## IDIOPATHIC SCOLIOSIS: ETIOPATHOGENISIS AND DYNAMIC CORRECTIVE BRACE TREATMENT PRINCIPLES

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This article is based on my experience over 22 years treating Children at the Sheffield Children's Hospital, Sheffield, UK and the work of Dr Charles Hilaire Rivard MD and Dr Christine Coillard MD at Ste Justine Hospital, Montreal, Canada with whom I have had the privilege to work with over the past six years.

Flexible Dynamic Corrective Bracing (SpineCor $\mathbb{R}^1$ ) is principally prescribed for Idiopathic Scoliosis patients with a Cobb angle between 15° and 50° and Risser sign 0 to 2. The brace is fitted on the patient in accordance to a sub-classification of the traditional SRS<sup>2</sup> definition of curve types. The brace is prescribed to be worn by the patients 20 hours per day until they have reached maturity, with radiological evaluations performed prior to and immediately following the fitting of the brace, and every 4 to 6 months thereafter.

Dynamic Corrective Bracing was designed, developed and tested for the treatment of idiopathic scoliosis.

The new therapeutic approach is based on a new concept upon the etiology and pathogenesis of idiopathic scoliosis. It is a pathology of the neuro-musculoskeletal system in growth and maturation. The cause is genetic and the pathogenesis involves a three-dimensional deformation of the spine, postural disorganisation, unsynchronised growth and particular movement patterns of the body.

In order to obtain an accurate diagnosis that would specify a particular class and subclass for the patient, the evaluation combines a clinical exam, radiological and postural evaluation.

A specific corrective movement is performed and the brace is applied according to specific curve classification instructions. The moderate tension in the elastic bands allows the repetition and amplification of the corrective movement as the child undertakes everyday activities. This results in progressive curve reduction. The brace is worn 20 hours out of 24, the four hours out of the brace must not be taken at once; usually the patient divides them into two breaks: morning and evening. Sports are to be encouraged and done while wearing the brace. To obtain a neuro-muscular integration of the new strategy of movement, the average duration of the treatment is 18 months. Due to the progressive changes, absence of external support during the treatment and intact muscles, there is no loss of correction after the brace discontinuation. Physical therapy is not a necessity with Dynamic Corrective Brace treatment as the brace itself may be considered physiotherapy 20 hours out of 24. However, to complement the action of the brace and reinforce the principles of the treatment, a curve specific physiotherapy program may be recommended. It is essential for the

physiotherapists to learn the treatment principles and understand this new treatment approach before attempting to treat patients.

In 1992, Dr Charles Hilaire Rivard MD, Professor of Orthopaedic Surgery at Ste Justine Hospital Montreal and the University of Montreal secured significant grant monies for scoliosis research. His team of sixty-five scientists was headed by Dr Christine Coillard a Paediatric Orthopaedic Surgeon from Paris, France specialising in the treatment of scoliosis. The objectives of the research team were to gain a better understanding of idiopathic scoliosis: it's etiopathogenesis, early diagnosis, progression factors and develop new and better treatments.

Dr Coillard and the team of researchers at Ste Justine Hospital knew right from the beginning the importance of understanding how and why idiopathic scoliosis progressed in order to treat it effectively. Previous treatments have all been impaired with poor studies and limited follow-up. Dr Coillard describes idiopathic scoliosis as a neuromuscular skeletal pathology since there is clear evidence of abnormality in all three areas in advanced cases. There are, however, to date no generally accepted publications detailing the precise causation and subsequent sequence of events in the disease process. Early on in the research (1), animal studies were carried out to gain better understanding of the evolution of scoliotic curves and the factors that determine progression.

The chicken was chosen as the ideal model to study scoliosis due to its high natural rate of scoliosis and its short growth period. Additionally, the natural rate of scoliosis in chickens could easily be increased to around 80% by performing a pinealectomy, preventing production of melatonin. The absence of melatonin produces a peak of somatatrophin secretion, which in turns regulates the "ready" growth plates. The growth of each vertebra via its four growth plates one anterior, one posterior and two laterals are never perfectly symmetrical. With normal hormonal control mechanisms in place and with growth within normal variables, any mild asymmetry is quickly compensated by growth of the contra lateral side. Such mild oscillation does not produce a deformity and is a part of normal development. If the hormonal control mechanisms are compromised and particularly when growth is fast, recuperation of the contra lateral side may not be full, creating vertebral deformity and subsequent scoliosis. The study of scoliotic curve development in chickens shows specific localised vertebral deformity at or around the apex of the major structural curve (Figures 1 & 2). These studies clearly show the bone deformity of the 3 dimensional and the initiating factor in scoliotic curve development.

