ETIOLOGY OF IDIOPATHIC SCOLIOSIS: AN UNSYNCHRONIZED GROWTH OR WHY A SYSTEM CAN TURN CHAOTIC

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ABSTRACT

An unsynchronised bone growth, temporal fault of genetic origin, focused at a given spinal segment, prompts the formation, a priori transient, or discordant vertebrae that modify the dynamic internal equilibrium of the spine. In rapid growth and maturation situations, this leads to the dysfunction of the neuro-musculo-skeletal and scoliotic deformation.

INTRODUCTION

Scoliosis has been recognised for centuries. However, in most cases it is considered ≪idiopathic≫. Nevertheless, many etiopathogenic hypotheses have been advanced, but they lack conclusive proof. Thus, no definite aetiology for idiopathic scoliosis is certain.

All the hypotheses surely contain a certain element of truth, but two key obstacles seem to hinder any clear progress in this field. First, research and reflections on this ides have targeted one element of the neuro-musculo-skeletal system (NMSS), or some component of it likely to interact with the NMSS – such as bones, muscles, nerves, hormones or neurotransmitters… without really taking into account the growth and development of the entire system itself. This approach is well guided but inadequate, to the extent the NMSS is an entity in itself of which the growth and function obviously rely on the integrity and proper functioning of each of its individual elements, because it is an integrated system. The second problem is to presume that visible spinal deformity necessarily depends on a relatively recent or original lesion of one of the NMSS components.

However, there is another possibility that has never really been considered. This is that none of the system's components are initially injured, but there is a <phase difference>, a temporal defect that can possibly deviate the system from its growth and original function. Let's theorise about such a temporal phase difference within the spinal column itself caused by asynchronised vertebral growth and maturation. Let's also analyse the consequences using our current knowledge, considering the NMSS as an integrated system. The temporal shift translates itself as a theoretically temporary disruption of the vertebrae's shape, itself being a spatial shift. The cohesion between each NMSS element is faulty; therefore, the NMSS is progressively dragged into a self-maintained dysfunction or a chaotic functioning. This NMSS chaotic functioning can perpetuate itself or not. All depending on the context in which it happens, permitting or not the NMSS to attain a new equilibrium. If this equilibrium is attained, the initial disruption can go more or less unnoticed. If not, the scoliotic deformation appears and worsens as long as the elements of the cause and context are not modified. This hypothesis refers to a scientifically well-known fact being that minor initial differences can lead to considerable final differences. This is called the <sensitive dependence to initial conditions>.

Three questions must be raised to understand this approach:

- Why is the worsening of the scoliosis closely related to maturation and to rapid bursts of growth?
- What inferences may be drawn from the family or hereditary nature of this pathology?
- What is the relationship between worsening of the curvature and the spinal function?

Obviously, the answers to these questions are not simple. All aspects of the answer must be considered to organise the thought process and put forward some conclusions. It is based on data found in old or recent literature. Only the thought process is original and innovative. It must be noted that the basic data used are scientifically recognised. Only a few cautious extrapolations are occasionally made. The data employed comes from a variety of fields including: embryology, anthropology, genetics, endocrinology, biomechanics and neurosciences. The logic is based on principles of Chaos Theory – now commonly applied in many fields.

EMBRYOLOGICAL DATA

The formation, organisation and functioning of various organs and tissues must be studies to understand the growing and the adult organism. Among other things, such a study provides a fabulous overview of the child's development. Progress over the past few years in the field of genetics has shown the importance of the genetic program. This program determines the development

mode and the type of vital functions that occur in each individual. The program is just as unique as a fingerprint – although the way it expresses itself may come under various degrees of environmental influence. It is very important to realise that embryological development is the first way in which this program asserts itself but not the last.

A general review of the spinal column embryology will not be done here. But there exists, important information that has been ignored or overlooked in the literature. This information can be grouped into three points:

- Segmentation of the vertebra
- The temporospatial pattern of development, and
- Cranio-caudal vertebral segmentation

1. SEGMENTATION OF THE VERTEBRA

Embryologically, the cervical, thoracic and lumbar vertebrae – plus the first few sacral vertebrae – are made up of four portions. The first portion includes the anterior part of the vertebral corpus in front of the future neurocentral cartilage. This portion is very likely subject to the notochord influence.

