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REVIEW ARTICLE

Idiopathic scoliosis and the vestibular system

Ammar H. Hawasli · Timothy E. Hullar · Ian G. Dorward

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Abstract
Purpose Despite its high prevalence, the etiology underlying idiopathic scoliosis remains unclear. Although initial scrutiny has focused on genetic, biochemical, biomechanical, nutritional and congenital causes, there is growing evidence that aberrations in the vestibular system may play a role in the etiology of scoliosis. In this article, we discuss putative mechanisms for adolescent idiopathic scoliosis and review the current evidence supporting a role for the vestibular system in adolescent idiopathic scoliosis.
Methods A comprehensive search of the English literature was performed using PubMed (http://www.ncbi.nlm.nih.gov/pubmed). Research articles studying interactions between adolescent idiopathic scoliosis and the vestibular system were selected and evaluated for inclusion in a literature review.
Results Eighteen manuscripts of level 3–4 clinical evidence to support an association between adolescent idiopathic scoliosis (AIS) and dysfunction of the vestibular system were identified. These studies include data from physiologic and morphologic studies in humans. Clinical data are supported by animal model studies to suggest a causative link between the vestibular system and AIS.
Conclusions Clinical data and a limited number of animal model studies suggest a causative role of the vestibular system in AIS, although this association has not been reproduced in all studies.

Keywords Scoliosis · Vestibular System · Adolescent idiopathic scoliosis · Labyrinth · Vestibular

Abbreviations

AIS	Adolescent idiopathic scoliosis
MRI	Magnetic resonance imaging
VOR	Vestibulo-ocular reflex
VCR	Vestibulocollic reflex

Introduction

Adolescent idiopathic scoliosis (AIS) affects 1–3 % of children aged 10–16 and causes problems as back pain, pulmonary restriction, reduced mobility, and disfigurement [6, 42]. Treatment options include serial observation and bracing, but ultimately many patients will require operative intervention, which typically involves extensive, costly, and potentially morbid spinal instrumentation procedures. At present, the disease is thought to represent a multifactorial condition stemming from genetic, biochemical, nutritional, and biomechanical influences. Scoliosis may also be affected by select neurologic and neuromuscular influences such as cerebral palsy, paralysis and muscular dystrophies [14, 20]. One intriguing possibility is that sensory dysfunction may be another contributing cause. In particular, some evidence suggests that dysfunction of the vestibular system may be a contributing factor to the

A. H. Hawasli (✉) · I. G. Dorward
Department of Neurosurgery, Washington University School of Medicine, 660 South Euclid Avenue, Campus Box 8057, Saint Louis, MO 63110, USA
e-mail: Hawaslia@wudosis.wustl.edu

T. E. Hullar
Department of Otolaryngology, Washington University School of Medicine, St. Louis, MO, USA

I. G. Dorward
Department of Orthopaedic Surgery, Washington University School of Medicine, St. Louis, MO, USA

development of AIS. Here, we discuss a potential vestibular mechanism for AIS and review the current human and animal data supporting a role for the vestibular system in the etiology of adolescent idiopathic scoliosis.

Putative mechanism for AIS The vestibular apparatus is a special sensory system that assists in spatial orientation and balance. Together with the cochlea, it comprises the labyrinth that is housed in the otic capsule of the temporal bones bilaterally [29]. Each vestibular system contains five inertial sensors: three semicircular canals and two otolith organs (Fig. 1). The semicircular canals sense rotations of the head. Each canal is a fluid-filled tube opening at both ends into the vestibule. During movement, inertial forces affect hair cells which change the firing rates of primary vestibular afferent neurons projecting to the brainstem. The direction and inertial forces modulate the activity of this system, which allows the vestibular system to respond to accelerations in any direction. The three canals are oriented orthogonally to each other so that a rotation in any direction can be represented as the summed response from all three canals [29].

"Otolith" end organs, the utricle and saccule, reside within the vestibule and sense linear accelerations of the head, which can be due to linear movement ("translation") of the head in space or the effect of gravity ("tilt"). As with the hair cells of the semicircular canals, acceleration in one direction increases the firing rate of vestibular afferents leading from the otolith organs and accelerations in the other direction reduce the firing rate.

