limbs of the TD children (TDL) (the left or right limb was randomly selected), and the involved (IL) lower limbs of the children with CP. The data from the uninvolved limbs of CP children were not analyzed in this study. For each phase, a mixed linear ANCOVA with repeated-measures was used to model the excursion (dependent variable) as a function of a covariate (coactivation index) and the type of lower limb (independent variable), and as a function of subject heterogeneity (random variable). To describe the relationship between joint excursion and coactivation, the term "positive" is used to indicate that the joint excursion increased when the coactivation index increased, while "negative" indicates that the joint excursion decreased when the coactivation index increased.

All statistical analyses were carried out using R 2.13 (R Development Core Team, 2011) for Windows XP. The significance level was set at 0.05. Post-hoc tests were performed using Bonferroni adjustments.

3. Results

There were no significant differences between the TD children and children with CP for age ($P = 0.78$), height ($P = 0.57$) or body mass ($P = 0.91$). The gait speeds of the TD participants were 0.89 ± 0.14 m/s for slow gait, 1.14 ± 0.17 m/s for spontaneous gait, and 1.47 ± 0.20 m/s for fast gait. The gait speeds of the CP participants were 0.69 ± 0.19 m/s for slow gait, 1.01 ± 0.20 m/s for spontaneous gait, and 1.49 ± 0.23 for fast gait.

3.1. Hip Kinematics

A positive relationship was found between hip extension excursion (H_2–H_3) and coactivation in the RF/ST muscle pair in the IL, but not in the TDL. Conversely, a negative relationship was found between hip flexion excursion (H_5–H_3) and coactivation in RF/ST in the TDL, but not in the IL (see Table 1).

3.2. Knee Kinematics

3.2.1. RF/ST muscle coactivation

Negative relationships were found between RF/ST coactivation and knee extension in stance excursion (K_2–K_3) and knee flexion excursion

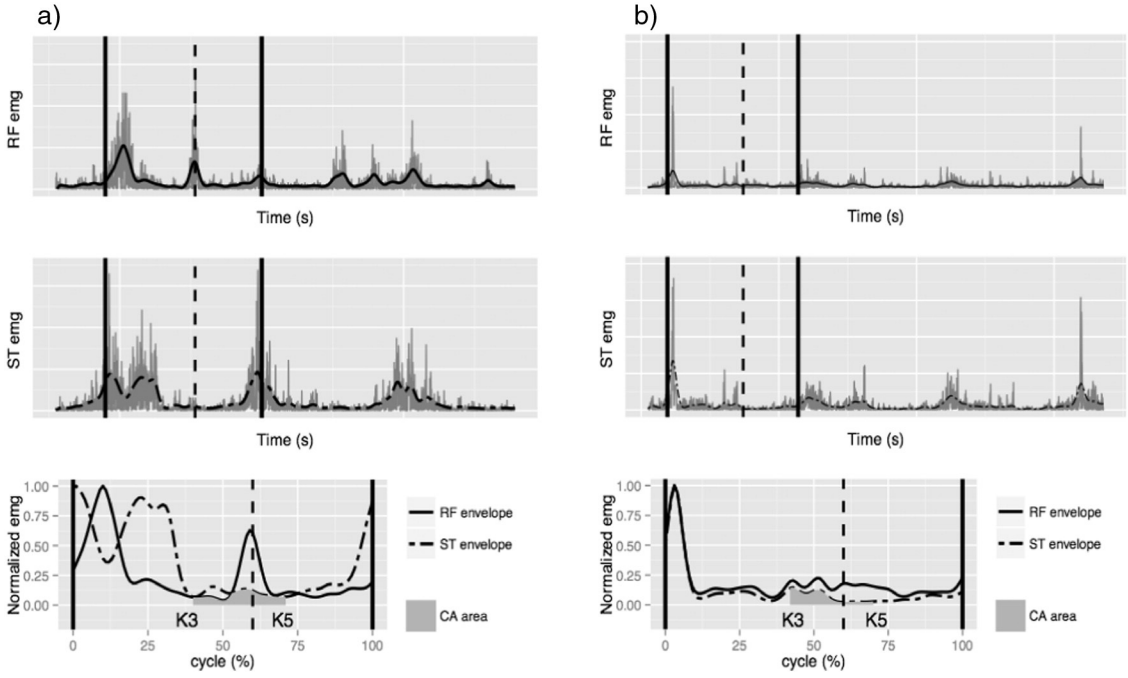

Fig. 2. EMG processing for a) a typically developing child and b) a child with hemiplegic cerebral palsy. Part 1 (top and middle): examples of dynamic electromyographic (EMG) signals during two consecutive strides. The rectified EMG signals of the rectus femoris (RF) and semitendinosus (ST) appear in gray. The full bars indicate initial contact and the dashed bars indicate foot-off. Part 2 (bottom): the EMG envelopes of rectus femoris (RF, full line), and semitendinosus (ST, dashed line), are presented for one stride. The EMG envelopes were normalized with respect to the maximal value of the EMG from the same muscle across all gait cycles. The gray area (the surface under the minimal EMG curve during the stride) corresponds to the coactivation index, in this example for the knee flexion from late stance to initial swing excursion (K_5–K_3).

