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Impairments restricted knee flexion during gait in a child with cerebral palsy

ABSTRACT

Introduction: A child with cerebral palsy has multiple coexisting deficits that limit functional abilities such as walking. Their interaction shapes the kinematic gait pattern by modulating both the range and shape of the knee flexion movement during the swing phase. The significance of the effect of specific impairments on knee joint flexion range-of-motion is not clear.
Aim: The aim of this study is to comprehensively analyse the effect of coexisting deficits on the range and speed of flexion at the knee joint in the sagittal plane in a child with cerebral palsy.
Material and methods: In 132 patients (M = 76; F = 56; age: 11 ± 4) with spastic cerebral palsy, lower limb joint range-of-motion, selective motor control, strength and spasticity were assessed during the clinical examination. The range of knee flexion in the terminal stance phase, pre-swing and initial swing (TSt-ISw) was assessed during the laboratory three-dimensional gait analysis.
Results: The TSt-ISw knee flexion movement was most strongly (R_S = –0.28) dependent on knee extensor hypertonia, similarly as in the KSt stance phase (R_S = –0.22) and in the KSw swing phase (R_S = –0.22). The velocity of flexion (V) was most strongly correlated with knee extensor muscle hypertonia (R_S = –0.32) and successively with selective control of hip flexor movement (R_S = –0.28), knee extensor strength (R_S = –0.23) and plantar flexor hypertonia (R_S = –0.21).
Conclusions: The range-of-motion of knee flexion in the sagittal plane depends on the hypertonia of the knee extensor muscles in both the TSt-ISw and KSt and KSw phases.
Velocity increase depends on the occurrence of knee extensor muscle hypertonia, selective control of hip flexor movement, knee extensor strength and plantar flexor hypertonia.

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KEY WORDS: kinematic gait pattern; selective motor control; spasticity; muscle strength; gait analysis

INTRODUCTION

Patients with cerebral palsy (CP), as a result of damage to the central nervous system, present a range of impairments that limit their functional abilities. Hypertonia (HYP), muscle weakness (FM) and reduced selective motor control (SEL) are the most commonly cited factors associated with locomotor impairment in a child with CP [1, 2]. The consequence of the presented abnormalities is a pathological gait pattern and a consequent reduction in gait efficiency and quality [3–6]. Treatment of the patient, aimed at functional improvement, is based on data collected during a clinical examination and the three-dimensional gait analysis.

The most commonly described abnormalities in the movement of the knee joint in the sagittal plane relate to its insufficient or excessive flexion. The flexion of the knee joint in the sagittal plane during at gait cycle is observed twice: a slight flexion that serves as a shock absorber at the beginning of the stance phase [3, 6, 7] and a maximum flexion during the initial swing (ISw), which increases from the maximum knee extension seen during the terminal stance phase (TSt), reaching approximately 60°, covering a period of approximately 40% to 70% of the gait cycle. The second phase of

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knee flexion consists of two periods comprising flexion in stance (KSt) and in swing (KSw). Reduction in flexion range-of-motion has a significant impact on gait efficiency by shortening steps and reducing walking velocity [6].

The most common causes limiting the normal flexion range-of-motion include hypertonia (abnormal muscle tone), reduced selective motor control and muscle strength control, the coexistence of which is characteristic of patients with CP. A detailed biomechanical analysis of the gait pattern and the involvement of muscle groups during specific phases of the gait cycle allows potential pathological factors modulating movement in the knee joint to be identified. In addition, walking with self-paced velocity is the result of the combination of active control processes with the dynamic properties of the human body, which are mediated by the biomechanical coupling between the individual body parts in the standing position together with their visco-elastic properties [3, 6, 8, 9]. In the remainder of this article, these factors will be referred to in the simplified form as "dynamic".

The TSt-ISw knee joint flexion occurs during both the stance phase, where the knee flexion results from moving the body weight forward with the foot stabilised on the ground, and the swing phase, mainly based on a pendulum motion.

During the KSt stance (TSt terminal stance and PSw pre-swing phases), there is dynamic passive flexion of the knee and hip joints resulting from the forward movement of the body weight with the foot stabilised on the ground. This is accompanied by transition between pronation along the foot with passive stretching and activation of plantar flexors in preparation for toe-off. Stretching of the knee and hip extensors and plantar flexors can cause spasticity in these muscle groups and hypertonia inhibiting flexion movement and limiting toe-off performance.

The flexion of the knee during the KSw phase is mainly related to the dynamic coupling (double pendulum) between the thigh (which is the upper part of the double pendulum) and the lower leg (which is the lower part of the pendulum). The lower leg moves forward with the movement of the thigh (resulting from the weight transfer in the direction of movement), with possible participation of the hip flexor and knee extensor muscles and intentional (at will) activation of the dorsal flexor muscles and stretch of the plantar flexors of the foot [3, 6, 8, 9].

