Sensorimotor Control Impairment in Young Adults With Idiopathic Scoliosis Compared With Healthy Controls

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Abstract

Objective: It has been hypothesized that the impaired sensorimotor control observed in adolescents with idiopathic scoliosis (IS) may be related more to the onset of scoliosis than to the maturation of sensory systems or sensorimotor control mechanisms. The objective of this study was to assess sensorimotor control in adults diagnosed with IS in adolescence versus healthy controls.

Methods: The study included 20 young adults 20 to 24 years of age (10 healthy controls and 10 diagnosed with adolescent IS but not treated for it). Binaural bipolar galvanic vestibular stimulation (GVS) was delivered to assess sensorimotor control. Vertical forces under each foot and upper body kinematics along the frontal plane were measured before GVS (2-second window), during GVS (2-second window), immediately after the cessation of GVS (1-second window), and during the following 2 seconds. Balance control was assessed by calculating the root mean square values of vertical forces and upper body kinematics.

Results: Compared with healthy controls, the IS group showed greater body sway upon GVS; the amplitude of this sway was even greater immediately after the cessation of GVS—an outcome requiring sensorimotor control.

Conclusion: Compared with normal controls, adults who had been diagnosed with IS in adolescence showed altered balance control immediately following GVS. This finding suggests that dysfunctional sensorimotor control may be related to the onset of scoliosis rather than to a transient suboptimal development of the sensory systems or sensorimotor control mechanisms. (J Manipulative Physiol Ther 2016;39:473-479)

Key Indexing Terms: *Scoliosis; Postural Balance; Neurobehavioral Manifestations; Sensation Disorders; Sensorimotor Feedback*

Introduction

Adolescent idiopathic scoliosis (AIS) is a tridimensional deformity of the spine that occurs during puberty, with a female-to-male ratio of 8 to 2 and a prevalence of 1% to 3%.[1-3] Most cases of scoliosis are not linked to any known cause and are consequently described as idiopathic.[4] It has been suggested that AIS involves genetic factors because of its higher prevalence in the children of individuals with scoliosis (27%) and its higher incidence among girls than among boys.[1,5-7] Genetic expression with the influence of environmental and lifestyle factors[8] could involve dysfunction at the biomechanical[9] (eg, asymmetrical bone growth), neurophysiological[10] (eg, sensory processing or sensorimotor transformation impairment), or molecular[11] (eg, melatonin signaling malfunction) levels. Because multiple factors are involved, it is difficult to pinpoint a particular cause of scoliosis onset or progression. A better understanding of the mechanisms leading to AIS inception and progression would help to improve the targeting of treatment.

With regard to the neurophysiological hypothesis, the vestibular system is worth considering as a potential risk factor in the onset of scoliosis.[4,12,13] For instance, because the vestibular nuclei are located in the brain stem and the lateral vestibulospinal tract controls axial muscles, an alteration in the

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brain stem during body growth (ie, the preadolescent and adolescent periods) may translate into abnormal activation of trunk muscles, causing spinal deformities.[4,14] This suggestion has been supported by results from the study of animal models. For example, scoliosis has developed in some rats (25%) with selective lesions of the brain stem structures (eg, the lateral vestibular nucleus) and in *Xenopus* with unilateral removal of a vestibular apparatus.[15,16] The possible link between these lesions and spinal deformities results from continuous asymmetrical axial muscle activity.[17] Impairment of vestibular processing has already been established in patients with AIS at the level of cognitive integration. For instance, in the dark, after rotating their bodies along the vertical axis, patients with AIS underestimate the magnitude of body rotation more than do controls.[12] However, their vestibulo-ocular reflex gains were similar to those observed in healthy participants. As a result, it has been suggested that alterations in vestibular information from the cerebellum to the vestibular cortical network or impairment of the cortical mechanisms that process vestibular signals could be implicated in the onset of AIS.[12] Sensorimotor control in adolescent patients with scoliosis has recently been compared with that in healthy adolescents.[18] While standing with their eyes closed, adolescents with mild (Cobb angle <30°) or severe (Cobb angle >30°) spinal deformities showed greater body sway than healthy participants during galvanic vestibular stimulation (GVS) or immediately thereafter. It is known that during or following sensory alteration, sensory reweighting is required to control balance.[19,20] Consequently, it is necessary for the sensorimotor control mechanisms to dynamically reweigh vestibular and somatosensory information and select motor commands that improve balance control. Impaired balance control implies that the sensorimotor control of adolescents with scoliosis is altered compared with that of controls.

Human balance control relies on feedback mechanisms that generate corrective motor commands based on body motions detected mainly by somatosensory, visual, and vestibular sensory systems. Because this information may be unavailable (with the eyes closed) or inaccurate (upon GVS), the brain must weigh sensory data—according to their relative reliability—to accurately select appropriate motor commands to control balance.[21] This is the sensorimotor control problem; it is addressed by altering the reliability of vestibular information and quantifying the amplitude of the vestibular-evoked postural response. Binaural GVS is an efficient way of altering the firing rates of all vestibular afferents, with a preference for the irregular afferents.[21,22] Such stimulation creates an illusion of movement toward the cathode, which evokes lateral body sway toward the anode.[22,23] In absence of visual information during GVS, and to reduce body sway, the brain must assign more weight to proprioceptive than vestibular data.[19] If, during or immediately following GVS, the central nervous system does not appropriately adjust the weight of proprioceptive and vestibular data, balance control will be impaired.[24]

Multisensory integration depends on the maturation of the nervous system.[25] For instance, it is known that individual sensory systems mature first on their own before multisensory integration becomes effective.[26] From childhood to adulthood, the privileged sensory system changes over the years.[27] For example, when somatosensory information alone or in conjunction with visual information is altered, balance control in children younger than 15 years of age is impaired compared with that in individuals older than age 20.[28] Therefore, it appears that effective sensorimotor control is not completely developed before age 20.[29,30] According to this observation, it is possible that the sensorimotor control impairment in adolescents with idiopathic scoliosis (IS) entails suboptimal maturation of the sensory or sensorimotor mechanisms. In that case, because balance control is drastically improved by age 20 despite conflicting sensory conditions, it should be expected that adults with IS would not demonstrate impaired sensorimotor control compared with age-matched controls. The objective of this study was to assess the sensorimotor control of adults who developed scoliosis during adolescence. We hypothesized that sensorimotor control impairment observed in adolescents with IS might be related to the onset of scoliosis rather than reflecting delayed maturation of the sensory or sensorimotor control mechanisms. Thus, it was expected that, during or immediately following GVS, adults with IS would demonstrate impaired balance control compared with healthy individuals without IS.

Methods

Participants

Our study included 20 participants (Table 1). Ten individuals without known history of scoliosis and without signs of scoliosis (Adam forward bend screening test and other visual inspection were performed) formed the control (CTR) group. The IS group consisted of 10 individuals who had been diagnosed with scoliosis by an orthopedic surgeon while they were adolescents. Because of their mild scoliosis, these patients did not receive any surgical or bracing treatment during adolescence, nor did they follow any specific rehabilitation exercises.[31-33] After protocol approval by the local biomedical ethics committee Comités d'éthique de la recherche avec des êtres humains de l'Université Laval (CÉRUL) (#2009-248), all participants gave their informed written consent prior to participating in the study. They were recruited through advertisements sent via various university mailing lists. Individuals with nonidiopathic scoliosis or a history of neurological disease were excluded.

Data Recording and Analysis

We previously adopted this experimental protocol to assess the sensorimotor control mechanisms of adolescents with IS.[24]