On the imitation of CP gait patterns by healthy subjects

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ABSTRACT

The comparison of gait imitated by healthy subjects with real pathological CP gaits is expected to contribute to a better distinction between primary deviations directly induced by neurological troubles and secondary compensatory deviations in relation with the biomechanics of the pathological gait. However, the ability of healthy subjects for imitating typical CP gaits such as “jump” or “crouch” gaits still remains to be determined. The present study proposes to investigate healthy subjects imitating these typical CP gait patterns.

10 healthy adult subjects performed three types of gait: one “normal” and two imitated “jump” and “crouch” gaits. Kinematics and kinetics of the hip, knee and ankle were computed in the sagittal plane. Rectified normalized EMG was also analysed. Our data were compared with reference data. For the statistical analysis, the coefficient of multicorrelation has been used. It has been demonstrated that healthy subjects were able to voluntarily modify their gait pattern with a high level of intra-session and inter-subject reproducibility as quantified by a CMC values higher than 0.76 for all parameters. The comparison with literature reference data showed that healthy subjects not could perfectly reproduce a CP gait, however could only simulate the main characteristics of “crouch” and “jump” gaits pattern. As a perspective, pathological gaits imitated by healthy subjects could be used as valuable additional material to analyse the relationship between a voluntarily modified posture and the altered muscle activation to explore a new paradigm on pathological gait pattern analysis and musculoskeletal modelling.

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1. Introduction

Cerebral palsy (CP) is a disability, defined as a persistent disorder of movement and posture due to a non-progressive lesion in a developing brain [1]. During childhood, cerebral palsy children’s ability to walk progressively worsens over time because of impairments in muscle activation that result in musculoskeletal impairments [2]. For patients with spastic diplegia, the most common form of CP is defined by hypertonia and spasticity in the lower extremities muscles and various gait patterns e.g. true equinus gait, jump gait, crouch gait have been clinically classified according to joint trajectories observed during the gait cycle [3–5]. In young diplegic patients and for the earlier independent walking stage, “jump” gait is the most common gait pattern observed [3]. Jump gait is mainly characterized by an excessively flexed knee and hip in an early stance phase that are normally extended in mid-stance and late-stance, but never reach full extension [4]. The jump gait phase is usually preceded by a “true equinus” gait with excessive ankle plantar flexion throughout stance and the hips and knees extended, which can be hidden by recurvatum knee development [3]. Later, “crouch” gait becomes the most characteristic gait for patients with diplegic cerebral palsy [6,7]. Crouch gait is characterized by an increased knee flexion through the stance phase, with variable alignment in the swing phase [4].

Clinically, various corrective treatments attempting to control and to improve the efficiency of pathological gaits seen in patients with cerebral palsy are proposed and are based on information obtained during clinical evaluation and gait analysis exams [8]. Classically, data gathered from gait analysis are compared to a healthy population with normal gait in order to determine gait deviations of CP [9]. Most of these gait deviations reflect the impact of the pathological posture adopted by the CP patient caused by the primary deviation related to the neurological disorders (hypertonia, spasticity, lack of selectivity and paresis) but also secondary deviations due to postural or muscle compensations, and bone deformities caused by muscle force unbalance on the bones during growth [10]. Determining the primary cause of these gait abnormalities is very important for clinicians, because once determined, a more appropriate corrective treatment can be applied, especially for the efficiency of surgical interventions in the management of CP [8].
Table 1: Intra-session mean and standard deviation of CMC values for ankle, knee and hip flexion extension angles and sagittal moments, as well as normalized rectified EMG of Gastrocnemius, Rectus Femoris, Biceps Femoris, and Tibialis Anterior.

<table>
<thead>
<tr>
<th>Flexion extension</th>
<th>Sagittal moment</th>
<th>Normalized rectified EMG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle</td>
<td>Ankle</td>
<td>Hip</td>
</tr>
<tr>
<td>Normal gait</td>
<td>0.97 (0.025)</td>
<td>0.97 (0.044)</td>
</tr>
<tr>
<td>Simulated jump</td>
<td>0.96 (0.024)</td>
<td>0.94 (0.040)</td>
</tr>
<tr>
<td>Simulated crouch gait</td>
<td>0.96 (0.078)</td>
<td>0.90 (0.035)</td>
</tr>
</tbody>
</table>

A complementary approach consists in comparing CP gaits relatively to those of healthy subject imitating characteristic CP patterns. Previous studies analysed the effects of voluntary crouched and/or equinus postures [11] as well as the voluntary unilateral toe-walking [12]. As stated by Romkes and Brunner [12], comparing gaits of healthy subjects imitating CP with real CP gaits contributes to a better understanding of the pathological gait. These authors also conclude on the ability to distinguish primary deviations in muscle activity directly related with the underlying neurological pathology of CP with compensatory deviations induced by the biomechanics of the pathological gait.

