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Joint stiffness and gait pattern evaluation in children with Down syndrome

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Abstract

Hypotonia, ligament laxity and motor alterations are characteristic for patients with Down syndrome (DS). The purpose of this study was the evaluation of typical gait pattern of subjects with Down syndrome and the quantification of their joint stiffness, connected with ligament laxity and hypotonia, as a possible compensation.

98 children with DS (mean age: 11.7 years; range: 6–15 years) and 30 healthy children (control group (CG); mean age: 11 years; range: 5–13 years) underwent full 3D gait analysis at self-selected speed.

Subjects with DS walked with more hip flexion during the whole gait cycle, knee flexion in stance phase, a limitation of the knee range of motion, and plantarflexion of the ankle at initial contact. Ankle power was limited as evident in terminal stance and pre-swing, represented by a low propulsive capacity at push-off, too. Hip joint stiffness was increased in general in patients with DS versus normal subjects while ankle joint stiffness revealed a lower value instead.

Keywords: Gait analysis; Down syndrome; Gait pattern; Joint stiffness

1. Introduction

Down syndrome (DS) is the most common non-inherited cause of mental impairment and occurs in 1 out of 1000 live births [1] as a result of the presence of all or a portion of an extra copy of chromosome 21. There are a number of medical problems that are associated with the syndrome, including cardiac and respiratory conditions. Motor disability is widespread among individuals with DS. It includes longer motion and reaction times, balance and postural deficits, and cocontraction of agonist and antagonist muscles [2,3]. These deficits may have a causal link to delays in achieving motor development milestones in children. The motor dysfunction in individuals with DS involves impaired muscle control, which is frequently referred to as ''clumsiness'' by parents and health professionals [4]. The neuropathological basis for motor dysfunction in DS is unknown, but cerebellar dysfunction, delayed myelination, as well as proprioceptive and vestibular deficits have been suggested as possible causes [6,7]. The delay in motor development in DS is linked to the generalized muscle hypotonia and ligament laxity that is characteristic of the condition [5].

Early physiotherapy focuses on facilitating motor control and coordination in order to achieve developmental milestones. Once walking is established (which is often delayed by an average of 12–18 months) [8,9] regular physiotherapy is usually discontinued. There are, however, numerous reports in the literature suggesting that children with DS begin to develop orthopedic problems early in childhood and would benefit from specific biomechanical assessment and

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management. Caselli et al. [10] reported that walking in children and adolescents with DS was characterized by a ''Chaplinesque'' pattern with external rotation of the hips, increased knee flexion and valgus, and external rotation of the tibia. In childhood, pes planovalgus with marked pronation of the foot was observed, which could impact on postural stability and ambulation.

Foot deformity and resulting impeded function has been described as lever arm dysfunction in patients with neuromuscular disorders [20]. In adolescents and adults with DS, hallux valgus, hammer toe deformities, plantar fasciitis, and early onset of foot arthritis associated with severe flat feet were also observed, which impair ambulation and cause further dysfunction [21].

Parker and Bronks [11] studied the gait pattern of six young children (mean age: 4.2 years) with DS using video analysis. Poor heel–toe rocking during the stance phase and exaggerated abduction of the lower limb to facilitate foot clearance were observed. The gait pattern of 63 children with DS showed prolonged hip flexion during the gait cycle, an increase of knee flexion in the sagittal plane at initial contact and significant changes in ankle movement during the gait cycle [12]. These findings fit with the lever arm dysfunction in other disorders where inadequate plantarflexion results in crouch. The gait is further characterized by a significant decrease in plantarflexor moments and of absorbed and generated ankle power [12]. These abnormalities may reflect muscle hypotonia, ligament laxity, weakness of the plantarflexors and dysfunction of the foot as a lever arm.

While the specific orthopaedic and biomechanical limitations have been clearly identified, little is known about the nature of the relationship between muscle hypotonia, ligament laxity and the resulting joint stiffness in children with DS. The purpose of this study was to document the gait characteristics of children with DS and to quantify the hip and ankle joint stiffness that characterize gait in individuals with DS.

2. Patients

Ninety-eight children with DS (mean age: 11.7 years; range: 6–15 years) and 30 healthy ones (control group: CG; mean age: 11 years; range: 5–13 years) participated in this study. All patients were independent ambulators. The characteristics of the subjects are listed in Table 1.

The parents of all children provided informed consent to participate in the study and this study was approved by the Ethical Committee of the hospital IRCCS ''San Raffaele-Pisana'', Rome, Italy.

Table 1
Characteristics (mean ± S.D.) of analyzed subjects

Subjects	Height (cm)	Weight (kg)
CG	132.44 ± 10.92	29.81 ± 6.10
DS	141.69 ± 12.96	47.65 ± 14.13

3. Methods

Three-dimensional kinematic data were obtained using a 12-camera optoelectronic system with passive markers (ELITE 2002, BTS, Milan, Italy [13], sampling rate of 100 Hz). Two force platforms (Kistler, Winterthur, CH), embedded in the walkway were used to obtain kinetics. All trials were videotaped using a video system, synchronized with the optoelectronic system and force platforms (Videocontroller, BTS, Milan, Italy). Seventeen passive markers were placed according to Davis [15].

All subjects were asked to walk barefoot at their self-selected speed along a 10 m walkway. Six trials were collected for each subject. Kinematic and kinetic data were computed using Euler angles and Euler's equations of motion, respectively [15]. The kinematic and kinetic data of the hip, knee and ankle joints in the sagittal plane were studied, as they represent push-off capacity. All the graphs were normalized for percentage of gait cycle.

Temporal spatial parameters were compared between the two groups. Hip, knee, ankle joint kinematics and kinetics (range of motion, maximum/minimum of flexion extension angles values during gait cycle instants, hip, knee and ankle flexion extension joint moments, generated ankle power) were analyzed.

In order to evaluate the effect of ligament laxity and hypotonia on joint kinetics and kinematics, hip and ankle joint stiffness (hip joint stiffness: K_h; ankle joint stiffness: K_a) were expressed by plotting the values of flexion–extension moment versus flexion–extension angle over the gait cycle interval (Fig. 1a and b). The interval between the 10% and 30% (corresponding to the second rocker) of gait cycle was selected and the linear regression was fitted (Fig. 1c and d); the angular coefficient of linear regression corresponded to the joint stiffness index as described in previous studies [16,17]. Knee stiffness was not included in this study because of the lack of linear relation between kinematics and kinetics. As the weight differed significantly between the two groups (BMI: DS, 27.44 ± 3.8 kg/m^2; CG, 21.5 ± 1.49 kg/m^2) (Table 1), the kinetic data were normalized for weight. The individual mean and standard deviation of the parameters of interest were calculated before the mean and standard deviation of the groups.

Kinematic and kinetic parameters were compared using the Student's *t*-test (parametric data) or the Wilcoxon test (non-parametric data). Statistical significance was set at $p < 0.05$.

4. Results

4.1. Gait evaluation

Subjects with DS showed a significant decrease in gait speed (0.42 ± 0.08 s-1; $p < 0.05$) and stride length (0.29 ± 0.04; $p < 0.05$) in comparison with the control group (gait speed: 0.85 ± 0.06 s-1; stride length: 0.89 ± 0.09).

Sagittal hip kinematics (Fig. 2a) showed more hip flexion in DS patients (initial contact: DS, $37.0 \pm 8.1^\circ$; CG,