ACTA ORTHOPAEDICA SCANDINAVICA
Supplementum No. 150

From the Department of Orthopaedic Surgery,
University of Göteborg, Sweden
(Head: Professor Alf Nachemson)

# STUDIES IN IDIOPATHIC SCOLIOSIS

*relevant to etiology, conservative and operative treatment*

By
ANDERS NORDWALL

MUNKSGAARD
COPENHAGEN 1973

Printed in Sweden by
Göteborgs Offsettryckeri AB, Surte 1973

ISBN 87 16 01446 4

CONTENTS

PART TWO: RESULTS OF CONSERVATIVE TREATMENT WITH THE MILWAUKEE BRACE AND OPERATIVE TREATMENT WITH A TWO-STAGE HARRINGTON ROD PROCEDURE

## ACKNOWLEDGEMENTS

I especially want to express gratitude to Professor Alf Nachemson, my highly esteemed teacher and chief. He introduced me to the field of scoliosis, and suggested the subjects of the present study. All the routine clinical work on which the investigation is based was carried out by Professor Nachemson, and his great enthusiasm and interest in research and clinical work on scoliosis has been most stimulating. I am deeply grateful for his advice and continuous encouragement throughout this study.

My sincere thanks are also due to my former chief Professor Bertil Stener for making available time and laboratories that made this work possible, and for his careful and skilful teaching of orthopaedic surgery.

I am greatly indebted to Professor Andrus Viidik, Aarhus, for kindly making available to me laboratory facilities for chemical and heat denaturation studies, and for valuable advice and criticism in the field of collagen research.

Furthermore, I wish to express my gratitude to Tekn. Dr. Lars Sonnerup, Chalmers University of Technology, who advised me on the biomechanical theories and details of the first part of this work, to Dr. Alice Maroudas, Imperial College of Science and Technology, London, for help in performing mucopolysaccharide analyses. I also want to thank Tekn. Dr. Gösta Elfström, Chalmers University of Technology, and Med. Dr. Stig Willner, Malmö, for valuable assistance in the preparation of this publication.

Likewise I am indebted to Eng. Hans Hellström, Mrs. Birgitta Runze and Mrs. Ingrid Melkersson for their laboratory assistance and willing cooperation, to Miss Lena Karling for programming and processing the data, to Mr. Karl Sandell for the photographic work, and to Mrs. Gunnel Blinge, Miss Berit Mattsson and Mrs. Lena Toll for their excellent secretarial work.

I thank Anders Odén, M. Sc., for his supervision of the statistical analyses, and my sincere thanks are also due to Mrs. Lois Goldie-Carlson who revised the English text.

The experimental investigation was supported by grants from the Swedish Medical Research Council (project no:s B70-17X-2808-01 and B71-17X-2808-02) and from Förstamajblommans Riksförbund, to which I am greatly indebted. The clinical study could be performed due to generous supports to the scoliosis unit during the last years from the following foundations: Greta och Einar Askers Stiftelse, Göteborgs Läkaresällskap, Kronprinsessan Lovisas Förening för barnasjukvård, Riksföreningen för Trafik och Polioskadade, Axel Tielmans minnesfond, and the University of Göteborg.

# PREFACE

Structural scoliosis is one of the "classical" diseases of orthopaedics. Known since ancient times it has always presented difficult problems of treatment. Treatment should preferrably be based on a concept of the etiology of the disease. In spite of considerable research into the field of scoliosis in general, and its etiology in particular, it is a disappointing fact that still more than 80 per cent of all cases with structural scoliosis are labelled "idiopathic".

Although investigations in the etiology of scoliosis have not yet been successful in clarifying the nature of the disease, treatment problems have been tackled for centuries. However, it is not until lately that reasonably effective treatment methods have been introduced. The Milwaukee brace, constructured by Blount,has proved to be an effective means of controlling scoliosis below $50^{o}$ with comparatively little encroachment in the life of the patients. Curves above $50^{o}$ can be corrected by an operation with the Harrington instruments. There are still, however, several questions of interest to the orthopaedic surgeon dealing with these problems.

This work is presented with the purpose to test one of the common theories of etiology and to analyse the different factors of importance for the result after conservative as well as operative treatment in a larger consecutive series of patients with idiopathic scoliosis.