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Table 1
Values of the slopes associated with the mixed linear models predicting joint excursion as a function of coactivation index (CA) of rectus femoris/semitendinosus (RF/ST).

		Coactivation effects	
		Slope (joint excursion/coactivation)	
		TDL	IL
RF/ST	Hip extension	−0.54 (1.35), $P = 1$	6.86 (1.77), $P < 0.001$
	Hip flexion	−5.21 (0.85), $P < 0.001$	−3.96 (2.11), $P = 0.182$
	Knee loading	−0.24 (0.12), $P = 0.115$	−0.19 (0.07), $P = 0.013$
	Knee extensionin stance	−1.41 (0.34), $P < 0.001$	0.38 (0,77), $P = 1$
	Knee flexion from late stance to initial swing	−2.13 (0.85), $P = 0.036$	0.48 (1.16), $P = 1$
	Knee extensionin swing	0.25 (0.72), $P = 1$	−1.09 (0.65), $P = 0.283$

EMG data from the RF/ST muscle pair are presented in relation with the joint excursions of the hip and knee in the sagittal plane. Data are given as means (standard deviation) with the *P*-values of the mixed linear models.

from late stance to initial swing (K_5–K_3), in the TDL only. Conversely, in the IL, a negative relationship was found between coactivation and knee loading (K_2–K_1) (see Table 1).

3.2.2. VM/ST muscle coactivation

There was a negative relationship between VM/ST coactivation and knee loading (K_2–K_1) in both TDL and IL. A negative relationship was also found for knee extension in stance (K_2–K_3), in the TDL only (see Table 2).

3.3. Ankle Kinematics

Significant relationships were found between coactivation of the TA/SO muscles and ankle motion for all phases analyzed in the TDL, and for two phases in the IL. The relationships were negative for plantarflexion from initial contact to foot-flat (A_1–A_2) in the TDL, dorsiflexion in stance (A_3–A_2) in the TDL, and dorsiflexion in swing (A_{10}–A_5) in both the TDL and IL. Conversely, for ankle plantarflexion during push-off, opposite relationships were found: positive in the TDL and negative in the IL (see Table 3).

4. Discussion

This study used mixed linear models to determine the relationships between joint kinematics and muscle coactivation during gait in TD children and children with unilateral CP. Most of these relationships were negative in TD children, confirming the study hypothesis that muscle coactivation stiffens joints during gait. Ankle plantarflexion during push-off, which increases with muscle coactivation in TA/SO, appears as a noticeable exception. In contrast, in children with CP, few relationships could be evidenced. Some were negative and consistent with the results in TD children, while some were missing, and one was even opposite (ankle plantarflexion during push-off). These results highlight that in children with CP, coactivation is a complex phenomenon, largely not elucidated. Moreover, the scarcity of relationships between coactivation and joint excursions in the involved limbs indicates that alternative mechanisms, such as paresis and muscle contracture, could take a large part in the reduced movement amplitude during gait in children with CP.

The role of each of the symptoms of the upper motor neuron syndrome, referred to as "primary abnormalities" in children with CP (Gage et al., 2009), in the alteration of the gait pattern and reduction of gait performance is still a matter of debate. The deleterious role of spasticity and of cocontraction has been questioned in several studies. For instance, in a study of children with spastic diplegia, spasticity was not found to influence gait and overall function, while strength parameters were (Ross and Engsberg, 2007). Regarding coactivation, Damiano et al. (2000) found no relationship between coactivation in the knee flexor and extensor muscles and strength or functional performance (such as gait speed) in children with spastic CP. Likewise, studies of post-stroke hemiparetic gait have suggested that the observed increased muscle coactivation could be, at least in part, an adaptive mechanism rather than an impairment in the pattern generating mechanisms (Lamontagne et al., 2000; Den Otter et al., 2007). It is important to determine if muscle coactivation is a primary abnormality (impairment) or a tertiary abnormality (compensation) as it should only be treated if it is considered to be a primary abnormality (Gage et al., 2009). This is crucial, all the more so as new treatments to reduce muscle coactivation are currently emerging. For example, locomotor training has been shown to improve muscle coordination during gait (Routson et al., 2013). Biofeedback via a myoelectric computer interface has been proposed by Wright et al. (2013) to reduce muscle CA in the upper limb of stroke survivors. Earlier studies have shown that the injection of botulinum toxin in the ankle plantarflexors is effective in reducing coactivation during active ankle dorsiflexion (Tang et al., 2012). Since treatments for muscle coactivation can be proposed, future studies should determine the effect of such treatments on function and establish treatment indications.