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Two other parts are lateral – one to the right, the other to the left – behind the neurocentral cartilage, with the exception of the spinous process. These paired, theoretically symmetrical, entities are under the prevailing influence of the peripheral and autonomous nervous systems. As such, they are more particularly exposed to environmental influences that shall be discussed later. Between these three anterior and lateral portions, and covering them cranially and caudally, a three-dimensional growth structure appears. It corresponds to a <layer> of growth cartilage that covers top and bottom of the entire vertebral corpus and is morphologically linked to the neurocentral cartilage. Accordingly, this three-dimensional growth cartilage that will ensure the volumetric expansion of the vertebral corpus and in fact the length of the pedicle, is a morphological, but an unsynchronised functional entity. It is obvious that cranio-caudal growth differentiates itself through time from <horizontal> growth. This reveals a vertebra, depending on the period of observation that is higher than massive, or vice versa. Moreover, depending on whether the vertebra in question will become a lumbar or a thoracic vertebrae, the same growth structure must permit to ensure different resultant massivity – an thus a vertical-horizontal differential growth.

The point must be emphasised that these first three portions have the same mesodermal and somatic origins, but their evolution and control are quite different. The close relationship between cells originating from the neural crest and cells making up the lateral portions should be noted.

The fourth portion is represented from the spinous process originating in meso-ectodermal contact and closely linked to the embryo closure process. In the eitopathology of scoliosis, it presents little interest in itself, but maintains its place in the patter of spinal column development.

2. THE TEMPOROSPATIAL PATTERN OF SPINAL COLUMN DEVELOPMENT

One of the best ways of discovering and realising the existence of a temporo-spatial pattern is to study the appearance of vertebral ossification in the cartilaginous embryo. Whatever vertebrate is studied, it is clear that the progression of vertebral ossification occurs independently for each of the four previously described portions in a sequence not cranio-caudal. Naturally, differences may appear depending on the vertebrate studies, given, for example, the final number of vertebrae. However, if we refer to somites rather than formed vertebrae, it is more than likely that the temporospatial pattern of ossification is exactly the same. This is because it is quite certainly dependent on ancestral segmentation, found particularly in the homeobox genes' organisation.

In the anterior position, ossification begins in a group of three to five vertebrae bridging the thoracolumbar junction. The progression occurs cranially and caudally at different paces and by groups of an average four vertebrae. The manner in which ossification points appear by group is still unclear, although there are two main hypotheses. One is the appearance of ossification points, proceeding from one vertebra to another with temporal pauses defining the groups. The other is, it involves in reality ossification activity within the cartilaginous organisation of a group genetically predefined in space. This phenomenon occurs over a short period of time, at an irregular rate, making its observation harder. During this period, and often starting earlier in males, ossification of the lateral portions begins in the cervico-thoracic and cranio-cervical junctions by groups of two and then four vertebrae. It progresses bi-directionally at different paces. It is worth noting that in animals it is not rare for an ossification group of a few right or left lateral portions to precede controlateral portions for a certain period of time and then to subsequently resynchronise themselves. This phenomenon is termed ≪UNSYNCHRONISED GROWTH≫. For a variable period of time and with respect to a variable number or ≪space ≫ of vertebra, a TEMPOROSPATIAL lag occurs. This phenomenon is temporal, but its immediate consequence is a spatial disorder of the three-dimensional symmetry. The consequences of this unsynchronised development in the embryonic context are most often minimal or non-existent as the abnormality is short-term, transient, without vertebral volume alteration and occurs in a positive environment in terms of gravity, constraints, movement, etc. But the consequences can be quite different if this phenomenon recurs repeatedly over a longer period of time, altering the volume of one or more lateral portions and in an unfavourable context.

The ossification of the fourth portion occurs last, for once following apparently a cranio-caudal progression. This means there is an important time lag between ossification of the diametrically opposed anterior and posterior portions. In the study of growth what impact this lag can have on sagittal curvatures of the spinal column can be seen.

