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The aetiology of idiopathic scoliosis: biomechanical and neuromuscular factors

Received: 4 March 1999
Revised: 27 January 2000
Accepted: 4 February 2000

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Abstract The aetiology of adolescent idiopathic scoliosis (AIS) remains an enigma. In the literature there are two opinions: one believes a deviating growth pattern is responsible for the condition – patients with AIS tend to be growing faster/be taller – while the other opinion assumes that the growth pattern is normal, but its presence is necessary to allow the development of the scoliosis. We discuss the two stage hypothesis: the natural history of AIS involves an initial stage in which a

small curve develops due to a small defect in the neuromuscular control system and a second stage during adolescent growth in which the scoliotic curve is exacerbated by biomechanical factors.

Key words Scoliosis, aetiology · Scoliosis, pathogenesis · Scoliosis, neuromuscular · Scoliosis, growth

Introduction

Idiopathic scoliosis is a deformity of the torso, characterised by lateral deviation and axial rotation of the spine. Although good anatomic descriptions of the structural changes seen in scoliosis were first made by the ancient Greeks, we have not as yet elucidated its pathogenesis.

The deformity always develops from a straight spine into a curved spine, usually accompanied by a rib cage deformity, during the growth period in general, and in particular in the rapid growth period.

In the growing scoliotic spine, the loss of mechanical stability results in deformation of the vertebral bodies and ribs. The eventual magnitude of an idiopathic scoliotic curve varies and is unpredictable. The extent of the alterations in the shape of the vertebrae and ribs is strongly related to the severity of the scoliotic curve. Pain is a rare symptom, and the patient seems unaware of his or her condition. The search for an aetiological explanation of idiopathic scoliosis has included virtually all aspects of medicine. The frequency with which scoliosis is found in many different neurologic syndromes has led to repeated attempts to find a neurologic cause for idiopathic scoliosis.

Neurologic disease with motor weakness is a well-recognised cause of scoliosis [31, 36, 43], while scoliosis is also frequently found in association with disease affecting afferent function [37]. Although some workers favour a neuromuscular basis for the condition, others believe that asymmetrical growth is the primary aetiological factor [13, 14, 16, 17, 21, 54]. It is unlikely that there is one single aetiological agent. This paper analyses the different factors of relevance, combining the findings, and proposes a possible model for the pathomechanics.

Aetiology

A strong familial history is usual, and a hereditary transmission suggesting an autosomal dominant or multifactorial defect has been described [103, 104]. Whatever factors underlie the aetiology, they ultimately express themselves in the biomechanical changes that define scoliotic curve progression [6, 15, 54, 68]. For over 100 years the association between idiopathic scoliosis and vertebral growth has been debated [5, 10, 12, 21–23]. A large number of studies on growth differences between normal and scoliotic girls have been conducted. Unfortunately, the re-

sults of these studies were not consistent. Willner observed a taller mean standing height in girls with scoliosis compared to healthy controls [99–101]. These findings were supported by other cross-sectional studies [9, 35, 44, 47, 58, 60, 61, 80].

Loncar-Dusek et al. demonstrated a higher peak velocity for scoliotic children [45]. Moreover, Goldberg et al. and Ylikowski et al. reported that girls with adolescent idiopathic scoliosis (AIS) have an earlier growth spurt and earlier attainment of adult height compared to healthy nonscoliotic controls [32, 108]. This is in marked contrast to many other reports, which found no difference in growth pattern or height between AIS patients and nonscoliotic controls [19, 88, 90, 91]. However, one should keep in mind that most of the studies on growth differences between scoliotic and nonscoliotic girls mentioned above were based either on length measurements of the sitting height, without correction for the error introduced by the scoliotic deformity itself, or were corrected using the method described by Bjure et al. [8]. This method overestimates the real length of the spine, and may not be valid for curves of 30° or less Cobb angle, since they had no patients with such mild curves in their material [81].