In our study, we propose to extend the previous research scope to the most common pathological “jump” and “crouch” gaits as observed in patients with diplegic cerebral palsy. The aim of this study is to assess the ability for healthy subjects to imitate CP gait kinematics and their consequences on kinetics and EMG parameters. Results should contribute to evaluate the relevance for using pathological-like gait types in CP gait analysis and clinical diagnosis.

2. Materials and methods

Ten healthy adult volunteers (six females, four males; 22–36 years), without any neurological or orthopaedic troubles, were recruited for this study. Subjects were asked to perform a normal gait and to simulate pathological “jump” and “crouch” gait as observed in CP patients with spastic diplegia. Instructions has been given by the examiner for the imitation of “jump” and severe form of “crouch” gaits, and a training session followed until the volunteer felt ready to perform a reproducible pathological-like gait. All simulated gait patterns were performed at self-selected comfortable walking speed.

A standard clinical protocol with fifteen reflective markers placed on the lower extremities in accordance with the Helen–Hays marker set was used [13]. The motion capture was performed using 6 MX3 and 7 T160 Vicon cameras (Vicon, Oxford Metrics, Oxford, UK), synchronized with two AMTI force plates (AMTI, Boston, MA, USA). The muscles activities of the lower extremities were recorded with a Noraxon electromyography system (Noraxon, Scottsdale, AZ, USA). Small surface EMG electrodes were placed over the Rectus Femoris, the lateral Gastrocnemius, Biceps Femoris, and Tibialis Anterior muscles according to the SENIAM recommendations [14]. The 3D markers’ tracking was sampled at 100 Hz, EMG and force plates at 1000 Hz.

Six gait trials were performed for each condition such as at least 10 temporal normalized gait cycles were used to determine the different gait parameters. Sagittal gait kinematics and kinetics data of lower limbs were computed using the “Plug-in-Gait” biomechanical model [13]. These data were taken from the ten successful gait cycles for each of the conditions and averaged within subjects in order to be representative of the specific condition and used in the statistical analysis. EMG signals were rectified, and then normalized by the maximal value. After normalization of the gait cycle by 100% and interpolation at each 2% of the gait cycle [15], the inter subject coefficient of multi correlation (CMC) was computed to quantify the inter-subject similarity [16] of the kinematics, kinetics and EMG data. Our results were compared with literature data that had been obtained using similar protocols for investigating healthy normal gait [9,12,15,17–19] and CP gait [3,5,20,21] by the calculation of the CMC coefficient. In addition, we used the 3 clusters of “crouch” gaits (mild, moderate and severe) differentiated by Rozumalski and Schwartz [5]. For all calculations the MATLAB software package (MathWorks Inc., Natick, MA, USA) has been used.

3. Results

The healthy subjects performed reproducible intra-session gaits with mean intra-session CMC values of 0.95 for the flexion–extension angles, 0.97 for the sagittal moments and 0.92 for normalized rectified EMG (Table 1). They also achieved similar gait and mean inter-subject CMC values were indeed of 0.96 for normal gait and 0.91 for simulated gait patterns (Table 2). Only the flexion–extension of the knee during the simulated “crouch” gait presented slightly more inter-subject variability, as attested by a mean inter-subject CMC value of 0.76. The normalized rectified EMG of Gastrocnemius, Rectus Femoris, Biceps Femoris and Tibialis Anterior presented an excellent inter-subject reproducibility, with a mean inter-subject CMC value of 0.90 whatever the type of gait.

For the normal gait, the gait was comparable to that observed in the literature. A mean CMC of 0.92 for kinematics data, a mean CMC of 0.93 for kinetics data and a mean CMC of 0.89 for normalized rectified EMG were obtained by comparing the data of the present study with those of the literature (Table 3).

Table 2: Inter-subject CMC values for ankle, knee and hip flexion extension and sagittal moments, as well as normalized rectified EMG of Gastrocnemius, Rectus Femoris, Biceps Femoris, and Tibialis Anterior.

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<td>0.98</td>
</tr>
<tr>
<td>Simulated jump gait</td>
<td>0.90</td>
<td>0.93</td>
</tr>
<tr>
<td>Simulated crouch gait</td>
<td>0.95</td>
<td>0.76</td>
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The simulated CP pathological gait produced significantly different kinematics, kinetics and EMG patterns compared to "normal" gait (Figs. 1–3) but their comparison with reference data of the imitated pathological gait was contrasted.