# PART I

## PHYSICAL PROPERTIES OF COLLAGENOUS STRUCTURES IN PATIENTS WITH IDIOPATHIC SCOLIOSIS

CHAPTER ONE

# INCIDENCE AND ETIOLOGY OF SCOLIOSIS

## INTRODUCTION

Structural scoliosis is one of the major spinal diseases in growing children. Extensive research is carried out to study scoliosis and its causes. Despite this, the etiology and the pathogenesis are to a large extent still unknown. Cases of idiopathic scoliosis amount to approximately 80 per cent of all types of scoliosis (Cobb 1948, Hertel and Würfel 1969, Goldstein 1971, Krol et al 1972). Wynne-Davies (1968) estimated the incidence of idiopathic scoliosis to be approximate 4 pro mille for adolescent girls and about 1/10 of this for boys. In Minnesota, a prevalence rate of at least 1.33 pro mille was found for scoliosis requiring referral to an orthopaedic surgeon (Kane and Moe 1970). Brooks et al (1973) recently reported a study on 841 adolescent school children with a scoliosis incidence of 11 per cent roentgenographically verified. Approximately 2 per cent of the adult population has some kind of scoliosis, only in about 0.5 per cent does this exceed 20 degrees. (Shands and Eisberg 1955, Bouillet and Vincent 1967).

## THEORIES OF ETIOLOGY

James (1970) pointed out, that the only really known cause of scoliosis is congenital malformations of the vertebrae. In paralytic scoliosis with asymmetrical muscle paresis, it is also possible to understand the cause of scoliosis. In idiopathic scoliosis, however, the etiology is not known, nor are the factors known, which sometimes cause the scoliosis to increase, while sometimes even in growing individuals the scoliosis does not change much. Several comprehensive reviews have been published on the research in different fields to explore the pathogenesis and etiology of idiopathic scoliosis such as Waugh (1966), Bouillet and Vincent (1967) and recently Willner (1972). The factors particularly studied in connection with a possible etiology to idiopathic scoliosis are briefly reviewed in the following.

## Vertebrae and epiphyses

Farkas (1954) considered the vertebrae to be a primary cause to scoliosis in that there was a pathological separation of the epiphyseal ring from the vertebral cortex. This he considered would cause scoliosis by a growth disturbance of the vertebral body.

Most other authors have considered the changes of the vertebrae to be secondary. A review in this particular field was published by Karaharju (1967). He also performed an experimental study, provoking scoliosis in rabbits and obtained secondary structural changes in the vertebrae. He found that these changes partly were caused by a passive deformation of the vertebrae, and partly also were caused by a compensatory growth process, which tried to balance the lateral deviation and pressure (according to Hueter-Volkmann's law). The structural changes thus seem to be secondary to external factors outside the vertebrae, which cause the primary deformation. This was also confirmed by others (Liszka 1961, Michelsson 1965) and also by Enneking and Harrington (1969), who investigated the articular cartilage of the intervertebral articular processes. Also, the end epiphysis and the neurocentral epiphysis (believed to affect the rotational deformity) have been examined (Bisgard and Musselman 1940, Knutsson 1966, Mineiro 1969). Vertebral deformation can be secondary to different injuries resulting in scoliosis when predominantly unilateral. Radiation for tumours in the growing spine often results in deformed vertebrae and scoliosis. (Arkin et al 1950, Katzman et al 1969). Vertebral fractures in children usually do not causescoliosis, however (Horal et al 1972).

## Neuromuscular factors

A recognized and possible cause of scoliosis seems to be muscular imbalance, either caused by a primary muscle disease or secondary to a neurological disorder in the upper or lower motor neuron (Gruca 1958, Movshovich 1960). Roaf (1958) discussed the possibility of a rotatory muscle imbalance. Hirano (1972) using electron-microscopy studied paraspinal muscles. The changes in these muscles were not exclusively found in idiopathic scoliosis, and did not differ significantly on the convex or concave side. Also Badger (1969) and Hoppenfeld and Spiro (1973) reported histochemical changes in these muscles. It is difficult to determine whether these changes are primary or secondary. Studies in patients with poliomyelitis and scoliosis (James 1967) have shown that the latissimus dorsi muscles, the intercostals and the lateral abdominal muscles were weaker on the convex side of the scoliosis. This was also reported by Garret, Perry and Nichel (1961). The paralysis of the muscles of the extremities or the midline muscles did not show any relation to the side of the scoliosis convexity according to James (1967).

