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A CONTRIBUTION TO THE DISCUSSION OF THE BIOLOGICAL CAUSE OF IDIOPATHIC SCOLIOSIS

By

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Idiopathic scoliosis had been thoroughly studied by a number of clinical workers (*Harrenstein, James et al., Nicoladoni, Roaf, Scott & Morgan, Lindermann, Rathke, Jentschura & Mau*). Included in this definition are those scolioses which appear during the growing years and where no direct cause can be demonstrated. The scoliosis can begin during the first three years of life or later, the curvatures often increase to severe deformities, but in some cases there is a spontaneous return to normal. The progressive development is shown in Fig. 1 which is derived from the publication of *James*. The curvatures appear most often in the lower part of the thoracic spine followed by compensatory curvatures, but there are also other kinds. Various stabilising operations have been tried to prevent progression but the results have often been unsuccessful. Since the biological cause of idiopathic scoliosis is unknown, it has not been possible to carry out an adequate causal treatment.

A number of points are presented in the present paper which are worthy of consideration in attempts to find the cause of idiopathic scoliosis.

The normal growth of a vertebra.

A vertebra consists both anatomically and biologically of two different parts: body and arch.

The vertebral body is analogous to the bones of the extremities, increasing continuously during the whole period of growth in length, breadth and depth. This growth ends first at 18–20 years of age.

The vertebral arch on the other hand is analogous to the cranium. It grows at the same rate as the cranium. The part of the arch which

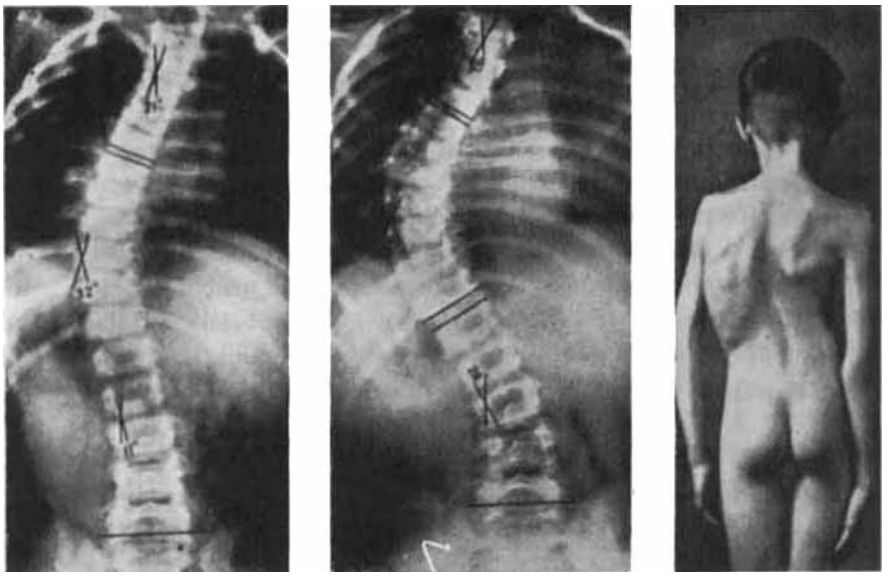


Fig. 1.
Idiopathic scoliosis in a boy at 2 and 8 years of age.
(Taken from James' publication).

constitutes the spinal canal and surrounds the spinal medulla is thus analogous to the cranium which surrounds the brain. Apart from the various processes of the arch, which continue their growth during the development of the individual, the ring around the spinal canal follows at quite a different pace. As early as 1–2 years of age, both halves of the arch have become unified in bone. As a result of this, the continued growth, leading to increase in breadth of the spinal canal, occurs exclusively in the neurocentral junction. This growth occurs rapidly during the first 3–5 years of life and then more slowly until 10 years of age when it is complete (*Knutsson*). Thus the ring of the arch has attained its final size by about 10 years of age.

There is a risk of conflict in this double development of a vertebra. According to the literature, growth in the neurocentral junction is complete between 3 and 8 years of age. This wide time limit may seem strange. This can, however, be explained if completion occurs at different ages in different parts of the vertebral column. No systematic anatomical study of the time of completion in different vertebrae has been carried out and a roentgenological assessment is impossible in practice. I have examined two publications in which it is possible to decide from

the films whether the join is complete or incomplete. These show that in one child, 6½ years of age, the join was incomplete in vertebra Th2, Th4 and Th8, but, on the other hand complete in Th10, L1 and L2 (*Nicoladoni*). In another child of 3½ years the join was incomplete in vertebrae Th3 and Th7 but complete in C4 and L4 (*Engelmann*). These few observations are in favour of completion occurring at different times in different parts of the vertebral column and furthermore that it occurs last in the thoracic spine. Thus the harmonious development of the vertebral column even up to 8 years of age probably depends on the paired neuro-central junctions growing absolutely similarly and being complete at the same time. On the other hand, if growth is not uniform, an asymmetry between arch and vertebral body is to be expected.