For simulated “jump” gait, there are clear differences for the sagittal rotation of the knee between the imitated gait of the present study and reference data from the literature (Fig. 1). During the loading response of the stance phase reference data presented indeed a knee flexion that appeared earlier than ours whereas during the terminal swing our subjects presented less knee flexion (Figs. 1–3). These differences were quantified by CMC values of 0.67 and 0.68 (Table 3).

For “crouch” gait and according to the Rozumalski and Schwartz cluster analysis [5], our subjects simulated a severe form of CP “crouch” gait. This is confirmed by the CMC values that were greater when comparing the “crouch gait” kinematics of our subjects with those of the severe “crouch” gait group from Rozumalski and Schwartz’ study (Table 3). Nevertheless, sagittal rotation of the knee presented more amplitude compared to reference data, this difference being confirmed by CMC values of 0.63 and 0.67 (Table 2). For the sagittal moments, only the hip moment demonstrated a major difference with the data from Lin et al. [20] (CMC = 0.62). Moreover, the data of Lin et al. [20] presented a clear alteration of the curve shape for “crouch” gait,

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**Table 3**

CMC values comparing current study data vs. literature data.

<table>
<thead>
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<tbody>
<tr>
<td></td>
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<td>Knee</td>
<td>Hip</td>
</tr>
<tr>
<td>Normal gait</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Gage [9]</td>
<td>0.84</td>
<td>0.99</td>
<td>0.95</td>
</tr>
<tr>
<td>Vaughan [19]</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Ganley and Powers [17]</td>
<td>0.73</td>
<td>0.98</td>
<td>0.97</td>
</tr>
<tr>
<td>Romkes and Brunner [12]</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Schache and Baker [18]</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Schwartz and Rozumalski [15]</td>
<td>0.89</td>
<td>0.99</td>
<td>0.94</td>
</tr>
<tr>
<td>Simulated jump gait</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lin et al. [20]</td>
<td>0.80</td>
<td>0.67</td>
<td>0.97</td>
</tr>
<tr>
<td>Rodda et al. [3]</td>
<td>0.84</td>
<td>0.66</td>
<td>0.91</td>
</tr>
<tr>
<td>Simulated crouch gait</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Lin et al. [20]</td>
<td>0.83</td>
<td>0.85</td>
<td>0.90</td>
</tr>
<tr>
<td>Rodda et al. [3]</td>
<td>0.93</td>
<td>0.59</td>
<td>0.96</td>
</tr>
<tr>
<td>Rozumalski and Schwartz [5] (mild)</td>
<td>0.44</td>
<td>0</td>
<td>0.61</td>
</tr>
<tr>
<td>Rozumalski and Schwartz [5] (moderate)</td>
<td>0.93</td>
<td>0.60</td>
<td>0.98</td>
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<tr>
<td>Rozumalski and Schwartz [5] (severe)</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>Steele et al. [21]</td>
<td>–</td>
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**Fig. 1.** Ankle, knee and hip flexion-extension for normal and simulated jump and crouch gaits.
although our subjects demonstrated only a reduction in amplitude. The direct consequence of the difference in kinematics and kinetics is obviously a change of the muscle activation also observable in EMG activation patterns of Fig. 3 and quantified by low CMC values in Table 3. However, only few reference data are available for this case.

4. Discussion

The present study has investigated the kinematics, kinetics, and EMG parameters of healthy subjects imitating CP gait patterns. It has been demonstrated that healthy subjects are able to simulate gait patterns in a reproducible manner. However, in comparison...
with available reference data which commonly characterize CP gait, some variations have been observed.

Previous studies using simulated pathological gait had already suggested that healthy subjects were able to simulate pathological gait patterns [11,12]. We also noticed a full understanding of the instruction for the imitation with an intra-session CMC value up to 0.83 and an inter-subject CMC value of up to 0.76 for all parameters, and found no evidence for any level of imitation experience of the volunteer. Here we have pursued these studies by enrolling the investigations to “crouch” and “jump” gaits because these gaits are the most common gaits in spastic diplegia CP children [4,8]. Equinus gait has not been investigated as healthy subjects are unable to achieve it correctly. The combination of planar flexion and extension of the knees and hips was not comfortable to be imitated [12]. In the present study, a complete set of kinematic, kinetic and muscle activation parameters has been analysed, and CMC parameters were computed to determine the inter-subject variability of the imitation as well as the deviation with respect to published CP gait reference data. Our study is based on the analysis of sagittal joint rotations, sagittal moments and EMG. These parameters are commonly used to characterize gait patterns of healthy subjects but also those of CP subjects [6,15,22] and demonstrated a high level of inter-study reliability [23,31]. Although some authors suggest that the use of joint kinematics and kinetics data on the transverse and frontal planes could complete the gait analysis, these data demonstrated only a low level of inter-study reproducibility [23].