In TD children, a negative relationship was found between muscle coactivation and the following movements: hip flexion, knee loading (only for the VM/ST pair), knee extension in stance, knee flexion from late stance to initial swing (RF/ST only), ankle plantarflexion from initial contact to foot-flat, ankle dorsiflexion in stance, and ankle dorsiflexion in swing. These findings are in accordance with the theory that the role of muscle coactivation is to stiffen joints (Winter, 2009) and

Table 2
Values of the slopes associated with the mixed linear models predicting joint excursion as a function of coactivation index (CA) of vastus medialis/semitendinosus (VM/ST).

		Coactivation effects	
		Slope (joint excursion/coactivation)	
		TDL	IL
VM/ST	Knee loading	-0.36 (0.12), $P = 0.012$	−0.23 (0.08), $P = 0.007$
	Knee extensionin stance	−2.53 (0.61), $P < 0.001$	−0.33 (0.78), $P = 1$
	Knee flexion from late stance to initial swing	−2.17 (0.97), $P = 0.075$	0.21 (1.28), $P = 1$
	Knee extensionin swing	−0.46 (0.72), $P = 1$	0.33 (0.85), $P = 1$

EMG data from the VM/ST muscle pair are presented in relation with the joint excursions of the knee in the sagittal plane. Data are given as means (standard deviation) with the *P*-values of the mixed linear models.

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Table 3
Values of the slopes associated with the mixed linear models predicting joint excursion as a function of coactivation index (CA) of tibialis anterior/soleus (TA/SO).

		Coactivation effects	
		Slope (joint excursion/coactivation)	
		TDL	IL
TA/SO	Ankle plantarfexion from initial contact to foot-flat	−0.05 (0.01), $P = 0.006$	−0.06 (0.05), $P = 0.559$
	Ankle dorsiflexionin stance	−3.17 (0.63), $P < 0.001$	1.11 (2.09), $P = 1$
	Ankle plantarflexion during push-off	2.95 (0.71), $P < 0.001$	−1.61 (0.66), $P = 0.047$
	Ankle dorsiflexionin swing	−3.78 (0.85), $P < 0.001$	−0.56 (0.15), $P = 0.001$

EMG data from the TA/SO muscle pair are presented in relation with the joint excursions of the ankle in the sagittal plane. Data are given as means (standard deviation) with the *P*-values of the mixed linear models.

suggest that muscle coactivation could be a physiological means of reducing movement amplitude, and therefore adjusting joint motion during gait. This role of muscle coactivation is well known in the upper limb (Valero-Cuevas, 2005). In the lower limb, coactivation has been particularly described at the knee joint around initial contact. It is considered to be a means of achieving knee stability during the large moments of force which occur during the loading response (Perry, 1992). The results of the present study showed that the degree of muscle coactivation was associated with some kinematic parameters of the knee joint during both the stance and swing phases of gait. Interestingly, coactivation of RF and ST was associated with decreased knee flexion from late stance to initial swing, while coactivation of VM and ST was associated with reduced knee loading amplitude. This result shows that different patterns of coactivation occur in the muscles of the anterior and posterior compartments of the thigh during the gait cycle to produce appropriate joint kinematics. It has previously been shown that the rectus femoris differs from the other heads of the quadriceps by its specific timing (around toe-off), and by its mechanical action (limb advancement at the beginning of the swing phase) (Nene et al., 2004 Aug). According to our results, the specificity of the rectus femoris among the quadriceps would also stand concerning coactivation with the medial hamstrings and its biomechanical effect.

One unexpected result was that ankle plantarflexion excursion during push-off increased with the degree of TA/SO coactivation in TD children. This appears counter-intuitive, as coactivation is thought to stiffen joints. Muscle coactivation could be needed for the generation of power around the ankle joint during push-off, potentially to ensure a stable forefoot fulcrum. It has been demonstrated that muscle force production and muscle cocontraction can be regulated independently in persons with normal motor control (Basmajian and De Luca, 1985). Simultaneous increases in force production with increasing cocontraction of the thigh muscles have also previously been demonstrated at the knee joint (Damiano et al., 2000) during isometric contractions in children with CP. Increasing coactivation stiffens the joint, while the parallel increase in agonist activity creates the net moment and therefore the joint motion (Damiano et al., 2000). This postulated mechanism appeared to be disrupted in the children with hemiplegic CP in the present study since a negative relationship was found between coactivation in the TA/SO and ankle plantarflexion during push-off.