Growth conflict in paired neuro-central junctions.

After the age of 1–2 years, the symmetrical shape of the arch is completely dependent on growth in the neuro-central junction. If this takes place at a different rate and leads to a premature synostosis on one side, the vertebral body is twisted out of its harmonious relationship with the vertebrae above and below. Such a unilateral retardation in growth leads to the distance between the joint processes and body of the vertebra being continually shorter during the time of growth than on the opposite side. The conditions required for an normal development of the vertebral column are that growth in the neuro-central junctions occurs at the same rate on both sides (Fig. 2).

This problem has been dealt with by *Nicoladoni*. In a series of dead children with infantile scoliosis aged 1, 6, 6½ and 7 years, he has studied the neuro-central junction. This was without exception completely open on the concave part of the scoliotic curvature and completely closed on its convex part, or more open and more closed respectively.

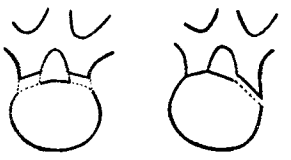
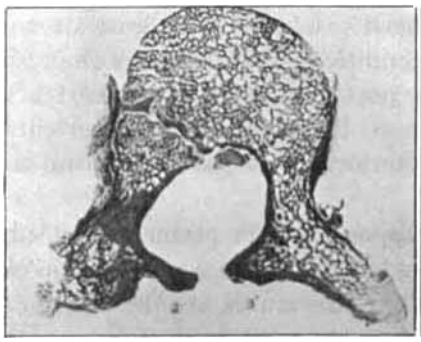


Fig. 2.
Diagram of the effect of unequal growth on the neuro-central junctions. The vertebral body rotated towards the side where premature synostosis occurs and thus producing scoliotic curvature with the convexity towards this side.



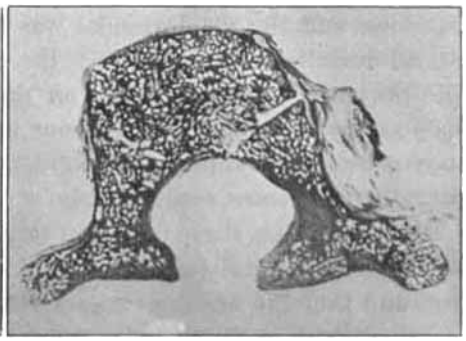


Fig. 3. *Fig. 4.*

Fig. 3. Th8 in a 6½ years old child with a scoliosis convex to the right. The neuro-central junction is completely open on the concave side of the curvature but fused with bone on the convex side. The vertebral body is rotated towards the convex side. (Taken from Nicoladoni's publication).

Fig. 4. Th2 in the same child as in Fig. 3, situated in the scoliotic curvature convex towards the left. The neuro-central junction is completely open on the concave side and almost closed on the convex side. The vertebral body is rotated towards the convex side. (Taken from Nicoladoni's publication).

Thus growth was more rapid on the concave side causing a prolongation of the peduncle which in its turn produced a rotation of the vertebral body towards the convex side.

In one of *Nicoladoni's* cases, the 8th thoracic vertebra, which was at the apex of the curvature convex to the right, showed a completely synostosed neuro-central junction on the convex side and one completely open on the concave side (Fig. 3). The vertebral body is rotated towards the convex side. This change must be ascribed to a retarted and premature growth in the neuro-central junction on the convex side. The compensatory curvature higher up with Th2 as the apex and with the convexity to the left, shows the opposite asymmetry in both neuro-central junctions (Fig. 4). If the change in Th8 is considered as primary, then the change in Th2 is the biological reaction by which the underlying curvature is compensated for.

Schede came to the conclusion that the characteristic change in the scoliotic vertebra can only be explained by an injury to the neuro-central junction. He has suggested rickets as a possible cause.