Normal Chicken Spine



(Figure 1)

Chicken spine with mild scoliotic deformity



(Figure 2)

The initial localised deformity T7 being primary with the major deformity with secondary partially deformation of T6 & T8. This pattern of primary 3 dimensional deformities on one vertebra plus half of the vertebrae above and half of the one below is typical in both chicken and human idiopathic scoliosis curve development. The rest of the vertebrae are disorientated but not deformed. The localised bone deformity starts as a delay of ossification in the posterior-lateral part of one vertebra, which produces a tilt of the overlaying vertebra. The first curve to appear is the overlaying one as a consequence of the tilt and has limited potential to progress (compensatory curve). The second and the major curve is that containing the deformed vertebra. The major vertebral deformity will usually be located around the apex of the major curve but not always! The localised deformity is often under estimated when judged on x-ray.



When the bone deformity is large (Figure 3), there are significant limitations to conservative treatment. In such cases, curves will be very stiff and have a bad prognosis. Laying xrays (supine/prone) can be very helpful in determining the flexibility and therefore the extent of bone deformity in an individual case. Comparison of laying (supine/prone nonweight bearing) versus erect X-ray Cobb angles can be used to calculate the potential reducibility of a particular curve. This method has been found to be more reliable than the use of lateral bending films or clinical evaluations, which are subject to much greater intra user reliability.

(Figure 3)



(Figure 4)

(Figure 5)

The above images show the remarkable similarity between the chickens vertebra deformities (Figure 4) compared to the human vertebra CT images (Figure 5). Clearly, in both cases, the deformities are 3-dimensional demonstrating:

i) Lateral wedging

ii) Anterior/Posterior wedging

iii) Rotational deformity prescribing an arc from the spinus process through the vertebral body

These features of the vertebral deformation match perfectly with the 3-dimensional patterns seen in scoliotic spinal deformity. The characteristic spinal rotation seen in idiopathic scoliosis, originates from the localised rotational deformity in the primary vertebra; rotation in the rest of the spine is disorientation as a result of this. The anterior/posterior wedging results in modification of the sagital spinal profile typically seen in idiopathic scoliosis. Changes in kyphosis/lordosis and vertebral rotation give an indication as to the degree of bone deformity and therefore the risk of progression. These factors may not initially relate to frontal Cobb angle magnitude but provide evidence of high progression risk in an immature spine. Lateral wedging may be evidenced by intercostal asymmetry and rib vertebral angle differences and may also indicate high progression risk



(Figure 6)

Dr Coillard's concept of idiopathic scoliosis etiopathogenesis (10) is not new, Dr Hoffa proposed something similar in 1902, describing scoliosis as an apical vertebra posterior wall asymmetrical collapsing (Figure 6). Whilst it is clear that idiopathic scoliosis is a complex multifactoral neuromuscular skeletal disorder, we conclude from our research that growth disharmony resulting in localised vertebral deformity is the initiating factor as well as a significant but not the only progression factor.

The posture and movement in scoliotic patients is never normal. Part of the research project (2,3) involved studying the kinametrics of the spine and posture in scoliotic patients. The amplitude of movement in opposite directions for scoliotic patients is not surprisingly unbalanced (Figures 7 & 8).



AMPLITUDE OF MOVEMENTS

(Figure 8) Control Subject

Studies using Vicom Motion analysis (2,3) (Figure 8) reveal very specific postural abnormalities relating to each type of curve. Comparison of X-rays and postural analysis from hundreds of patients led to the development of a new scoliosis classification system taking into account not only the 3-dimensional aspect of each spinal deformity, but the global postural disorganisation. The true importance of this combined curve and postural classification will become clear later when the dynamic treatment concept is explained.



(Figure 9)

Despite the posture and movement of scoliotic patients being abnormal, not one of them feels crooked. This is because scoliosis develops gradually and the motor control centers have time to adapt to the information received from muscles and ligaments. The neuromuscular integration of the bad posture and movement strategy are reinforced over time creating one of the vicious circles of scoliosis (figure 9).



(Figure 10)

The muscles attached to the affected spine have their length; strength and direction altered preventing them from actively working to correct the progressing deformity (Figure 10).



(Figure 11)





Clearly in an advanced case there is bone deformity (Figures 11 & 12), however, localised vertebral bone deformity is present early on and is proposed to be the initialising factor in the development of all idiopathic scoliosis cases.

The genetic basis of vertebral deformation has been attributed to the homeobox genes (13,14,15). It is hypothesised by Dr Coillard that a genetic temporal fault in these genes results in the delayed ossification of either the lateral or anterior part of the vertebra and subsequently creates the initial vertebral deformity. Hormonal maturation, growth velocity and mechanical factors associated with gravity and deformity can also play a significant role in the progression of scoliosis.



(Figure 13)

In Dr Coillard's etiopathogenic concept (Figure 13), the initiating factor in idiopathic scoliosis is a genetic temporal fault. Whilst the specific location of the genetic fault is not known, there is strong evidence to suggest that it lies within the Hox Gene (13,14,15). In addition the control mechanism for growth plate regulation is not known but there is evidence to suggest that hormonal factors do influence progression of scoliotic curves (16).

