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Stiff-knee gait in cerebral palsy: How do patients adapt to uneven ground?

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ABSTRACT

Patients with cerebral palsy frequently experience foot dragging and tripping during walking due to reduced toe clearance mostly caused by a lack of adequate knee flexion in swing (stiff-knee gait). The aim of this study was to investigate adaptive mechanism to an uneven surface in stiff-knee walkers with cerebral palsy. Sixteen patients with bilateral cerebral palsy, GMFCS I–II and stiff-knee gait, mean age 14.1 (SD = 6.2) years, were compared to 13 healthy controls with mean age 13.5 (SD = 4.8) years. Gait analysis including EMG was performed under even and uneven surface conditions. Similar strategies to improve leg clearance were found in patients as well as in controls. Both adapted with significantly reduced speed and cadence, increased outward foot rotation, knee and hip flexion as well as anterior pelvic tilt. Therefore cerebral palsy and stiff-knee gait did not affect the adaptation capacity on the uneven surface.

On the uneven surface an average increase in knee flexion of 7° (SD = 3°) and 12° (SD = 5°) was observed in controls and patients with cerebral palsy, respectively. Although rectus femoris activity was increased in patients with cerebral palsy, they were able to increase their knee flexion during swing. The results of this study suggest that walking on uneven surface has the potential to improve knee flexion in stiff-knee walkers. Therefore training on uneven surface could be used as a conservative treatment regime alone, in combination with Botulinum neurotoxin or in the rehabilitation of surgery.

Introduction

Patients with cerebral palsy frequently experience foot dragging and tripping during walking. This is due to reduced toe clearance mostly caused by a lack of adequate knee flexion in swing (stiff-knee gait) [1]. In daily life patients with stiff-knee gait might be particularly impaired during walking on uneven terrain, when their knee flexion to increase toe clearance is limited. When walking on uneven ground, healthy subjects were shown to increase hip and knee flexion and ankle dorsiflexion during swing phase [2,3], in order to increase their toe clearance. Since patients with stiff-knee gait have limited knee flexion ability, they might use hip flexion and ankle dorsiflexion or other mechanisms such as circumduction or pelvic hike to assist their toe clearance [4].

Up to now, the exact physiopathological mechanisms of stiff-knee gait are only partly understood. Several causes in isolation or combination were mentioned in the literature: Apart from hip flexor weakness and over activity of the m. gastrocnemius during terminal stance [5], the most commonly cause is a prolonged swing phase rectus femoris activity [4,6,7]. The interference of the rectus femoris over activity was experimentally confirmed by a study which measured an increase in knee flexion after rectus femoris motor branch block [8]. More recently it was shown that increased activity of rectus femoris particular during pre-swing phase was responsible for an inadequate knee flexion in swing [9,10].

Recognition of the involvement of the rectus femoris in stiff-knee gait in children with cerebral palsy has led to conservative or surgical treatments targeted at this muscle: surgical distal transfer, distal release, or Botulinum neurotoxin (BoNT) injections [11]. Significant improvement in knee flexion during swing and a reduction in energy cost of walking were observed after BoNT injections into the rectus femoris muscle in adult stroke patients with stiff knee gait [5]. However, the effect in children with CP seems to be low [11]. Distal rectus femoris transfer was documented to be an effective procedure for stiff-knee gait. This procedure mostly creates a long-lasting increase of peak knee flexion in swing phase [12]. In summary, current treatment strategies of stiff-knee gait targeting the rectus seemed to be successful for the majority of the cases. However it appears that a

Abbreviations: BSCP, bilateral spastic cerebral palsy; CON, typically developed controls; EMG, electromyography; iEMG, integrated electromyography signal; BoNT, Botulinum neurotoxin; MAS, modified Ashworth scale.

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number of patients (between 18% and 33%) showed a permanently poor treatment response [12,13]. These poor responders suggest that rectus might not always be the underlying primary cause of stiff-knee gait and that this aspect must require further research. In particular it would be informative to know whether stiff-knee gait affects walking on uneven surface, and how patients compensate their reduced toe clearance for this situation. Both may help to better understand the underlying causes of stiff-knee gait to improve safe and efficient daily life walking that is frequently challenged by uneven surface conditions.

Therefore, the hypotheses were that first, patients with stiff knee gait face great difficulties when walking on uneven surface, namely displaying more reduction in walking speed and step length than controls while also widening their steps to improve balance. Second, knee flexion in stiff knee gait walkers cannot be further increased on uneven surface and they use other mechanisms: hip flexion, ankle dorsiflexion, circumduction, external foot progression and pelvic hike to assist their toe clearance.