As the two labyrinths are mirror images of each other, a head rotation or linear acceleration that increases firing rate in one labyrinth will decrease the firing rate from the other. Thus, when the head turns to the left, the firing rate of afferents from the left horizontal semicircular canal increases and that from the right semicircular canal decreases. These asymmetric inputs are then compared by the central nervous system to determine the direction and amount of rotational and linear acceleration. This information drives two fundamental reflexes that are frequently tested, vestibulo-ocular reflex (VOR) and vestibulocollic reflex (VCR). VOR uses the signals provided by the vestibular system to move the eyes in the head so that they stay pointing the same direction regardless of head movement. This system allows a visual image to remain stable on the retina, eliminating the symptom of "oscillopsia." VOR eye motions are mediated by the different firing rates in right and left semicircular canals. VCR stabilizes the head on the body. It uses vestibular input to alter tension in the cervical musculature in response to head motions. VCR serves to resist passive head motions but is "turned off" by the brain during volitional head motion.

Examining the vestibular system relies on measuring the output of each of these reflexes in response to a testing stimulus that induces a vestibular asymmetry. An example is rotational chair testing, where a patient's eyes are measured while sitting in a chair and turning slowly in the dark. The speed of the head is compared to the speed

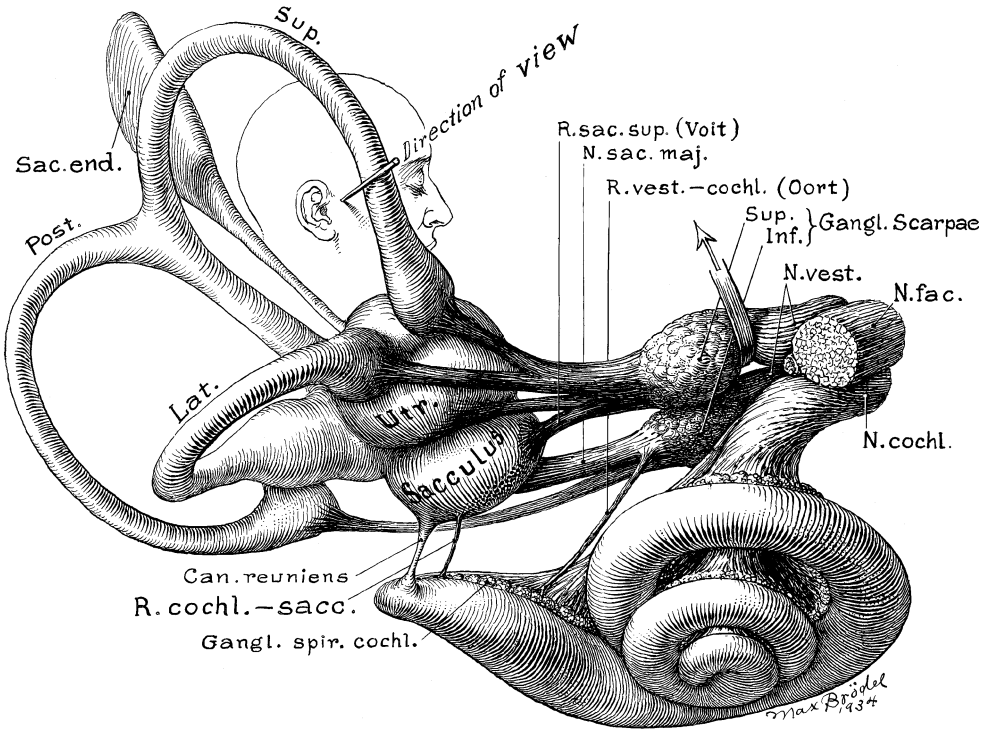

Fig. 1 The labyrinth (*right side*). The structures shown form the membranous labyrinth, which is filled with endolymphatic fluid. The membranous labyrinth sits inside the bony labyrinth, with the utricle and saccule together occupying the vestibule. The space between the membranous and bony labyrinth is filled with perilymphatic fluid. The semicircular canals sense rotations and the utricle and saccule sense linear accelerations. (Original illustration #933 in the Walters Collection of the Max Brödel Archives, Department of Art as Applied to Medicine, The Johns Hopkins University School of Medicine, Baltimore, Maryland, USA)

of the eyes moving to compensate for head motion. The ratio between eye and head velocity depends on the particular stimulus conditions, but when decreased can indicate vestibular weakness. Alternatively, caloric testing modulates the firing rates of afferents by introducing warm or cold water into the external auditory canal. Higher firing rates in the warmer ear relative to the cooler ear are interpreted as a rotation to the warmer side, in which the VOR counteracts by moving the eyes to the cooler side. This movement continues until the eyes reach the limit of their mobility and cannot turn any farther. A rapid saccade brings them back toward the warmer side, producing nystagmus. The intensity of nystagmus generated by the VOR depends on the vestibular system sensitivity, which is measured using electrical potentials recorded around the eye (electronystagmography) or video recordings of the eyes (videonystagmography). If the otolith organs on one side are weaker than the other, the brain may interpret the result as a tilt of the head. In response, the VOR rolls the eyes in the orbit (about the corneoretinal axis) to keep the visual world stable and horizontal. The amount of this roll is difficult to measure, so often the otolith system is measured by the subjective visual vertical test, where the subject sits in the dark and turns a light bar until it seems vertical. A deviation of more than a few degrees from true vertical indicates asymmetric otolith function.