In the IL of children with CP, a negative relationship was found only between coactivation and joint excursion during knee loading (RF/ST and VM/ST), ankle plantarflexion during push-off, and ankle dorsiflexion in swing. The positive relationship between hip extension and coactivation in the RF/ST muscle pair was specific to CP children and highlights a specific coordination of the thigh muscles during the stance phase. The other joint excursions were not influenced by muscle coactivation. Of particular importance is the fact that there was no relationship between coactivation of RF and ST or VM and ST and knee flexion from late stance to initial swing in this group. This suggests that coactivation of these muscle pairs is not a cause of stiff knee gait in children with hemiplegic CP. Winters et al. (1987) hypothesized that the simultaneous contraction of the knee flexor and extensor muscles limits knee flexion during swing in hemiplegic children with a flexed, stiff-knee ("group III"), however the results of the present study do not support this. Nevertheless, it must be taken into account that the children with CP in this study did not all have stiff-knee gait. It is possible that in a larger sample of selected group III children, the influence of RF/ST or VM/ST coactivation on knee flexion from late stance to initial swing would have been significant. It would be interesting to investigate this relationship in a selected sample of children with hemiplegic CP and stiff-knee gait. If confirmed, the lack of a relationship would suggest that stiff knee gait is mostly caused by other mechanisms, such as reduced ankle and/or hip power generation in late stance (Goldberg et al., 2003 Aug).

Another impairment which frequently occurs in hemiplegic CP is spastic drop foot (Winters et al., 1987). Our results showed that there was a relationship between coactivation of the ankle dorsi/plantar flexors and excursion of ankle dorsiflexion in swing in the IL. This suggests that coactivation of TA and SO could be a cause of spastic drop foot of children with hemiplegic CP. Therefore, the use of a treatment that reduces muscle coactivation, targeting the shank muscles, appears to be relevant given our results.

4.1. Study Limitations

4.1.1. Population

The children with cerebral palsy included in the present study had a GMFCS level of I, and they habitually walked without orthoses. The results therefore cannot be forecasted to more severely impaired children with unilateral CP or to children with spastic diplegia.

4.1.2. EMG processing

The frequency at which the rectified EMG data is low pass filtered determines the shape of the obtained envelope. A recent review failed to establish recommendations regarding the most appropriate filter frequency for the computation of coactivation indexes (Rosa et al., 2014). Previous studies have used frequencies ranging from 3 Hz to 25 Hz. We chose a frequency of 8.9 Hz since it has been previously demonstrated that this frequency retains 95% of the power of the signal, even for data acquired during fast gait (Shiavi et al., 1998).

Previous studies of pathological muscle coactivation during gait normalized the EMG data either by peak EMG activity during maximum voluntary contractions or peak/mean EMG values during gait (Rosa et al., 2014). Some authors even chose not to normalize EMG data (Lamontagne et al., 2000). The reliability and validity of the classical MVC method for the evaluation of maximal motor output in children or adults with spastic paresis have been questioned (Burden and Bartlett, 1999; Lamontagne et al., 2000). Moreover, mean ensemble and peak ensemble values (maximum value reached within a period), have both been considered as feasible methods for the normalization data in subjects with neurological disorders (Yang and Winter, 1984). A potential drawback of the normalization method used in the present study (peak EMG value obtained during gait) is that it is not possible to assume that this maximal value represents the maximal neural drive. Because the EMG–force relationship during dynamic movements is not linear in children with cerebral palsy, our results are based on the

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assumption that the maximum EMG value across all trials represents the same percentage of maximum effort in all muscles. However, there is currently no agreement on the best normalization procedure to be used (Rosa et al., 2014).

4.1.3. Statistics

The relationship between hip flexion excursion and muscle coactivation was significant in the TDL but not in the IL despite the fact that the slopes were similar in both groups (see Table 1). This result should therefore be interpreted with caution as it may be due to the higher variability of the data in the IL.

It must also be kept in mind when interpreting the results of this study that the sample sizes were relatively small and that there is a lack of reference data for comparison and thus the power of the study cannot be determined.

5. Conclusions

The results of the present study show that muscle coactivation is largely associated with reduced active motion of the corresponding joints during gait in TD children, arguing for the stiffening role of muscle coactivation in healthy children. Contrary, the relationships are much more mitigated in the involved limb of children with hemiplegic CP, challenging the common belief that muscle coactivation during gait is one of the major causes of reduced active range of motion. The phenomenon of coactivation, still puzzling, appears to be disrupted in children with CP, as a result of disordered motor control. These results demonstrate the need for further studies in order to unravel the relationships between the different symptoms of the upper motor neuron syndrome (paresis, spasticity, coactivation and loss of selectivity), and the actual effects of impairments on gait parameters and functional performance.

7. Conflict of Interest

The authors do not declare any conflict of interest.

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