Control of the generation of intentional muscle activity may be affected by SEL and FM deficits, while stretching the muscle may induce HYP as a result of the hyperexcitability of the stretch reflexes associated with spasticity. In addition, limiting the passive flexion range-of-motion the knee joint can have an impact on limiting range-of-motion.

Both previous studies on the flexion limitation and its treatment focus mainly on single deficits without analysing the whole picture of a patient with CP. Previous studies indicate a dependence of flexion range-of-motion on the *rectus femoris* muscle: its spasticity [8, 10], length [9] and abnormal activity [4, 5]. Consequently, treatment focuses mainly on transferring or lengthening the *rectus femoris* muscle [11]. Other potential factors affecting the behaviour of the knee during this phase of the gait cycle are overlooked. In particular, the effect of impairments was not differentiated according to the gait phase (stance/swing), where they could potentially be related to the generation of muscle activity or to muscle stretching.

The aim of this study is to comprehensively analyse the impact of coexisting deficits on the range-of-motion and velocity of knee flexion in the sagittal plane in the TSt-ISw phases in a child with cerebral palsy.

The flexion range-of-motion during gait was assessed by 3D movement analysis and impairments were analysed by clinical examination.

MATERIAL AND METHODS

The study group consisted of 132 patients (M = 76; F = 56; age: 11 ± 4) with a diagnosis of cerebral palsy. Data were collected at the Motion Analysis Laboratory of the Poznan University of Medical Sciences. Patients were eligible for the study if: (1) they were diagnosed with CP; (2) did not receive botulinum toxin or underwent surgical treatment a minimum of 6 months prior to the study; (3) their complete kinematic and clinical data were collected; and (4) if they were found to be able to walk independently (without a third party) without or with orthopaedic equipment (crutches, canes, walker). All patients or their legal guardians consented to the study.

REPORT

Data including gait analysis and clinical examination were collected once according to the following scheme:

1. 3D gait analysis:

   Three-dimensional motion analysis was carried out using an 8-camera system from Vicon Motion Systems Ltd, (6 Bonita and 2 Vero cameras; Oxford, United Kingdom) with a sampling rate of 120 Hz. Markers reflecting infrared radiation were placed on the patient according to the standard Plug-in-Gait model. Patients walked a 10-metre stretch barefoot at self-paced velocity individually preferred speed. Kinematic data were recorded to calculate the knee flexion range-of-motion in the sagittal plane in the terminal stance (TSt), pre-swing (PSw) and initial swing (ISw) phases, as well as its velocity at toe-off.
2. The clinical examination included an assessment of:
   - Range-of-motion (ROM): clinical assessment of passive range of motion was performed using a hand-held goniometer; the data obtained were compared with normative data; if the angular value deviated from the norm, we concluded the presence of a impairment [12];
   - Muscle hypertonia (HYP): muscle responses to high-velocity stretching were assessed using a modified Ashworth scale and scored from 0 (normal muscle tone) to 4 (limb immobilised in flexion or extension); a score > 0 was considered the presence of a deficit [13];
   - Muscle strength (FM): muscle strength was assessed using the 5-grade Lovett's scale, where 0 means no tension and 5 means normal muscle strength; as the patients studied were paediatric patients, in whom the distinction between a value of 4 and 5 is not meaningful [14], we took the presence of a impairment to be a score of ≤ 3 [12];
   - Selective motor control (SEL): was assessed using the Trost scale from 0 (no isolated joint movement) through 1 (partial selectivity of movement) to 2 — ability to perform isolated joint movement; we considered a score ≤ 1 as the presence of a deficit [15].

The tests were carried out by therapists with many years of experience working with patients with cerebral palsy.

THE ANALYSIS OF THE OBTAINED DATA

The primary outcome data was the knee flexion range-of-motion in the sagittal plane during the TSt-ISw phases. The flexion range--of-motion was also analysed, divided into the KSt and the KSw. In addition, the velocity of the flexion at the toe-off (V) was analysed (Fig. 1).

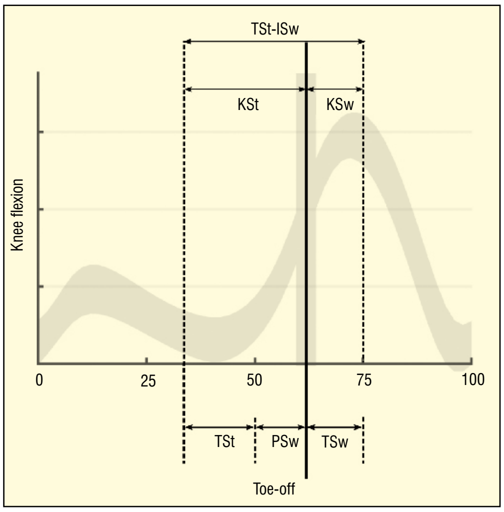

Figure 1. Kinematic pattern of knee joint movement in the sagittal plane; KSt — knee flexion in the stance phase; KSw — knee flexion in the swing phase; TSt-ISw — knee flexion including the stance and swing phases