With the knowledge of the definitely completed growth of the arch at about 10 years of age, and the subsequently continuous growth in height of the vertebral body, it should be possible to explain the progress of the scoliosis. After the vertebral body has rotated in a lateral

direction and the displacement has been combined with lordosis and lateral deviation (*Sommerville*), the conditions of loading are changed. The pressure becomes greater on the posterior areas of the vertebral body surfaces than on the anterior areas. By this means, the vertebral body grows more rapidly in height anteriorly than posteriorly and the curvature increases continuously.

Because of this, there is reason to suppose that the premature closure of the neuro-central junction on one side can initiate a scoliotic curvature and that the adjacent compensatory curvatures are the biological reaction which in their turn produce unequal growth in the other neuro-central junctions.

One can ask oneself if there is any plausible explanation of why the differing growth in the paired neuro-central junctions appears particularly in the thoracic region. There is a reason to think that growth in the neuro-central junction in this part of the vertebral column continues longest and that closure here can be delayed until 8 years of age. This situation may provide a disposing cause.

CONCLUSION

Idiopathic scoliosis consists of rotation, lordosis and lateral deviation. There is evidence that rotation can be elicited by varying rates of growth in the paired neuro-central junctions (*Nicoladoni*). Growth in the neuro-central junction is complete at different times in different parts of the vertebral column, between 3 and 8 years. Some observations are are in favour of its continuing longer in the thoracic region than in other parts of the vertebral column. In discussing the origin of idiopathic scoliosis one may ask whether a retarded growth in one of the paired neuro-central junctions can initiate a scoliosis from the rotation of the vertebral body out of its harmonic relationship to the other vertebral bodies. Rotation occurs towards the side in which growth in the neuro-central junction is retarded and the scoliosis is convex on that side (*Nicoladoni*). From this there exists a theoretical possibility of inhibiting an increasing rotation during the period of growth by an epiphysiodesis in the neuro-central junction on the concave side of the curvature. In order for such an epiphysiodesis to be effective, it must be carried out at an early stage, before the age of 3–5 years. In such a way it is theoretically possible to counteract continued rotation, and thus the continued increase in the curvature, which depends on continued growth in height of the vertebral bodies, is inhibited.

Further research into this problem is required. For this purpose, an injury to a neuro-central junction could be produced which would lead to premature synotosis, to see if a scoliotic curvature would develop. There is no such isolated lesion published, without simultaneous lesions of growth zones of the vertebral bodies (*Pacher*).

SUMMARY

In spite of extensive clinical and experimental research, the biological cause of idiopathic scoliosis is still unknown. There is evidence for an unequal growth in the paired neuro-central junctions which can lead to rotation of the vertebral body out of its symmetrical relationship in the vertebral column. This seems to be one of the ways in which scoliosis can arise. The progress of the scoliosis which follows can probably be explained by the continued growth of the vertebral bodies. Further experimental research, concentrated on isolated neuro-central junctions, is required, to try to produce premature synostosis and to study the effect.

RESUME

Malgré des recherches cliniques et expérimentales très poussées, la cause biologique de la scoliose idiopathique est toujours inconnue. On a constaté une croissance inégale des jonctions neuro-centrales accouplées qui peut conduire à une rotation du corps vertébral hors de son rapport symétrique dans la colonne vertébrale. Cela semble être l'une des manières dont la scoliose peut se produire. Le progrès de la scoliose peut s'expliquer par la croissance continue des corps vertébraux. Il est nécessaire de poursuivre la recherche expérimentale en la concentrant sur des jonctions neuro-centrales isolées pour essayer de produire des synostoses prématurées et d'en étudier l'effet.

ZUSAMMENFASSUNG

Trotz ausgedehnter klinischer und experimenteller Forschung ist die biologische Ursache der idiopathischen Skoliose noch immer unbekannt. Es bestehen Anzeichen für ein ungleiches Wachstum der gepaarten neuro-centralen Verbindungen, das zu einer Herausdrehung des Wirbelkörpers aus seiner symetrischen Lage in der Wirbelsäule führen kann. Dies scheint eine der Möglichkeiten zu sein, die eine Skoliose hervorrufen kann. Das folgende Fortschreiten der Skoliose kann wahrscheinlich durch das fortgesetzte Wachstum des Wirbelkörpers

erklärt werden. Weitere Untersuchung, die ihre Aufmerksamheit besonders auf die neuro-centralen Verbindungen richten und versuchen sollte vorzeitige Synostosen hervorzubringen und deren Wirkung zu studieren, ist nötig.

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