3. CRANIO-CAUDAL SEGMENTATION

Observation of different phenomena of embryological growth and maturation of the spinal column illustrates the lack of homogeneity of this structure's cellular activity. This activity is not subject to the laws of chance. Depending on the space-time factor considered, some regions are more active than others and follow each other in a clearly predetermined reproducible order that is not purely cranio-caudal. There is a temporospatial growth pattern and vertebral maturation that takes particular account of the vulnerability of certain areas to leisional agents intervening at different points in time. Over this same period, other organs, such as the heart or kidney, for example, can be involved, creating well-recognised lesional associations. But when a disorder is not due to an outside agent but to a disturbance of the very framework of the structure itself – as is probably the case with unsynchronised development – only the concerned structure is afflicted to various degrees and levels based on the type and extend of the pattern disturbance. If the abnormality involved in the unsynchronised development is a spatial one, the problem is temporal and thus fleeting. If there are any consequences, they can only be observed after the fact. Otherwise, the disturbance will go unnoticed unless it is recurrent and is specifically sought.

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Unknowns remain in the spinal column's temporo- spatial pattern development. However, we already can say that, generally, the thoracolumbar junction, for example, develops much sooner than the upper thoracic region, which matures late and slowly. The corollary of this aspect, for example, is that any abnormality of the upper thoracic region has a potentially greater impact than the same abnormality appearing in the thoracolumbar or lumbar region. Thus, I find it much more logical to consider all spinal pathology such as scoliosis not in relation to anatomical segmentation, but in relation to the pattern of development of the spinal column. This allows a definition of the different regions based on ≪functional≫ segmentation. Such segmentation is inevitably linked to the genetic program and adds the needed temporal dimension to the understanding of scoliosis.

The embryology of the spinal column illustrates the existence of a temporospatial development that does not follow a cranio-caudal order and defines functional segments distinct of morphological anatomical segments.

The spinal column, incidentally, ossifies after the skeleton as a whole. Except for the skull, which also has its own ossification pattern, ossification begins with the limbs, following with the pelvis, continuing in the rib cage and concluding in the spinal column. It follows a periphero-central progression. It shall be seen that the growth and maturation of the child's skeleton follow this same sequence.

It should be noted that the growth and maturation of the musculo-ligamentary system, the neurological system and the hormonal system occur in parallel with each other and inevitably interfere with the growth and maturation of the skeletal system.

ANTHROPOLOGY AND THE STUDY OF GROWTH

Longitudinal studies of a child's growth are very length, difficult and rare – but are quite interesting. They are generally not aimed at studying skeletal growth or morphological maturation in a whole. It must therefore combine various studies to arrive at the global vision that is interesting.

What stands out is that, outside the extraordinary longitudinal and volumetric intrauterine growth, there are two major periods of growth and one of maturation. The first such growth period occurs in the two or three first years of life. The second covers preadolescence, intermingled with maturation of the adolescent whose morphology is suddenly perturbed. Each period includes a number of (probably two or three) growth spurts during which the growth rate in the preadolescent accelerates and can achieve 7 to 10mm per month in height. Outside these spurts, height increases about 5mm per month, to return to 2 to 3mm per month outside periods of rapid growth. As for the skeleton, longitudinal growth prevails during the first period of growth. This is also true during the second period, but it is immediately followed by a volumetric increase, particularly in girls whose pelvis expands, whose chest becomes larger and whose bones become more massive. The slim and willowy preadolescent girl becomes a woman. The same process is also true with boys, but the proportions are quite different. During these two periods of rapid growth, idiopathic scoliosis worsens most, particularly among girls. However, after maturity, reversing the condition is unlikely, contrary to the first period of growth.

It is also important to note the change in ratio of different body segments during these growth spurts that renders a pattern of temporospatial growth. Very generally, each spurt starts by affecting the limbs, then the pelvis, next the chest and finally the spinal column. This order resembles very closely the order in which embryological ossification appears. It is not a very broad leap of logic to imagine the pre-established genetic program is the same. While an unsynchronised development may occur during the prenatal period, why would they not recur after birth? The context is quite different and the consequences more likely.

