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Kinetics of compensatory gait in persons with myelomeningocele

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Abstract

This study investigated the kinetic strategy and compensatory mechanisms during self-ambulatory gait in children with lumbo-sacral myelomeningocele. Thirty-one children with mid-lumbar to low-sacral myelomeningocele who walked without aids and 21 control children were evaluated by three-dimensional gait analysis. Joint moments in all planes at the hip and knee and sagittal moments at the ankle, as well as joint power and work done at all three joints, were analyzed. Joint moment capacity lost due to plantarflexor and dorsiflexor weakness was provided instead by orthotic support, but other joints were loaded more to compensate for the weakness at the ankles and restricted ankle motion. Subjects with total plantarflexor and dorsiflexor paresis and strength in the hip abductors had more knee extensor loading due to plantarflexor weakness and dorsiflexion angle of the orthotic ankle joint. The subjects with orthoses also generated more power at the hip to supplement the power generation lost to plantarflexor weakness and fixed ankles. The most determinant muscle whose paresis changes gait kinetics was the hip abductor. Hip abductor weakness resulted in a characteristic pattern where the hips displayed an eccentric adduction moment, mediating energy transfer into the lower limbs, and the hips replaced the knees as power absorbers in early stance. Joint moment, power and work analyses complement a kinematic analysis to provide a complete picture of how children who have muscle paresis recruit stronger muscle groups to compensate for weaker ones.

Keywords: Motion analysis; Spina bifida; Joint moments; Biomechanics; Pathological gait

1. Introduction

Myelomeningocele (MMC), a congenital birth defect, which can affect sensory and motor function, often results in muscle paresis proportional to ascending spinal lesion level. An altered gait pattern, dependent on muscle strength or spinal lesion level, has been described [1–5]. Joint moments during gait have been described for some groups of MMC at the knee and/or ankle [4,6–11,12]. Major goals of most of these studies have been to describe characteristic gait kinematics with specific lesion levels and to determine the cause of knee pain in MMC [6–8,11] by investigating coronal and sagittal plane knee moments.

Major gait deviations in all planes have been observed in children with MMC who have weakness in the plantarflexors where increased knee flexion [1,2,4,10], increased anterior pelvic tilt [1,2,4,10], altered [1,4] or exaggerated pelvic frontal and transverse movement [2,10] and increased trunk rotation [1,13] were characteristic observations. Hip abductor weakness, however, produced the largest change in gait strategy [1,4,13–15] with a completely different pattern of trunk and pelvic rotation with large lateral trunk sway, hip abduction during stance and pelvic hike. By using such a kinematic pattern, one can align the centre of mass over the hip joint [1,5,14] in a hip abductor-avoidance mechanism and progress their centres of mass forward.

Kinetic studies in myelomeningocele have so far been mainly aimed at determining the effects of orthoses [6,16–19], investigating knee pain [8,20] and studying external valgus moment at the knee, whether actual [9] or ‘apparent valgus thrust,’ where knee flexion combined with internal hip rotation results in the appearance of knee valgus [7,11]. Many studies have reported increased or persistent knee flexion angle and/or increased knee extension moment in MMC compared to normal [4,9,15], increasing

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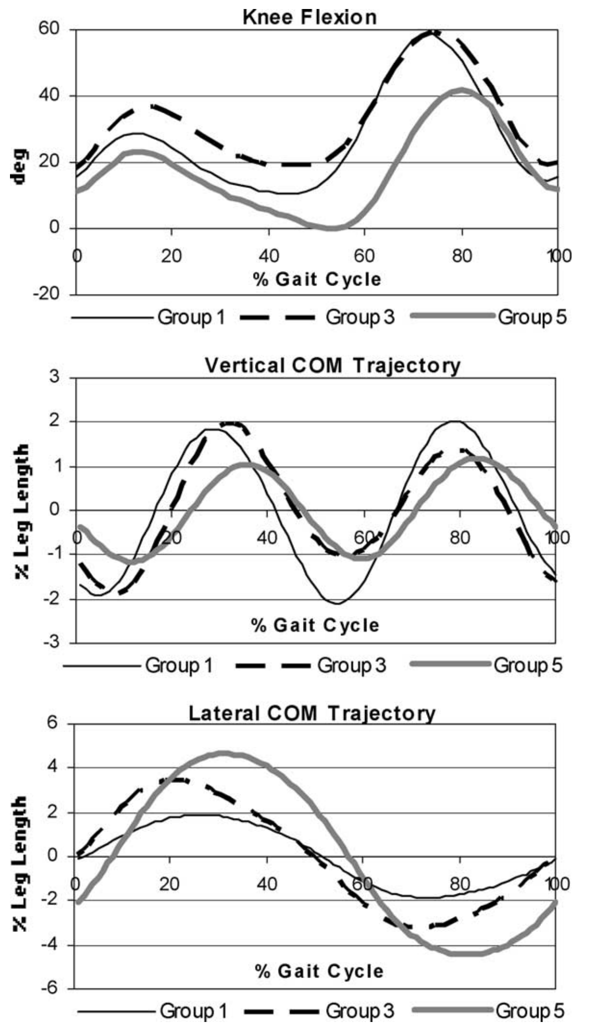

