Aetiology of idiopathic scoliosis:

Current biomedical research and osteopathic theories

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Abstract

The question of the aetiology of the three-dimensional spinal deformity of idiopathic scoliosis (IS) has not been answered so far. Current biomedical studies and osteopathic theories that offer possible explanations for the development of IS are reviewed to identify similarities or diametrical differences of the two approaches. The results of the biomedical studies show various possible influences on the development of IS: Genetic factors, structural anomalies, anatomical asymmetrical patterns, neurological dysfunctions. But none of them seems to be completely accepted in biomedical research.

Osteopathic theories for the aetiology of IS are dysfunctions in the embryology, trauma indicated by the birth process, SSB-dysfunctions, dysfuncions on a bony-, membranous-, or fluid-level induced by traumata. Also fascial distortions are discussed as possibilities that initiate the development of scoliosis. The known osteopathic theories for the development of IS are not based on empirical studies and the so called “dysfunctions” are diagnosed via palpation so that the reliability of the postulated theories therefore is doubtful.

Similarities between biomedicine and osteopathy can be found in some hypotheses about possible causes of IS like disturbances in the embryology or in the last months of pregnancy but in both cases they have so far not been proven by clinical research.

In order to find an answer to the question of the aetiology of idiopathic scoliosis further interdisciplinary studies are needed.

Key words: Aetiology of IS, biomedical studies, osteopathic theories, similarities or diametral contradictions

Eidesstattliche Erklärung

„Ich erkläre hiermit an Eides Statt, dass ich die vorliegende Arbeit selbständig angefertigt habe. Die aus fremden Quellen direkt oder indirekt übernommenen Gedanken sind als solche kenntlich gemacht. Die Arbeit wurde bisher weder in gleicher noch in ähnlicher Form einer anderen Prüfungsbehörde vorgelegt und auch noch nicht veröffentlicht.“

……. …….
Ort, Datum (Mona Lüftinger)

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1. Introduction

Scoliosis – as a condition that affects the spines of many children, teenagers and adults – has for a long time been an important research area in orthopedics and the focus of various kinds of clinical studies over the last few decades. The word scoliosis derives from the Greek σκολιος and means curvature. Hippocrates was one of the first to describe and discuss various normal and abnormal curvatures of the spine. He treated scoliosis using traction but regarded this treatment as difficult and ineffective. (Moe 1978). Galenus first used the terms kyphosis, lordosis and scoliosis.

Ever since Greek and Roman times a series of new discoveries has resulted from research in various fields. Some of these discoveries are still of great importance today, e.g. Meijer's discovery of 1866. Meijer found out that kyphosis is an important feature of a non-progressive scoliosis. Other examples are the preventive exam by Adams (1882) or the discovery of the relevance of the torsion (Jach 1892). Nevertheless, the aetiology or pathogenesis of idiopathic scoliosis remains unclear to this day.

In my own work as an osteopath I have met a large number of adolescent scoliosis patients and have thus been repeatedly confronted with this disease and the questions related to it. The techniques I chose for the osteopathic treatment of these patients were diverse and very individual. Although the results were on average very good (reduction or at least prevention of progression of spine curvature measured in Cobb degrees) I could never find a clear pattern with AIS patients. I have asked myself repeatedly: When and where did scoliosis originate? Can the development really be explained by a primary lesion or is it a multifactorial pathophysiology in which the development of scoliosis results from an accumulation of various kinds of dysfunctions at different points in time? This leads to the question of the causality of idiopathic scoliosis as a research topic.

The aim of this dissertation is to take a close look at the aetiology of idiopathic scoliosis and to review the results of the current biomedical research and osteopathic theories. Furthermore I will discuss similarities and diametrical contradictions between certain results of biomedical research and osteopathic theories with regard to the development of idiopathic scoliosis. Thus I would like to improve my osteopathic way of thinking and working with regard to the treatment of adolescent scoliosis patients.

To reach this aim I would like to give you a brief overview of the history of diagnosis and treatment of scoliosis in chapter 2. Chapter 3, 4 and 5 will deal with the basic elements of scoliosis like the pathology, the current status of diagnostics and the possibilities of treatment. In chapter 6 I will present the possible causes of scoliosis from a biomedical viewpoint and in chapter 7 osteopathic theories regarding scoliosis will be explained. In

chapter 8 I will compare the biomedical and the osteopathic viewpoints and point out similarities and contradictions between the two.

2. History of scoliosis

This chapter will give an overview of the history of scoliosis diagnosis and scoliosis therapy from antiquity to the 21st century.

The history of the diagnosis and treatment of spinal column diseases goes back to antiquity. The spinal column has been a research field of great variety and depth for many physicians for over 2 000 years, with new insights and knowledge constantly being gained in the areas of anatomy, physiology, pathology, and therapy.

Fig. 1: Hippocrates

Hipppocrates of Kos (approx. 460-375 B.C.) is considered the founder of academic medicine in Europe. Famous quotes from Hippocrates show that he had clearly recognized the importance of the spinal column over 2 000 years ago.
He wrote on joints, bone fractures, and rachiotherapy. His functional anatomic descriptions mention displacement of joints and vertebrae under the term ‘parathemata’, which he felt was the source of many diseases. Hippocrates was the first to describe treatments suggesting the beginings of manual therapy. He used traction and pressure to correct dislocations, bone fractures and malcurvatures of the spinal column.

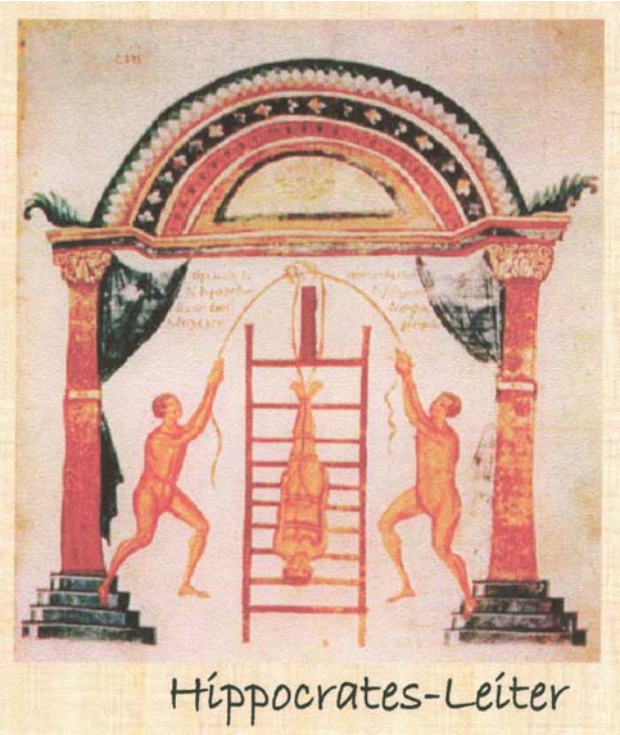

Fig. 2: Hippocrates' ladder

Appolonius of Kitium (1st cent. B.C.) was the first to ilustrate some of Hippocrates' works. The pictures show procedures for repositioning bone fractures and joint luxations as well as manipulative, corrective procedures to be carried out on the spine under traction.

Galen of Pergamon (129-199 A.D.) was a Roman physician of Greek origin who influenced the development of academic surgery. His papers on osteology, myology, the structure of the sceletal system, and descriptions of the cerebral and spinal cord nerves are significant. The knowledge gained thereby provided a considerable foundation for the training of surgeons over the course of the following centuries.

Oreibasios (326-403), was a Greek surgeon from Pergamon and the author of a collection of medical works the "synagogia latrike", a 72-volume encyclopedia, mainly based on the works of Galen and other Greek surgeons. The illustrations also include examples of extension treatment.

Guido Guidi (1491-1547) was a professor of surgery at the famous "College de France" in Paris. He was the author of a detailed illustrated work on diseases of the spinal column an their treatment.

Ambroise Paré (1510-1590) a French surgeon is considered the founder of modern surgery and treatment with prostheses and supportive orthotic devices. He developed the first supporting corsets made of iron plates to correct spinal deformities.

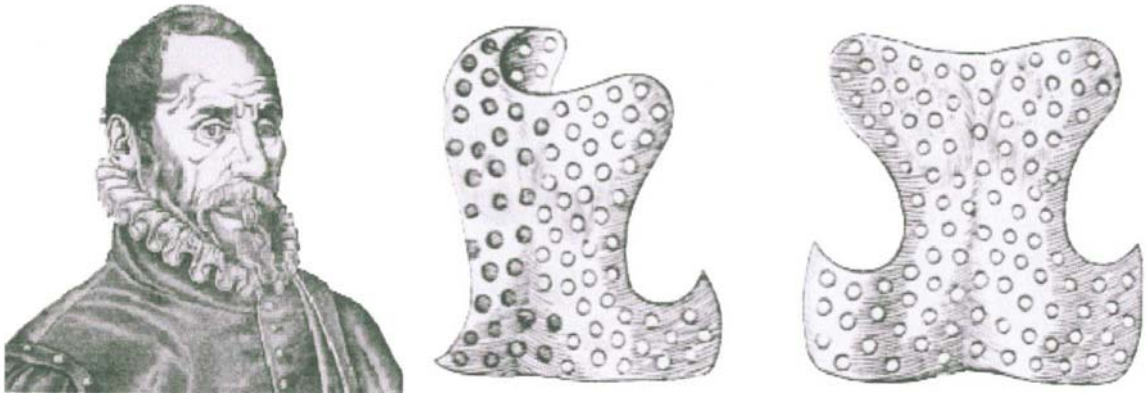

Fig. 3: Ambroise Parè (supporting corsets made of iron plate)

Andreas Vesal, called Vesalius, (1515-1564), a Flemish anatomist and surgeon of German origin was the founder of modern academic anatomy with his work “De humani corporis fabrica libri septem“. Contrary to Galen’s works, his anatomical drawings were based on dissections he performed on executed prisoners.

Fig. 4: A.Vesal, “De humanis corporis“

Wilhelm Fabry, called Fabricius von Hilden (1560-1634), was a famous German surgeon. He is regarded as the founder of academic medical surgery and the first to describe a scoliotic spinal column in his work “Der Abriß des Rückgrads“.

Fig. 5: Hildanus, “Abriss des Rückgrads”

Francis Glisson (1597-1677), an English anatomist, was the first to describe rachitis in detail – a bone disease very common at the time caused by a vitamin D deficiency, where a bone metabolism disorder caused skeletal deformations, in particular scoliosis. He invented an extension treatment for this disease where tensile force was exerted on the spinal column unsing a padded leather sling fixed at the chin and the back of the head to straighten the spinal curve.

In the following decades, the development of devices for extension treatment and orthotic devices for correction of spinal column curvatures continued.

Around 1762 Augustin Roux developed various different orthotic devices for the correction of spinal column curvatures.

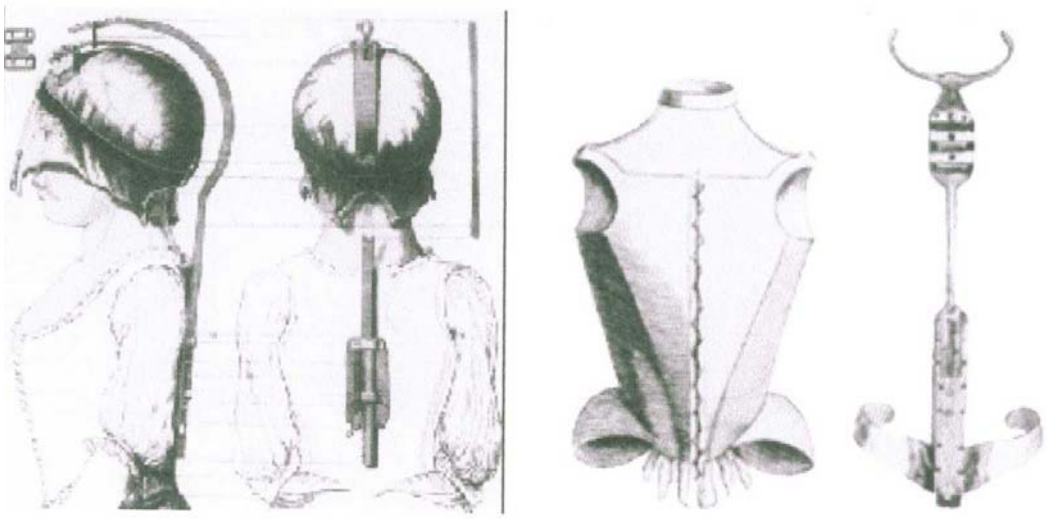

Fig. 6: Roux corrective orthotic device

In 1783 Le Vacher and Sheldrake designed further corrective devices for straightening the deformed spinal column.

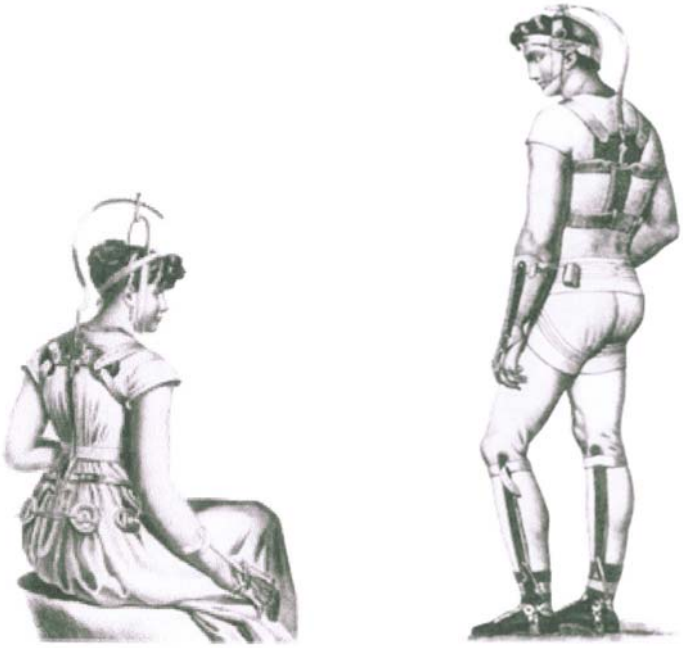

Fig. 7: Le Vacher and Sheldrake corrective orthotic devices

In 1835 J. Hossard designed the first corset that could be mechanically adjusted to correct spinal column curvatures. Up to the beginning of the 20th century, the diagnostics and therapy of spinal column diseases was characterized mainly by clinical examination and the therapeutic application of various braces and extension treatments.

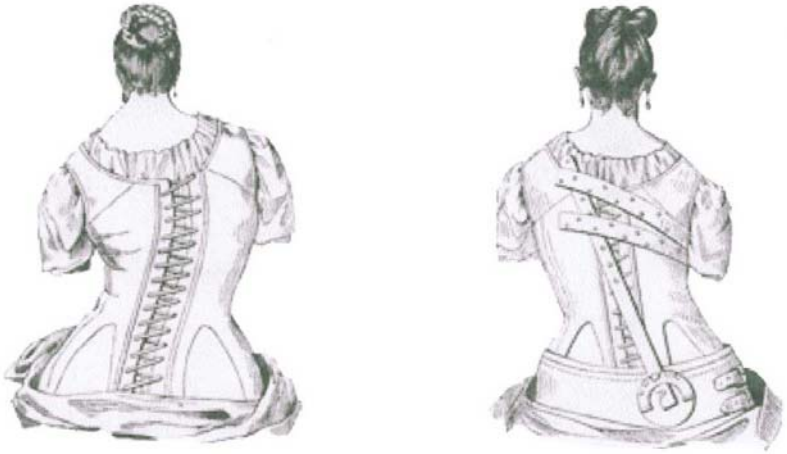

Fig. 8: Hossard corrective orthotic devices

Jules Guerin (1801-1886), a Belgian surgeon was the first to practice “myotomie rachidienne”, a transection of the paraspinal muscles to treat scoliosis, since he considered a dysbalance in the paraspinal muscles to be the cause of the development of scoliosis. The corrective results were poor and the method was not practiced for long.

Wilhelm Conrad Röntgen (1845-1923), professor of physics in Würzburg, made an important step forward in diagnostics with his discovery of an as yet unknown type of radiation while experimenting with cathode ray tubes in November 1895. He called them X-rays. In January 1901 he presented his discovery in public and revolutionized diagnostics. Spinal column images in 2 planes first became possible in 1925, facilitating appropriate conclusions concerning structural spinal column findings.

In 1902 L. Wullstein published “Die Skoliose in ihrer Behandlung und Entstehung“ describing his clinical and experimental research and thus contributed considerably to the understanding of scoliosis.