Vestibular asymmetries can result from a variety of conditions. Acute unilateral loss is interpreted by the brain as a dramatic rotation of the head, activating the VOR and causing nystagmus, vertigo, and postural instability. Over time, however, the brain can accommodate partially to chronic asymmetry and these symptoms become much less pronounced. Changes in the responsiveness of brainstem and cerebellar neurons to peripheral input form an important mechanism for this compensatory process. In some cases, even patients with complete unilateral vestibular loss have few symptoms except during particularly rapid head motions.

Although we know much about compensation in the VOR to asymmetric vestibular input, we understand much less about how changes in the VCR and reflexive circuits affect muscle tension lower in the spine. The vestibular system can profoundly shift perceived verticality, head yaw/position and other balance-related functions, and therefore can induce a neurogenic trunk shift or torsional effect on the spine by activating particular muscle groups asymmetrically. Some evidence suggests that this muscle asymmetry alone could induce a scoliotic change [4, 5, 14–16]. If this suggestion is true, vestibular asymmetry leads to asymmetric recruitment of paraspinous musculature and, in turn, may lead to scoliosis.

Methods

A comprehensive search of the English literature was performed using PubMed (http://www.ncbi.nlm.nih.gov/pubmed). Search-engine key works included “adolescent idiopathic scoliosis” or “AIS” and “vestibular,” “vestibular system,” or “labyrinth.” Search-engine results were confirmed through the Google Scholar Search Engine (http://scholar.google.com/) where no additional citations were discovered. The search initially produced a total of 57 results which included duplicates. Inclusion criteria for the literature review included unique research articles that evaluated interactions between adolescent idiopathic scoliosis and the vestibular system. Final selection yielded 18 research articles.

Results

Scoliosis and vestibular responsiveness

A link has long been suspected between AIS and dysfunction in the vestibular organs. Early investigations identified a significantly increased prevalence of spontaneous and positional nystagmus among patients with AIS [32]. In a series of prospective studies, Sahlstrend et al. [32, 33] reported that AIS patients display increased sensitivity to caloric tests on the side ipsilateral to their curve and increased postural sway during caloric stimulation.

Cakrt et al. [2] demonstrated significantly altered perception of subjective visual vertical in AIS patients versus controls. Off-vertical axis rotation is a similar test, but is able to isolate the function of the otoliths in each ear. Wiener-Vacher et al. [43] identified that that AIS was associated with a significantly abnormal off-vertical axis rotation. Mallau et al. [26] discovered loss of head yaw strategies in AIS patients. Lion et al. [19] found that AIS patients with Cobb angle greater than 15 degrees had altered visuo-oculomotor function, as tested by saccadic latency and velocity.

Several studies have demonstrated altered posturography and postural sway under both static and dynamic conditions, [8–10, 15] with various types of scoliotic curves associating with balance perturbations to differing degrees [7]. By investigating the interplay between the visual, proprioceptive, and vestibular systems, multiple groups have independently found that patients with scoliosis demonstrate increased body sway and poorer balance control under situations of visual or proprioceptive challenge, or in the presence of underlying abnormal proprioceptive responses [10, 16, 18, 39, 40]. In addition to changes that can be attributed to reflexive function, scoliotic patients also show impaired cognitive integration of

vestibular signals compared with normal controls [38]. In summary, the clinical data provide level III-IV evidence (Table 1) that there is an association between vestibular abnormalities and scoliosis.

