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LONGITUDINAL STUDY OF RIB AND SPINE GROWTH AND PROGRESSION IN IDIOPATHIC SCOLIOSIS

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Abstract

In order to examine relationships between growth and progression of adolescent idiopathic scoliosis (AIS), two studies were performed. In the first, stereo-radiographic measurements of rib length asymmetry in a group of patients with AIS (some progressive) were made. There was no evidence of rib length asymmetry increasing either with age or with increasing spinal curvature; rather the asymmetry often reduced with time in those patients with rib asymmetry initially. In the second (analytical) study the idea that growing vertebrae and discs become more wedged with growth because of asymmetric loading ('Heuter-Volkmann mechanism') was tested by formulating a linear relationship between stress and growth attenuation. Adolescent growth of 10% produced only 1% increase in wedging angle for initial wedge angles in the range of 1° to 10° per motion segment. These studies present evidence against both rib asymmetric growth and stress modulation of vertebral growth as responsible for scoliosis progression.

1. INTRODUCTION

Previously published findings on rib length asymmetry show that asymmetries occur in different curve types and that the magnitude of this asymmetry does not correlate with the magnitude of the scoliosis (Stokes et al., 1989). Normelli et al. (1985) also reported rib asymmetry in clinical series. An animal model showing scoliosis associated with rib growth asymmetry was reported by Agadir et al. (1988). The production of a small lateral spinal curvature, together with apical vertebral rotation in the transverse plane was demonstrated in a finite element model by Stokes and Laible (1990). Rib length asymmetries could be a manifestation of an overall growth asymmetry in the trunk, which may also appear as asymmetries of the upper arm, as found by Dangerfield & Burwell (1980). This work shows asymmetric lengths of the humeri in patients with right thoracic curves, and the magnitude of the asymmetries were very similar to those reported for the ribs.

There is much clinical and experimental evidence that growth is disturbed in patients with idiopathic scoliosis (Archer & Dickson, 1985; Drummond & Rogala, 1980; Gross et al. 1983; Nicolopoulos et al. 1985; Nordwall & Willner, 1975; Skogland & Miller, 1981; Tjobjorn et al. 1988; Veldhuizen et al. 1986; Willner, 1974, 1975). The regulation of growth which normally results in a skeleton of quite accurate symmetry can be disturbed by denervation (Dietz, 1989; King, 1961), by mechanical stress (Arkin & Katz, 1956; Gooding & Neuhauser, 1965; Hall-Craggs & Lawrence, 1969; Hert & Liskova, 1964; Heuter, 1862; Moreland, 1980; Porter, 1978; Strobino et al. 1952; Volkmann, 1862), by neuromuscular pathology (Gullickson

et al. 1950; Ratcliffe, 1959), altered blood flow (Treuta & Amato, 1960) and by injuries to the periosteum (Jenkins et al. 1975; Solá et al. 1963). The role of growth and growth asymmetry in the etiology and progression of idiopathic scoliosis remains unknown.

The growth of ribs, which are flat bones, is incompletely described, in contrast with substantial published information on the behavior of growth cartilages in long bones. Snellman (1973) reviewed literature on rib growth, but found "no comprehensive description", and some disagreement about the relative importance of endochondral ossification anteriorly, the epiphyseal growth plate between the head and the tuberculum, and apposition/resorbsion in the body (Barer & Jowsey, 1967; Epker & Frost, 1965). Snellman's measurements of rib growth in pigs showed that longitudinal growth at the costal cartilage was about four times greater than the epiphyseal growth. He examined how growth and remodelling was affected in a scoliosis model, but this did little to explain how mechanical factors influence the growth process.

Growth in the vertebral body occurs in the cartilage of the end plates (Dickson & Deacon, 1987). Growth in these cartilages is influenced by biomechanical loading. Roaf (1960) hypothesized that a "vicious circle" develops in the progression of scoliosis deformity. The concave side of a curve is subjected to greater compression load and this decelerates growth of the vertebral bodies compared with the convex side, according to the principles described by Heuter (1862) and Volkmann (1862). Roaf (1963) developed a rationale for the placement of staples on the vertebral bodies for treatment of scoliosis with varying degrees of kyphosis and lordosis. However, there are several unanswered questions about the scientific basis for this theory of scoliosis progression. For instance, the normal spinal curvatures (kyphosis and lordosis) in the sagittal plane are of similar magnitude to the curvature of scoliosis which is at risk to progress. Why do the normal kyphosis and lordosis not progress into hyperkyphosis and hyperlordosis?