In 1911 Fred Albee was the first to carry out fusion surgery in tuberculosis patients with deformed spinal columns. The rigidification was achieved by fusing the vertebral arches by inserting bone material from the shinbone after the spinous processes were split.
In 1920 the surgeon Wreden introduced a surgical technique for the treatment of scoliosis involving metal implants.

In 1931 Russel Hibbs published the results of over 300 scoliosis operations in which he had carried out a spondylodesis (rigidification) based on the method worked out by Albee. He made use of the spinous processes and fine bone chips from the vertrebral arches as autologous bone material for spondylodesis. He also sclerosed the vertebreal arch joints in the area of spondylodesis.

In 1951 Max Lange published a scoliosis surgery technique in an orthopedic surgery textbook employing internal fixation with Küntscher nails to stabilize the corrected spine. The nails were attached to the spinous processes of the vertebrae.

In 1962 Paul R. Harrington, an American orthopedic surgeon, developed and introduced an set of instruments, the Harrington rod system, that revolutionized surgical scoliosis therapy. The main concept revolves around the insertion of a convex compression rod and a concave distracion rod to compensate and stabilize the scoliotic curvature of the spinal column.
Many patients with idiopathic, congenital, and neuromuscular scoliosis have been successfully treated using this technique. Many modifications and further developments of Harrington's innovation have been done since.

Pierre Stagnara (1917-1995), a French orthopedist, played an important role in the development of scoliosis therapy from 1950-1984. He recognized the importance of the removal of the costal hump and the resulting increase in spinal column flexibility. He also developed the technique of "greffe anterieur", i.e. anterior support chip, to support spinal column stability in the surgical treatment of scoliosis combined with a severe kyphosis. He improved radiological evaluation of kyphosing scolioses with the introduction of a special angled x-ray image, where the late is held parallel to the median surface of the costal hump.

In 1975 Luque introduced a further development of the Harrington instruments for posterior segmental fixation of the spinal column. In this method, the scoliotic curve is compensated and stabilized with 2 individually curved metal rods attached to the vertebral arches with wire slings after the yellow ligaments have geen severd. The advantage of this method was the

high level of post-surgical biomechanical stability achieved: Practically no follow-up brace therapy was required. The drawback was the very high risk of neurological complications.

In 1973 Dwyer developed a surgical access route from the front (anterior or ventral) for the correction of scoliosis. The preparatory work for the ventral access breakthrough was done by Hodgson and Stock, who had operated on many tuberculosis patients via transpleural-retroperitoneal access, thus establishing a standardized access to the lumbar spine and lower thoracic spine.
This ventral access approach reduced the number of neurological complications, the fusion length was shortened, and stability was improved by means of intercorporeal fusion. Drawbacks of this method consisted of the necessity of several months of follow-up treatment in a brace and the lack of derotation of the vertebrae.

In 1975 Klaus Zielke presented the ventral derotation spondylodesis (VDS) method, developed on the basis of the Dwyer method. The main improvement was a new compression technique and use of a derotator. Using this method, excellent results were obtained in the frontal plane. The drawback was that the method paid too little attention to the sagittal profile of the spinal column with the elimination of the lumbar lordosis.

In 1984 Yves Cotrel und Jean Dubousset invented a surgical method based on the Luque method. The objective of the procedure was a three-dimensional correction of the spinal column by applying translation, distraction, and compression for improved derotation and an improved sagittal profile of the scoliotic spinal column in combination with primary stability. This basic concept is still the basis for dorsal instrumentation of scoliosis as it is done today. The neurological complications resulting from Luque's procedure with wire cerclages around the vertebral arches were avoided in this method, since the two metal rods are directly inserted into the bone using hooks and screws placed in the vertebral pedicles. The disadvantage of the method was the insufficient restoration of the sagittal profile of the spinal column and frequent decompensation of the non-instrumented sections of the spinal column.

Physiotherapeutical Approach

A systematic orthopedic remedial gymnastics was only beginning to establish at the end of the 19th century. In special clinics corsets were applied in long sessions, supervised by doctors and additionally patients were made to use special devices and do gymnastic exercises.

Rudolf Klapp (1873-1949), a surgeon from Cologne, invented a set of exercises for scoliosis patients. The spine is mobilized and the spine muscles are strengthened and stretched by performing specific exercises at creep speed while standing on hands and knees with leather braces applied to knees and hands.

Around 1905 Albert Hoffa introduced bend-and-stretch-exercises to scoliosis therapy. At the beginning of the 20th century the gymnastics method after Ling was widespread. Resistance exercises were done in sitting and standing positions as well as lying on the back and front and hanging.

In 1913 Oldevig noticed the drawback of having to work one to one only in therapy sessions and introduced new techniques using straps so the patient could work actively on his own.

Max Lange regarded scoliosis as a disorder of the muscular equilibrium which he over-corrected by using resistance devices.

August Blenke (1913) viewed the Klapp crawling exercises rather critically and held the opinion that scoliosis should only be treated individually, according to the specific situation and condition of each patient.

Katharina Schroth (1894-1985) invented the three-dimensional scoliosis therapy. Based upon her own experience she developed a holistic therapeutical method by combining specific correction techniques with a special corrective breathing technique called „Dreh-Winkel-Atmung“.

Vaclav Vojta started from the idea that with the help of facilitation of reflex movements the muscular dysbalance of scoliosis patients can be compensated by central mechanisms. He first treated spastic children and in the 1960s he started to apply his concept to scoliosis patients as well.

3. Pathology of scoliosis

This chapter will present the fundamental basics of the pathology of scoliosis, starting with the definition, followed by the division of idiopathic scoliosis and the classification of scoliosis according to their aetiology will be presented.

3.1. Definition of scoliosis

Scoliosis can be defined as a partly fixated lateral curvature of the spine which cannot be completely straightened up again (Meister 1980).
Idiopathic scoliosis is a (partly) fixated lateral curvature of one or more parts of the spine, which co-occurs with a rotation, a torsion, and a structural change of the vertebrae (Humpke 2002).
Scoliosis is a lateral curvature of the spine which represents a rotational malalignment of one vertebra on another. Rotation and side-bending occur to opposite sides. Ribs are rotated posteriorly and are prominent on the convex side of the curve. The positional strain is exacerbated in forward flexion, producing a rib hump (Jane Carreiro 2003).
Structural scolioses are fixated lateral curvatures of the spine (Lindemann 1957). They result from intrinsic changes in the anatomy of one vertebra or several vertebrae and/or the surrounding tissue, and lead to an irreversible restriction in spine movement in one or more directions. In this case a complete correction of the spinal curvature through a conservative method is no longer possible.
The most striking sign of a structural scoliosis is the fixated rotation of one or more vertebrae, the deformity of these vertebrae, a bulge in the loin or a rib hump.
You need to distinguish between a rotation and a torsion of the vertebrae. Rotation refers to a rotation of single vertebrae against each other in their craniocaudal axis (Ebenbichler 1994). A torsion, by contrast, refers to the torsion of the bodies of vertebra of two consecutive vertebrae and the helical/spiral torsion of the final parts of the spine as a whole.
Three components of the torsion can be distinguished: the rotatory moment in the axial plane, the lateralisation between the vertebrae in the frontal plane, and the hyperextension in the sagittal plane (Pedriolle 1985).
X-rays (Pedriolle et al. 1984), clinical (Mau 1982) as well as experimental examinations (Dickinson et al. 1984) showed that the patients' vertebral body growth plates are ventrally higher than dorsally, which leads to a consecutive lordosis at the height of the scoliotic apex.

In addition to this asymmetry of the spine there is very often an asymmetry of the spine in the frontal plane. In a growth spurt – idiopathic scoliosis always being an illness brought on by growth – strain and flexion of the spine bring about scoliosis with a torsion.

Unlike scolioses of known aetiologies, idiopathic scoliosis occurs without any obvious cause before the onset of bone maturation (Heine 1992, Perdriolle and Vidal 1985). Idiopathic scoliosis accounts for the largest part of scolioses vis-á-vis those scolioses with known causes (i.e. 80-90%).

Scoliosis is diagnosed by full-length standing spine X-rays. These x-rays are then assessed through measuring the Cobb angle (Cobb 1948), the vertebral rotation, and through ascertaining bone maturation.

Curvatures of less than ten degrees according to Cobb are not regarded as scolioses.

Females are affected by idiopathic scoliosis more often than males in a proportion of 4:1. Admittedly, with curvatures below 10 degrees, the male-female distribution is equal, but the stronger the curvature gets, the more marked is the predominance of the female sex (Weinstein 1985).

Statements about progress show that small curvatures have been known to take a favourable course (Brooks et al. 1975, Rogala et al. 1978). Curvatures of a larger degree tend proportionally towards an increased likelihood of progress (Lonstein and Carlson 1984).

The degrees of curvature are classified by the U.S. American Scoliosis Research Society according to the angle as follows:

Grade 1	Curvature angle between 5 and 20 degrees
Grade 2	Curvature angle between 21 and 30 degrees
Grade 3	Curvature angle between 31 and 50 degrees
Grade 4	Curvature angle between 51 and 75 degrees
Grade 5	Curvature angle between 76 and 100 degrees
Grade 6	Curvature angle between 191 and 125 degrees
Grade 7	Curvature angle above 125 degrees

3.2. Classification of idiopathic scoliosis

Through localising the curvature the following groups of idiopathic scoliosis can be distinguished:

Thoracic scolioses: The vertex lies above and including Th2, with semi-thoracic cases down to Th3, and with thoracic scolioses down to Th10.

Thoracolumbar scolioses: The vertex can be localised in Th11-12.

Lumbar scolioses: The vertex lies between L1 and L4.

Lumbosacral scolioses: The vertex lies in L5 or in the sacrum (Ebenbichler et al. 1994)

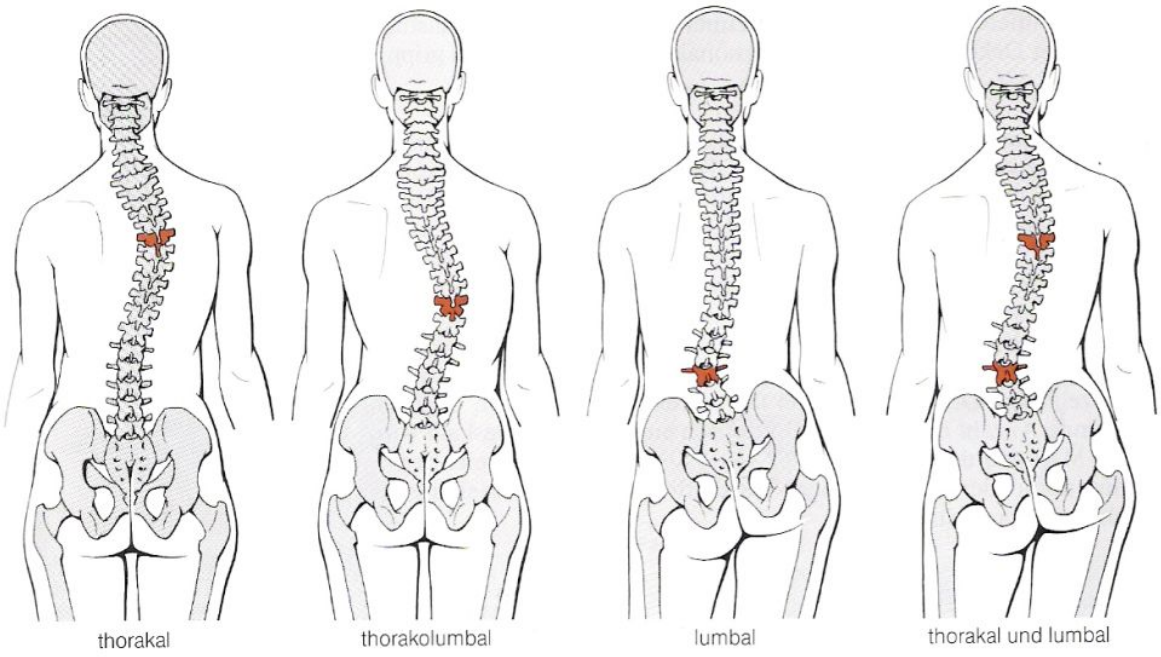

Fig. 9: Classification of scoliosis

3.2.1. Three-curved scolioses

With three-curved scoliosis the main curvature is in the thoracic area, with an additional compensatory lumbar curvature which is not significant. The position of the pelvis in the frontal plane is balanced. During the bend test, the loin-pelvis block is hardly asymmetrical. The distribution of weight on the legs appears to be even (Weiß and Rigo 2001).

3.2.2. Four-curved scolioses

With four-curved scoliosis, there is a thoracic curvature of varying extent and a marked lumbar curvature which exceeds the midline of the body, and enters caudally into a lumbosacral compensatory curvature (Weiß and Rigo 2001).

3.3. Division according to age of manifestation of scoliosis

Congenital scoliosis: 0-2 years
Infantile scoliosis: 3-7 years
Juvenile scoliosis: 7 years to onset of puberty
Adolescent scoliosis: puberty to epiphyseal closure

3.4. Classification of scoliosis according to their aetiology

Congenital scoliosis: failure of formation (hemivertebrae), failure of segmentation (unilateral bar)
Idiopathic scoliosis: infantile, juvenile, adolescent
Neuromuscular scoliosis: cerebral palsy, spinal muscular atrophy, Syringomyelia, spinal cord trauma, spinal cord tumor, Friedreich's ataxia
Myopathic scoliosis: muscular dystrophy
Mesenchymal scoliosis: Marfan's syndrome, Ehler-Danlos syndrome
Other causes: leg-length inequality, hysterical, metabolic, soft tissue contractures, osteochondrodystrophies (Niethard 1992).

4. Diagnostics

This chapter will give you an overview of the current diagnostic methods from general clinical assessment, to metrical assessment and diagnostic imaging techniques.

4.1. Clinical parameters

General clinical assessment:
As a result of the lateral curvature of the spine there is a deviation of the spinal process line from the straight line, the shifting of trunk mass, an asymmetrical position of the shoulder blade, and an asymmetrical shape of the waist triangles. Through the rotation of thoracic vertebrae and the adjoining ribs a rib hump and a concave flattening of the thorax. In the lumbar area a loin bulge can be seen instead of the rib hump, which is caused by the rotation of lumbar vertebrae and the emerging paraspinal muscles.
With medium and servere scolioses the trunk asymmetry can already be seen in standing position. The bend test constitutes another position for diagnoses, and because of maximum kyphosis of the thoracic and lumbar spine even allows for diagnosing smaller trunk asymmetries (Adams 1882).

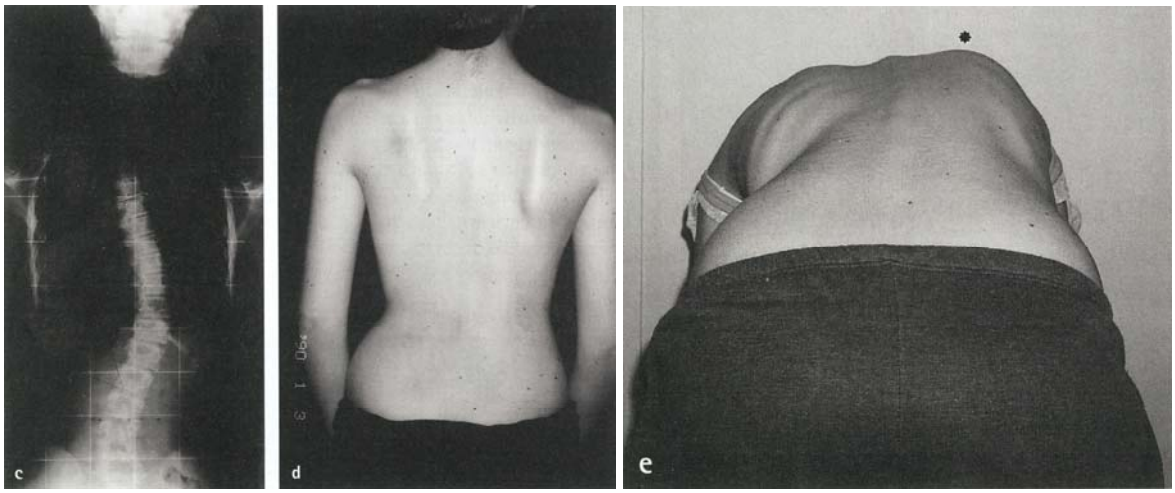

Fig. 10: Bend test

4.2. Metrical assessment

There are various metrical diagnostic methods which serve to ascertain the severity of the curvature and are of some prognostic value.
Whether a spine is statically compensated or decompensated can be determined by dropping a perpendicular from processus spinosus C7 to the rima ani. If the perpendicular does not fall through the rima ani, the curvature of the spine can regarded as decompensated. The deviation from the rima ani will be measured, documented, and matched with the corresponding degree of severity.
In order to clinically assess trunk asymmetries a measurement instrument which was designed according to the principle by Bunnell (1984) is used. This scoliometer is placed above the spinous processes at the level of maximal paraspinal prominence. Through the resulting inclination, the corresponding angular dimension is shown on a scale. In addition to this specific diagnosis, chest expansion and lung capacity are ascertained.

