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A procedure to detect abnormal sensorimotor control in adolescents with idiopathic scoliosis



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ABSTRACT

This work identifies, among adolescents with idiopathic scoliosis, those demonstrating impaired sensorimotor control through a classification procedure comparing the amplitude of their vestibular-evoked postural responses. The sensorimotor control of healthy adolescents ($n = 17$) and adolescents with idiopathic scoliosis ($n = 52$) with either mild (Cobb angle $\geq 15^\circ$ and $\leq 30^\circ$) or severe (Cobb angle $> 30^\circ$) spine deformation was assessed through galvanic vestibular stimulation. A classification procedure sorted out adolescents with idiopathic scoliosis whether the amplitude of their vestibular-evoked postural response was dissimilar or similar to controls. Compared to controls, galvanic vestibular stimulation evoked larger postural response in adolescents with idiopathic scoliosis. Nonetheless, the classification procedure revealed that only 42.5% of all patients showed impaired sensorimotor control. Consequently, identifying patients with sensorimotor control impairment would allow to apply personalized treatments, help clinicians to establish prognosis and hopefully improve the condition of patients with adolescent idiopathic scoliosis.

1. Introduction

Scoliosis is the most frequent deformity of the spine occurring in adolescents [1]. According to various authors, adolescent idiopathic scoliosis (AIS) is a multifactorial condition involving a dysfunction in the central nervous system, an alteration in the control of hormonal and metabolic functions, an asymmetrical skeletal growth, or an abnormal loading of the spine [2]. The multifactorial model of AIS implies that the population of patient with AIS is heterogeneous. Thus, treating each patient with AIS using the same conservative treatment to limit the progression of spine deformation regardless of the patient's specific physiopathology has shown mitigate success [3–5]. A better understanding of the physiopathology could enhance the benefit of conservative treatment. As an example, scoliosis specific exercises (SSEs) could improve trunk sensorimotor control, improve respiratory function and potentially reduces spine deformation or curve progression [6–8]. Although SSEs looks promising, the conclusion of a Cochrane systematic review suggests that there is a lack of high-quality evidence to recommend the use of scoliosis-specific exercise for AIS [9]. It is possible that applying such SSEs only to patients with abnormal sensorimotor control would improve the benefit of the SSEs. Consequently,

identifying patients with sensorimotor control impairment would allow to apply personalized treatments and hopefully improve AIS patient's condition. Furthermore, knowing that a subset of patients has sensorimotor control impairment could be beneficial for geneticists [10]. In these patients, the causative gene of AIS could be related to the development of brain networks involved in sensorimotor control. Therefore, geneticist could target specific genes.

For decades, much work has been done to classify the types of spine deformation. For instance, the King and Lenke classifications are used to identify the best method of treatment for each curve pattern [11]. About a decade ago, classification of patients with AIS based on functional in vitro assays was elaborated [12]. Three different groups were formed according to their responsiveness to melatonin or a nucleotide (i.e., Gpp(NH)p). Identifying the distinct mutations causing alteration in melatonin signal transduction could lead to new treatment targeting specific gene. Furthermore, previous studies, through the analysis of trunk accelerometry signals have uncovered different aspects of the locomotor pattern in healthy and pathological populations [13]. In a similar way, we believe that developing a procedure identifying patients with AIS having sensorimotor control impairment is worthwhile, as specific sensorimotor treatments could be beneficial for these

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Table 1

Clinical and demographic characteristics of the participants. Clinical group data is reported using bold case, data from subgroups issued from the classification discussed in this article is reported with regular case. Mean (standard deviation) are presented when appropriate.

| Group | CTR | AIS-M total | AIS-M normal | AIS-M abnormal | AIS-S total | AIS-S normal | AIS-S abnormal |
|--------------------|-------------------------------|-------------------------------|------------------------|------------------------|-------------------------------|------------------------|------------------------|
| Age (years) | 14.4 [13.0 15.8] | 14.8 [13.9 15.6] | 14.6 [13.5 15.7] | 15.0 [13.5 16.5] | 15.2 [14.4 15.9] | 15.5 [14.6 16.3] | 14.8 [13.2 16.4] |
| Weight (kg) | 50.0 [43.7 56.2] | 54.4 [49.5 59.3] | 55.5 [48.9 62.1] | 52.8 [43.6 62.1] | 58.2 [50.4 66.0] | 56.9 [47.1 66.8] | 59.7 [44.6 74.8] |
| Height (cm) | 160.7 [157.5 163.9] | 164.6 [159.6 169.6] | 167.0 [161.0 173.0] | 161.0 [151.0 171.0] | 164.7 [159.5 169.9] | 165.6 [159.2 172.0] | 163.6 [153.2 174.0] |
| Cobb angle (°) | | 20.4 [18.8 22.1] | 21.8 [20.3 23.2] | 17.8 [14.1 21.6] | 40.4 [35.4 45.4] | 41.6 [33.7 49.6] | 38.9 [31.7 46.1] |
| Risser sign | | 3.8 [3.2 4.4] | 3.6 [2.8 4.5] | 4.3 [3.2 5.3] | 4.1 [3.5 4.6] | 4.4 [4.0 4.9] | 3.6 [2.4 9] |
| Menarche (years) | 11.1 | 10.6 | 10.1 | 11.4 | 10.8 | 12.7 | 8.9 |
| not yet | [8.9 13.3] | [8.0 13.3] | [6.3 13.9] | [6.7 16.1] | [8.1 13.5] | [11.3 14.1] | [3.2 14.5] |
| Other Conservative | 1 | 3 | 2 | 2 | 2 | 0 | 2 |
| Brace | 0 | 3 | 3 | 0 | 4 | 2 | 2 |
| Surgery | 0 | 9 | 5 | 4 | 16 | 9 | 7 |
| Male | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Female | 3 | 3 | 2 | 1 | 6 | 4 | 2 |
| Total | 14 | 17 | 10 | 7 | 14 | 7 | 7 |
| | 17 | 20 | 12 | 8 | 20 | 11 | 9 |