The advocates of a deviating growth pattern explain the initiation of idiopathic scoliosis as the result of a greater tendency of taller and more slender spines to buckle out of the sagittal plane under loading. [16, 17, 54, 82]. Deacon, Dickson and co-workers reported that the height of the anterior vertebral body of the apical vertebra in scoliotic patients was significantly greater than the posterior vertebral body height [13, 14, 16, 17, 54]. This lordosis at bony level was an important basis for their theory that thoracic lordosis, which is caused by a relative overgrowth of the anterior part of the vertebral body, triggers the initiation of scoliosis by buckling. In a three-dimensionally rendered CT scan study we have previously described the vertebral and rib deformities in idiopathic scoliosis [96]. The observed vertebral deformities suggest that these are caused by bone remodelling due to an imbalance between forces in the anterior and posterior spinal column [52, 96] (Fig. 1). In our study, we also noted a minimal wedge deformation in the local sagittal plane in certain apical vertebrae, as mentioned by Deacon and Dickson, but it is questionable whether this deformation in the sagittal plane is a primary aetiological phenomenon, as they suggest, or whether it is rather a secondary phenomenon, comparable to the other vertebral deformations. In an earlier study, we could not demonstrate a significant difference in the growth increments and spinal dimensions of vertebral bodies involved in the scoliotic curve compared with the rest of the vertebral column, and the growth increments were similar to those reported in the literature for normal girls [90, 91]. Nor could a difference in spinal flexibility be established between patients with idiopathic scoliosis and controls [51, 90, 92]. Furthermore, no proof of the “Euler theory”, that idiopathic

Fig. 1A, B Force pattern in the scoliotic spine. The compressive forces of the anterior column result in a force driving the apical vertebral body out of the midline (A), whereas the tension forces of the posterior column result in a force attempting to keep the posterior complex in the normal position (B)

scoliosis is the result of buckling under load, has ever been given. The mechanical behaviour of such a complex and highly non-linear structure as the human vertebral column is very difficult to analyse. Using a new finite element model of the spine, we have previously examined this buckling theory [66, 67]. Judging from the results of this finite element study, buckling can not initiate idiopathic scoliosis, because the characteristic coupling of lateral deviation and axial rotation is absent. Mechanical and computer models of the spine are frequently used to analyse the mechanisms by which scoliosis is initiated and aggravated. In a relatively simple stable physical model of the trunk, progression of the scoliosis due to growth can be shown [56, 59].

However, not all scolioses worsen, as the tendency to progress is balanced by neuromuscular factors and remodelling.

Neuromuscular factors

Awareness of the position of the body in space is a highly developed sense in humans. It is the result of input from the vestibular, visual and proprioceptive neural pathways. In recent years, strong evidence has been found for the idea that, for visuomotor co-ordination and exploration of space, the brain uses abstract, neural representations of space interposed between sensory input and motor output. These neural representations seem to be organised in non-retinal, body-centred and/or world-centred co-ordinates [4, 83]. Spatial information in non-retinal co-ordinates al-

lows the subject to determine the body position with respect to visual space, which is a necessary prerequisite for accurate behaviour in space. To obtain such a frame of reference, the information coded in co-ordinates of the peripheral sensory organs must be transformed and integrated.

Defective postural equilibrium has been proposed as a contributing factor in the development of scoliosis [34]. In this regard, defects in visual and vestibular input have been studied extensively as a possible genesis of idiopathic scoliosis. [39–41, 55, 73–77].

Vestibulo-ocular reflex changes may be viewed as a function of asymmetrical control of reflex gain, which is disturbed further during any postural task requiring control of the body motion in the presence of visual fixation. Hence, postural instability is ascribed to the conflict between visual and vestibular information within the higher central nervous system (CNS) centres, which can integrate and calibrate converging sensory data for perception and control of postural movement [39–41]. Proprioceptive input from joints, ligaments and tendons has been recognised as an integral contribution to the body's postural equilibrium [34]. Defects in the muscle spindle system and tone in the spinal muscles have been implicated in scoliosis [7, 42, 48–50, 98, 107].

