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ORIGINAL RESEARCH

Mechanisms contributing to gait speed and metabolic cost in children with unilateral cerebral palsy

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Abstract
Background: Gait speed and metabolic cost are indicators of functional capacity in children with cerebral palsy. Uncovering their mechanisms helps guide therapeutic actions.
Objectives: To investigate the contributions of energy-generating and energy-conserving mechanisms to gait speed and metabolic cost of children with unilateral cerebral palsy.
Methods: Data on eccentric and concentric muscle work, co-contraction, elastic torque and vertical stiffness of the affected-limb, forcing torque of the non-affected limb, gait speed and metabolic cost were collected from 14 children with unilateral cerebral palsy, aged 6–12 years. Analyses included two groups of multiple regression models. The first group of models tested the association between each dependent variable (i.e., speed and metabolic cost) and the independent variables that met the input criteria. The second group verified the contribution of the non-selected biomechanical variables on the predictors of the first model.
Results: Gait speed ($R^2 = 0.80$) was predicted by elastic torque ($\beta = 0.62$; 95%CI: 0.60, 0.63), vertical stiffness ($\beta = -0.477$; 95%CI: -0.479, -0.474) and knee co-contraction ($\beta = 0.27$; 95%CI: -1.96, 2.49). The production of eccentric work by the affected limb proved relevant in adjusting the vertical stiffness ($R^2 = 0.42$; $\beta = -0.64$; 95%CI: 0.86, -0.42); elastic torque of the affected-leg was associated with impulsive torque of the non-affected leg ($R^2 = 0.31$; $\beta = 0.55$; 95%CI: 0.46, 0.64). Metabolic cost of gait ($R^2 = 0.48$) was partially predicted by knee co-contraction ($\beta = 0.69$; 95%CI: 0.685, 0.694).
Conclusions: The chain of associations revealed by the two steps models helped uncover the mechanisms involved in the locomotion of children with unilateral cerebral palsy. Intervention that changes specific energy conserving and generating mechanisms may improve gait of these children.

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Introduction

Gait supports the independence of children with cerebral palsy (CP) in different environments.[1] Previous studies identified speed and metabolic cost of gait as indicators of the functional capacity of these children.[2,3] Such variables are intrinsically related: an increase in gait speed increases the metabolic cost of gait.[4,5] Compared to typically developing children, children with CP exhibit higher metabolic cost when walking,[6,7] although their gait speed is slower.[8]

Gait speed is the product of stride frequency and stride length.[9] There are two ways in which gait speed can be increased: via an increase in stride length through increased generation of power or via an increase in cadence through increased stiffness of lower limbs' tissues.[9,10] Power generation capacity of lower limbs in children with unilateral CP (UCP) is known to be lower than that of typically developing peers.[11] As a result, their ability to increase gait speed by increasing their stride length is reduced.[12,13] Increasing cadence, therefore, becomes the most viable strategy for increasing their gait speed.[2] To increase cadence, children with UCP must actively increase stiffness, which could theoretically result in higher metabolic cost of gait.[14,15]

Several factors are associated with gait speed and energetic cost in children with UCP.[4,12,16,17] For example, a positive association between lower limb muscle strength and gait speed of children with spastic UCP has been consistently documented.[18,19] Leg extensor muscle strength, however, only partially explains the energetic cost of gait in these children.[17] Unnithan et al.[14] claimed that children with CP's increased metabolic cost of walking could be attributed to higher levels of muscular co-contraction.[14] It has been proposed that co-contraction restricts the ability to increase stride length when walking, reducing speed and negatively impacting gait metabolic cost.[14] On the other hand, muscle co-contraction could increase joint stiffness, ensuring greater conservation of mechanical energy.[12,13,16] As a result, there is no consensus regarding the role of co-contraction on gait speed or energetic cost of children with UCP.[12]

Despite the interest in identifying factors that influence gait speed and energetic cost in children with UCP, their essential determinants have not yet been uncovered. Theoretical models of gait of children with UCP identify mechanisms related to decreased speed and increased metabolic cost.[10,13,20] For example, the biomechanical model of inverted pendulum with springs, proposed by Fonseca et al.,[13] describes energy generating and conserving mechanisms that are fundamental to gait. This model captures overall gait adaptations used by children with UCP and may help understand specific determinants of gait speed and metabolic cost.

Child's ability to generate power (e.g., impulsive torque) or to conserve energy stored in elastic tissues (e.g., elastic torque) is essential for walking.[13,21] These generating and conserving mechanisms are related, respectively, to concentric work produced by the legs[22] and the ability to control joint stiffness via co-contraction or eccentric work.[23] Understanding the role of these factors, which are modifiable by means of intervention, may support the development of effective therapeutic strategies. This study aimed to investigate how mechanisms related to the ability to generate power and conserve energy contribute to gait speed and metabolic cost in children with spastic UCP.

Methods

Participants

Participants were 17 children with UCP. Inclusion criteria were: medical diagnosis of spastic hemiplegic CP, age 6–12 years, independent ambulation (i.e., Gross Motor Function Classification System – GMFCS levels I or II), not participating in a muscle strengthening program, not having contraindications to exercise, not having had botulinum toxin application or used serial plaster casts in the 6 months prior to evaluation and not having undergone surgery up to twelve months before evaluation. Three children were excluded due to technical problems during data collection, resulting in a group of 14 children (Table 1). The mean age of participants was 7.78 years (standard deviation [SD] = 1.31 years). Four participants had undergone Achilles tendon lengthening surgery more than a year previously, 5 received botulinum toxin application to the calf more than 6 months before the study, and 3 had never used bracing. The University Research Ethics Review Committee approved this study's procedures (ETIC: 585/08 – Universidade Federal de Minas Gerais, Belo Horizonte, MG, Brazil). Children's guardians signed informed consent forms authorizing participation.

Instruments and procedures

In this cross-sectional study, one trained examiner performed all data collection. In addition to the primary outcomes (gait speed and metabolic cost), the following variables related to energy generation and conservation were measured and/or calculated: concentric and eccentric lower limb muscle work, co-contraction of the affected leg muscles, vertical body stiffness, impulsive torque of the non-affected leg and elastic torque of the affected leg. Children remained barefoot and without orthotic devices in all measurements.

Children's body weight, height, total leg length and length of the thigh, shank and foot were measured. Each child was equipped with a portable gas analyzer (Cosmed K4 b^2) to measure oxygen (O^2) consumption and calculate the metabolic cost. Data were collected during five minutes at rest and five minutes at self-selected speed. Muscle activity and kinematic parameters were evaluated simultaneously during gait. Active surface electrodes recorded the activity of the gluteus maximus, rectus femoris, vastus lateralis, biceps femoris, tibialis anterior and gastrocnemius muscles. The electromyographic signal was transmitted wirelessly to an ME6000 electromyograph (Mega Eletronics®, Finland) with a sampling frequency of 1000 Hz. To normalize the signals obtained during gait, the children performed maximal voluntary contractions (MVCs).

A motion analysis system (Qualisys ProReflex®, Gothenburg, Sweden) with 6 cameras captured kinematic gait parameters at 120 Hz. Passive markers were placed on the head (2 on the temporal bones in front of the ear and 1 on the frontal bone in the glabellar region), trunk (2 on the body of the clavicles and 1 on the body of the sternum),

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