Methodology

Participants and procedure

Sixteen patients with bilateral spastic cerebral palsy (BSCP) who had been referred to the Gait Analysis Laboratory at the Orthopaedic Hospital in Aschau for evaluation of their gait and assessment of possible orthotic or orthopaedic interventions, volunteered to participate in this study. Inclusion criteria were first, that patients were able to walk without assistance or assistive devices (GMFCS I&II) and second, stiff-knee gait. Stiff-knee gait was defined, according to an established criterion from the literature, by three of four gait parameters that were two SD outside those of typically developed controls [14]. The gait parameters were reduced peak knee flexion in swing, reduced knee range of motion measured from toe off to peak knee flexion in swing, reduced total range of motion of knee flexion during gait, and delayed timing of peak knee flexion during swing. Only those legs that fulfilled these criteria were included. None of the patients had BoNT treatment or casting within the last 6 month. Further patients diagnosed visual or perceptual impairments were excluded. Patients mean age was 14.1 (SD = 6.2) [6,26] years, mean bodyweight was 45.6 (SD = 19.6) kg, mean body height 150 (SD = 22.4) cm, mean BMI 19.3 (SD = 4.24) kg/m m, 14/16 were males, 25/32 legs fulfilled the stiff gait criteria and were included. For those legs manual clinical examination of passive range of motion revealed knee extension between −20° extension deficit and 5° hyperextension with a mean of −6° (SD = 10°). Mean passive knee flexion was 133° (SD = 14°) [105°, 150°] Ankle dorsiflexion was on average 4° (SD = 9°) [−15°, 20°], passive hip flexion 111° (SD = 15°) [90°, 140°] and passive hip abduction 22° (SD = 11°) [10°, 45°]. Spasticity graded by the Modified Ashworth Scale (MAS) was 1.9 (SD = 0.9) [1.0, 4.0] for m. rectus femoris (Duncan–Ely) and 1.2 (SD = 1.0) [0.0, 3.0] for the triceps surae (supine with extended knees). All prior surgeries were more than 2 years ago. In detail of 25 limbs 4 had calf muscle lengthening, 2 knee flexor lengthening, 4 psoas release, 5 adductor tenotomies, 3 supracondylar derotation osteotomies.

Thirteen typically developed controls (CON) mean age 13.5 (SD = 4.8) [7,22] years, mean bodyweight 43.8 (SD = 14.2) kg, mean body height 153 (SD = 16.1) cm, mean BMI 18.3 (SD = 2.87) kg/m m, 7/13 males, volunteered to participate. All participants provided written consent, as approved by the ethics committee of the University of Salzburg. All walking trials were performed ambulating barefoot. Participants had to walk on even ground and on an uneven surface (Terrasensa, Hübner GmbH, Kassel, Germany) of 7 m length within the gait laboratory of 15 m length. The uneven surface shown in Fig. 1 consisted of 28 squared floor panels (50 × 50 × 8 cm). It is made of polyurethane with a patented surface relief system that is comfortably shock-absorbing.

The order of even and uneven surface was randomized. On the uneven surface three walking trials were used for familiarization. Instrumented gait analysis was performed using an 8-camera Vicon System (Vicon Inc., Oxford, UK) using the Plug-in-Gait marker-set and model. EMG of six muscles was bilaterally captured with the Telemyo 16 channel EMG system (Neurodata GmbH, Vienna, Austria). Selected muscles were gluteus medius, rectus femoris, medial hamstrings, vastus medialis, gastrocnemius medialis and tibialis anterior.

Data processing and statistical analysis

Spatio-temporal parameters walking speed, cadence, step length and step width were calculated to quantify the performance of gait under the two surface conditions. Spatio-temporal parameters were given in non-dimensional units to correct differences in leg lengths [15].

To show the amount of foot clearance, the peak vertical position of the toe marker during swing phase of gait was analyzed. This was contrary to the literature that typically defined foot clearance as the minimum of the vertical distance during mid-swing [16]. Since BSCP patients compared to CON did not show a clearly detectable minimum in mid-swing (Fig. 2) the clearly defined peak value over the whole swing phase was chosen.

To investigate the kinematic adaptations, selected segment or joint angles were evaluated that might have the potential to interfere with leg clearance during swing. These were peak pelvis anterior tilt, hip flexion, knee flexion, ankle dorsiflexion, pelvic drop, hip abduction and foot inward progression. Foot inward progression was selected as peak value at initial swing; all other

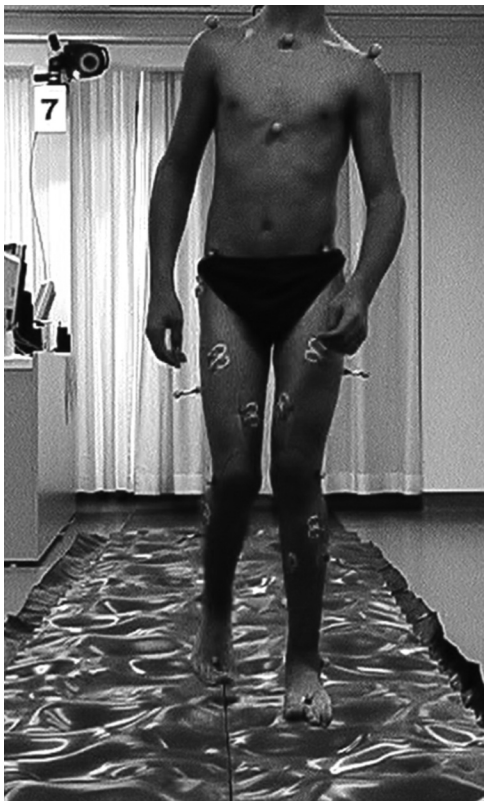

Fig. 1. Patient with BSCP walking over the uneven surface of 7 m length within in the gait laboratory.