Neural pathways involving visual, vestibular and proprioceptive afferents all have discrete interconnections in the brainstem. A lesion in this anatomical location could affect all three pathways. Congenital lesions in this area are associated with scoliosis [89], and scoliosis has been successfully induced by damaging this area [20]. Experimentally created defects in the vestibular system of a rat resulted in delayed posture and motor development [29]. Previous studies of CNS function in AIS have suggested that altered cerebral cortical/subcortical function [41, 55, 63, 73] or hemispherical dominance [24] may be related to the aetiology of AIS. Patients with scoliosis and primary alteration of the motor system, so-called neuromuscular scoliosis, are known to have a curve morphology and natural history very different from that of the "typical" idiopathic curve. Magnetic resonance imaging studies of the brain stem in adolescent idiopathic scoliosis by Geissele et al. show an asymmetry in the ventral pons or medulla in a number of patients [30].

The paraspinal muscles have been implicated by several investigators as a possible causative factor in the production and progression of adolescent idiopathic scoliosis [25–27, 85, 105].

An increased myoelectric response on the convex side of the curve, near its apex, was the main finding reported by various authors [2, 3, 11, 33, 38, 69, 70, 78, 85, 102, 105, 110], but not all agreed on the meaning of these findings. In early reports a fatigue mechanism was suggested [70], while others explained the difference as an effect of the stretching of the erector spinae muscles on the convex side [11]. This view was supported by the finding of a

stretch reflex (H-reflex) that was more sensitive to vibration and hammer tapping on the spinous processes in larger curves [42]. Others believed that the increased myoelectric activities on the convex side were only a secondary effect of the muscles adapting to a higher load demand in larger curves [110]. This would be consistent with the reported findings of differences in the morphology of the paravertebral muscles between the left and right sides [78, 85, 102, 105]. However, the increase of type 1 muscle fibres on the convex side can be explained on the basis of muscle denervation [27, 93, 94, 110], produced by an alteration of the motor drive arising at the spinal cord level, either from altered sensory input at the same level [64, 65, 87] or from a central mechanism [7, 20, 53, 73, 98].

The ocular and postural control systems reach maturation in early adult life [28, 57, 79]. Children in the age range of 9–16 years exhibit delay in the complete development of smooth pursuit and optokinetic nystagmus; moreover, they demonstrate a delay in tasks that couple

Fig. 2 A cortical model. Faulty sensory information from visual, vestibular, and somatic sensors or faulty interpretation within the central nervous system results in an altered body-spatial orientation, leading to sensory rearrangement to restore perceptual dysfunction. Motor adaptation of the axial motor system ensues when the subject maintains modified perceptual analysis of proprioceptive information describing an erect spine. A new motor control strategy is adopted, resulting in the structural scoliotic deformity

the vestibular and visual systems, particularly those requiring voluntary suppression of the vestibulo-ocular reflex [40]. This behaviour is required to ensure optimal visual acuity in phase with head motion. Incomplete maturation of visual and visuo-vestibular functioning is ascribed to inefficient extraretinal processing of perceptual information by cortical structures within the CNS, e.g. to delayed development of perception of the position of visual images in space [40, 79, 106, 109]. The maturation of the ocular and postural control systems coincides with the secondary rapid growth period. We propose, therefore, that the most likely cause of idiopathic scoliosis includes a neuromuscular condition and an asymmetry of the transversospinalis muscles, produced by alteration of the motor drive at the spinal cord level, either from altered sensory input at the same level or from a central mechanism, which may produce enough lateral deviation and axial rotation to embarrass the delicate balance of forces in the region, so producing an idiopathic scoliosis (Fig. 2).

Growth disturbance may not be a primary cause of idiopathic scoliosis, but it certainly plays a prominent part in the progression of this deformity.