patients or help geneticists to target specific genes [14,15].

In a study investigating brain activation through functional MRI, when compared to controls, patients with AIS demonstrated a greater increase in blood oxygenation in contralateral supplementary motor area when performing a motor task with either hand. These patients also showed a larger interhemispheric asymmetry index than controls. These results support the suggestion that sensorimotor control impairment could be involved in the pathogenesis of AIS [16]. Further, the assessment of vestibular function revealed that some patients with AIS have cognitive vestibular information processing impairments [17]. It has been observed that patients with AIS underestimated whole body rotation, in absence of visual information, more than control participants despite normal vestibulo-ocular reflex [18]. Consequently, the authors suggested that impairment in the integration of vestibular information rather than peripheral organ dysfunction could be related to scoliosis onset. Nonetheless, impairment at the cerebellum or brainstem level has been suggested as visuo-oculomotor dysfunction was observed in patients with AIS [19]. Overall, despite some divergences, results from these studies suggest that patients with AIS have sensorimotor control impairments. Although the magnitude of the sensorimotor control impairment does not scale with spine deformation amplitude [20], it is possible that scoliosis onset is preceded by sensorimotor control impairments that last during curve progression. Bipolar binaural galvanic vestibular stimulation (GVS) can elicit a vestibular evoked postural response in the frontal plane. GVS is a tool for probing vestibular function and assessing its role in balance control as it stimulates the labyrinth receptors directly. It increases the firing rate of the vestibular afferents on the cathode side and decreases afferent firing on the anode side [21]. This alteration in the firing rate of the vestibular afferents creates a vestibular error signal that the brain interprets as an unplanned body movement towards the activated labyrinth. Thus, a postural response in the opposite direction is observed [22]. Consequently, the analysis of the amplitude of the vestibular-evoked postural response provides valuable information for studying sensorimotor control.

Because various factors are likely involved in scoliosis onset, we hypothesized that only a fraction of patients with AIS could demonstrate abnormal sensorimotor control. Identifying these patients would allow the development of patient’s oriented-treatment that could reduce spine deformation or curve progression. Consequently, we developed a classification procedure allowing the identification of patients with abnormal vestibular-evoked postural response from those with

postural responses like healthy controls.

2. Materials and methods

2.1. Participants

This study population includes data from a previously published manuscript investigating sensorimotor control in AIS patients (n = 52, 16 healthy controls, 16 and 20 patients with severe and mild spine deformation, respectively) [20]. Overall, the classification procedure was developed using a data set of 57 adolescents (45 females and 12 males; 40 AIS patients and 17 healthy controls).

Participants were aged between 10 and 18 years old and were divided into three different groups. Patients with AIS were grouped per the severity of their spine deformations (i.e., Cobb’s angle) measured by an orthopedic surgeon through X-ray. The Cobb’s angle represents the angle that measures the curvature of the spine along the frontal plane. The Cobb angle is defined as the angle formed between a line drawn parallel to the superior and inferior endplates that are the most tilted towards each other. The magnitude of the scoliotic curves was measured on radiographs. Twenty participants with a Cobb angle larger than 30° were assigned to the severe AIS group (AIS-S) and twenty participants having a Cobb angle larger than 15° and smaller than 30° formed the mild AIS group (AIS-M). These two groups were formed to determine if severity of spine deformation could be related to sensorimotor control impairment. Patients with non-idiopathic scoliosis or known neurological disease were excluded. Seventeen healthy age-matched participants with no known spine deformation were part of the control group (CTR). A description of the participants is provided in Table 1. Both AIS groups had similar skeletal maturity as no group difference was observed for the Risser sign (Mann-Whitney U Test, p = 0.53). Age, height, weight and age of menarche were similar between the three groups (ps > 0.05). Parents and adolescents gave their written informed consent per the institutional review board.

2.2. Data acquisition

Balance control was measured using two force platforms (AMTI, model BP400600NC-1000, Watertown, MA, USA), and upper-body kinematics was acquired using an electromagnetic system (Polhemus – model Liberty 240/8, Colchester, VT, USA). In order to track upper-body kinematics, one sensor was located on the 7th cervical vertebra

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