4.3. Diagnostic imaging techniques

X-ray diagnostics complement clinical assessment, and serves the purposes of ascertaining status, observing progress, and of checking obtained correction results.
X-ray screenings of scolioses consist of two full-length standing spine radiographs, with one being a postanterior radiograph, the other a lateral radiograph, in order to obtain a three-dimensional picture of the scope of scoliosis. An evaluation of these total standing spine X-rays starts with ascertaining lateral spine curvature according to Cobb, and of the vertebral rotation after Pedriolle's (1985) or after Raimondi's technique (Weiss 1995).

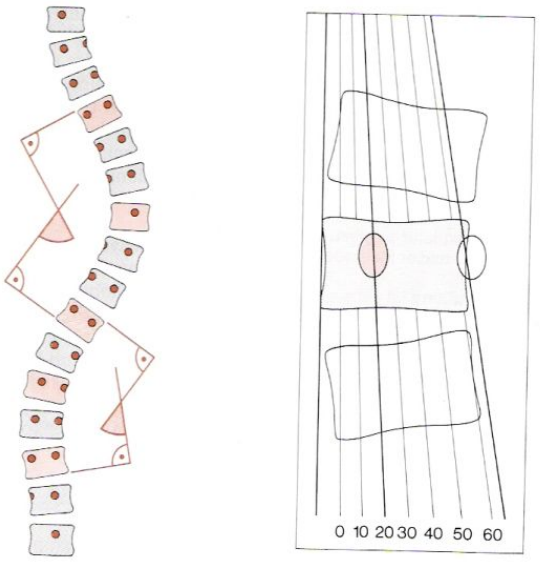

Fig. 11: Cobb curve

For checking progression, prognosis and treatment of scoliosis, an assessment of bone maturity is crucial. For this purpose, the ossification of the epiphyses in the wrist joints, the ossification of the ring apophyses, and above all the ossification of the iliac crest apophysis as described by Risser (1958) are assessed. Children before the onset of menstruation or of mutation are usually placed in Risser Stadium 0, which leaves room for the entire pubertal growth spurt. With Risser Stadium 3 the main phase of growth is completed, and the prognosis gets considerably more favourable. With Risser Stadium 5 growth is complete.

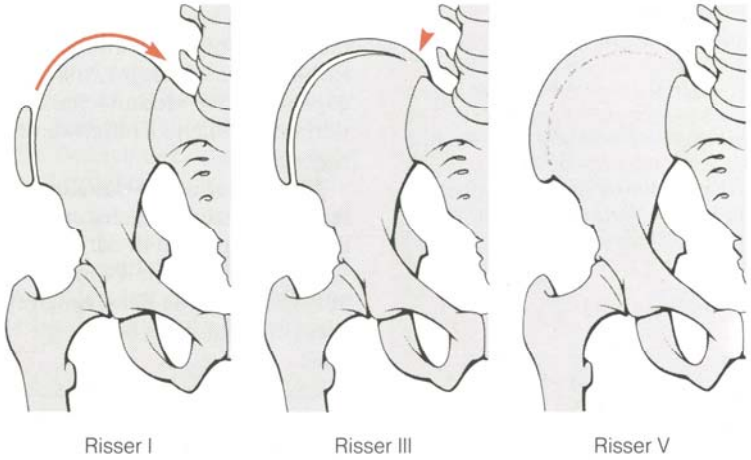

Fig. 12: Risser grades

5. Treatment options in scoliosis

In this chapter current treatment options are being discussed, starting with conservative treatments, followed by orthopedic methods like ortheses and finally operations with the aim of correcting the curvature.

For identifying the best kind of treatment it is important to know the aetiology of scoliosis (various progression tendencies), the patient's age (for remaining spine growth), and the scope of deformity.
Treatment is tripartite. With incipient scoliosis (up to 20 degrees after Cobb) physiotherapy is carried out. Scolioses between approximately 20 and 50 degrees are treated by wearing a corset or bracing in addition to physiotherapy. If there is a curvature of more than 50 degrees after Cobb, an operation is recommended.
This three-stage plan for treatment shows how important it is to diagnose scoliosis already early on, as with incipient growth deformities less invasive methods are feasible (Niethard and Pfeil 1992).

5.1. Conservative treatments

Osteopathy offers a wide spectrum for treating scoliosis through techniques which regulate strain in various tissue structures and planes. Structural, visceral and cranio-sacral techniques are applied according to diagnostic findings on individual cases of scoliosis. It is the overall aim to reduce the rigidity of scoliosis, to balance out dysbalances caused by strain in myofascial, ligament and membrane tissue, to harmonise cranio-sacral dysfunctions, to improve metabolism in general, and thus to reduce the curvature degree of the spine, to stop or slow down the progression of scoliosis, and to prevent restrictions in the cardio-pulmonary tract. According to studies by Mandl-Weber (2000), and Phillipi et al. (2004) osteopathic treatment leads to better therapy results with scoliosis than in control groups treated with traditional methods.

The three-dimensional scoliosis therapy according to Katharina Schroth is an active therapy concept, in which specific correction mechanisms and corrective breathing (Dreh-Winkel-Atmung) are meant to influence scoliosis through a change in body image.

This is the leading therapy concept, next to treatments based on developmental kinesiology like Vojta (which is used to treat scoliosis in its early stages), and general physiotherapy.

5.2. Orthopaedic methods

5.2.1. Ortheses

Bracing is an invasive but usually inevitable form of therapy, which is indicated with scolioses between 25 and 40 degrees after Cobb (Lohnstein and Carlson 1984).
Since scoliosis is a growth deformity, it is recommended to wear the brace 23 hours a day in order to reduce the progression of scoliosis. The correct fit of the brace needs to be checked every four months (Ebenbichler et al. 1994), and the brace needs to be worn till the end of the bone growth phase (Risser V).
The various forms of braces can be summarised as follows:
the Milwaukee brace
the Chenau brace
the Boston brace
the Lyon brace (Stagnara brace)
Bending brace
Wilmington brace

EDF-plaster cast is an extension, derotation and flexion plaster which is nowadays only rarely used with severe scolioses after preceding extension treatment on the Cotrel table.

Electrotherapy stimulation of convex musculature cannot be recommended any longer, as clinical studies have shown that slight corrections of the scoliotic spine were achieved only initially (O'Donell et al. 1988).

5.2.2. Operative treatment

Operative treatment is indicated with adolescent patients suffering from idiopathic scoliosis with a curvature of more than 40-45 degrees after Cobb, and adult patients with curvatures of more than 50 degrees after Cobb.
Pre-operative traction procedures are used in order to interoperatively facilitate as secure and good a correction of scoliosis as possible. Ventral and dorsal invasions are either

employed in isolation or in combination in scoliosis surgery, with the aim of correcting the curvature in the frontal as well as the sagittal plane. Spinal fusion (reinforcement of certain spinal segments) is an obligatory part of every scoliosis operation. In the past, intraoperative correction was achieved with plaster casts, while now correction and stabilisation are achieved through metal rods.
The most common operative procedures are as follows:

Spinal fusion (spondylosyndesis) according to Harrington
Luque-Instrumentation
Operation accordting to Cotel and Dubousset
ISOLA-Instrumentation (Niethard 1992)

6. Causes of scoliosis in a biomedical view

In this chapter I would like to give you an overview of the biomedical studies in current aetiology research on idiopathic scoliosis, and their results as a basis for analyzing similarities and differences between biomedical research and osteopathic theories.

During the 19th century, three main concepts of causation of IS emerged:
myopathic (Guerin)
malpostural (Lovett)
osteopathic (Schulthess).

The latest research shows that there are a lot of causes for IS being discussed, like genetic factors, structural anomalies, anatomical asymmetric patterns and neurological dysfunctions.

The question of aetiology must be answered if logical preventive and therapeutic measures are to be devised. The word aetiology is used to embrace all aspects of causation for IS although the discussion here relates more to pathogenesis and pathomechanism than to aetiology.

The following studies I am going to present should give an overview of the different discussions and hypotheses regarding the development of idiopathic scoliosis.

6.1. Genetic and epigenetic factors

One of the questions discussed in research on idiopathic scoliosis is if there is a genetic determination or an association between structural genes encoded for different elements of the extracellular matrix. The following two studies are related to this question.

Zaidman et al. (2006) proved a genetic determination of idiopathic scoliosis: Previous studies of Axenovich et al. (1996) of representative samples of pedigrees (360 families in which the proband had II-IV grade IS) had proved major gene control of high grade forms of this pathology. Zaidman et al. (2006) included a search for markers of gene pathology and found them in proteoglycans – the most important parts of the growth plate cartilage matrix. The change in aggrecan gene (they are in all zones of the growth plate) results in disturbance of the growth plate main functions, such as metabolic diffusion, barrier and signal transduction,

chondroblast contact interactions, regulation of cell and matrix reproduction by type of contact inhibition or stimulation of proliferative activity. In this study the point of asymmetry is explained as follows: each vertebral anlage forms automatically, and gene regulation also occurs automatically. Depending on localization of mutant gene expression, either right-side or left-side scoliotic deformity forms.

Pathogenetic mechanism of IS is a manifestation of spine deformity during the time of intensive growth, which is based on an asymmetrical growth disorder (Zaidman et al. 2006). Zaidman et al. (2006) postulated that the process of development assumes the presence of multilevel system of cell specialization, which is a time parameter of the mutant gene switching. Disruption of aggrecan gene transcription occurs in periods of intensive growth; therefore cell matrix reproduction is violated. Aggrecan provides a diffusion of metabolics and a disruption results in fibrotization and dystrophic degeneration of the disc.

From their results Zaidman et al. (2006) defined IS as a “genetically dependent spinal deformity inherited by autosomal-dominant type, with incomplete gender- and age-related penetrance of genotype. Pathogenetic mechanism of spine deformity formation is a mutation in aggrecan gene which encodes synthesis and modification of lateral parts of vertebral bodies” (ZAIDMAN et al., 2006, 16).

In another study Miller et al. (1996) provided the evidence of an association between structural genes encoded for different elements of the extracellular matrix in adolescents from the same family where at least one adolescent had IS. They recruited 96 individuals from 11 different families, 52 of them diagnosed with IS. The authors could not establish an association of genes, including the gene defining the collagen type I, between the members of the same family. No association could be determined between individuals of different families and they also excluded the genes linked to the cause of IS. They affirmed that as scoliosis is transmitted between individuals of the same family, research in larger populations including more families should be incorporated.

6.2. Structural anomalies in idiopathic scoliosis

In this chapter I would like to present some studies dealing with structural anomalies found in idiopathic scoliosis (IS). There are some studies which present imbalance in the connective tissue like elastic fiber imbalance in the intervertebral disc (Yu and Fairbank 2005) or collagen fibers imbalance of the annulus fibrosus in conjunction with spinal growth (Heidari et al. 2003) in IS. A further study shows a unilateral postponement of growth of ligamentum flavum and intertransverse ligament (Van der Plaats 2007).

Yu and Fairbank (2005) investigated the elastic fiber network organization in scoliotic discs in comparison to that in normal human intervertebral discs (IVD), which I will briefly summarise in the following.
33 human IVD were obtained from patients aged 12 to 22 years, undergoing surgery for idiopathic or neuromuscular scoliosis and 2 non-scoliotic patients, one aged 12 after astrocytoma tumor, and the other 17 years old after a spinal trauma. They took snap frozen slices in a radial profile of the disc. The organization of the network varies in the different region of the disc. The organization and the distribution of this network indicate that it fulfils a mechanical role. The elastic fiber network of the non-scoliotic patient's discs reveals a high level of organization. The elastic fibers seen in the idiopathic scoliotic disc tissues appeared sparse and less well organized. There also appears to be a difference between the organization of collagen and elastic fiber network in idiopathic and neuromuscular scoliosis.
The neuromuscular patient discs are more disorganized and elastic fibers appear to be less dense. Also markedly more disorganized fibers were found in ligamentum flavum of some idiopathic scoliosis patients (Yu and Fairbank 2005).

Heidari et al. (2003) also focused on the role of collagen fiber imbalance of the anulus fibrosus but they also wanted to find out in their study if there is a conjunction between spinal deformity and degree of fiber imbalance and if this is influenced by axial growth.
The results support the hypothesis of the project and imply that a greater fiber imbalance will cause a more severe curve. During adolescent growth the curve becomes severe, indicating that, while the induced rotation is independent of fiber elongation, the resulting deformity is directly influenced by the magnitude of the vertical translation or growth.
The study is presented in a mathematical model of the contribution of the collagenous anulus to the spinal deformity. The model was used to study the effect of the fiber imbalance in scoliosis initiation and progression.
The final curvature is influenced by axial growth and fiber ratio, with higher fiber imbalance resulting in more severe spinal deformity.

Van der Plaats et al. (2007) investigated the asymmetrically altered growth in IS by means of a model study. They developed a new finite element model to simulate the mechanical behavior of the human spine, composed by the vertebrae, intervertebral disc, facet joints, spinal ligaments and mm rotators.

The following three theories were analyzed:
1. Buckling of the vertebral column

The underlying assumption is that buckling phenomenon causes an initial lateral curvature with axial rotation, which progresses by growth. Dickson (1992), and Millner and Dickson (1996) mentioned buckling as an initiation of IS by using the linear Euler buckling theory. But this was a linear model that did not include ligaments, articulations or muscles.

2. The theory of Veldhuizen and Herman was “assuming unilaterally postponement in growth of muscles, resulting in asymmetric muscle properties that could lead to asymmetric behavior of the spine.” (Van der Plaats et al., 2007, 1207)

3. Van der Plaats et al.'s own theory was that unilateral postponement of growth of mm rotators (MMR) or of ligamentum flavum and intertransverse ligament could initiate the development of IS (Van der Plaats et al., 2007).

Their model was validated by the earlier stiffness data provided by Panjabi et al. (1976). After a small correction of the prestrain of some ligaments and the mm rotators the model was valid. To investigate the buckling of the human spine as a possible initiation of IS instability of the spine due to an axial downward force was examined.

In the second hypothesis study the postponement in growth was translated in the numerical model in an asymmetrical stiffness. The spine resulting deformation was analyzed for the presence of the coupling of lateral deviation and axial rotation that is characteristic for scoliosis after the spine was loaded axially.
The result of the studies by Van der Plaats et al. (2007) showed that only unilateral postponement of growth of ligamentum flavum and intertransverse ligament appeared to initiate scoliosis. Buckling did not initiate scoliosis, nor did asymmetric stiffness of mm rotators.

6.3. Anatomical asymmetric patterns in idiopathic scoliosis

This chapter deals with studies in which asymmetrical features in the morphology of idiopathic scoliosis were found. Anatomical asymmetric patterns were found in occlusion (Ben-Bassat et al. 2006), and in the sacropelvic morphology (Karski et al. 2006) of idiopathic scoliosis. Mau et al. (1979) described the pattern as “syndrome of contractures” found in newborns and babies. Years later Karski et al. (1995-2006) analyzed children with “syndrome of contractures”, and noted its relevance to some clinical symptoms in children with scoliosis.

The occlusions of patients with idiopathic scoliosis and random controls were examined by Ben-Bassat et al. (2006) to elucidate possible relationships between these two conditions.
96 female and male scoliosis patients aged 13.9 ± 3.5 years, and 703 school aged children in a control group of random were examined by an orthopedic and an orthodontic.
They looked for the

molar relationship
canine relationship
upper midline deviation
lower midline deviation
anterior crossbite
posterior crossbite

Only in A/P dimension the frequency of asymmetrical molar relationships was identical in the scoliosis and the control groups. In the groups of patients with idiopathic scoliosis compared with random controls almost all other parameters of occlusal asymmetry were significantly more prevalent.

Ben-Bassat et al. (2006) showed in their study that patients with idiopathic scoliosis have more asymmetrical features of malocclusion than a random group, and that early detection of asymmetric malocclusion can sound the alarm about possible underlying orthopedic problems.
In the orthopedic literature, Floman (1998) indicated a possible connection between thoracic scoliosis and restricted neck motion in a report of 6 AIS patients. Floman found a marked limitation of neck flexion although the radiological examination including MRI of the entire spine failed to disclose the mechanism which caused the limitation of neck motion. And the discussion is if such a restriction in neck motion has a secondary influence on scoliosis or/and occlusion (Floman 1998).