Curve progression

Scoliosis does not always follow the same course [95]. Some cases are progressive while others remain stable. The deformity may continue to progress as long as vertebral growth potential remains. This growth is completed when the apophyseal vertebral rings have fused to the vertebral bodies. However, the deformity may progress after skeletal maturity, because of degeneration of the disk and the fibrocartilage at load transfer points on the concave side of the curve, although this progression will be at the very low rate of 1° or 2° a year [22, 23]. In the literature it was demonstrated that the expected spinal growth at the moment that the initial curve is diagnosed is of crucial importance for the further development of scoliosis [46]. In a recent study, we demonstrated that progression of an idiopathic scoliotic curve correlates with periods of moderate and rapid growth, measured on successive radiographs [97]. The variations in growth speed across individuals, as seen in our study, may explain the variations in expression of AIS, together with other factors such as the type of curve. Various theories exist about the biomechanical mechanism responsible for scoliosis progression during spinal growth. It was suggested that asymmetrical growth of the apical vertebral bodies due to chronic axial asymmetrical loading on the physes, according to the Hueter-Volkman law, may result in scoliosis progression [1, 62]. Stokes et al. quantified the relationship between the degree of a symmetrical loading and the degree of asymmetrical growth in a rat-tail model and confirmed that vertebral wedging results from asymmetric growth in the physes [86].

In our study, there was a strong correlation between the degree of apical vertebral deformation (wedging) and the degree of lateral deviation (Cobb angle), meaning that more vertebral deformation was found in the severe curves. However, we have not found a direct relation between curve progression and an increase in wedging in progressive scoliosis. Others have stressed the importance of the posterior musculo-ligamentous structures of the spinal column, which have a strong tendency to shorten. It was postulated that the tethering tendency of the musculo-ligamentous structures of the posterior compared to the rapid growth of the anterior spinal column will result in curve progression and the complex geometry of scoliosis [56, 71, 72, 84].

A failure of the supportive musculo-ligamentous structures and/or their neuromuscular control system for stabilising the spine may explain the occurrence of progression in AIS. Lack of feedback, inappropriate feedback, or faulty programming within the CNS, due to pathology, may be an important contributing factor in curve progression [18].

Currently, there is a growing interest in a two stage hypothesis: the natural history of AIS involves an initial stage in which a small curve develops due to a small defect in the neuromuscular control system and a second stage, during adolescent growth, in which the scoliotic curve is exacerbated by biomechanical factors, whereas neurological dysfunction may play a role in the extent of progression during normal growth [18, 82]. Secondary to the scoliosis, a force system arises which may be held responsible for the geometrical and morphological characteristics of adolescent idiopathic scoliosis [52, 96].

Summary

The aetiology of idiopathic scoliosis is unknown. There are many conflicting theories on the aetiology and pathogenesis of this condition. We propose that the most likely cause of idiopathic scoliosis is neuromuscular. Asymmetry of the transversospinalis muscles may produce enough lateral deviation and axial rotation to disturb the delicate balance of forces in the region, thus producing a scoliotic deformity. This asymmetry of the transversospinalis muscles may be produced by alteration of the motor drive arising at the spinal cord level, either from altered sensory input at the same level or from a central mechanism, and "the altered muscle pull" is the final common pathway for the production of a scoliotic curve.

Secondary structural changes will develop in the vertebrae and ribs. A biomechanical explanation for these intravertebral deformities can be found in an imbalance between the forces of growth of the anterior and posterior column. The demonstrated rib deformities suggest an adaptation to rotation and displacement forces imposed by the scoliotic spine.

We discussed the possibility that asymmetrical muscle pull results in asymmetrical growth, increasing the deformity on an iterative basis, providing an explanation for progression. The progression of idiopathic scoliosis dur-

ing the second growth period correlates mainly with the growth velocity, and is determined in rate and extent by the neurological dysfunction.

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