One other fact is clearly detectable, particularly during the rapid growth period of preadolescence. It involves variations in sagittal spinal column curvatures. These curvatures, which are notable at the start of the rapid growth period, gradually and alternately wane – lumbar lordosis and then thoracic kyphosis – to gradually reappear at the end of the same period. There is as yet no satisfactory explanation for this odd phenomenon, but we can easily understand it if we consider the previously described phase difference in vertebral ossification. If the growth of the anterior part of the vertebral corpus precedes that of the posterior arc, the curves vary as described. We should note that, during the period in which curvatures diminish, conditions of static and dynamic equilibrium of the spinal column necessarily vary.

Minor growth disorders can be easily observed. This is the case with small differences in limb size. They are very often temporary concerning the lower limbs but more often definitive when it comes to the upper limbs, lacking a clear relationship with the dominant limb. Also, minor asymmetries of the pelvis, sometimes oblique, appear as well as those of the thorax. In short, 30 to 40% of the population is not perfectly symmetrical – either temporarily or permanently. Depending on the severity of these skeletal asymmetries and their evolutive or non-evolutive nature, they will be considered either pathologies or deviations from normality. Obviously, the spinal column is a median and unique structure. But each unit that makes it up – the vertebra – must not be considered an entity unto itself. Rather, each such unit should be seen as being composed of four parts, two of which are not medians and which make up a generally symmetrical pair. However, as other permanent or temporary asymmetries of varying significance exist in other structural pairs of the skeleton, why should we not also find some in the vertebra? Because of the functional segmentation of the spinal column, these asymmetries could only concern two to five consecutive vertebrae resulting necessarily in a regional disturbance. Even if it seems quite unlikely that two adjoining regions could be affected, two or

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three non-contiguous regions could be subject to the same kind of disturbance, such as vertebral congenital deformities.

It should be noted that patients suffering from idiopathic scoliosis are mostly ectomorph, which implies that the mesodermal drifts are less developed and thus the skeleton is less massive.

GENETICS

Research advances of the past few years have highlighted, among other things, two types of results that are the most interesting.

The first concerns homeobox genes. These « ancestral ≫ genes are relatively constant in all vertebrates. At a very early stage, they define the future, function and polarity of cells and are thus critical to the organisation and function of vertebrate component structures. These genes come into action according to a well-defined and very likely immutable temporospatial programme. Whether or not they fade during the embryological period, the imprinted temporospatial order persists. Moreover, a study of these genes reveals a segmentation of the body still inadequately understood in humans, but which suggests there is a certain autonomy of each segment and the transition between two segments is sometimes less clear than assumed. This would cause harmful abnormalities of the junction. These transitional zones do not necessarily seem to correspond – at least in terms of the spinal column – with anatomical junctions, but rather the segmentation that appears during vertebral ossification. In other words, it appears important to:

- Consider that the observed temporospatial sequence of development and growth observed may depend on a set genetic programme the human species shares with other species; and
- Imaging that there is a "functional" segmentation more apt at explaining disturbances of a median structure such as the spinal column. This genetic segmentation, in the present state of our knowledge, seems principally related to the homeobox genes of type Hox.

The Hox genes represent a family of homeobox genes particularly implicated in the development of the vertebral column. Each one of these genes is responsible for at least two somites, a vertebra plus half of the immediately superior and inferior adjacent vertebra. This corresponds to the anatomical observations of major vertebral deformations localised on a segment superposed in the primary scoliotic curves. There is only one step to take to suppose that there exists a tight line between a genetic segment and the pathological curve centred here.

On the other hand, it seems that certain species, like homosapiens, are particularly predisposed to asymmetry. Certain Homeobox genes express themselves to the right and to the left, or inversely, provoking a temporary asymmetric development of paired structures. This corresponds well with observations of asymmetrical ossification of the lateral aspects of the same vertebra during the embryology of the vertebral column, or the asynchronous growth of the lower limbs frequently observed n the young. This asynchronisation normally not "prejudiced", could perfectly become, following the context of hormonal impregnation, the period of growth and the biomechanical status, transforming an apparent non-significant temporal anomaly, into a permanent deformation definitively fixed by the phenomenon's of maturation at the end of growth.