Karski et al. (2006) presented another possible aetiology of IS in the context of anatomical asymmetrical pattern. They postulated that the malformations of skeletal system can already be taking place in the last months of pregnancy. The deformations are called “syndrome of contractures“, (“Siebener Kontrakturen Syndrom“).
This syndrome has been described by Mau (1979; 1982), Hensinger (1979), Howorth (1977) and others. The causes of the “syndrome of contractures” can be related with fetus itself or with mother conditions (Karski 2006).
Mau gave a detailed description of “Siebener Kontrakturen Syndrom”:

1. scull deformity/plagiocephaly – flattening of left forehead and temple regions, left chick atrophy, eyes asymmetry, nose and ears deformations

2. torticollis- usually left sided. Can be related with plagiocephaly and lack of proper head positioning, and also with primary shortening of stenocleidomastoideus muscle, torticollis with tumor neonatorym
3. scoliosis infantilis – usually right convex lumbo-thoracis curve.
   This type of spine deformity was during many years improperly added to the group of idiopathic scoliosis. This scoliosis usually recedes spontaneusly (disappearence 80% to 100%).
4. contracture of adductor muscles of the left hip. Untreated contracture can lead to developement of hip dysplasia, which primarily can be obseved only at 10% of newbornes.
   The remaining 90% of dysplasia are cases of secondary deformity resulting from the contracture and are classified as „developemental hip dysplasia“ (DDH). Untreated contracture of adductors enlarges dysplasia.
5. contracture of the abductor muscle of the right hip described as Haltungsschwäche by Mau. This contracture may cause oblique positioning of pelvic bone observed at hip joint X-ray picture of babies and young children. With time it may lead to disturbances of biomechanics (asymmetry during gait, asymmetry in growth and developement) and „permanent habit of standing on free only on the right leg“ (the right leg is stronger and more stable due to the contracture!) which in result leads to developement of the so called idiopathic scoliosis (Karski; 1995-2006) .
6. pelvic bone asymmetry – the abduction contacture can influence the pelvis positioning visible during X-ray examination for hip joint screening
7. feet deformities - such as: pes equino-varus, pes equino-valgus, pes calcaneo-valgus, or pes calcaneo-valgus adductus.
   (KARSKI et al., 2006, 34)

Further Karski et al. (2006) reported that the most common first fetus position is left-sided (80-85% of all pregnancies).

The fetus' body, meaning head, trunk and pelvis, is pressed to the left side of mother's spine, and this may result in some typical deformations (primarily unfixed) of the skeletal system.

Karski et al. (2006) focused in their study on newborns. The analysis was conducted in 1999-2001 on 300 histories of babies and children. The age of the children was 3 weeks to 12 months.

In 97 children from this group they noted different symptoms of "syndrome of contractures" (74 girls and 23 boys). The "syndrome of contractures" of the left side was noted in 55 children, of the right side in 42. The relation of left to right was different from fetus positioning (85% :15%).

The analysis by another consulting specialist showed that most of these children were from first pregnancies (80%), mothers had small bellies during pregnancy (mothers' report), and usually the newborns at birth were heavier or longer than normal.

Karski et al. (2006) also analyzed children already diagnosed with scoliosis. The analysis was conducted on 100 histories of children aged 5 to 8 years. In 20 of them they noted abduction contracture of the right hip ranging from 5 to 10 degrees, or adduction movement from 0 degrees, but the left hip at the same time the adduction was 35-50 degrees. In these children, initial stages of the so called idiopathic scoliosis were noted at X-ray examination.

The second group of 80 children with primary “syndrome of contractures“ showed only limitation of right hip adduction in comparison to the left one; adduction of the right hip 10-25 degree, of the left hip 35-50 degree.
In these children Karski et al. (2006) noted initial stages of the socalled idiopathic scoliosis – lumbar left convex, or sacro-lumbar left convex, or lumbo-thoracic left convex.

In another study concerning an anatomical asymmetrical pattern in IS, Mac-Thiong et al. (2006) compared the sacropelvic morphology between normal adolescents and AIS.
There were 27 normal adolescents (normal group), 10 boys and 17 girls, and 29 in the AIS group, 5 boys and 24 girls, aged between 11-15,8 years. The mean Cobb angle of the primary curve was 30-73 degrees.
By radiographs of the spine and complete sacropelvis they examined postero-anterior (PA) and lateral standing (LAT). Based on 26 anatomical landmarks on the PA radiograph and 13 on LAT radiograph and 19 sagittal parameters of the pelvis are computed automatically.
These parameters were used to characterize the complete pelvic morphology for each subject.

The results of this study showed that in the sagittal plane there was no significant difference in sacropelvic morphology between the two groups. Significant differences between AIS subjects and controls were found for coronal parameters involving pelvic height and width (Mac-Thiong et al. 2006).

The significantly different parameters were found in height measurements of the pelvis, which is in the right pelvic length (0,045) and in the right iliac height (0,014). Different parameters were also found in width measurements of the left pubic length (<10), of the right pubic length (0,010), in the left obturator foramen width (0,007), in bicristal distance (0,006), in bituberal distance (0,005), in biacetabular distance (0,001), in pubic symphysis width (0,003), in pelvic inlet (<10), and in the subpubic angle (0,041).

6.4. Neurological dysfunctions in idiopathic scoliosis

In this chapter I would like to present studies which analyze neurological dysfunctions in connection with the aetiology of IS. Hypotheses like maturational delay of the CNS involving undetected neuromuscular dysfunction (Burwell et al. 2006a), and disturbances in the longitudinal growth from bones which results in anomalous extra-spinal left-right skeletal

length asymmetries in the upper limbs, periapical ribs, ilia and lower limbs in AIS were postulated. It is also being discussed if genes and the environment (nature/nurture) may interact pre- and/or post-natally to explain the deformity of AIS (Burwell et al. 2006b).
Differences in dynamic balance between AIS and healthy children were found by Filipovic and Viskic-Stalec (2005). They did investigations and analyses of neurological differences between healthy children and children suffering from AIS. Their study showed weak postural control mechanism and proprioception in AIS. A trend that tonsilar ectopia appeared more often in IS patients, especially those with thoracic or thoraco-lumbar curves, was detected in MRI studies by Sun et al. (2006). Increased tension in the spinal cord which induces the development of IS is hypothized by Royo-Salvador (1996).

Burwell et al. (2006a) tried to explain the aetiologic theories of AIS in their neurodevelopmental concept of maturational delay of the CNS.
The current thinking then was that a defect of central control or processing in the central nervous system (CNS) affects a growing spine with a primary pathology involving the hind brain. How the CNS may be involved in curve progression is still unknown. It is generally considered to result from neuromuscular activity acting on the spine and trunk. But in the absence of evidence either way, curve progression may equally result from a failure of the CNS to control a curve-initiating process at a time of rapid adolescent growth. This may be the result if there is a maturational delay of the CNS body schema.
Burwell et al. (2006a) postulated four theoretical requirements about the CNS body schema concept for the development of AIS.

1. Curve-initiation process produced by left-right spinal asymmetry caused by vertebral body growth plates, or possibility periapical rib length asymmetry as a relative concave rib overgrowth or neuromuscular mechanism may cause the left – right asymmetry and initiate scoliosis in some subjects.
2. Rapid spinal elongation in adolescent growth spurt, principally of vertebral body growth plates under the influence of steroid hormones particularly estrogen.
3. Maturational delay of CNS body schema, this causes neuromuscular adjustments to a deforming and rapidly elongating spine. There is also a requirement that focal brain atrophy in progressive AIS will be shown.
4. Upright posture and movement of spine and trunk suggests in scoliosis curve progression.
   (BURWELL et al., 2006a, 74-75)

A study by Arkin (1949) stated that rest in bed may halt the progress of IS in children. He kept over 30 scoliotic children in bed for 22 hours a day. Except for one case, no progress was noted after 3 months.

Burwell et al. (2006a) introduced 4 hypotheses to explain where in the CNS body schema concept maturational delay may arise and cause AIS:

1. Impaired sensory input; the basic problem may lie in muscle spindles or other endings. In AIS patients they found abnormal reflex processing which may be associated with delay in maturation of the CNS body schema.
2. Primarily in the brain; parts of the brain that may contribute to maturational delay of the CNS body schema include in the parietal lobe, the somatosensory cortex (personal space of "self"), temporparietal junction, temporal lobes, frontal lobes, and visual cortex (extra personal space).
3. Impaired motor output; in a study by Herman et al (1985) it is shown that processing of vestibular signals within the CNS yielded the highest degree of correlation with curve magnitude. They considered that IS was a motor control problem. A higher level CNS disturbance was thought to be responsible for visuo-spatial perceptual impairment, motor adaption and learning deficits. These lead to a recalibration of proprioceptive signals from axial musculature causing IS.
4. Relation to the NOTOM hypothesis; the CNS body schema concept can be viewed as resulting from a abnormality in neuroosseous timing of maturation (NOTOM)
(BURWELL et al., 2006a, 76)

In a further study, Burwell et al. (2006b) developed theories about disturbances in the longitudinal growth of paired bones (long limb bones, ribs, ilia) and united paired bones (vertebrae, sternum, skull, mandibulae).

They postulated that it is evident that human vertebral body growth plates like other physes, during their years of functional activity liberate cascades of cells that respond symmetrically to successive hormones during growth. Hormones are secreted as postnatal development proceeds. These are in fetal life insulin and IGF-1, in the early postnatal life: growth hormone; and in puberty steroids including estrogen and androgens. Receptors on the cell surface or nucleus respond to these hormones.

Burwell et al. (2006b) claimed that genetic and environmental factors may disturb symmetry control in separate and united enantiomorph bones. These factors are said to be acting directly or indirectly on developing skeletal primordial in early embryonic life as a very complex disorder of differential growth in the skeleton.

Burwell et al. (2006b) also reported that progressive Adolescent Idiopathic Scoliosis (AIS), which mainly affects girls is generally attributed to relative anterior spinal overgrowth from a mechanical mechanism (torsion) during the adolescent growth spurt, and that established biological risk factors for the development of AIS are growth velocity and potential residual spinal growth assessed by maturity indicators.

Goldberg et al. (2000) reviewed left-right directional asymmetries in AIS and wrote: "Scoliosis is not a disease or group of diseases but a symptom or sign of environmental stress,

significant enough to overwhelm the intrinsic stability of the morphological genom. As such there is no specific etiology but a large number of precipitating stressors“ (GOLDBERG et al., 2000, 327).

There are also chemical risk factors and some dietary factors discussed by Mac Master et al. (2004). They reported evidence that some infants exposed to indoor swimming pools in the first years of life show an association with progressive AIS and in controls spinosus process asymmetry.

Barker et al. (2002) showed that the origins of important chronic system diseases of adult life including stroke, coronary heart disease and type2 diabetes as well as rates of aging, may lie in fetal responses to the intra uterine environment. It is termed the “fetal origins hypothesis“, and has led to national medical research projects being developed in the UK and USA.

The breaking of bilateral symmetry was obtained in a further study by Burwell et al. (2006c), in which they focused on mechanisms initiated in embryonic life including a disturbance of bilateral (left-right or mirror-image) symmetry highly conserved in vertebrates. They stated that normal external bilateral symmetry of vertebrates results from a default process involving mesodermal somites. The normal internal asymmetry of the heart, lungs, gut with its glands, major blood vessels is also highly conserved among vertebrates. It results from the breaking of the initial bilateral symmetry by a binary asymmetry switch mechanism producing asymmetric gene expression around the embryonic node and/or in the lateral plate mesoderm. In the mouse, this switch occurs during gastrulation by cilia, driving a leftward flow of fluid and morphogens at the embryonic node (nodal flow) that favors precursors of the heart, great vessels, and viscera of the left. The hypothesis of this study is that an anomaly of the binary asymmetry switch explains the excess of right/left thoracic AIS. They think that there is evidence that vertebrates within their bilateralized shell retain an archaic left-right asymmetric visceral body organization evident in thoracic and abdominal organs (Burwell et al. 2006c).

Kouwenhoven et al. (2007) presented in a cross sectional magnetic resonance imagine study, vertebral rotation measurements of the normal, non-scoliotic spine of persons with a complete mirror image reversal of the internal body organs, called situs inversus totalis. The results showed in the normal spine of humans with situs inversus totalis a pre-existent pattern of vertebral rotation opposite of what is seen in humans with normal organ anatomy, that is a predominant rotation to the left side of the mid and lower thoracic vertebrae, and to the right side of the upper thoracic and lumbar vertebrae.

Also focusing on neurological dysfunction in IS, Filipovic and Viskic-Stalec (2006) observed the mobility capabilities of AIS persons (total 38) and a control group (total 36). In the AIS group there were 21 persons with a Cobb angle < 25° and 17 persons with a Cobb angle > 26°.

The age in all groups was between 9-14 years; there were 2 different step tests applied; the left and the right step test. The tests were distinguished according to step on a 16inch tall bench and step down onto the platform for the base reaction force within 15 seconds in a normal rhythm. The amplitudes of force were considered by electromyography from muscles (m gluteus maximus sinister and dexter, mm erector spinae sinister and dexter) and by the platform for the base force reaction.

The results of this study showed that there was a significant difference between AIS and the healthy group on the left step test, and that the pathological form of AIS highly affects dynamic balance.

The values of the lumbar erector muscles and right gluteus maximus, and the side-to-side reaction of the platform are more pronounced than the other variables. There was no significant difference between the various Cobb angles in the AIS groups.

Filipovic and Viskic-Stalec (2006) presented that AIS affect dynamic balance and illustrated the compensational functioning of mobility, especially when there is a lack of normal mobility forms and there are weak postural control mechanism and proprioception.

Another study by Weiss and Lehmkuhl (1996) also determined that persons with AIS had the ability to stand, walk, run, and jump over barriers, but the movements involved are not economical, and that such participants tend to tire sooner, be slower and less coordinated than the healthy ones. Their movement function is reduced.

Dynamic balance is affected in AIS patients (Filipovic and Viskic-Stalec, 2006) which may indicate disturbances in the cerebellum, Sun et al. (2006) investigated the position of the cerebellar tonsils in AIS patients by a MRI-study.

The group comprised 205 AIS patients with a Cobb angle of more than 40 degrees, consisting of 27 boys and 178 girls. In the control group there were 86 healthy adolescents, 43 boys and 43 girls.

All patients were aged from 12–18 years. MRI examinations of the whole spine from foramen magnum to the sacrum were performed in both groups.

The connecting line between the basion and the opisthion (BO line) of the occiput were drawn, representing the level of the foramen magnum.

Then the most inferior part of cerebellum or tips of the cerebellar tonsils was selected for further measurements. The perpendicular distance from the inferior part of the cerebellar tonsil to the BO line was determined.

The study by Sun et al. (2006) showed that AIS patients had lower positions of cerebellar tonsils. Extend of tonsilar ectopia of more than 2 mm and 5 mm below the BO line was found in 13.3% (27/205). The incidences of tonsilar ectopia in AIS were 35.1% (72/205) in controls 5.8% (5/86).

In AIS patients they found no significant correlation between the tonsil positions and curve severity. The most frequent incidence of tonsilar ectopia was 62.5% in patients with a double thoracic curve, 39.3% in a right thoracic and left lumbar curve, 37.3% in a right thoracic curve, 36.4% in a thoraco-lumbar curve and 21.6% in a lumbar curve.

In the patients with a left thoracic curve, the tonsil position was identified to be 3.2 mm above the BO line. Patients with a lumbar curve had a significantly lower incidence than with thoracic or thoraco-lumbar curves. The results of Sun et al. (2006) also suggest that there might be associations of proprioception defects with tonsilar ectopia in AIS.