Baseline data from the analysis included biomechanically justified deficits that could limit the TSt-ISw flexion range-of-motion: hypertonia of the knee joint flexors (HYP_Kfl) and extensors (HYP_Kex) and ankle plantar flexors (HYP_Afl), muscle weakness of the knee joint extensors (FM_Kex) and hip flexors (FM_Hfl), knee extension contracture (ROM_Kex), restricted selective control of knee extensors (SEL_Kex) and hip flexors (SEL_Hfl). The abbreviations are as follows:

- Deficit: hypertonia (HYP), muscle weakness (FM), selective motor control (SEL) limitation, passive range-of--motion (ROM);
- Joints: knee (K), hip (H), ankle (A);
- Direction of movement: flexion (fl), extension (ex).

STATISTICAL ANALYSIS

The data were not normally distributed and their interrelationships were assessed using Spearman's correlation. Statistical analysis was performed using Statistica (Statistica 13, StatSoft Ltd), with a statistical significance level of $p \leq 0.05$.

RESULTS

RELATIONSHIP BETWEEN ASSESSED IMPAIRMENTS AND KNEE JOINT MOVEMENT IN THE SAGITTAL PLANE

Analysing the whole phase of the TSt-ISw knee flexion, the strongest relationship (R_S = –0.28) was found for knee joint extensor hypertonia, where an increase in hypertonia

Table 1. Correlation between the knee flexion range-of-motion TSt-ISw and flexion velocity, and selected deficits (HYP, SEL, FM, ROM)

Deficit	TSt-ISw		KSw		KSt		V	
	R_S	p	R_S	p	R_S	p	R_S	p
ROM_Kex	0.05	0.433	0.13	0.029	–0.04	0.478	–0.02	0.688
HYP_Afl	–0.06	0.322	0.01	0.841	–0.11	0.077	**–0.21**	< 0.001
HYP_Kex	**–0.28**	< 0.001	**–0.22**	< 0.001	**–0.22**	< 0.001	**–0.32**	< 0.001
HYP_Kfl	–0.10	0.116	–0.10	0.094	–0.04	0.489	–0.19	0.001
FM_Hfl	0.01	0.827	0.10	0.115	0.00	0.954	0.14	0.025
FM_Kex	0.12	0.043	0.03	0.582	0.19	0.002	**0.23**	< 0.001
SEL_Hfl	0.19	0.002	0.10	0.095	0.19	0.002	**0.28**	< 0.001
SEL_Kex	0.09	0.140	0.03	0.584	0.17	0.005	0.18	0.003

R_S, p — Spearman's R coefficient and probability, respectively; Afl — ankle plantar flexors; FM — muscle strength; Hfl — hip flexors; HYP — muscle hypertonia; ISw — initial swing; Kex — knee extensors; Kfl — knee flexors; KSt — knee in the stance phase; KSw — knee in the swing phase; ROM — range of motion; SEL — selective motor control; TSt — terminal stance phase; V — velocity

led to a reduction in flexion range-of-motion; both during the Kst stance phase (R_S = –0.22) and the KSw swing phase (R_S = –0.22).

Flexion velocity increase at toe-off (V) was most strongly correlated with knee extensor muscle hypertonia (R_S = –0.32) (as hypertonia increased, velocity decreased) and successively with selective motor control of the hip flexors (R_S = –0. 28) (reduction in selective movement control led to reduced velocity), knee extensor strength (R_S = –0.23) (weakened muscle strength was associated with reduced velocity), and plantar flexor hypertonia (R_S = –0.21) (the more severe the hypertonia, the lower the velocity).

Statistically significant R_S correlation coefficients between the baseline data were in the range of weak correlations from 0.12 to 0.32. Taking into account that $R_S < 0.2$ despite $p \le 0.05$ is not considered an indicator of a linear relationship, only correlations with $R_S \ge 0.2$ were included in the further analysis (Tab. 1).

DISCUSSION

The aim of this study was to determine the factors influencing the knee flexion range-of-motion and velocity (at toe-off) during the late stance phase and initial swing. The results of this study indicate that the primary deficit limiting knee flexion and velocity of movement is the occurrence of knee extensor muscle hypertonia. It influences both the movement in the stance and swing phase and its velocity (V).