It is easy to imagine that the biomechanical factors could become predominating when the scoliotic deformation is severe, but for a long time with little influence. Interestingly, only the vertebra concerned with the scoliosis, could be affected, and not all of the vertebra. Incidentally, only vertebrates are affected by scoliosis, but to our knowledge, not all vertebrates. Quadrupeds seem relatively free of it, however, up to 30% of fowls, such as chickens and ducks, spontaneously suffer from scoliosis – it is quite tempting to relate this to being pseudo-bipeds. But certain fish are also afflicted! Under these circumstances, it becomes more reasonable to think that the species can be a supportive medium – particularly certain species – but not determinant, other factors being necessarily present.

While many more years of research are required before understanding the exact role of homeobox genes, this promising approach brings hope for better understanding of certain pathologies linked to development.

Other results are also of note and concern the processes of development, maturation and healing. The processes of maturation and healing parallel that of development, but at a different time space. This is true, for example, with healing following a fracture. This process parallels the embryonic development framework in repairing the injured bone. The speed of the process matches the youth of the patient. This is another example of the persistence of original frameworks in constantly repeated processes identical to themselves. It should be noted that the concept of the biological clock determining individual life cycles comes into play at this stage. These cycles, modulating the genetic expression, are a function of age and environmental conditions at a given time.

Therefore, if we are able to determine the genetic mechanisms that determine the temporospatial order of the spinal column's ossification; for example, a much more rapid definition could be made for the pattern of growth for this structure and truly understand the unquestionable relationship between scoliosis and growth.

For the moment, emphasis must be placed on the importance of genetic programming which defines the spinal column's bone development along with the hormonal, neurological and muscular development that have an indirect major impact on this bone development.

ENDOCRINOLOGY

In this section, the hormonal mechanisms that prevail during bone growth and remodelling shall not be discussed. Rather,

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the impact of a defined hormonal environment on the growth of the spinal column shall be discussed.

First of all, emphasis is placed on the "biological clock" role of the pineal gland or epiphysis cerebri. This intracerebral gland, embryologically linked to the cells of the neural crest, secretes melatonin, among other things. Genetically programmed, the pineal glad is also able to react to any kinds of information coming from the environment (temperature, vibration, light…). This most certainly shapes and modifies its functioning. Its main secretion, melatonin, regulates secretion of paracrine or endocrine hormones and enzymes according to complicated and intricate cycles. It acts in a variable manner and can be inhibitory or stimulating. For example, a drop in peripheral melatonin levels around the time of adolescence is accompanied by a rise in circulating growth hormones whose impact on bone growth is known. What is less well known is the impact of melatonin on the chondrocyte growth plates. But, whatever the situation, it seems that, depending on the chondrocyte's own state of maturity, it may or may not be able to react to changes in its biochemical environment that are linked to changes in the hormonal environment. Thus, even if the biological clock is perfectly set and permits adequate hormonal modifications, the meant-to-be receptive cells must be receptive in a synchronised manner. Maturation of the tissues is genetically dependent. With the genetic programme having a temporospatial aspect, it is not difficult to imagine that, for the set of vertebrae to react to changes in the hormonal environment, the new conditions must persist long enough and the set of chondrocytes must be able to respond in a synchronised manner to permit harmonious growth of all vertebrae. Any disturbance of the rhythm results in desynchronisation and a relatively focused lack of cohesion for a varying period of time. This can cause discordant growth of the vertebra. The resulting deformity is theoretically temporary, but it can become permanent depending on the context. If the unsynchronised development is of genetic origin, we can circumvent the problem, theoretically, by delaying the effects of the drop in melatonin by an artificial cyclic rise in circulating melatonin levels to enable the dysrhythmia to disappear (by spontaneous resynchronisation as a result of maintenance of the programme) and thereby eliminate or limit the impact of resulting discordant growth.

It must be recalled that the change in the hormonal environment around the period of adolescence affects the musculo-ligamentary system providing a certain laxity to it, with two types of consequences. One is positive because bone growth in encouraged by reducing preload on growth plates whose equilibrium is restored after an increase in volume. The other is negative because of the temporary loss of load from preloaded systems like the joints – especially the poly-articulated spinal system, whose static and dynamic stability decreases. This subject will be re-examined further where, the apparent stiffness of preadolescents and adolescents incidentally, lie quite readily in strange positions that are only possible through the modification of their natural laxity and inherent muscle tone.