While Sun et al. (2006) in their MRI study showed the lower positions of the cerebellar tonsil in AIS, Royo-Salvador (1996) had hypothized already years before that tensions between cranium and sacrum are not transmitted through the dura mater spinalis but via the spinal cord, that this induces the lower position of the cerebellar tonsils, and also causes the development of scoliosis. He noticed an abnormal increase in tension of the medullar traction in IS and syringomyelia patients. According to Royo-Salvador (1996), an abnormal intensity of the medullar traction leads to the following effects on the cranial level: caudal traction at the truncus cerebralis and rise of tension of the surrounding dural meninges and the periostal attachment of the meninges, e.g. tentorium cerebella. The tonsillae cerebella are drawn to inferior and compressed, resulting in a deformation of the 4th ventricle, an increase in the basal cranium angle, a deformation of the clivus as well as an approximation of the pars petrosa (os temporale) and os sacrum. The cerebellar hemispheres are pushed into the fossa cranialis posterior, which leads to a deformation of the foramen magnum.
On a cervical level the caudal traction on C1/C2 level is related to an anteroposterior force and thus related to a posterior swinging movement of the dens of C2. In the cervical area the nerve tissue is most affected, resulting in compression, ischemia and necrosis.
In the thoracic region the result of the study provides an explanation for the development of idiopathic scoliosis. The spine tries to reduce the tension caused by the spinal cord. The formation of a thoracic curvature reduces the effect of the medullar traction. An abnormal traction of the spinal cord especially in the thoracic area leads to the development of scoliosis. They are caused by compressions that develop increasingly during the growth

period. Increase in pressure on the vertebral body growth plates (plaques epiphysaire) decelerates the growth of this part of the vertebra relative to the other parts and thus causes a deformation of the vertebral column.
The rising pressure leads to an increase in the density and the trabeculae inside the vertebra. An increased intensity of the medullar traction leads to lowering of the conus medullaris in the lumbo-sacral region and a rise in tension at the filum terminale. A rise in tension at the filum terminal can affect the dural bag and harm it. According to Royo-Salvador (1996), a power transmission takes place via the spinal cord from the os sacrum to the inside of the cranium.

6.5. Other relevant studies

In this chapter I will present other relevant studies dealing with the aetiology of scoliosis from a biomedical view. The contents of these studies emphasise various aspects of scoliosis. They deal with visual deficiency (Grivas et al. 2006), handedness and spinal deformity (Goldberg et al. 2006), the specific morphological manifestations in idiopathic scoliosis (Sevastik 2006), the degree of mineralization in IS (Yeung et al. 2006), and the incidence and outcome of scoliosis in children with pleural infection (Mukherjee et al. 2006).

Grivas et al. (2006) investigated if there is an association between visual deficiency and IS. 26 totally blind Greek women aged 20-67 were screened for scoliosis. In the forward bending test using the Pruijis scoliometer, 11 of 26 women had more than 7 degrees and this was a cut off criterion for radiological examination.

In 11 of 26 persons, they identified an average Cobb angle of 19 degrees (range 12-28). Thoracolumbar was the most common type of curve identified (9 out of 11; 6 were to the right, and 3 to the left). Furthermore, Grivas et al. (2006) recorded the circadian rhythm of the probands. None of the blind women reported any sleeping difficulties, and they had a normal circadian rhythm related to a 24-hour day. The result showed a prevalence of scoliosis in women with visual impairment at 42.3% against that of the general population in Greece of 2.9%.

Grivas et al. (2006) concluded that there is an association between idiopathic scoliosis and postural control of the trunk in which vision is involved. The results of the study also suggest that melatonin might be involved in the pathogenesis of human idiopathic scoliosis in a way that could explain the higher prevalence of scoliosis in people with visual impairment.

The melatonin overproduction in these people leads to late menarche and longer exposure to possible coexisting detrimental factors in the pathogenesis of idiopathic scoliosis (Grivas et al. 2006).

Another topic discussed with regard to the aetiology of scoliosis is if there is a connection between handedness and spinal deformity. Because of the hypothesis that handedness causes curve patterns in scoliosis or vice versa, Goldberg et al. (2006) did a study with patients from the computerized scoliosis database. Altogether they examined 1,477 patients in five groups (minor asymmetry, AIS, juvenile idiopathic scoliosis, infantile idiopathic scoliosis, and congenital vertebral anomalies). The data recorded included gender, diagnosis, curve pattern and preferred writing hand.

The group with the minor asymmetry (500 in total, not confirmed by radiograph) showed that the incidence of left-handedness was 10% in boys as well as in girls, and does not differ from that observed in the wider human population. Statistically there was a significant association between hand preference and lateralization of asymmetry.

In the group with AIS (total 673, confirmed by radiograph) the handedness in boys does not differ from the population mean and does not correlate with scoliosis patterns. The incidence in left-handedness in girls with AIS was 6.9% which is less than that for the normal population. There was also a statistically significant correlation between scoliosis patterns and hand preference.

In the group with juvenile idiopathic scoliosis (total 102, and age 4-10 years), the handedness in boys was the same as the population mean and did not correlate with scoliosis patterns. The incidence of left-handedness in girls was 16.7%, which is significantly more than that for the normal population. Left thoracic patterns in girls were 6.7% and therefore much lower than usual (20%) but did not correlate with hand preference.

In the group with infantile idiopathic scoliosis (total 50, and age < 4 years), the incidence of left-handedness in boys was 26%, in girls 48%, which is significantly higher than that in the normal population. The curve pattern does not match the usual 80% right thoracic. There was no correlation between handedness and curve pattern.

In the group with congenital vertebral anomaly (total 152); the girls had an increased incidence of left-handedness (18%) but there was no correlation between curve pattern and handedness in boys and girls.

The results of a study by Milenkovic et al. (2004) showed that left-handedness was significantly associated with scoliosis in girls after screening 2,546 schoolchildren. But they made no reference to radiological investigations. There was no association found in the study by Goldberg et al. (2006), so maybe Milenkovic's study was based on physical examination only.

To suppose that handedness can determine a posture that is so detrimental as to cause real spine deformity is to simply not understand how scoliosis evolves. Furthermore, Goldberg et al. (2006) concluded that the non-correlation between hand-preference and scoliosis pattern refutes this concept.

In a review which Sevastik (2006) called "the thoracospinal concept" he postulated that the ethiopathogenesis of the pathological complex of the right convex female adolescent scoliosis demands research from new standpoints. He showed that the majority of all cases of idiopathic scoliosis are characterized by a combination of specific morphological manifestations and signs of physiological abnormalities.

Sevastik (2006) defines the specific morphological manifestations as follows:

1. Laterality and pattern of the curve
Right to left convex curves in infantile idiopathic scoliosis is 86 to 14 with 11 % resolving, 42 % are thoracic, 16% lumbosacral and lumbar and 22% are double primary.
2. Height and weight
Girls with double primary and thoracolumbal-curved adolescent idiopathic scoliosis have been reported to be significantly taller at the time of menarche than girls with right convex thoracal curves. The weight of the girls with all curve patterns was significantly lower than that of the controls, essentially in infantile idiopathic scoliosis and juvenile idiopathic scoliosis.

The specific physiological characteristics according to Sevastik (2006) are:
1. Gender: the mean reported percentage of girls-to-boys ratio is 8 to 52 in IIS, 76 to 24 in JIS, and 85 to 15 in AIS.

2. Menarche: AIS girls with a double primary or thoracolumbar curve were significantly older and taller than the control girls or girls with right convex thoracal curves at the time of menarche.

3. Sympathetic dysfunction: the vascularity of the breasts in girls with AIS and matched normal controls was measured. In normal girls there was no significant difference in the AIS group while the vascularity of the left breast on the concavity of the thoracic curve was significantly increased as compared with the right one.

4. Abnormalities of muscle fibers and platelets

A decreased number of type II fibers in paravertebral muscles was reported by Yarom et al. (1979) and an increased number of type I fibers on the concavity rather than the convexity of the curve were found.
Also larger platelets than normal, decreased activity of intracellular contractile proteins with decreased platelet aggregation and increased platelet calmodulin levels were reported by Lowe et al. (2000), for patients with AIS.

5. Osteoporosis
Reports by Cheng et al. (1997) provided the evidence that girls with AIS are affected by osteoporosis which persists until late in life (Velis et al. 1989).

In his review article Sevastik (2006) also mentioned an investigation of the impact of increased vascularity of the concave hemithorax and rib length measurement. The ribs were measured from cadaveric specimens of 10 elderly women, 8 men with normal spines, and 15 women with scoliosis.
The result showed that in the scoliotic group the rib length was significantly different from the normal one. The ribs on the concave side were 4-7mm longer than those on the convex side (Sevastik 2006).

In the same review Sevastik (2006) also mentions another study by Yarom et al. (1979), done on growing rabbits to determine that IS is induced by rib length asymmetry. Yarom et al. (1979) shortened the ribs of growing rabbits with the result that the scoliosis developed progressively with a concavity to the side of the shortened ribs.

The studies led to two different results: Sevastik (2006) postulated the longer ribs on the concave side, in the other study by Yarom et al. (1979) the progression of scoliosis appeared with a concavity to the shortened ribs. They clearly do not correspond to each other.

Another factor discussed in the context of the aetiology of IS is the lower degree of mineralization found in IS. Yeung et al. (2006) observed the degree of mineralization of the bone matrix in the radius.

78 girls with AIS aged 15-18 were recruited from a scoliosis clinic; they were divided into two groups: AIS girls with a Cobb angle between 20-40° and more than 40°. The control group consisted of 45 age-matched healthy girls. The midshaft and the distal region of non-dominant radius were measured with a high-precision peripheral quantitative computed tomography. The length of the radius as the distance from the radiale to the stylion was also measured.

The cortical bone mineral density (cBMD) of AIS girls was significantly lower than that of the control subjects by 1.7%; there was no significant difference in the length of the radius.
Yeung et al. (2006) explained the lower degree of mineralization of cortical bone in AIS girls by the rate of bone modeling during puberty. When remodeling activity is high, there will be more unmineralized osteoid and more “young“ bone matrix, which has not yet completed mineralization.

It indirectly indicates that AIS girls may have a higher remodeling rate to cope with rapid growth. During pubertal growth, the bone modeling of the cortical bone is mainly through membranous ossification which regulates the bone size and the quantity of bone. The low cortical BMD in AIS girls indicates the presence of an abnormality in membranous ossification (Yeung et al. 2006).

With reference to the fascia system and the influence of this connective tissue I would like to present a study which shows the incidence and outcome of scoliosis in children with pleural infection. Mukherjee et al. (2007) ascertain the incidence and outcome of secondary scoliosis associated with parapneumonic effusion/empyema. In a pedriatic respiratory center in London they reviewed 122 children in a 3-year period 2002-2005 with a diagnosis of pleural infection by digitalized erect chest radiographs. Two observers measured the Cobb angle of the thoracic spine. The children were aged between 4 months to 15.8 years. There were 73 boys and 49 girls, all receiving intravenous antibiotics, and chest drains were inserted in 103/122 (84%). In all the cases, there was a single thoracic curve with the direction of scoliosis towards the side of the unaffected lung. There was no association between the presence of scoliosis, age, gender, size or type of effusion, and inflammatory marker. Overall, there were 87/122 (71%) children with a scoliosis from 10-30° at some stage during

the admission. At follow-up, 6 (5%) had a mild residual scoliosis but all subsequently resolved.

Mukherjee et al. (2007) further reported that whilst pain from pleuritic irritation or a drain may be an important factor it cannot be the only one since the children receive major analgesia and non steroidal anti-inflammatory drugs for most or all of the time their drain was in place.

Summary

The unknown aetiology of idiopathic scoliosis has generated numerous hypotheses supported by clinical observations, the results of experimental studies or by pathophysiological concepts. To sum up the results of these studies and critically consider should be the aim of this conclusion.

Genetic factors: A deviation of the vertebral column is found in IS but is also present in other syndromes (Marfan syndrome, etc.). With the aid of genetics, mutations of certain genes have been held responsible (Zaidman et al. 2006) for the emergence of these pathologies but further research is required to prove this.

Structural anomalies: Differences in the organization of connective tissue in IS were found by Yu and Fairbank (2005). The elastic fiber network appeared less dense und unorganized in the IVD and also in the ligamentum flavum. The model studies showed that axial growth and higher fiber imbalance result in more severe spinal deformity (Heidari et al. 2003). Also unilateral postponement of growth of ligamentum flavum and intertransverse ligament appeared to initiate scoliosis (Van der Plaats et al. 2007). It is not at all clear, however, whether these defects are primary or secondary, whether function governs structure or vice versa.

Asymmetrical patterns: More asymmetrical features of malocclusion in IS than in healthy children were found (Ben-Bassat et al. 2006). Also significant differences in the pelvic height and width in AIS were shown (Mac-Thiong et al. 2006). In the study by Karski et al. (2006), further asymmetries like scull deformities, torticollis, hipabductor and hipadductor contractures, pelvic bone asymmetries and feet deformities were postulated. The cause of the development of these asymmetries which are maybe initiating scoliosis is unclear. Hypotheses are discussed that deformations were already taking place in the last months of pregnancy (Karski et al. 2006).

To evaluate the danger of oncoming scoliosis, newborns, babies and children should be examined in detail so that early detection can sound the alarm about possible underlying orthopedic problems.

Neurological dysfunctions: In one study AIS coincided with lower positions of cerebellar tonsils with a possibility of an association of proprioception defects (Sun et al. 2006). Abnormal reflex processing which may be associated in maturation of the CNS and abnormality in neuro-osseous timing of maturation are discussed (Burwell et al. 2006a). Disturbances in the longitudinal growth of bones caused by genetic and environmental factors which disturb symmetry control in the growth of the bones are described (Burwell et al. 2006b). According to Royo-Salvador (1996), significantly more tension in the medulla traction leads to more tension in the dural membranes and causes a deformation in the foramen magnum and initiates scoliosis in the thoracic spine. Also the breaking of the initial bilateral symmetry by a binary asymmetry switch mechanism producing asymmetric gene expression around the embryonic node and/or in the lateral plate mesoderm are discussed (Burwell et al. 2006c).
Cheng et al. (1997) concluded also that there is a clear association between AIS and generalized osteopenia and opined that intra-skeletal mechanisms can contribute to the pathogenesis of AIS.
Lowe et al. (2000) reported that some of these abnormalities appear to be related to a defect in the cell-membrane in patients with AIS.
Multiple pathological biochemical and histological changes in IS patients have been presented in studies by Cheng et al. 1997; Yeung et al. 2006; Lowe et al. 2000 and others. The importance of the research of these complex and probably multi-factorial processes seems obvious.

It is likely that the cause of scoliosis is multi-factorial with the factors assuming different degrees of importance in individual patients. In the study titled "Incidence and outcome of scoliosis in children with pleural infection" (Mukherjee et al. 2007), it is stated that inflammatory processes in the trunk could be a possible cause in the development of scoliosis at some stage. It is interesting to follow up and investigate over a longer period of time if during the time of growth there would be a further incidence for the development of scoliosis.

Altogether, the current stage of knowledge of the aetiology of idiopathic scoliosis is based on the results of studies like the ones presented above but none of the existing studies offers a comprehensive explanation of the causes of scoliosis.

7. Osteopathic theories for the aetiology of scoliosis

This chapter deals with osteopathic models which can serve to explain the development of idiopathic scoliosis.

Literature research regarding this topic has not yielded much data.

(Research and personal communication at the European School of Osteopathy in Maidstone and at the Wiener Schule für Osteopathie / Vienna School of Osteopathy (WSO); literature search: MEDLINE, EMBASE, CINAHL, BIOSIS Previews, EBM Data Bases, and Osteopathic Research Web).

The following discussion is based on personal communication and on some statements in the osteopathic literature about the aetiology of idiopathic scoliosis.

First I would like to comment on osteopathic terms like "dysfunction", "midline", and "SSB torsion". These terms are, especially within the cranial concept of osteopathy, "created terms", which are based on palpation experience and on accounts by experienced osteopaths. There is hardly any empirical evidence of causal relations which are postulated for the cranial sphere.

The term "dysfunction" is used frequently in osteopathy and also in this chapter of my survey, and describes a "Veränderung der physiologischen Beziehung innerhalb eines Gewebes oder zwischen verschiedenen Organstrukturen“ (a change in the physiological links within tissue or between distinct organ structures; LIEM 2001, 10). Mechanical or physiological reactions to external or internal influences occur on osseous, fascial, muscular, visceral or fluidal levels, according to Liem (2001).

Liem (2001) also lists these influences which are genetic, intrauterine or traumatic impacts, the aftermaths of operations, dental invasion, illness, dietary or environmental influences. These lead first to subtle restrictions in movement in the tissue affected and to disruptions of physiological processes (Liem 2001).

“Dysfunction” in this paper refers to the disruption of a physiological process, which osteopathy takes for granted but which cannot be proven biomedically, and which may lead to the development of idiopathic scoliosis.

Finally, I would like to report the results of my personal communication with three experienced osteopaths who teach at the WSO as well as the results of my literature research regarding the development of idiopathic scoliosis, as postulated by the following authors: Blechschmidt (1982, 2002), Carreiro (2003), Frymann (2007), Fossum (2003), Liem (1998, 2001, 2006), Magoun (1973), Möckel (2006), Sergueef (1995) and Zink (1979).