The unsynchronised osseous growth resulting from a fault in the growth plate regulation system results in a functional unit deformation. The initial vertebral deformity is specifically confined to one vertebra plus half of the superior and half of the inferior vertebra. The boney deformation is 3-dimensional showing lateral wedging, anterior posterior wedging, rotational deformity of the body, spineous processes and transverse processes. These deformities can be evidenced by CT scans but are not easily seen on conventional X-rays until the deformity is extreme. Once the vertebral deformation is significant enough there is rupturing in the internal preloaded spine destabilising it. Once a curve is over 20<sup>o</sup> mechanical factors and resulting dysfunction of the neuromuscular system together with maturation can play a very significant role in rapidly progressing scoliotic curves.

Dynamic corrective bracing was developed around Dr Coillard's etiopathogenic concept (10) to minimise the progression factors and reverse the deformity. Dynamic Corrective Bracing has four modes of action.

i) Dynamic opening of curves (reductional Cobb angles): Changes in the compression and tensile loading of the vertebral growth plates can limit, stabilise or even reverse the vertebral deformity (10,11), one of the major progression factors in idiopathic scoliosis. Research (10, 11) has shown that dynamic reduction of Cobb angles (alternative compression and tension on

growth plates) during growth has a greater effect in arresting or reversing progression than static Cobb reduction.

ii) Normalisation of postural disorganisation by globally and dynamically over-correcting the posture.

iii) Neuromuscular re-education: A specific new movement strategy (corrective movement) is repeatedly performed by the patient through normal activities of daily living, whilst wearing the dynamic brace. This provides progressive improvement of posture and Cobb angles over time within the limits of the patient's level of skeletal maturing and deformity.

iv) Neuromuscular integration: Over time the new movement strategy becomes integrated into the brain overwriting the previous abnormal posture. The corrective movements resultant new posture and Cobb angles are maintained post bracing with no loss of correction over time. Long-term studies demonstrate extremely stabile results post dynamic corrective brace treatment two years and more post bracing (19).



(Figure 14)

Radiological Classification: Conventional classification of idiopathic scoliosis is based on a radiological evaluation in the P/A view and different types are identified according to the position of the apex without any consideration of the sagital view. This classification provides only partial information even though scoliosis is known as a 3-dimensional deformation of the spine associated with postural disorganisation. When comparing X-rays among patients classified as the same, several differences in the morphological aspects of the curvature and other characteristics may be noted. Clinically, the differences in posture for these patients are obvious enough to reconsider if they are indeed of the same type of scoliosis. This has led to the development of subclasses of the conventional classification of scoliosis patients. A classification that reflects the **3-dimensional deformation** of the spine and the **associated postural disorganisation** is therefore essential. Observation of specific parameters, by combining frontal and sagital X-rays, in order to get the maximum 3D information is involved.

- Tilt / rotation / version for each vertebra.
- Tilt / rotation / version for the shoulder girdle / thorax / pelvic girdle.
- P/A and lateral shift
- Modifications in the sagittal plane of the thoracic, thoracolumbar and lumbar segments.
- Anteversion / retroversion / antepulsion / retropulsion.

Corrective Movement Principle(6): For each of the new scoliosis classifications based on both 3-dimensional radiological and postural data a specific "corrective movement" has been developed. "Corrective movements" are global postural changes that give optimal Cobb reduction for each curve classification. Each global corrective movement is made up of one or more of the principal movements (Figure 15)



(Figure 15)

The corrective movements in general over-correct the curve specific abnormal postural features, in order to correct the posture, which in turn correct the spinal deformation. The following images (Figures 16-23) illustrate in the case of a Right Thoracic Type 1 curve how a global corrective movement strategy can affect the spinal deformity.



In summary, dynamic corrective brace treatment of idiopathic scoliosis utilises radically different principles to those of conventional rigid bracing. Effective utilisation of dynamic corrective bracing almost demands you forget conventional theory before it is possible to fully understand this new treatment approach.

To really change the natural progression of idiopathic scoliosis, it is essential to reduce the curvature enough to eliminate the negative impact of abnormal biomechanics and growth. In such circumstances, it is possible to achieve a complete or almost complete correction of moderate curves, if the treatment is started before the main growth spurt (before Risser 1 and menarche). In curves over 30 degrees of Cobb angle, or when the treatment is started during or after the main growth spurt, the goal of the treatment is a stabilisation of the deformity. Therapeutic success is possible in more than 80% of cases. Reference reducibility can be calculated as early as at 3-4 months of treatment, which is useful in defining the prognosis. However, for individual prognosis, the impact of the severity of the bone deformation, pattern of the growth and compliance must be considered.

Initial bracing results and even those well into treatment are not directly comparable to rigid bracing. True comparisons of dynamic versus rigid bracing may only be made post treatment, the most dramatic differences being noted at 2 years plus post treatment. Long-term treatment results (19) show benefits over rigid bracing with true stable curve reductions maintained five years plus post-treatment.

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#### Footnotes:

<sup>1</sup>SpineCor® is patented worldwide and is the <u>only</u> Dynamic Corrective Brace for the treatment of idiopathic scoliosis utilizing Dr Coillard's Radiological Classification and Corrective Movement Principle. The SpineCor® Dynamic Corrective Brace is manufactured exclusively by; The SpineCorporation Limited, Chesterfield, United Kingdom S41 9RN.

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