Parenthetically, for pregnant women, the modification of the hormonal environment permits a certain known laxity of the ligaments. This is beneficial during pregnancy and especially during delivery. During this period, if a woman suffers from scoliosis, it worsens notably, and then returns to its prior state about 6 months after delivery. This lets us forge the link between the hormonal environment, musculo-ligamentary laxity and stability of the spinal system. If we add potentially discordant growth phenomena to this picture, we arrive at a situation that places the spinal system at the limits of chaos.

BIOMECHANICS

1. STATIC ASPECTS

First of all, the spinal column is a poly-articulated, preloaded system. The disk located between two vertebrae behaves somewhat like a hydraulic shock absorber with a perpetual dissociating effect. It resists deformation under acute loads and compresses in response to long-term loads, like gravity and growth. The disk's effect is partially equalised by the proximity of the capsulo-ligamentary system. The latter serves to initiate the preload permitting the system's stability and mobility. The bone itself adapts to the loads to which two key mechanisms are subjected. One is the orientation of bone trabeculae along lines of maximum loads (bone remodelling) in response to long-term loads. The other is blood pressure within the vertebral body that can vary very quickly in response to acute loads. We should note the preload effect is a growth modulator. Any rise in load hinders growth. Any decrease releases it momentarily. Alternation of the two has a stimulating effect. Injuries may appear in response to excessive loads. If the cause of the injury is brutal, a bone fracture will occur before the disk ruptures or the ligament tears. If the cause of the trauma – which is not necessarily a load, but a repetitive movement or other condition involving an internal or external change – is gradual and long lasting, the disk will give way before the bone. In all events, all internal changes of the spinal system will bring the system to either a new static balance or to a more or less long-term collapse. This also depends on the ability of the NMSS to adapt – meaning the response of the neurological and muscular systems to new conditions. Thus, unsynchronised growth over four to five vertebral levels can result in a minimal deformity, but still sufficient to alter the intraspinal preloads in this same area and cause a change in static and dynamic balance without an initial visible morphological change of the spinal column. We do not need severe apex and pre-apex deformities to occur in scoliotic curvatures for the NMSS to be disturbed. This is particularly so if, at the same time, spinal stability declines along with a reduction in sagittal curvatures of the spine (sagittal curvatures being recognised as key elements in the biomechanical stability of the spine).

Furthermore, as in all complex systems, a minor disturbance can trigger a gradually destructive dysfunction. Maintenance of a paranormal initial condition and the addition of internal

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and external modifications such as the modification of the ligamentary constraints, inadequate neurological response and muscular dysfunction could result in chaotic functioning of the NMSS, resulting in a change in balance and a progressive deterioration.

It should be noted that the fact that scoliotic patients tend to be ectomorph means mesodermal derivatives are in some respects underdeveloped. Thus their skeleton and muscles are longer and frailer than they are strong or stocky. This does not necessarily result in abnormal functionality, but in the event of disturbance, the NMSS stability is diminished, making greater the risk of deterioration.

However, without movement or placement under a load, the NMSS has little risk of collapsing. A reclining position over an extended period of time was and furthermore remains in some countries the best treatment for scoliosis, which worsens little or not at all under such conditions.

2. DYNAMIC ASPECTS

All of the vertebrae in a single spinal column do not have the same anatomical shape. The size, shape and orientation of the articular apophysis of vertebra vary. Some bear ribs, others do not and, depending on the region, muscular insertions are quite different. This is common anatomical information but it still helps a better understanding of why the various movements are not distributed uniformly across the spinal column. It should be noted, for example, that torsion motions generally involve the middle thoracic spine. Lateral bending motions usually involve the lower thoracic region and upper lumbar region. Anterior flexion motions usually involve the lower lumbar region. Certain individual variations may apply, depending on the individual, the sex, the morphology and the age. But each person seems to embody both the pattern of motions of his or her species and his or her own personal composite pattern of motion. As a result, you recognise the step of your partner, or her/his gait in the dark. Also, a similar disturbance affecting the middle thoracic spine will result in the same kind of reaction, but not necessarily one of the same amplitude or of the same speed. If the disturbance appears in another area of the spine, the process is the same but the results different to the extent that certain movements (at least because of their main locations, their elective plane and their amplitudes) are more aggravating than others. Also, balance and intramuscular strategy, intra and extraspinal will be equally subject to modification following any change to an intraspinal mobility strategy. This can become an important factor in disequilibrium. It should be noted that the modification of muscular balance is closely linked to the type of main fibres of which each muscle is composed. These modifications in muscular strategy and balance largely depend on information delivered by the nervous system. What is the status of this information?