Personal communication was rather restricted contentwise as I had to raise my questions during breaks and classes, and I at this point still expected more substantial literature regarding this topic.

Patrick Van den Heede (2006), e.g., regards the development of idiopathic scoliosis as an embryonic dysfunction in the build-up of the brain and the heart. Hanneke Nusselein (2006), by contrast, answered that there are really many influences discussed, which may cause scoliosis, for instance that it starts in the embryology and the possibilities of dysfunctions in the time of development, or dysfunctions during the birth process, as SSB torsion, and/or dysfunctions in the pelvic region, especially in the sacrum. Also dysfunctions on a bony, membranous or fluid level during growing up have been discussed as possibly inducing the development of scoliosis.

Jane Carreiro (2005) stated – from a biomedical view – that it is unclear whether there is merely "one cause“ which initiates the development of scoliosis. Influences such as vestibular dysfunctions or cortical asymmetries and uneven leg lengths are discussed. Higher levels in platelet calmodulin in skeletally immature IS patients have also been found. Finally, she thought that we may need to view scoliosis as a symptom common to several pathologies, which is the same she postulated in her publication (Carreiro 2003).

Nusselein (2006) also stated in our conversation that the development of idiopathic scoliosis shows multifactoral origins whose primary cause can already be attributed to a dysfunction in embryology.
Scoliosis is defined as a partial structural lateral curvature of one or several parts of the spine which co-occurs with a rotation, torsion and structural alteration of the spine (Humpke 2002). Curvatures of less than 10 Cobb degrees are not considered scoliosis (Weinstein 1985), which means that in scoliosis there is a more severe asymmetry. It is not clear what the cause fort his increased deviation of the spine from the midline is.
According to Liem (2006), however, there is no complete symmetry in the body, the brain or the spine.
"Symmetry“ in the skeletal system is thus a sensitive topic, and the idea of "norm" behind deviation in the spine or the entire skeletal system should be reconsidered in light of new research findings. A MRI-study by Kouwenhoven et al. (2007) shows, for instance, that in humans the mid and lower thoracic vertebrae of the normal, non-scoliotic spine shows a pre-existent pattern of rotation to the right side. This rotational pattern is similar to what is seen in most of the prevalent types of adolescent idiopathic scoliosis and, therefore, probably plays

an important role in determining the direction of spinal curvature once scoliosis starts to develop. The cause of this pre-existent rotation, however, is unknown (Kouwenhoven et al. 2007). If this pre-existent pattern of rotation to the right side were the "norm" and only increased deviation of the spine induced a scoliosis, the question would arise as to which influences cause it.

"Symmetry“, "middle“, and "midline“ are expressions which will occur more frequently when dealing with my topic, and will therefore be investigated in greater detail by looking at select postulations. The term "midline", e.g., is used frequently in human embryology research and in osteopathy. The well-known human embryology scientist Erich Blechschmidt (1902-1992) assumed that every structure realigns itself around a certain primary midline or matrix of a fluid potency as e.g. the linea primitiva as a fluctuating midline or as one positioned in the chorda dorsalis. Blechschmidt (2002) states that spiraling development movements are always excentric in view of the midline. Furthermore, Blechschmidt (2002) describes structures of the midline which are an integrative point for asymmetries and fulfill a symmetry-stabilizing function. Blechschmidt did not explicitly comment on the development of scoliosis but hypothetically, and with a view at my topic, a dysfunction in this developmental stage can cause later scoliosis.
Emotions also orient themselves towards a certain midline, according to Liem (2006). Traumata and other factors can disturb the relationship of median and peripheral structures to a primary midline (Liem 2006).

In osteopathy, "midline" denotes an entity determined by palpation through experienced osteopaths which cannot be scientically proven, and is described differently by different people. Dunshirn's osteopathy master's thesis (2007) presented the various interpretations of the term “midline”. In this thesis by Dunshirn (2007), seven experienced osteopaths who have expert knowledge of midline osteopathy were interviewed and the analysis of the interviews revealed different possible approaches.

Accordingly, there are various definitions of the term, postulated by e.g. Patrick van den Heede (cf. Liem 2006), who differentiates the "midline" into:
-- a dorsal midline: neural tube; the point of equilibrium is the Sutherland fulcrum, in which exchange and memorization occur.
-- a middle, fluid midline, formerly ventral midline; from the chorda dorsalis to the sphenoid and the cellulae ethmoidales; point of reference: nasion to sacrum; point of equilibrium: the heart; serves as support of the body.

-- anterior midline: its development is caused the former two; nose-hyoid-sternum-xiphoid-linea alba-symphysis pubis; develops through the contact points of flushing-out dorsoventral growth movement.
Point of equilibrium: the hyoid; in the anterior midline the potential body manifests itself.
According to Van den Heede (cf. Liem, 2006), expansion occurs through the lateralization of the midline.
For the expansion to develop, the concentrical power of the midline is required as polarity.
Following a statement by Van den Heede (2006) that describes the cause of idiopathic scoliosis as a disruption in the build-up of the brain and the heart in embryology, the assumption suggests itself that a "dysfunction" in the middle fluid midline occurs.

James S. Jealous describes according to Liem (2006) the midline as a bioelectric line, which results from the development of the chorda dorsalis, and which represents a primary orientation for the spatial organization in the organism.

I would like to return to Van den Heede's assumption (2006) that the cause of idiopathic scoliosis can be found in a dysfunction in the build-up of the brain and the heart during embryology, and that this developmental stage can be located during gastrulation (14^{th}-21^{st} day) up to the eighth week, and provide a brief overview of embryology in this period, based on Rohen (2004):
Day 14-21
Organization of the primary trophoblast villi into secondary villi.
After the growing-in of embryonic blood vessels tertiary villi develop.
Reduplication and branching of chorionic villi which are anchored in the endometrium (decidua). Development of a consistent intervillous space which is filled with maternal blood.
In the epiblast of the germinal disc a primitive streak forms, as an infolding zone in which cells can commute between epiblast and hypoblast. The bottom of the amnionic cavity turns into the ectoderm of the embryo. At this stage, the definite S/I midline takes shape (superior-inferior midline). The midline develops in two parts: first, the caudal part (primitive streak and primitive cavity), then the cranial part (notochord and neural plate). These two parts meet in one point, the primitive node (a small bulge in the germinal disc, where the ectoderm aligns closely with the entoderm).
Determination of bilateral symmetry of the later embryonic body.
Development of the third amnionic layer (mesoderm) between epiblast (ectoderm), and the entoderm instead of the hypoblast.
Infolding of the notochordal canal ("gastrulation“) and induction of the neural tube (neurulation) in the ectoderm above. From a biodynamic perspective, the neural pit is

transformed into the neural tube, because the future pia-mater side of the cells grows faster than the ventral side. The occlusion of the cranial end of the neural tube is performed by the lamina terminalis.
This structure of the midline also exists in grown-ups as an anterior wall of the third ventricle, and turns into the pivotal point of all neural movements.
Division of the middle amnionic layer (mesoderm) into four parts:
1. Notochord (axial mesoderm, axis of the embryonic body)
2. Somites (paraxial mesoderm, cube-shaped "proto-vertebrae")
3. Somitic stems (intermediary mesoderm; they constitute the link between somites and lateral plates)
4. Lateral plates (somatopleura dorsal and splanchnopleura ventral)

Embryogenesis (22nd to 56th day) The large organ systems develop (Rohen, 2004):
Conversion of the flat germinal disc into a three-dimensional embryo, and development of the embryonic body (through developing the intraembryonic mesenchym).
Development of elementary functional systems (neural tube, gut tube, cardio-vascular system etc.)
Rudimentary limbs develop

4th week (Rohen, 2004)
22nd day a uniform heart tube is formed and starts to pulsate
24th day the neural tube is still open at both ends
26th day pharyngeal arches and pouches develop
28th day closure of the neural tube and development of the brain vesicle, the lens placode and the otic vesicle
Strong flexion of the embryonic body and differentiation of the umbilical chord.

In order to elaborate on the statement by Van den Heede (2006) even further, I would like to integrate brief descriptions by Blechschmidt, according to which the visage lies in the field between the brain and the heart ("das Antlitz im Wirkungsfeld zwischen Gehirn und Herz liegt“; BLECHSCHMIDT 2002, 56). Following Blechschmidt (2002), already in the fourth week, when the somites evolve, the dura tightly embraces the neural tube ventrally and thickens ventrally much more considerably than dorsally throughout development. Through this growth trait it causes the development of the main sensitive ganglia and the dorsal nerve root dorsally at the neural tube. The embryonic heart follows the diaphragm in a descending movement, observes Blechschmidt (2002), while the brain in its growth process increasingly

ascends. Blechschmidt describes further that the dura of the sinciput with the falx moves further away during the ascending growth process of the brain from the descending ligament system of the cervical viscera. Thus the facial connective tissue tightens muzzle-like between falx and hyoid, and a "long face" develops (Blechschmidt 2002).
Dysfunctions in this growth stage may give rise to the causality of the development of idiopathic scoliosis. Similarly, a dysfunction in this area can possibly explain the reinforced asymmetry of the cranium which is again hypothetically connected to the development of idiopathic scoliosis (Liem 1998).

5th week (Rohen, 2004)
Intensive development of the head (brain and sense organs)
Differentiation of the rudimentary thyroid and lungs
Development of the heart loop and pharyngeal arch artery
Development of the embryonic kidney from the uretheric buds and the metanephric tissue
Limb buds start to evolve (arm buds on day 33)
First spontaneous movements of limbs and torso

6th week (Rohen, 2004)
Strong flexure of the head (cervical flexure of the brain)
Extension of the telencephalon over the diencephalon
Rudiments of auricle and external auditory meatus
Pigmentation of rudimentary eyes
Development of the primary palate and nasal pits
Lung buds start to branch
Separation of heart atria and development of foramen ovale secundum
Development of uretheric buds and metanephric system
Rotation of the umbilical loop

7th week (Rohen, 2004)
Involution of the yolk sac, connection only via thin ductus omphaloentericus
Differentiation of ductus semicirculares and cochlea
Facial swellings fuse
Separation of the heart through septum aorticopulmonale und endocardial cushions
Occlusion of foramen interventriculare and formation of two ventricles
Physiological umbilical hernia through shift intestinal loops into the umbilicus
Differentiation of ductus paramesonephricus.

8th week: End of the embryonic period (Rohen, 2004)
Face develops human features (eye lids, auricle)
Back-formation of the cauda
Beginning differentiation of external genitalia
Elongation of the limbs and clear differentiation of fingers and toes
Reflectory reactions of the embryo to tactile stimuli

After this account of embryology I would like to move on to dysfunctions in the cranial area. In order to illustrate Nusselein's opinion, which among others refers to SSB-torsion as one of the possible causes of idiopathic scoliosis, the term SSB-torsion will be explained in greater detail. According to Liem, the synchondrose sphenobasilaris (SSB) ossifies between 18 and 25, but normally retains certain flexibility until old age (Liem 1998). SSB torsion is defined in osteopathy as the reverse rotation of os sphenoidale and os occiput around an antero-posterior axis (Liem 2001).

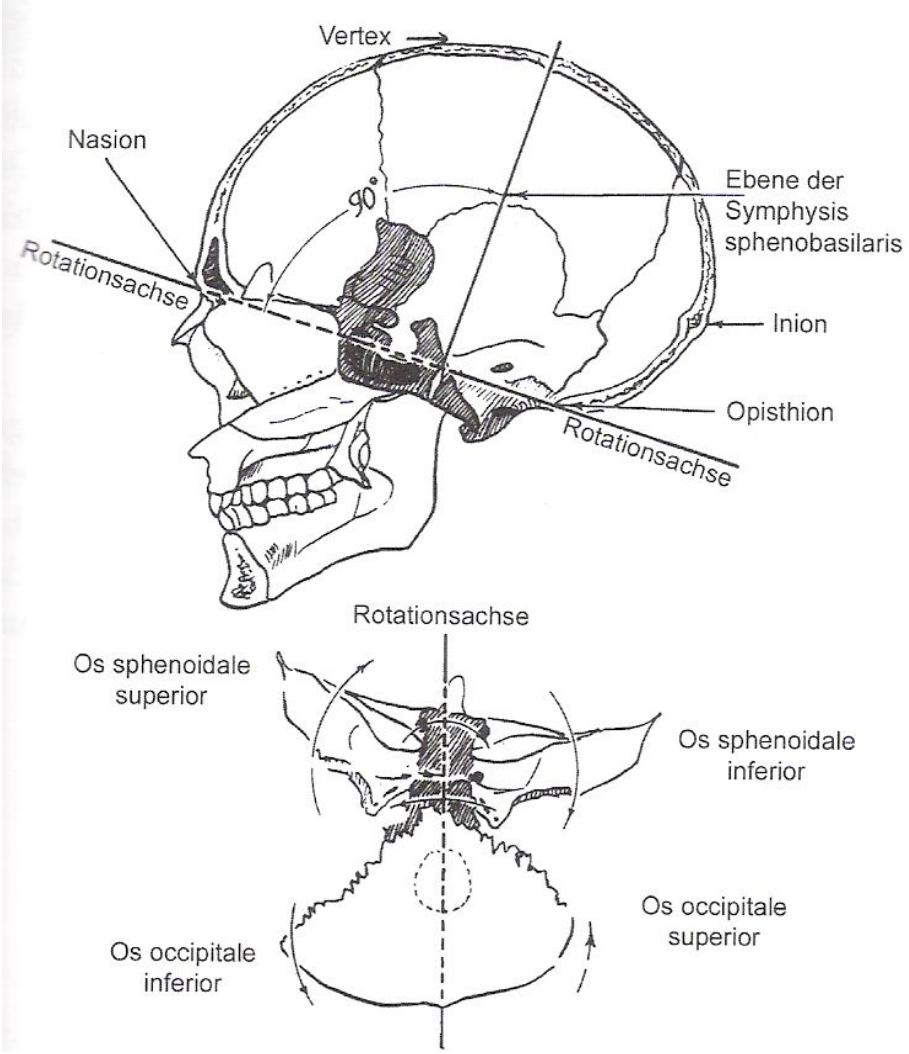

Fig. 13: Torsion-Strain

An SSB-torsion is diagnosed through palpation by osteopaths, which is why it is a rather vague model, not proven by any scientific test.

A master's thesis by Fuchs (2007) tried to find out shifts in the SSB (in 138 persons aged 20 to 86 years) by a computed tomography study in the sagittal plane. The results of this study presented that shifts of the SSB do exist, but to a large extent were minimal, and high-quality scans with specific investigations in the area of interest were required. In 4 cases Fuchs detected minimal changes in the position of the SSB but from an osteopathic view he could not see whether the presented changes were intraosseous lesions, torsions or side-bending/rotation dysfunctions of the SSB.

The causes of SSB-torsions are stated as primary traumatic, e.g. occurring at birth, or adaptative compensatory with disruptions in the muscular-fascial-skeletal system, with visceral or other disruptions (Liem 2001).

The possibility of a torsional dysfunction of the SSB at birth in the fetal engagement or dropping phase with simultaneous rotation is described by Nicette Sergueef (1995) as follows: Caused by antagonism of the os pubis against the squama occipitalis, a right-hand rotation of the entire cranium is initiated; uterus contractions press the head downwards, with the right side already lying lower at that time, which means that the left side has to move more around an a/p axis. If this is a large movement, it may lead to a torsional dysfunction of the SSB, which explains the development of scoliosis according to Nusselein (2006) and Liem (2001). Furthermore, Sergueef (1995) describes how protracted labor can result in SSB torsion through compression forces.

The use of a delivery forceps can also cause SSB torsion, Nusselein (2006) finds. In the subsequent development of the baby, infant, child, or youth, a fall on the os sacrum can lead to a torsional position of the sacrum, (complete ossification, according to Liem 2001, occurring between 20 and 25 years of age), and can, via the link through the dura mater spinalis, can cause an SSB torsion (Liem 2001 und Nusselein 2006). These assumptions are, however, not based on clinical studies.