Studies of electromyography have not yielded any conclusive results. However, most authors have reported an increased EMG-activity on the convex side of a progressing scoliosis, this is believed to be compensatory to the scoliosis. (Zuk 1965, Butterworth and James 1969). The suggestion, that idiopathic scoliosis would be caused by subclinical poliomyelitis seems less probable as it is evident, that a decreased frequency of polio because of vaccination campaigns has not resulted in a decreased incidence of idiopathic scoliosis (James 1970).

There is some evidence that nervous control of the balance and muscle tone can be of importance. Liszka (1961) divided unilateral spinal nerve roots in rabbits. This produced scoliosis convex to the operated side also if only the posterior nerve roots were sectioned. These experiments were repeated by MacEwen (1968), who divided posterior nerve roots and reported that scoliosis developed in a high percentage of the experimental animals. An interruption of the reflex arch was considered to be responsible for the deformity. The effect of this operation was later questioned by Alexander et al (1972), who considered scoliosis to be the effect of lesions of other structures in the vicinity (anterior root). Yamada et al (1969) investigated the postural mechanisms and the proprioceptive functions in scoliosis, and believed that there is a disturbance of these mechanisms in the central nervous system in these patients.

## Gravitational forces

Weight is generally accepted to be an important factor for the progress of scoliosis. Thus bedrest, or recumbency in a plaster cast has been a common procedure for patients with scoliosis, nowadays usually in conjunction with other treatment. Arkin (1964),treating patients for up to three years in recumbency in plaster casts,did note impairment in some cases. There are other factors than gravity that are important. Lucas and Bresler (1961) and Lucas (1970) compared the vertebral spine with a segmental column. This can be considered to be in an unstable equilibrium, depending on outer factors such as ribs, muscles and ligaments to stabilize it. Without being subjected to vertical load, there also is a complex of balanced forces and counterforces acting on the spine. Relatively small unbalanced forces might be sufficient to produce deformity of the initially straight spine (Lucas and Bresler 1961).

## Ligaments

The ligaments are important for the stability and equilibrium of the spine. The importance of the ligaments for the development of scoliosis has been discussed by a few authors. Langensköld and Michelsson (1962) in a series of experiments carefully investigated the different types of operative procedures on rabbits, which could cause scoliosis. Section of the costotransverse ligaments was the procedure that mostly caused scoliosis in these quadrupeds. Roaf (1966) pointed out that the predominant feature of scoliosis is excessive vertebral rotation, related to a relative lengthening of the anterior components of the spine, as compared to the posterior elements (the vertebral arches and the interspinal ligaments). Lindahl and Raeder (1962) considered rotation and lateral angulation possible because of an asymmetrical growth restriction of the spinal intertransverse ligaments. Schultz et al (1972) paid attention to the inter- and supraspinous ligaments and calculated in a mathematical analogue of the human vertebral column the changes required to bring a normal column into the geometrical configuration of idiopathic scoliosis. They found that changes in the length of the structures around the transverse processes gave a configuration resembling idiopathic scoliosis. A restricting tendency of the extreme posterior anatomic structures could be important in determining the shape of a scoliotic curve. No doubt, muscles and ligaments under tension tend to describe a straight line. This could by itself contribute to the rotational deformity in a curved spine. It has been found that the ligaments of the spine, both the posterior longitudinal ligament and the ligamentum flavum, are in a condition of constant strain (Åkerblom 1948, Tkaczuk 1968, Nachemson and Evans 1968).

## Growth

The milder scoliosis curves (up to 60 degrees) do not change much after bone maturity and end of growth, and there is a correlation between progression of scoliosis and growth rate. (Calvo 1957, Duval-Beaupère 1971). Risser (1964) noticed that there is an increase of approximately one degree per year after the spinal growth has ceased. This was also noticed by Collis and Ponseti (1969). Willner (1972) recently showed that girls with adolescent scoliosis were taller than the average girl at the same age, while the trunk–leg relationship was undisturbed.