THE NEUROSCIENCES

Neuroscience data are abundant and complicated. However, we retained the importance of feedback mechanism that send precise information of the joint function to the central nervous systems on bone growth should also be considered.

In general, any modification of the joint function is recorded by the nervous system, which tailors its response through the muscle. The effect can be protective and beneficial, but it can also establish and even amplify an intrarachidian dysfunction. This could be, for example, a muscular contracture (often observed in traumatic injuries) or a change in muscular balance that is often harmful over the long term (observed in chronic progressive disorders). When the cause can be treated early, a return to the norm occurs spontaneously. In contrast, elimination of the cause followed by a "proprioceptive" process of rehabilitation can eliminate the vicious circle: dysfunction disturbance of feedback information imbalance and vice versa. If asymmetrical segmentary vertebral growth occurs, the intra-spinal preload is even further modified to the extent ligamentary laxity and decreased spinal stability are also present. Among other things, this would thus produce abnormal play in the joints during movement. All conditions are thus present for this to add a lack of neurological control and thus of muscular balance. It may also be wondered what can be the impact of such a disturbance, while the neurological system is not mature and the body is quickly changing during periods of rapid growth. It is possible that abnormalities might make their marks over the long term, particularly in systems that regulate static and dynamic equilibrium.

The other aspect of the problem is direct control of the nervous system on bone growth. This is increasingly apparent in terms of the longer bones, but also probably present in terms of the vertebrae. We know the impact of the nervous system on embryological development of the lateral portions of the vertebrae. It would be no surprise for control to subsequently remain, permitting adaptation of bone growth to internal and external or environmental modifications incorporated by the central nervous system. If so, what would be beneficial in optimal conditions could become an additional cause of harm in preserving the integrity of the NMSS.

CONCLUSION

Naturally the lesion of any NMSS components, or of some other system interfering with it, could trigger a disorder severe enough to result in a scoliotic deformity. But if we add:

- The clear presence of a predisposing status.
- A neuro-muscular-skeletal system that is as complex as it is intricate and fragile.
- The development and growth of the NMSS, regulated by a predefined genetic program.

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- Rapid spurts of growth that upset the morphology and biology of the child, a major abnormality is not needed to trigger a dysfunction of the NMSS.

But no lesion is necessary to initiate a deformity. In science as well as in life, it is well known that a cascade of events can reach a critical point beyond which a small disruption can take gigantic proportions. A simple phase difference – even a temporary one – is enough to trigger the dysfunction of a complex system and thus leads to its gradual deterioration. An even minimal, discreet disturbance within a complex system – if the Chaos Theory is considered for example – can launch this system into what might appear to be incoherent disorder. However, it would actually be predictable and follow a dynamic order – it would bear evidence of a new unstable equilibrium. Accordingly, I think it is possible for an unsynchronised bone growth, a temporal defect of genetic origin, focused on a particular spinal section, to trigger the formation of discordant vertebrae that would alter the internal dynamic balance of the spine and, under conditions of rapid maturation and growth, result in a dysfunction of the NMSS. If major internal or external conditions such as a cessation of growth, change in hormonal environment or achievement of a new stable equilibrium, are not rapidly, spontaneously or artificially altered to break the vicious cycle that has been established, the NMSS gets caught up in a dysfunction of highly evolutive risks – leading to the severe spinal column deformities with which we are familiar.

Based on the Chaos Theory, this hypothesis is a reminder of the meteorological phenomenon humorously called butterfly effect: “The flutter of butterfly wings today in Peking creates eddies that can become next month’s storm in New York”.

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