Viola Frymann (2007) shows that a scoliotic spine in childhood or youth is very often the result of perinatal cranial scoliosis. In a study by Frymann in 1966 (cf. Frymann 2007), 1250 randomly chosen newborns were examined in order to investigate a possible connection between anatomical-physiological malfunctions in the cranio-sacral mechanism and its symptoms. She succeeded in demonstrating that 10% of the children had suffered perinatal or postnatal severe visible traumata in the head. In further 78%, experienced osteopaths could diagnose membranous joint strain in the cranium. Furthermore, in keeping with Frymann, it is assumed that there is a significant connection between SSB torsion with restrictions of the os temporale and circulatory and respiratory symptoms. These may be interesting findings, whose scientific value is doubtful, however, as the examination was done through palpation, which is not a reliable test method. Sommerfeld (2006), e.g. investigated the reliability of PRM-palpation carried out by two osteopaths on 49 healthy test probands, with the result that both inter-examiner as well as intra-examiner agreement could not be described beyond chance agreement, as the 95% limits of agreement showed the expected difference. Sommerfeld (2006) concluded that PRM-rates could not be palpated reliably and under certain conditions were influenced by the examiners' respiratory rates. Also Podlesnic (2006) investigated the reliability of the global listening as a general diagnostic tool. He performed this test on 15 test persons examined by 14 osteopaths. The results showed that for approximately half of the comparisons between the therapists, the k-

indices indicate only agreement on the level of chance, and he stated that the extent of the inter-examiner reliability is very heterogeneous for different pairs of osteopaths. Podlesnic (2006) concluded that the global listening test is not universally valid in the way it was performed.

Regarding the aetiology of scoliosis, Frymann (2007) as well as Liem (1998) postulated a connection between scoliosis capitis and the development of scoliosis. Arbuckle (1971) describes the unilateral flattening of the head on the occiput together with a flattening of the opposite side of the face as “scoliosis capitis”. For Arbuckle, a fetal defective positioning in the last months of pregnancy is to be held responsible fort his. The consequences of scoliosis capitis can be, in Liem's (1998) and Frymann's (2007) opinions, scoliosis, nervous and vascular symptoms, as well as pelvic and shoulder asymmetries. These signs normally only begin to show years later (Liem, 1998). Alternatively, according to Liem's assumption, after a new trauma (fall, accident etc.) years or decades later, neurological symptoms appear to be much stronger than warranted by the recent trauma.
In the same article Liem (1998) describes that intra-ossal dysfunction of the os sphenoidale, caused by forces on the sphenoid, (pre-, peri-, or post-natal) can lead to scoliosis. Furthermore he demonstrates that asymmetries of the cranium (intra-ossal or intra-sutural) cause changes in the cervical spine. These asymmetries can at first only be palpated as abnormal tensions in the head-neck area. When the child raises its head and tries to stand upright and walk, it also tries to keep its eyes and balance organ symmetrical, which will balance out scoliosis capitis and the dysfunction in the atlanto-occipital joint in another region, i.e. the spine (Liem, 1998; this explanatory model was also advocated by Hanneke Nusselein while teaching at the WSO in 2004). Liem (1998) observes, however, that at this time scoliosis is hardly visible yet. It will become more marked with increasing growth, and will often be diagnosed as idiopathic scoliosis by a doctor at a later time. According to assumptions by Liem (1998), Nusselein (2006) and other osteopaths, the tension dysbalances palpated by osteopaths can trigger IS before IS can be diagnosed biomedically. There is no scientific evidence for this, however.

Frymann (2007), Zink (1979), Nusselein (2006), Liem (1998), and other osteopaths maintain that certain cranial dysfunctions such as scoliosis capitis (Liem 1998) and SSB torsions (Liem 2001) can result in scoliosis.
What really causes scoliosis capitis or SSB-torsion is based on assumptions which cannot be scientifically proven. Nonetheless, in the book “Gesammelte Schriften von Viola Frymann” (Collected works by Viola Frymann; 2007), the illustrations of the heads of newborns which show increased asymmetries are impressive. These pictures are meant to help reconsider

impacts like the embryonic development forces described by Blechschmidt (2002), intrauterine influences (irregular position in the womb, uterine myoma, asymmetries of the mother's pelvis, premature contractions, premature entry into the birth canal, protracted compression forces), and the resulting asymmetries of the head (Sergueef 1995), as well as the impact of birth traumata and their consequences (Nusselein 2006, Liem 1998, Sergueef 1995).

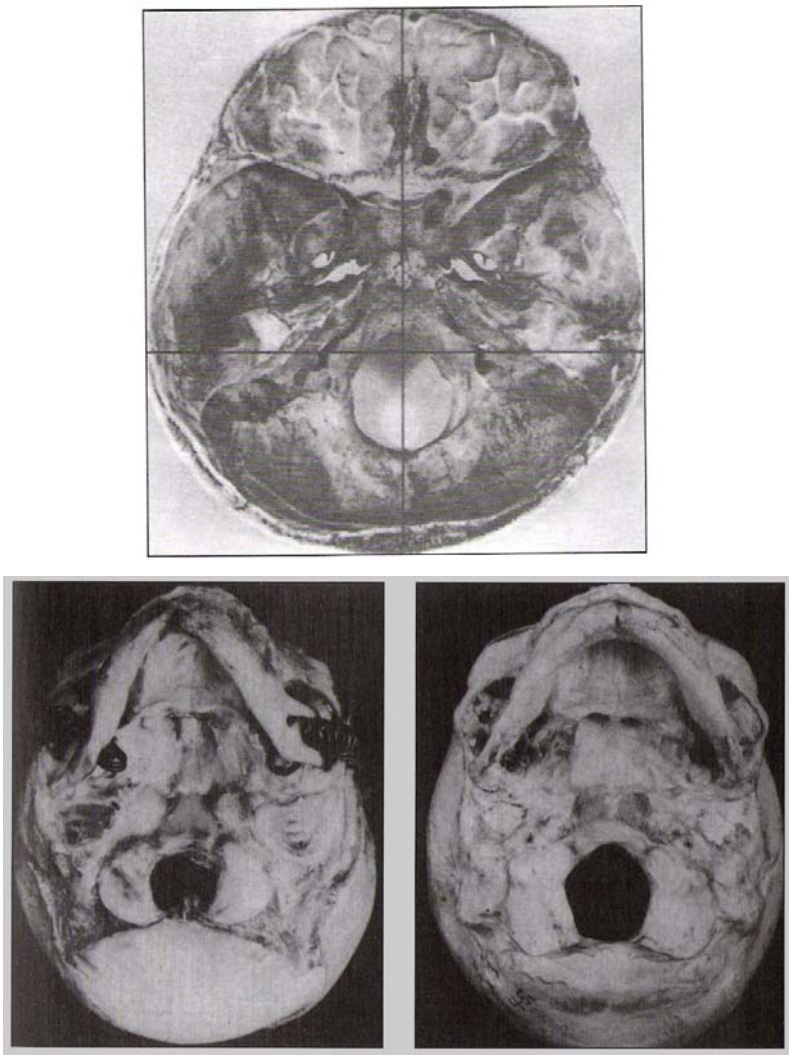

Fig. 14: Asymmetries of the cranium in newborns

In a pediatric osteopathic manual (Möckel, 2006), birth traumata or accidents are postulated as possible triggers of IS. The following dysfunction patterns are described in the chapter on osteopathic treatments in this osteopathic manual: SSB dysfunctions (SSB in the extension phase), compression of SSB, dysfunctions in the os sacrum und ossa ilia, inward rotation of the hip joints, costosternal and manubrial dysfunctions, muscular-fascial dysfunctions of the

abdomen, and segmental dysfunctions (costovertebral, ligamentary, vertebral, oder discus dysfunctions). Additionally, focal lesions in the embryo are depicted, in which all three germ layers (entoderm, ectoderm, mesoderm) are affected more or less strongly, thereby determining the degree of scoliosis. Similarly, disruptions in the development of the pericardium are cited as an example of visceral dysfunctions (Möckel 2006). Once more, the postulations by Möckel (2006) are based on personal observation and cannot be proven scientifically.

Also according to Liem (2001), disruptions in the muscular-fascial-skeletal system and visceral disruptions can result in SSB-torsion, which may explain the development of idiopathic scoliosis. The "interconnectedness" described is exemplary and is described by Liem (2001) as follows. He holds the opinion that a unilateral hypertonus M.longus capitis, M. rectus capitis anterior, M. rectus capitis lateralis, M.rectus capitis posterior major, M. semispinalis capitis and/or M. trapezius can cause torsion of the SSB, which may promote scoliosis. Unilateral tension of e.g. the anterior superficial fascia of the neck to the left (lamina cervicalis superficialis, fascia masseterica, parotidea and temporalis), and a unilateral tension of the fascia of the M. Trapezius to the right effects an SSB torsion to the right, according to Liem (2001). Dysfunctions of the intracranial and intraspinal membranous systems, which through their contact points on the os occipitale, os sphenoidale and os sacrum directly transfer their dysfunctions onto the SSB, also belong with this area.

According to a publication by Fossum (2003) about fascia and the osteo-articulatory system, every human adapts to the environment through his/her posture, which can be attributed to various factors like gravity, postural habits, macro and micro traumata, and other physiological and psychological stressors in the external and internal milieu. These adaptations, says Fossum (2003), occur on various planes and levels, e.g. the sagittal plane (changes in the anterior-posterior curvatures of spine and posture), the frontal plane (lateral curvatures of the spine caused by specific somatic dysfunctions, scoliosis, oblique position of the sacrum, and unequal leg length), and in the horizontal plane (rotation of body parts towards each other).
Fossum also states that "der schlimmste Feind der physiologischen Funktion, die Verdrehung der Faszienebenen ist“ (the worst enemy of the physiological function is the distorsion of the fascial planes; FOSSUM, 2003, 5). Preferred directions of fascial movements in the entire body in combination with the main regional compensatory changes which pertain to the fascial plane were recorded from earlier observations by Zink, Neider and TePoorten (cf. Zink, 1979). The common compensatory pattern (CCP) described by Zink (1979) consists of a string of myo-fascial torsions, which are compatible with a physiological

function until the primary organ system, the muscular-skeletal system is under stress. Zink (1979) believed that the lumbar spine of the growing child was especially vulnerable to repeated minor traumata that resulted in a twisting of the torso. He also felt that the ideal physiological pattern was best suited for locomotion, and that while the CCP was not as efficient a pattern, it was very adaptive. Implicit in these statements is the reasoning that during childhood development, as the infant attains the ability to crawl and then eventually to stand and walk children will adopt the more adaptive rotational pattern of the CCP. In other words, as a consequence of repeated minor traumata the lumbar spine develops a twist or bias of rotation. Then, through the reciprocating rotational motions of walking, this torsional bias is transmitted to other junctional regions of the spine. The regions of most restriction can be found in the so-called transitional zones which Zink (1979) considered “anatomical weak points.” Magoun (1973) also confirmed the fact that fascial tension and healthy alignment of the spine or fascial tension and skeletal system are closely linked.

Fossum (2003) states that the fascial torsion patterns include the longitudinal and horizontal organizational planes of the fascia, as well as their connections to adjacent parts of the cranium, the spine, and the pelvis. In this model, fasciae directly or indirectly influence the body's health via coordination with the muscular-skeletal system, via coordination with circulating body fluids, and by providing extensive passages to the nerves. Disruptions within the fascial areas can manifest themselves in venous and lymphatic stasis, in restricted breathing, abnormal reflexes, and a reduced range of movements (due to the impact on bony levers and joints). Moreover, Fossum (2003) claims, several visceral restrictions can be direct (mobility) and indirect (reflexion) consequences of the tension in the fascial areas.
Once more, the connections drawn by Fossum (2003) and Zink (1979) are only models and hypotheses which cannot be scientifically proven.

Following Liem (2001), there are several strings of muscular fascia such as the one from the os metatarsale I, via the caput fibulae, the tuber ischiadicum and the sacrum to occiput via M. peroneus, M. biceps femoris, lig. sacrotuberale, fascia sacrolumbale and M.erector spinae.
In this framework, tension dysbalances in these integrated myo-fascial chains can spur the development of scoliosis, possibly also brought forward by additional dysfunctions (growth spurt, stressors,…).
Also visceral disruptions like e.g. a ptosis of the stomach, have been found (cf. Liem 2001) to reach the SSB via fascial connections, which constitutes yet another explanatory model of idiopathic scoliosis.

The "Tensegrity-Model" invented by Buckminster-Fuller (cf. Liem 2006) is of further interest, albeit not explicitly mentioned or postulated in the context of scoliosis. It constitutes a structural system with rigid discontinuous structures which are connected by continuous tensile structures. Within this system, pressure is distributed discontinuously while traction forces are spread continuously. Tensegrity-structures cannot only be characterized by high stability but also by their ability to transmit mechanical energy and information throughout the entire system. Tensions are thus distributed continuously among all structural elements so that increasing tension in one element will always lead to increasing tension in other elements of the system as well. This increase in tension will be balanced out by an increase in compression in certain elements of the system. Thus the system stabilizes itself. From an osteopathic viewpoint, the boney skeletal system represents the rigid parts, while the muscles, ligaments, and fascia constitute the continuous tensile structures within the body. Ingber described this reciprocal tensile system at cellular level (Liem, 2006). Additionally, the psychoneurotic-immunological network deserves to be mentioned as it touches upon my topic, and may also influence the development of idiopathic scoliosis.

According to Liem (2006), psychoneuro-immunology can serve to explain that part of osteopathic interactions in which findings allow for the conclusion that emotions can be a pivotal point between body and mind. Biochemical substrates of emotions transmit information between brain structures and endocrinal, gastro-intestinal and immunological systems. Regarding the development of scoliosis, this aspect has not yet been sufficiently investigated but since it is known by now that protracted stress (distress) can weaken the immune system and even the gene expression of immunological cells (Liem 2006), psychoneuro-immunology could also influence the development of idiopathic scoliosis.

Conclusio:

Research findings regarding osteopathic explanatory models of the development of idiopathic scoliosis are vague and restricted to hypothetical assumptions.
The survey by Frymann et al. (2007) of 1250 newborns, in which anatomical disruptions in the cranio-sacral system are presented as related to the newborns' symptoms, shows that, among other factors, SSB torsions can be linked to circulatory and respiratory problems. “Scoliosis” was not mentioned explicitly as a symptom, however. It is hardly possible to diagnose an already existing increased deviation of the spine, unless it is an obvious one like scoliosis capitis, torticollis, or congenital scoliosis, since spine X-rays of newborns for mere research purposes cannot be justified ethically. Furthermore, there is an X-ray study by Greenman (1970) related to SSB dysfunctions and their symptoms as postulated in

osteopathy, in which he looked into the relationship between the sphenoid body and the basiooccput. The study was conducted with 25 patients and he was able to show abnormal relationships between these bones that demonstrated the lesions defined by Sutherland as flexion, extension, torsion, side-bending, vertical strain, and lateral strain. Greenman (1970) found no correlation with clinical symptoms as described in osteopathy, however.
In order to prove or disprove the symptoms resulting from SSB torsions (e.g. scoliosis) as they are postulated by osteopathy, further research needs to be done.
The theory that idiopathic scoliosis is caused by a dysfunction in embryonic development is a rather complicated guiding idea. The period of embryology alone with its complex processes, which could only be dealt with briefly here, constitutes a large field with many possible dysfunctions, and can thus only be grouped among the ”hypotheses about the development of idiopathic scoliosis"
Overall, there is little to no evidence of causal relationships in the development of IS and the explanatory models postulated in osteopathy. There is no scientific proof that embryonic developmental disruptions, birth traumata, SSB torsion, or cranial and sacro-pelvic dysfunctions in general, or visceral and fascial dysfunctions clearly lead to idiopathic scoliosis.

A good explanatory model for the complexities in dynamic processes and interactions is offered by the Tensegrity-Model. The extra-cellular matrix creates a continuum in our body which corresponds to the model's principles, establishing a connection between organs, structures and systems. A disruption in this system like those previously described may explain the development of scoliosis. This Tensegrity-Model, however, when applied to the human body, is also merely a model, and offers yet another possible hypothesis which cannot be scientifically proven.

This "lack of clarity" in the aetiology of scoliosis complicates osteopathic treatment on the one hand, but on the other hand offers a chance because of the manifold approaches to the complex connections and their specific osteopathic treatments, which may well lead to better results in IS treatment, as small-scale studies have already pointed out (Philippi et al. 2006, und Mandl-Weber 2000).
In order to render the therapy of idiopathic scoliosis even more efficient, osteopathy is required to carry out more studies and submit results which can be substantiated and thus better argued for.

8. Similarities or diametrical differences

In this chapter some biomedical results pertaining to the aetiology of idiopathic scoliosis will be juxtaposed with osteopathic explanatory models in order to show similarities or diametrical differences between the two.

