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Lower extremity joint stiffness during walking distinguishes children with and without autism

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ABSTRACT

How children with Autism Spectrum Disorder (ASD) and peers with typical development (TD) modulate lower extremity stiffness during walking could identify a mechanism for gait differences between groups. We quantified differences in lower extremity joint stiffness and linear impulses, along the vertical and anterior/posterior axes during over-ground walking in children with ASD compared to age- and gender-matched children with TD. Nine age- and gender-matched pairs of children, aged 5–12 years, completed the current study. Joint stiffness and linear impulses were computed in four sub-phases of stance: loading response, mid-stance, terminal stance, and pre-swing. The Model Statistic technique ($\alpha = 0.05$) was used to test for statistical significance between the matched-pairs for each variable and sub-phase. Furthermore, dependent *t*-tests ($\alpha = 0.05$) were utilized, at the group level, to determine whether significant differences existed between sub-phases. Results indicate that children with ASD may exhibit greater stiffness in pre-swing, and thus, produce inefficient propulsive forces during walking. We attribute these differences to sensory processing dysfunction previously observed in children with ASD.

1. Introduction

Autism Spectrum Disorder (ASD) is clinically characterized by core features such as deficits in social and language skills, movement stereotypy, restricted interests and hyper- and hyposensitivity to sensory stimuli (American Psychiatric Association, 2013; Baoi, 2010; Kim & Lord, 2013). However, recent hypotheses suggest movement quality should also be considered a core feature of the disorder (Dufek, Eggleston, Harry, & Hickman, 2017; Eggleston, Harry, Hickman, & Dufek, 2017; Hocking & Caeyenberghs, 2017; Moran, Foley, Parker, & Weiss, 2013) since motor dysfunction is present in as many 90% of children with ASD (David et al., 2009; Floris et al., 2016; Miyahara et al., 1997). Specifically, children with ASD were shown to exhibit both different movement patterns and lesser movement control over a variety of movements in comparison to children with typical development (TD) (Calhoun, Longworth, & Chester, 2011; Cook, 2016; Dufek et al., 2017; Eggleston et al., 2017; Fournier et al., 2010; Kindregan, Gallagher, & Gormley, 2015; May et al., 2016; Rinehart, Tonge, Bradshaw, et al., 2006; Rinehart, Tonge, Iansek, et al., 2006). Not only are these movement abilities different than their peers with TD, the degree of physical performance impairments is quite heterogeneous among children with ASD (Dufek et al., 2017; Eggleston et al., 2017). It has been stated that continued study of movement quality in this population could help explain the root cause of movement dysfunction in individuals with ASD (Fournier, Hass, Naik, Lodha, & Cauraugh, 2010; Hocking & Caeyenberghs, 2017; Kindregan et al., 2015; Mari, Castiello, Marks, Marraffa, & Prior, 2003; Moran et al.,

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2013; Weiss, Moran, Parker, & Foley, 2013) and provide a more comprehensive understanding of the disorder. Furthermore, continued experimentation relative to unique motor abilities may provide more detailed insight into specific intervention-responses among individuals with the disorder to improve overall treatment outcomes.

Gait abnormalities in children with ASD have been related to muscular weakness (Kindregan et al., 2015), hypotonia, akinesia, and bradykinesia (Damasio & Maurer, 1978; Kohen-Raz, Volkman, & Cohen, 1992). However, the specific neuro-musculo-skeletal parameter underlying the uniqueness of each child's developed movement pattern remains unknown. A potential variable to explain such unique physical presentations is lower extremity joint stiffness due to its importance during musculoskeletal movement performance (Butler, Crowell, & Davis, 2003) and its involvement in all human gait patterns. Joint stiffness is often quantified using a mass-spring model which describes the interaction between the body and the ground (Farley & Morgenroth, 1999) by dividing the torque about the joint by the angular displacement of the joint during a movement. As joint angular position changes during walking, the stiffness of the joint also changes (Farley & Morgenroth, 1999). Individuals with TD voluntarily modify joint stiffness in response to changes within the environment (Ferris & Farley, 1997; Ford, Myer, & Hewett, 2010) during a movement task. However, individuals with ASD might not possess the ability to appropriately modulate lower extremity joint stiffness due to deficits in movement planning and spatial awareness (dyspraxia) (Dziuk et al., 2007; MacNeil & Mostofsky, 2012), as well as sensory perception (Kern et al., 2006). Examining lower extremity joint stiffness may provide insight into the neurophysiological underpinnings of motor planning and control associated with this disorder. Understanding how children with ASD, relative to children with TD, modulate lower extremity stiffness could identify a mechanism for gait differences observed between children with ASD and their peers with TD.