Much of what is known about the interaction between mechanical forces and bone metabolism relates to the remodeling of mature bone, as opposed to the modeling of growing bone (Carter, 1987). One should distinguish between the effects of compression on longitudinal growth, torsion on rotational modeling, and bending on angular development. Strobino et al. (1952) placed springs across the tibial growth plates of calves, and it appeared that growth was relatively insensitive to compression forces of the order of body weight. Arkin and Katz (1956) reported a more sensitive response in growing rabbits, without evidence of a threshold level of load. They found elongation of limbs which were immobilized and relieved of weight-bearing. Tension forces applied across the growth plate produce a small acceleration of growth (Porter, 1978). It appears that growth cartilage placed under tension to accelerate growth requires fracture before significant growth acceleration occurs (Kenwright et al. 1990). However, Gooding and Neuhauser (1965) reported "tall vertebrae" in patients with paralysis and also in younger patients who had been treated surgically with posterior fusion of the spine. They argued that the relative unloading of the spine produced increased longitudinal growth.

In order to investigate some of the questions surrounding scoliosis progression and growth, two parallel studies were conducted. In the first, the changes in lengths of ribs were measured in patients with scoliosis to test the hypothesis that asymmetric development of the spine during growth would be accompanied by asymmetric development of the rib cage. In the second study the 'Heuter-Volkmann' hypothesis of mechanically-mediated scoliosis progression was studied by means of a mathematical model which assumed a relationship between local growth rate and mechanical stress.

2. METHODS

This paper presents a clinical study and a simulation study of the progression of scoliosis.

2.1 Clinical Series, Studied Longitudinally

A group of patients with idiopathic scoliosis in whom asymmetries of the rib lengths was reported previously (Stokes et al., 1989) was studied longitudinally. 39 patients were stereo-radiographed on 2 or more clinic visits (average 2.7 visits, duration of follow-up mean 1.7 years, range 0.3 - 4.7 years). The arc lengths of the ribs at T2 through T11 were measured with an estimated precision of 1.5% (sd). The length asymmetry was expressed as a percentage of the length at that level and averaged over the T2 to T11 levels. There were 14 patients with single right thoracic curves (mean Cobb =30°), 10 single left lumbar curves (mean Cobb =32°) and 10 with double curves (mean Cobb =29°). Five patients fitted none of these classifications (mean Cobb =20°).

2.2 Mathematical model of asymmetric load and asymmetric growth

Based on the concept that a small scoliosis deformity can be initiated by asymmetric growth, but that subsequent progression of this small scoliosis continues in the absence of continued (primary) asymmetric growth, a preliminary model of such progression was tested. Spinal compression, produced by muscle forces, produces lateral bending moments in the asymmetric spine and this is often invoked as the cause of scoliosis progression, based on the 'law' of Heuter (1862) and Volkmann (1862). This theory was tested quantitatively in

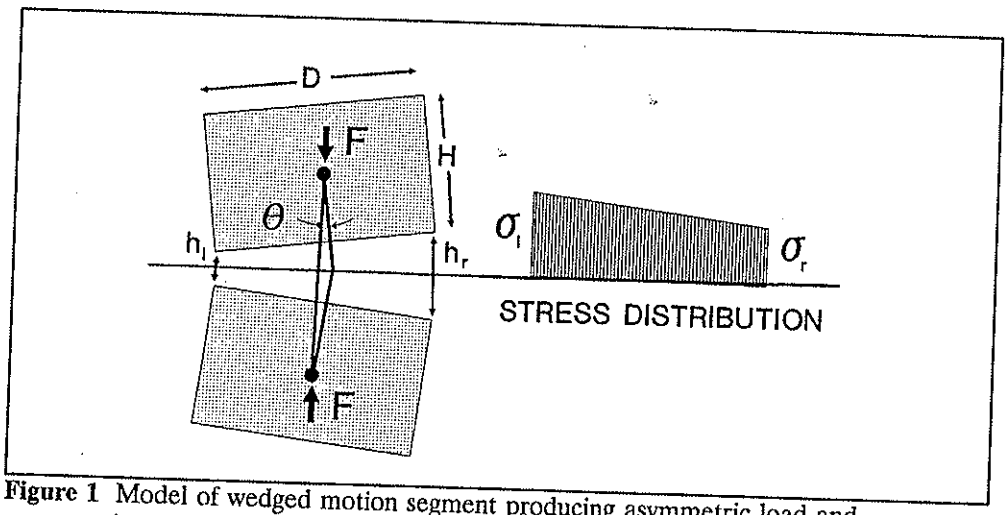

Figure 1 Model of wedged motion segment producing asymmetric load and asymmetric growth.

the model shown in Figure 1. In this model force is applied to a wedged motion segment. The stress distribution is assumed to be linearly distributed across the width of the endplates. The distribution of stress was calculated by assuming equilibrium of moments about the line joining the centers of the vertebral bodies.