Scoliosis and vestibular anatomy

The functional deficits in vestibular activity seen in scoliotic patients seem to have anatomic correlates [36, 37]. Rousie et al. [31] found correlations between basicranial labyrinthine measurements on MRI, labyrinth activity and lumbar scoliosis. Shi et al. [36, 37] demonstrated that subjects with AIS have 5 % shorter distances between centers of the lateral and superior semicircular canals and 6 % smaller posterior canals. Using different morphological techniques, Zeng et al. [45] found that vestibular canals are 9 % longer and 2 % thinner in people with AIS than controls. Rousie et al. [30] evaluated 445 patients with vestibular symptoms (95 of whom had scoliosis) and discovered that an abnormal direct connection between the lateral and posterior vestibular canal was found in 55 and 15 % of patients with and without scoliosis, respectively. Direct morphological vestibular studies also correlate with findings in downstream neural targets. Upon evaluating MRIs from 90 patients, Wang et al. [41] determined that the thickness of cerebral cortex in areas involved with

Table 1 Clinical evidence linking idiopathic scoliosis and the vestibular system

References	Design	Level of evidence	No. of subjects	Major finding
Sahlstrend et al. [32]	Case–control study	III	56	AIS is associated with spontaneous and positional nystagmus. AIS patients display increased sensitivity to caloric tests on the side ipsilateral to their curve
Sahlstrend et al. [33]	Case–control study	III	49	AIS patients display increased postural sway during caloric stimulation
Sahlstrend et al. [35]	Case–control study	III	40	Increasing scoliosis curvature by changing to the erect position did not affect labyrinthine sensitivity
Sahlstrend et al. [34]	Case–control study	III	52	Vestibular abnormalities cannot predict scoliosis progression in AIS
Cakrt et al. [2]	Case–control study	III	56	AIS patients display altered perception of subjective visual vertical
Mallau et al. [26]	Case–control study	III	33	AIS patients display loss of head yaw strategies
Wiener-Vacher et al. [43]	Case–control study	III	45	AIS is associated with abnormal off-vertical axis rotation
Gruber et al. [8]	Case–control study	III	46	AIS patients display a complex impairment in center of pressure sway, a measure of postural control
Guo et al. [9]	Case–control study	III	162	AIS is associated with increased latencies in somatosensory-evoked potentials and an abnormality in postural control
Haurmont et al. [10]	Case series	IV	65	Patients with Cobb angle >15° have worse balance control than those with smaller Cobb angles
Kuo et al. [15]	Case–control study	III	55	AIS patients show abnormalities in posture control patterns with asymmetric muscle activities
Gauchard et al. [7]	Case–control study	III	102	Different curve types display different balance control impairments
Simoneau et al. [38]	Case–control study	III	23	Scoliotic patients show impaired cognitive integration of vestibular signals compared with normal controls
Lao et al. [18]	Case series	IV	24	Somatosensory dysfunction in AIS patients impacts dynamic balance control
Kuo et al. [16]	Cross-sectional study	III	44	AIS disrupts balance control requiring compensatory muscle activity
Simoneau et al. [39]	Cross-sectional study	III	17	AIS patients display impaired scaling of balance control commands
Simoneau et al. [40]	Cross-sectional study	III	18	AIS patients depend more on ankle proprioception than controls for balance control
Lion et al. [19]	Cross-sectional study	III	53	AIS patients with Cobb angle >15° showed abnormal visuo-oculomotor functions: increased saccade latencies and decreased saccade velocities

vestibular function was different in patients with AIS. Morphological data from radiological sources support an association between vestibular abnormalities and scoliosis.

Limitation of current clinical data and radiographic data

Despite a long list of clinical and radiological studies to support an association between the vestibular system and AIS, much of the data relating scoliosis and vestibular dysfunction remain predominantly correlative. This association between vestibular dysfunction and scoliosis does not clarify whether one begets the other, as vestibular dysfunction may in fact just represent an epiphenomenon created as an individual adapts to a curved spine. Nonetheless, several studies to date have posited an etiopathological causality, whereby changes in the vestibular system precede and cause scoliosis. One study demonstrated that an increase in scoliosis curvature, caused by changing patients to the erect position, did not in turn affect the amount of labyrinthine sensitivity [35]. The initial Sahlstrend et al. studies suggested that differences in labyrinthine responses noted in AIS were likely due to primary vestibular etiologies rather than the scoliosis and suggested that such vestibular abnormalities may contribute to scoliosis through vestibular–spinal mechanisms. Furthermore, if scoliosis was the cause of vestibular dysfunction, rather than the other way around, there would need to be a mechanism whereby the vestibular system would receive inputs indicating the presence of a spinal deformity; in fact, there is not a well-described feedback system between the spine and the peripheral vestibular organs. For example, Joassin et al. [12] found no significant effects of complete thoracic spinal cord injuries on orientation of subjective visual vertical. Barra et al. [1] also found that verticality estimates in subjective visual vertical tests were not different for paraplegic patients versus normal controls.