8.1. Asymmetrical pattern

In the study by Karski et al. (2006), the syndrome of contractures according to Mau is presented. This describes scull deformities like the flattening of left forehead and temple regions, eye asymmetry, nose and ear deformation, torticollis, scoliosis infantilis, contracture of the left hip adductor muscles, contracture of the right hip abductor muscles, pelvis bone asymmetries, feet deformities. The hypothesis for these deformities was the conjunction with biomechanical aetiology of so-called idiopathic scoliosis caused by the fetus position during the last months of pregnancy.
The deformities described above and the increased deviations from symmetry can – from an osteopathic perspective – be explained by dysfunctions in embryology, intrauterine dysfunctons, or birth traumata. According to experienced osteopaths like Van den Heede, Nusselein or Liem, embryological dysfunctions can cause later idiopathic scoliosis. Birth traumata are also postulated as a cause for the development of idiopathic scoliosis (Sergueef, 1995; Liem, 2001).

In both fields (biomedicine and osteopathy) only hypotheses and models are being currently published. The causes of these deviations from symmetry which are already diagnosed in new-borns remain unclear.
Therefore newborns and babies require detailed examination to discover early symptoms of scoliosis or other skeletal malformations.

Ben-Bassat et al. (2006) showed in their study that patients with scoliosis have asymmetrical features of malocclusion compared with a random population. Also a theoretical model of a possible relationship between stresses on the cervical column and vertical occlusal alteration are discussed.
Malocclusion can be caused by embryologically induced cranial dysfunctions. The dysfunctions mentioned above like SSB torsion, SSB compression und SSB in extension do lead to idiopathic scoliosis but not to malocclusion, according to Liem (2001). SSB-

dysfunctions which co-occur with malocclusion are identified in the literature (Liem, 2001) as vertical strain and lateral flexion and rotation of SSB, which cannot be linked to the development of scoliosis, however.
According to Sergueef (1995), the birth phase of "shoulders being pushed out" can require considerable rotation and side-bending of the cervical spine, which in turn can lead to a dysfunctions in the cervical spine, the cervico-thoracic junction and the upper thoracic spine. Ben-Bassat et al. (2006) state that dysfunctions in the cervical spine can be linked to a malocclusion, which has been frequently diagnosed in scoliotic children and adolescents.

As indicated earlier, it is important to critically review the symptoms postulated for a SBB-dysfunction and initiate new research in this field.

Mac-Thiong et al. (2006) stated that the coronal sacropelvic morphology is distorted in AIS. Two hypotheses are discussed: the increased values of height and width parameters in AIS may be related to the altered growth rate (maturation), or may be due to an altered sacro-pelvic development which could be in association with the process involved in the progression of the spinal deformity.
Sacro-pelvic dysfunction can – according to osteopathic models – result from embryologically induced dysfunctions or be intrauterine (caused by "pathological" fetal positions; cf. Liem, 2001; Sergueef, 1995). Sergueef (1995) also postulated that distorsion patterns in membranes and fasciae, which reflect the recorded spiraling rotation of the birth canal, can induce dysfunctions in the sacro-pelvic area if it is a difficult birth. Intra-osseous dysfunctions or torsion of the sacrum, dysfunctions of the ossa ilia, dysfunctions of the hip joints, unequal leg length and/or traumata in this area during growth can lead to dysfunctions in the sacrum and the pelvis (Liem, 2001; Nusselein, 2006; Sergueef, 1995; Zink 1979). Sacro-pelvic dysfunctions can lead to the development of idiopathic scoliosis according to the views expressed by Liem (2001), Nusselein (2006), and Zink (1979). A possible model of the development of sacro-pelvic dysfunctions is offered by Schults et al. (1996); they showed in a study that pressure or tension in one area of the embryo results in increased secretion of connective tissue fibers in that area, and that these fibers tend to organize themselves along lines of tension. It is possible that dysfunctions in the forming forces during the embryonic phase and/or intrauterine dysbalances in the fetal phase lead to increased tensional dysbalances in connective tissue. Such dysbalances can already be diagnosed in newborns, Liem (2001) and Sergueef (1995) observed. Also fascial torsions in the sacro-pelvic area, which also match this explanatory model, can cause IS, according to observations by Fossum (2003), Liem (2001) and Möckel (2006).

The dysfunctions described in osteopathy are dysfunctions that are "diagnosed" through palpation at osseous, fascial, muscular, visceral or fluid levels, which cannot be scientifically proven. This study may serve to stress the importance of the fascial structure, which has recently been neglected in biomedicine, with its complex anatomical connections and physiological functions, and may initiate further research in this area.

8.2. Neurological Dysfunctions

Various studies have been presented in the field of neurological dysfunctions and their connection with the development of IS, in which vague links can be demonstrated.

Sun et al. (2006), e.g., found lower positions of the cerebellar tonsils in AIS patients. Filipovic and Viskic-Stalec (2006) stated in their study that AIS affects dynamic balance and illustrated the compensational functioning of mobility, especially when there is a lack of normal mobility forms and there are weak postural control mechanisms and proprioception.
Royo-Salvador (1996) postulated that an increased tonus of the medullary traction can result in the following changes: increased traction on the brain stem leads to increased tonus of the meninges, and, via their periosteal attachment, to increased traction in the tentorium cerebelli. Thus the tonsillae cerebelli are drawn lower and compressed. Furthermore, Royo-Salvador maintains that the cerebellar hemispheres are pressed into the fossa cranialis posterior, resulting in a deformation of the foramen magnum. In the cervical area of C1/C2, compression of the nervous tissue occurs, and in the thoracic area, especially during growth, idiopathic scoliosis develops. Royo-Salvador's explanation (1996) for this is that through the thoracic curvature medullary traction is relieved.
Carreiro (2005) stated in personal communication and also in her publication (2003) that hypotheses like vestibular dysfunction to cortical asymmetries are discussed.
Van den Heede (2006) sees in the development of idiopathic scoliosis an embryonic dysfunction in the build-up of the brain and the heart.
The statements by Carreiro (2005) and Van den Heede (2006) rest on very brief communication, and can in this form only serve as the starting point for further conclusions. Interviews with experienced osteopaths would probably have been more conducive.
In order to at least illustrate the connection between the three biomedical studies presented and the dysfunctions of the brain, and the cerebellum in particular, as discussed in osteopathy, I would like to refer to a brief overview of the physiology of the cerebellum.
The cerebellum is divided into three sections in keeping with its development and its cell structure, as elaborated by Van den Berg et al. (2000):

-- Vestibulocerebellum, whose function consists of integrating information yielded by the sense of balance into sensory motor activity. It has a close connection to the spinal chord, and contributes to the coordination of posture and locomotion.

-- Paleocerebellum or spinocerebellum is subject to influences from the spinal chord. Its function is to support posture and to coordinate posture and locomotion, whose effect evolves via the cerebellar cores along the nucleus ruber.

-- Neocerebellum or pontocerebellum occupies the largest part of the cerebellar hemisphere, and is closely connected via the bridge (pons) with various regions of the cerebral cortex. Its function ist he coordination of target and support motor activity.

The studies by Sun et al. (2006) about the "lower position of cerebellar tonsils“, the study by Royo-Salvador (1996) about "tonus increase in the spinal chord", which leads to the development of idiopathic scoliosis, and the study by Filipovic and Viskic-Stalec (2006), who postulated that in AIS-patients the dynamic balance and proprioception are reduced, all more or less explicitly refer to a dysfunction in the cerebellum.

When and where a cerebellal dysfunction occurs, and if a causal relationship can be established in IS development remains unclear, also in the studies mentioned above. Nonetheless, the results presented can be related to each other, and at least a hypothetical connection in IS development can be established.

9. Conclusion

The increased curvature of the spine was already diagnosed by Hippocrates (460-375 B.C.), who treated it with traction, but up to the 21st century the underlying causality of this illness has remained unclear. The present vast range of research into the aetiology of idiopathic scoliosis (IS) in the biomedical field reveals that various hypothesis are being discussed.
None of the studies, however, can solely claim to explain the cause of scoliosis (cf. among others, Goldberg et al. 2006, Miller et al. 1996, Sevastik et al. 2006 etc). The results show which structural, physiological, and functional changes have been found with IS but where the cause(s) of these changes lie, which result in an increased deviation of the spine, could not be clarified.
Biomedical hypotheses which imply that neurological dysfunctions lie at the root of the development of IS are increasingly being presented. Also in this area, however, there is no scientific evidence to support the tenability of these hypotheses. During my research I also found a tendency to report multiple pathogenesis for IS (Goldberg et al. 2006; Ben-Bassat et al. 2006; Heidari et al. 2003; and others). Thus Goldberg also concluded: “It may be associated that many pathological conditions and no specific pathology that belong to scoliosis alone has been identified” (GOLDBERG et al. 2006, 447).

Regarding osteopathic theories about the aetiology of idiopathic scoliosis I could only find few publications. Qualitative interviews would probably have been the more adequate method of data generation. Besides, I found that only models about the aetiology of scoliosis had been published which are not scientifically proven. In the study by Frymann (2007) briefly referred to, in which the connection of disruptions in the cranio-sacral mechanism with the symptoms in 1,250 newborns has been examined, the scale of the study is impressive while its reliability is rather dubious since the diagnostic method chosen is palpation. Thus, the dysfunction diagnosed by osteopathic lack any scientific basis, and in view of the recognition of our profession we need to reconsider the role of palpation as a diagnostic tool, proceed with more caution in our statements, and initiate as much research within osteopathy as possible.

In the chapter about "Similarities and diametrical differences", in which biomedical research results about the aetiology of IS were contrasted with osteopathic explanatory models, hypotheses can be found on either side; those from biomedicine are better substantiated by previous research, however.
In osteopathy, by contrast, there are no studies about this rather common clinical picture of idiopathic scoliosis. There are, however, several models which are plausible but not

scientifically proven or provable, since the causes mentioned in osteopathy like SSB torsion, dysfunctions of the sacrum, ileum or the hip joints, and dysfunctions on a bony, membranous, or fluid level, as well as fascial distorsions are not reliable.

Clearly, reliability is an important scientific factor, which ought to be given wider currency in osteopathy too, since this will improve the quality of "osteopathic doing and thinking". In this context I would like to quote the sentence Sommerfeld postulated in his master's theses: "The results of scientific-reliability testing can give certain support for clinical osteopathic acting“ (SOMMERFELD, 2006, 112).

The initial intention of this thesis – to gain more insight into the treatment of scoliotic patients through the results of recent biomedical research and through the osteopathic theories postulated about the aetiology of idiopathic scoliosis – has been eventually somewhat modified. Owing to the wide spectrum of hypotheses about the aetiology of IS on either side, yet more open questions have emerged.

My own experience in the treatment of idiopathic scoliosis in adolescents shows on average good results which can also be proven clinically by X-rays. Which of the osteopathic techniques applied in particular really does bring about change, and demonstrably improves or at least stabilizes the degree of scoliosis, remains unclear to me and requires studies which yield empirical evidence for causal relationships, especially in the field of cranial osteopathy. As Andrew Taylor Still already remarked, a successful man not only pursues theory, his motto is 'prove it'. ("Der erfolgreiche Mann verfolgt nicht nur die Theorie. Sein Motto heißt ausschließlich beweisen!“, STILL, 2002, w. Vorbemerkungen)

In order to obtain qualitatively better answers to the question about IS causality interdisciplinary studies in biomedicine and osteopathy are desirable.

10. Summary

This work has reviewed and analysed various current biomedical studies and osteopathic theories for the aetiology of idiopathic scoliosis.

Looking at possible genetic and epigenetic causes of IS, Zaidman et al. (2006) came to the conclusion that IS is a "genetically dependent spinal deformity inherited by autosomal-dominant type, with incomplete gender- and age related penetrance of genotype presented", while Miller et al. (1996) stated that no clear association could be determined that genes are linked to the cause of IS.

Some studies showed structural anomalies like imbalance of the connective tissue in IS patients. Fiber imbalance in the intervertebral disc and also in ligamantum flavum were stated by Yu and Fairbank (2005). Heidari et al. (2003) found out that higher fiber imbalance results in more severe spinal deformity. According to a model study by Van der Plaats et al. (2007), unilateral postponement of growth in os ligamantum flavum and intertransverse ligament appeared to initiate scoliosis. Above all, however, it is not clear whether these defects are primary or secondary, whether function governs structure or vice versa.

Other studies proved anatomical asymmetrical patterns in IS. Ben-Bassat et al. (2006) found more asymmetric features of malocclusion in IS patients. The “syndrome of contractures” was already diagnosed in newbornes and children by Karski et al. (2006). In these children they noted initial stages of IS and they concluded that the malformations of skeletal system can already be taking place in the last months of pregnancy. The sacropelvic morphology in the coronal plane of AIS patients showed significant differences in comparison to normal adolescents but it is unclear from which cause this asymmetric pattern do result.

In some studies neurological dysfunctions are hypothized to cause IS. Sun et al. (2006) proved that cerebellar tonsils have lower positions in AIS patients than in normal adolescents. Burwell et al. (2006a) hypothised that maturational delay in the CNS may arise and cause AIS. In a further study Burwell et al. (2006b) developed theories about disturbances in the longitudinal growth of paired (long limb bones, ribs, ilia) and united paired bones (vertebrae, sternum, skull, mandibulae). Differences in dynamic balance between AIS and healthy children are presented in a study by Filipovic and Viskic-Stalec (2006). An increase in tension in the spinal cord which further induces the developement of IS is presented in a study by Royo-Salvador (1996). Burwell et al. (2006) claimed that a disturbance of bilateral symmetry in embryonic life results from a default process involving mesodermal somites which causes the excess of right/left thoracic in AIS.

Other relevant studies looked at the connection of IS with visual defiency, a lower degree of mineralisation in IS and pleural infection.

The study by Goldberg (2006) on handedness did not show any evident connection between preferred hand and the development of IS.

Although a large number of studies has been done over the last few years, the aetiology of the three-dimensional deformity of idiopathic scoliosis remains unknown.

Osteopathic theories for the aetiology of scoliosis are scarce. The major source of information was personal communication with three experienced osteopaths.
Hypotheses like dysfunctions in the embryology are discussed by Van den Heede (2006), Nusselein (2006), and Möckel (2006). Malformation of skeletal system taking place in the later months of pregnancy which can induce the development of IS are discussed by Liem (1998), Nusselein (2006) and Sergueef (1995). Frymann (2007), Liem (2001), and Sergueef (1995) postulated that birth traumata can influence the incidence and outcome of scoliosis. In osteopathy SSB-torsions can indicate different symptoms, amongst them scoliosis. These dysfunctions in the SSB are based on palpational diagnostics which is a not reliable test method. Further dysfunctions in the sacropelvic region, ossa ilia, the hip joints, or distorsions costosternal and in the manubrium of the sternum are published in osteopathic literature. But there is no scientific proof for these hypotheses.
Distorsions in the myofascial system and visceral dysfuntions inducing the development of IS are also some of the evidence cited in osteopathic publications (Fossum 2003; Liem 2001; Magoun 1973; Zink 1979).

Several similarities and contradictions between the two views have been pointed out.
Anatomical asymmetrical patterns were diagnosed already in newborns by Karski et al. (2007) which they claim to be caused by the fetus position during the last months of pregnancy. Also osteopaths like Liem (1998, 2001), Möckel (2006), Nusselein (2006), Sergueef (1995), and Van den Heede (2006) stated that intrauterine dysfunctions can induce IS.
For both sides only hypotheses are presented and in-depth research needs to be done to help discover the aetiology of IS.
More asymmetrical features of occlusion in IS patients was proved by Ben-Bassat et al. (2006). From an osteopathic perspective, cranial dysfunctions can be caused by embryonic dysfunctions, birth traumatas or SSB-torsions. The postulated symptoms of the SSB-torsion (Liem, 2001), however, which are discussed in the context of the development of IS, do not agree with malocclusion. Mac-Thiong et al. (2006) claim that sacropelvic morphology is distorted in the coronal plane of AIS patients. The osteopathic theories for sacropelvic morphology are embryological dysfunctions, birth traumata or traumata inducing dysfunctions

on a bony, membranous or fluid level (Liem 2001; Nusselein 2006; Sergueef 1995; Zink 1979). But also in this case there is also no scientific base for osteopathic hypotheses.

With regard to the neurological dysfunction and its connection with the development of IS Sun et al. (2006) found anatomical features like lower positions of the cerebellar tonsils found in IS patients. Roya-Salvador (1996) postulated in his study that this is induced by an increased tension in the spinal cord, which also causes the development of scoliosis. Filipovic and Vaskic-Stalec (2006) showed that dynamic balance is affected in AIS patients and this seems also to indicate a dysfunction in the cerebellum. From an osteopathic point of view, Van den Heede (2006) stated that IS is caused by an embryologic dysfunction in the build-up of the brain and the heart.

Finally it has to be said, if and where the cause for a cerebellar dysfunction is involved and whether there is a context in the aetiology of IS remains unclear.

11. Table of Figures

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