Our study is based on the analysis of adult subjects although the CP population analysis in gait often concerns children. Moreover, in order to take into account the usual target patient population, reference data included gait motion analysis data of children. However, according to literature, 7-year-old children already have mature gait patterns and can be compared with adult gait [17]. This was confirmed in our study by CMC values quantifying the variability between the data from the literature and ours that were higher than 0.90 for normal gait. The fact that all gait imitations were performed by adults and not children is an obvious limitation to the current study, however previous observation and studies [12,17] suggest that this is a limited bias.

Conversely, we noticed variations between kinematics, kinetics and EMG results of the healthy group imitating CP gait in comparison to literature results from CP population. These variations can be explained by the fact that the gait of our healthy subjects simulated only the main characteristics of the CP gait patterns. As a matter of fact, modifying a gait pattern needs a learning phase, and the more complex the modifications, the longer the training time. Consequently, we chose the compromise to have an inter-subject reproducible gait pattern at the expense of a complex gait modification. For the “jump” gait, we told the subject to focus on having the ankle in equinus [3], but to increase his or her stability the subject tended to flex less his or her knees which explains differences in the muscle activation of the Biceps Femoris, Gastrocnemius and the sagittal knee rotation during the stance phase in comparison with the reference data. For the “crouch” gait, we specified a gait progression with excessive knee flexion [3]. Naturally, subjects compensated it by an excessive ankle dorsiflexion and the upper body stayed behind the feet with the consequence of no extension moment for the hip and an early activation of the Rectus Femoris. However, it is understood that imitation can never be perfect e.g. the bone deformities and joint dislocation can of course not be reproduced by the healthy subjects [12].

Another explanation can be found in the homogeneity of the gait pattern performed by the healthy subjects as attested by a high level of inter-subject CMC values. These gaits are compared with heterogeneous references from literature which present average values of various sizes and types of CP populations. The data of Lin et al. [20] derive from a population of 8 “crouch” gait subjects and 7 “jump” gait subjects, the data of Rodda et al. [3] of 38 “crouch” gait subjects and 28 “jump” gait subjects, and the data of Rozumalski and Schwartz [5] from a large population of 2956 CP children. This last study differentiated 5 clusters, mild to severe “crouch” gait patterns, based on kinematics parameters and suggested that there were several patterns for this pathological gait. However, such sharp demarcations between the various patterns are not trivial [3]. Our observation suggested that our subjects imitating “crouch” gaits fitted only with the severe cluster of “crouch” gait identified by Rozumalski and Schwartz [5], which suggests that only extreme characteristics of pathological gait could be imitated. Furthermore, CP is a consequence of unpredictable and irreproducible brain damage and for this reason most of the time, either, only case reports are available [24] or patient specific procedure are promoted [25,26].

Despite these contrasted results, imitating pathological gait can be considered as being sufficiently reproducible and comparable to enable calibration of the gait deviation index for a specific controlled alteration of the gait in relation with posture and muscle activation. Indeed, the gait deviation index (GDI) is a composite value based on various gait parameters used to globally quantify the global gait quality [15,27,28]. In practice, the GDI of CP children is compared with the GDI of normally walking healthy children. The differences observed certainly inform of the general state of the pathological gait, in particular of the gait type. However, this comparison does not give any information on the fact whether this pathological gait is “typical” for the pathological gait type. More sophisticated approaches are proposed based on cluster and PCA analysis [5] requiring however a large homogeneous sample population. Consequently, comparing the CP children gait with imitated pathological gaits by healthy subjects in order to disclose CP gait type related deviations could be an additional approach.

Moreover, gait being a sophisticated system of neurological control “normal” healthy gait is also remarkably efficient [9] and suggest an optimized process. The imitation of a pathological gait by healthy subject supplants the optimization paradigm of musculoskeletal coordination by compensation strategies to achieve a reasonable progression. Then the analysis of the simulated pathological gait can be used in musculoskeletal modelling to explore alternative criteria to solve the mathematical redundancy of musculoskeletal modelling for CP patients [29,30].

To conclude the imitation of CP gait patterns by healthy subjects appears to be an interesting additional material to analyse CP gait patterns. The results demonstrated that healthy subjects were able to reproduce modify their gait patterns in order to imitate the main characteristics of pathological gait patterns. The last two decades were dedicated to prove to the medical staff and society the benefit of gait analysis for CP patients, the next step will be to have reliable reference data based on large populations and multicentre sources. Therefore, as perspective, imitated pathological CP gait compared to CP cases could help to underline the relationship between a voluntarily modified posture and altered muscle activities and offer a alternative outlook for CP diagnosis and treatment where muscle dysfunctioning modifies the posture.

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Conflict of interest

There is no conflict of interest.

References