Knee flexion in the phases analysed starts in the middle phase of the terminal stance (where the knee is fully extended or where there is a change in knee movement velocity associated with a change in the direction of movement e.g. due to hyperextension) and reaches its maximum value (approx. 60°) in the initial swing phase [6] (Fig. 1). This leads to stretch of the knee extensor muscles inducing a stretch reflex and consequent hypertonia, which may limit the range-of-motion. Given that our results (negative R_S) indicate that the smaller the total knee flexion range-of-motion (as well as its components in the stance and swing phases), the greater the hypertonia of the knee extensor muscles, this suggests that hypertonia can be the main factor limiting these ranges (Tab. 1).

Our data shows that the velocity of movement at toe-off, (in addition to knee extensor hypertonia R_S = –0.32) the worse the selective control of hip flexor movement (R_S = 0.28) and lower knee extensor strength (R_S = 0.23).

Movement velocity (V) is related to the flexion range-of-motion in the phases following the separation of the foot from the ground. This suggests that the involvement of the muscles that flex the hip joint, thus also knee extensors (which can support hip flexion movement in this phase), is minimal. In addition, in normal gait, moving the limb forward and increasing knee flexion, thanks to adequate toe-off and forward movement of the thigh resulting from hip flexion, is based on a pendulum motion. This mechanism is often impaired in patients with CP. Celebral palsy patients walk more slowly, and the role of toe-off in moving the limb forward compared to hip flexion may be limited [6, 16]. In addition, it appears that the greater the velocity of the flexion movement, the greater (when stretching individual muscle groups in the ankle and knee joints) the probability of velocity-dependent hypertonia. Inhibition of movement and/or inadequate toe-off force leads to a restriction of the free forward movement of the limb. In addition, deficits in CP patients very often co-occur and their effects overlap. Spasticity is accompanied by muscle weakness and disorders of selective motor control [8, 17–21]. Muscle strength may be reduced due to damage to the central nervous system, abnormal coactivation of antagonistic muscles resulting from reciprocal activation instead of inhibition and abnormal recruitment of motor units which may be associated with spasticity [22, 23]. Selective motor control impaired due to damage to the descending pathways or hypersensitivity to stimuli, as well as the spread of reflexes can also be associated with spasticity [2].

The results of our study are a further argument pointing to the dominant role of knee extensor muscle hypertonia in limiting the flexion range-of-motion in the TSt-ISw phase. Unlike previous researchers, in our analysis, we have considered all potential deficits that may have an impact on limiting the range-of-motion.

Most of the previously presented work focuses on single factors causing abnormalities in gait pattern, restricting itself to an analysis of the characteristics of the rectus muscle. It was indicated that its spasticity may be a significant factor limiting the flexion range-of-motion [24, 25], although there is also research to the contrary [26]. It is also pointed out that the stimulation pattern may be important — the moment of activation of the rectus muscle which may limit the range-of-motion in the stance phase but has no effect on the swing phase [4, 5]. Research also shows the importance of its length [9]. Some authors point to the role of efficient toe-off as a factor modulating movement, although the significance of plantar flexor muscle activity for the swing phase is under discussion [16].

Improvements in the range-of-motion were mainly sought through lengthening or transferring the rectus muscle, so as to reduce its straightening effect on the knee joint in this phase of movement, and thereby improve the kinematics and movement velocity [27]. Meta-analysis shows the ineffectiveness of such treatment [28], although some authors present significant improvement after surgical treatment [27, 29]. These differences result from the way of selecting patients. Their functional status, muscle stimulation pattern and clinical condition are critical to achieving positive treatment outcomes.

The limitation of the work is basing the analysis on statistically significant but weak correlations between output data (R_S from 0.21 to 0.32). Our analyses used the results of clinical tests to assess existing deficits. It seems that applying objective instrumental methods in patient assessment could increase the reliability of the data obtained. In everyday clinical practice, instrumental methods are not used due to the considerable cost and time required for their use. As previous work has shown [14, 30], the use of clinical tests in the case of finding pathology or lack thereof (without deficit gradation assessment) can be a reliable method of patient assessment.

The analysis of individual factors that may impact the range-of-motion of knee flexion, and then basing the patient's therapy on these results seems to be an inadequate approach. As our comprehensive analysis of all possible factors of a range-of-motion has shown, it is affected by many deficits, the co-occurrence of which is typical for CP patients. It appears that establishing a gradation of the impact of each abnormality may lead to a better understanding of the causes of particular gait pathologies and, consequently, to better targeted therapeutic management. However, such an analysis does not seem to be possible using only the results of clinical tests without applying quantitative, objective and instrumental measurement methods for each of the deficits.

CONCLUSIONS

The knee flexion range-of-motion in the sagittal plane depends on the knee extensor muscle hypertonia both in the TSt-ISw phases and in the stance and swing phases.

Velocity of knee flexion at toe-off depends on the occurrence of knee extensor muscle hypertonia, selective control of hip flexor movement, knee extensor strength and plantar flexor hypertonia.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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