Despite the evidence supporting a potential causative role of vestibular dysfunction, the data linking scoliosis and the vestibular system have not been reproduced in all studies. For example, a prospective evaluation of AIS patients with severe curves demonstrated vestibular and postural dysfunction in only a fraction of subjects, leading the authors to suggest that no etiologic link existed [27]. Despite the association between AIS and aberrations in vestibular function, Sahlstrend et al. [34] did not find that vestibular abnormalities in stabilometry and electronystagmography can predict scoliosis progression. To further complicate interpretation, scoliosis has been associated with several other genetic, congenital and neuromuscular disorders [6, 20, 31, 42], suggesting that AIS may be a multifactorial disease spectrum with vestibular and other etiologies.

Animal models: a search for causality

Several animal studies have also examined the causal relationship between vestibular and scoliosis. De Waele et al. [3] evaluated the effect of selective lesions of otolith receptors in guinea pigs, finding that lesion induced scoliotic curvatures with rotation towards the contralateral side. X-rays of the head and spine after hemilabyrinthectomy showed head rotation due to cervical vertebra rotation and head tilt due to thoracic vertebra rotation. It was also noted that unilateral lesions of the otolithic and semicircular canal systems resulted in thoracic vertebra or head rotation, respectively. Although, these studies used adult animals, it is difficult to extrapolate their results to adolescent human. In a recent study using an aquatic tadpole—frog model, Lambert et al. [17] isolated the vestibular influence upon the growing spine and have reliably created scoliotic deformities through unilateral vestibular lesioning during a critical window in tadpole development. Labyrinthine end organs were removed in frog Xenopus larva and X-ray images with three-dimensional spinal reconstructions were obtained after metamorphosis into young adult frogs. They found that aqueous frogs develop scoliosis in coronal and sagittal planes and along the rotatory axis. The authors suggest that imbalanced activity in locomotor—postural control mechanisms may be a common mechanism for scoliosis. Scoliosis has also been created by pinealectomy in chickens [13, 21–23, 25] or bipedal rats (rendered so by forelimb amputation) [24, 44] and by variations of rib resections or asymmetric muscular stimulations in rabbits, lambs, or chickens, [4, 5, 28] though the applicability of these findings to the human condition of AIS remains questionable [11]. Combined, the current animal studies suggest that the vestibular system may play a major role in the creation of scoliotic deformities.

Discussion

Adolescent idiopathic scoliosis is a common disease with significant clinical and socioeconomic impact. Unfortunately, there is little consensus on the etiology underlying AIS. This review shows that several studies support an association between vestibular abnormalities and AIS. Limitations of this literature review include the fact that the search was restricted to the English language, the limited number of case–controlled studies and the paucity of studies showing causation.

At present, then, the literature is replete with evidence to link scoliosis with abnormalities of the vestibular system. However, additional studies are needed to adequately assess for causality. Novel clinical tools allow us

to further interrogate the vestibular system. Vestibular testing has advanced since prior studies have attempted to evaluate a link between vestibular dysfunction and the development of scoliosis. Dynamic assessments of otolithic function are now possible, enabling more direct assessment of the functional accuracy of the postural relationship to the true vertical in individual patients. As technology advances, new neuroradiological testing may also provide additional information. For future clinical studies, one may test if vestibular rehabilitation, including positional tasks, head movements and oculomotor exercises, can mitigate some of the vestibular abnormalities observed and the extent or progression of AIS. If vestibular rehabilitation shows an effect, research using medical vertigo management strategies (e.g., diuretics, vestibular suppressants or other medications) may be considered.

Basic science animal research using systems such as the frog model [17] offers new insights and may also address question of causality. Since AIS was associated with abnormalities in the cerebral cortex associated with vestibular function [41], future animal models could also target regions of the nervous system. Indeed, advanced animal models could allow for targeted manipulation of the vestibular system to induce and potentially reverse scoliosis. Additional clinical and basic science studies offer new opportunities to define an etiological link between the vestibular system and AIS.

Conclusion

Adolescent idiopathic scoliosis has historically been linked to genetic, biomechanical, nutritional and congenital causes. There is a significant volume of level 3–4 clinical evidence to support an association between AIS and dysfunction of the vestibular system. These studies include data from physiologic and morphologic studies in humans. Although this association has not been reproduced in all studies, clinical data are supported by a limited number of animal model studies to suggest a causative role of the vestibular system in AIS. Additional research is necessary to justify incorporating vestibular rehabilitation techniques (e.g., positional tasks, oculomotor exercises) within conservative AIS management.

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Conflict of interest The authors have no relevant conflicts of interest.

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