It was then proposed that the growth strain ϵ is related to local stress σ and overall growth δG by the relationship

$$\epsilon = \delta G(1 + \beta\sigma) \quad \text{where } \beta \text{ is an unknown constant.}$$

By assuming an overall growth δG of 10%, reduced by 1% by the average stress acting in the motion segment ($\delta G \beta \sigma_a$), the unknowns F and ß could be eliminated from further consideration. The increase $\delta\theta$ in wedging angle as a result of growth was then found to be

$$\delta\theta = \frac{(\sigma_r h_r - \sigma_l h)}{(50D(\sigma_r + \sigma_l))} = \frac{(h_r - k h_l)}{50D(1+k)}$$

where k is a function of the geometry of the model.

3. RESULTS

3.1 Clinical Series

Based on consistent trends over several visits, or a difference greater than measurement precision in the case of 2 visits only, 22 patients were classified as having asymmetry which reduced over time, in 10 patients the asymmetry increased, and there was no change in 7. The mean loss of rib asymmetry was 1.6% of rib length, from an average initial asymmetry of 4.8%. Although 15 of the 29 patients were undergoing brace treatment, 15 showed evidence of progression of scoliosis during the period of study, 7 improved and there was no change in 17. Measurable spine growth occurred in all patients under the age of 16 years. In 19 of the 38 patients the direction of change of rib asymmetry corresponded to the direction of change in the Cobb angle, which is the level of agreement expected by chance. In 7 of 15 progressing patients rib length asymmetry increased, and in 8 of these it decreased. Therefore, change in rib length did not correlate with change in spinal deformity.

3.2 Modelling study

This model failed to progress, in that the increase in 'wedging' was always two orders of magnitude less than the initial angulation. With an initial angulation of 1° in a motion segment, the increase in angle ($\delta\theta$) was 0.01° after a 10% overall growth. Increasing the initial angulation to 10° only produced an additional angulation of 0.1° with growth. Increasing the load on the spine or increasing the sensitivity of growth to compressive stress simply decelerated the longitudinal spinal growth in the model, with minimal increase in the frontal plane curvature.

4. DISCUSSION

These findings contradict the idea that progression of scoliosis results from asymmetric growth of ribs although an original rib asymmetry might have initiated the scoliosis. The alternative idea that asymmetric loading of the spine produces asymmetric growth and progression of an established deformity was studied in a mathematical model in which bone

growth rate of vertebrae was assumed to be linearly related to the compressive stress across the vertebral body. This model failed to demonstrate progression. In the absence of published data giving a quantitative relationship between stress and growth rate, this linear relationship, without a threshold of response was used.

There is a lack of information about how mechanical factors interact with growth of the immature skeleton (as opposed to some work on ossification and an extensive literature and much active research on mechanical influences on the remodelling of mature bone (Carter). Frost (1990) draws attention to the apparently inconsistent response of bone (as opposed to growth cartilage) to a mechanical compression. On one hand, a child with a malunion of a long bone which has resulted in a marked curvature will gradually remodel and straighten the diaphysis by a process through which tissue on the concave (compression) experiences apposition of bone, whereas the tension side experiences resorption. Conversely, the alignment of teeth can be altered by means of orthodontic braces in such a way that compressed bone resorbs while bone on the opposite side of the tooth socket (the tension side) advances. Clearly, there are complex processes underlying the modeling of immature bone. Biewener et al. (1986) found that ambient strain levels with ingrowing bones remain quite constant during the period of growth, but there were variations between different sites, indicating an underlying genetic control. Hall-Craggs & Lawrence (1969) rabbits, which had been stapled at one growth cartilage, produced accelerated growth at the growth cartilage at the other end of the bone. Cook et al. (1983) used a mathematical model to study progression of tibia vara. They found that for angulations greater than 10 degrees, the growth cartilage on the lateral side would experience tensile forces. This degree of angulation across several vertebrae would add up to a scoliosis with a large Cobb angle. From a mechanical point of view, the difference in the force transmission between the convex and concave side of an angular deformity must increase as the angulation of the deformity increases, and this may produce an effective threshold when rapid progression is to be expected. Development of the rotational deformity in the spine complicates this process further. The growth plates of long bones are also responsive to torsional loads (Arkin & Katz, Moreland), but the origin of such forces in the spine during scoliosis progression is not clear. As yet, the clinical observation (Lonstein & Carlson 1984) that the risk of progression of scoliosis is a combination both of curve magnitude and amount of residual growth cannot be explained quantitatively by mechanical principles.

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