Increased joint stiffness is thought to be a compensatory strategy employed to maintain stability about the joint, as observed in individuals with knee osteoarthritis (Gustafson, Gorman, Fitzgerald, & Farrokhi, 2016). However, insufficient joint stiffness can lead to excessive and unnecessary joint motion (Butler et al., 2003) due to a lack of dynamic joint stability (Ford et al., 2010). Increased stiffness could also lead to increased ground reaction force (GRF) impulse magnitudes, due to either increased torque or decreased angular motion about a joint. The combination of increased GRF impulse magnitudes and excessive joint motion could lead to issues relating to balance and stability challenges (Ament et al., 2015; Bugnariu et al., 2013; Memari et al., 2013) which may result in a trip or fall. Specific to ASD, increased joint stiffness might be employed at distal joints, such as the ankle, as a protective mechanism to reduce the risk of falling when the foot is in direct contact with the ground. Moreover, increased distal joints stiffness during the early portion of stance may provide evidence for a lesser ability to control the distal segments in anticipation for impact with the ground. The large number of significant differences previously observed in both vertical and anterior/posterior GRF trajectories during stance phase between children with ASD and their peers with TD (Dufek et al., 2017) indicates children with ASD might modulate their lower extremity joint kinematics prior to, and immediately upon ground contact in comparison to their peers with TD who adapt stiffness to dynamic changes in terrain or motor task. Because the foot is the only body part contacting the ground during bipedal walking, children with ASD may have a neurologic strategy to cautiously modulate the ankle joint stiffness because it is the most distal larger joint in the lower extremity which is responsible for ankle strategy balance response. These anticipated occurrences could explain the greater number of significant differences previously observed between children with ASD and matched peers with TD in hip joint angular positions versus ankle joint angular positions (Dufek et al., 2017).

The purpose of this investigation was to compare lower extremity joint stiffness between children with ASD and children with TD during the stance phase of over-ground walking using a matched-pair design. A secondary purpose was to determine whether differences in lower extremity joint stiffness coincided with different GRF magnitudes, as defined by GRF impulse. It was hypothesized that (a) greater magnitudes of joint stiffness would be observed in children with ASD, and (b) greater GRF impulse magnitudes would coincide with greater joint stiffness magnitudes. These hypotheses were based upon scientific speculation and the known presence of dyspraxia (Dziuk et al., 2007; MacNeil & Mostofsky, 2012), decreased coordination and smoothness during walking (Rinehart, Tonge, Bradshaw, et al., 2006; Rinehart, Tonge, Iansek, et al., 2006), and deficits of sensory perception (Kern et al., 2006) among children with ASD in comparison to children with TD.

2. Methods

2.1. Participants

Nine children with ASD and nine children with TD (5–12 years of age) participated in this study (14 males, 4 females; 9.0 ± 2.3 years, 1.4 ± 0.2 m, and 34.2 ± 14.0 kg, ASD; and 8.9 ± 2.1 years, 1.4 ± 0.2 m, and 36.3 ± 10.2 kg, TD). Participants were recruited from the community population through recruitment flyers which were dispersed to therapeutic clinical offering services to individuals with ASD. Further recruitment was conducted through the host university list-serve email list soliciting participants with ASD as well as children with TD. Each child with ASD was required to have a clinical diagnosis of the disorder, and each child's parent(s) verbally verified the diagnosis. Children with TD were age- and gender-matched to a study-enrolled child with ASD. Varied gait patterns were not excluded to best replicate the heterogeneous presentations among children with ASD (Calhoun et al., 2011; Eggleston et al., 2017; Eggleston, Landers, Bates, Nagelhout, & Dufek, 2018; Kindregan et al., 2015). Parental consent and child assent were obtained prior to completing any study related activities as approved by the Institutional Review Board (protocol number: 710824), and in accordance with the Declaration of Helsinki. An *a priori* sample-size estimation was not performed due to the matched-pair study design in which a single child with ASD was compared to a single matched control with TD. In such a design, a greater number of trials or observations afford greater statistical power independent of the sample size (Bates, Dufek, James, Harry, & Eggleston, 2016).

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