Fig. 1. Previously-reported sagittal plane knee kinematics and centre of mass (COM) excursions in the vertical and lateral directions during the gait cycle in Group 1 (weak but present plantarflexors and dorsiflexors), Group 3 (total absence of plantarflexor and dorsiflexor strength, some weakness in hip abduction and extension) and Group 5 (total absence of plantarflexion, dorsiflexion, abduction and hip extension strength). Group 3 had the most knee flexion in stance, while Groups 1 and especially 5 had more knee extension in mid-stance. Groups 1 and 3 had higher vertical COM peak-to-peak excursions than Group 5. The lateral COM excursion, however, increased from Groups 1 to 3 to 5. Data obtained from Gutierrez et al. [1,5].

with advancing muscle paresis [4,7,10]. In a recent study with the same patient population as the present study, the groups who had no abductor strength were observed to have more knee extension during stance than those with abductor strength when walking in orthoses [1] (Fig. 1). The groups with weak abductors displaced their centres of mass laterally much more than the groups with abductor strength [5] (Fig. 1), displacing their centres of mass laterally over their hip joints. This finding contrasted those from other centres who found no increase in the lateral centre of mass displacement in children with weak abductors [14] whose subjects were described as moving the pelvis and hip medially as the trunk moved laterally. This may reflect different orthotic philosophy.

Ankle–foot orthoses have been observed to prevent excessive tibial advancement for subjects with weak plantarflexors [10,17,18,21], but the other major lower limb muscle groups are not as easily compensated for without changes in gait kinematics. The altered gait pattern, combined with orthotic support, provides the support and the forward progression, yet these mechanisms are not fully understood. Investigations with knee–ankle–foot orthoses are less common in the literature. It has been reported, however, that similar trunk and pelvis motions [13] and heart rate [22] were observed in children with MMC and weak abductors tested in both ankle–foot and knee–ankle–foot orthoses.

The aims of this study were to determine to what extent successive muscle paresis in the lower limbs alters the kinetic gait strategies at the ankles, hips and knees, and to explain the kinetic mechanisms of the compensatory movements in subjects who lack lower body strength and use a different gait strategy from normal to progress forward. Furthermore, this study aimed to identify which muscle groups' pareses are most determinant in gait kinetic alterations, and to investigate the kinetic mechanisms of gait in children who lack abductor strength, revealing a gait pattern which has little been described in the literature.

2. Methods

Ethical approval for this study was obtained from Karolinska Institutet Ethics Committee. Participation was voluntary.

2.1. Subjects and classifications

A consecutive series of 45 ambulatory children with MMC born between 1985 and 1995 was recruited for the study between 2001 and 2003. Four children chose not to participate, two children could not be reached, and one had moved away from the area. Of the 38 children tested, the following exclusion criteria were established: thoraco- or cervicocele and inability to walk independently without the use of a walking aid. Five of the remaining subjects were excluded: four who were not able to walk without a walking aid and one with a cervicocele. No kinetic data were obtained in two subjects, whose data were then eliminated, leaving a total of 31 subjects in this study (mean age 10.3 years, Table 1). Twenty-one healthy controls were also tested (mean age 10.4 years, Table 2). Muscle function was assessed using a standard manual muscle test (0–5 scale)[23]: in grade (0) no contractile activity was felt, (1) muscle activity in a gravity-eliminated position, (2) all or partial range of motion in a gravity-eliminated position, (3) could perform the movement through the full range of motion but not withstand resistance, (4) the subject could hold the position against moderate to strong resistance and had full range of motion, and (5) the subject could hold the