## Heredity

In recent years, there has been an increasing interest in the heredity of scoliosis. It seems probable that idiopathic scoliosis is an inherited disease, where the mode of inheritance is dominant and sex-linked but with an incomplete penetrance and variable expressivity (De George and Fischer 1967, Wynne-Davies 1968, MacEwen and Cowell 1970, Cowell et al 1972). The sex-linkage is to the X-chromosome: father to daughter, mother to son or daughter (De George and Fischer 1967, James 1970). Boury (1963) reported an anomaly in the fifth chromosome in three children with idiopathic scoliosis, however, this has not been verified by others (Anders 1966, Bouillet and Vincent 1967). The Scoliosis Research Society recently discussed the substitution of the label "idiopathic scoliosis" with "genetic" scoliosis.

## Metabolic and chemical factors

In idiopathic scoliosis, there seems to be a dominant heredity for the disease. This is also the case in those diseases termed inborn errors of metabolism such as Marfan's syndrome, Ehlers-Danlos syndrome and osteogenesis imperfecta. All these have an autosomal dominant heredity like idiopathic scoliosis, while homocystinuria is recessively inherited (Mc Kusick 1966, Beighton and Horan 1969). These diseases have been studied and parallels have been drawn with idiopathic scoliosis. Thus, Dent (1968) and Beals (1969) point out that patients with homocystinuria often have scoliosis. Homocystinuria is a disease with a defect in the metabolism of homocystine, where this substance accumulates in the tissue because of an enzymatic defect. Scoliosis is often a part of the clinical picture in the patient. Analysis of skin collagen has shown this to be defective in these patients (Harris and Sjoerdsma 1966). Homocystinuria has features clinically similar to those of Marfan's syndrome (Brenton et al 1972), which also is a disease, where scoliosis often is part of a total syndrome. There seems to exist some similar features in Marfan's syndrome and in idiopathic scoliosis, such as growth disturbances (Sinclair et al 1960, Willner 1972),and furthermore, the ocular symptoms in patients with Marfan's syndrome can be compared with the anomalies of the pectinate ligaments in the anterior chamber of the eye, which have been found in patients with idiopathic scoliosis (Ponseti et al 1960). In patients with Marfan's syndrome, like in patients with idiopathic scoliosis, there is an increased excretion of essential aminoacids in the urine.

Related to these diseases is also the Ehlers-Danlos syndrome, where sometimes scoliosis occurs. This disease is characterized by increased laxity of the joints, and hyperelasticity of the skin. Beighton (1969, 1971) considered the joint laxity of the ligaments to be the basic cause of the joint defect.

Osteogenesis imperfecta is another disease, where scoliosis sometimes occurs. This disease seems to be related to the above mentioned, with defects in the collagenous tissue (Bleckman et al 1971).

In all these diseases, the basic defect is considered to be in the collagenous tissue proper, with a decreased number of cross-links between the collagen molecules. (Mc Kusick 1966, Scarpelli and Goodman 1968, Beighton 1971, Viidik and Örndahl 1973).

There is a wide variation of the degree by which the individuals may be affected in these diseases and the incidence of complications also varies. There are no absolute criteria available for making the diagnosis.

Scoliosis can be produced experimentally in laboratory animals by a systemic change of the tissues by feeding the animals a diet rich in sweet peas (lathyrus odoratus). Sweet peas contain the molecule beta-aminoproprionitril (BAPN) and in vertebrates cause extensive damage in mesenchymal tissues, including skeletal deformities, hernias and dissecting aneurysms of the aorta. Scoliosis is a feature in most of these animals. (Ponseti 1957, 1959, Amato and Bombelli 1959.) Ponseti has described the pathogenesis of scoliosis caused by lathyrism, which also was extensively reviewed by Tanzer (1965). It has been shown, that collagen has a decreased tensile strength and that there is a decrease in the resistance of the ligamentous attachment to bone against rupture. (Levene and Gross 1959, Fry et al 1962, Zweymüller and Plenk 1968).

Thus, scoliosis is a part of the general disease picture in experimentally produced lathyrism as well as in a number of hereditary diseases with inborn errors of metabolism. For these reasons, there has been a great interest in the metabolism of patients with idiopathic scoliosis. It has been speculated, that this disease would be a form of inborn error of metabolism, where the only clinical sign of the disease is a curvature of the spine. Several authors have studied the metabolism in patients with idiopathic scoliosis, with special reference to collagen and the ground substance.

Stearns et al (1955) reported in idiopathic scoliosis patients a serious disturbance of the protein metabolism with an accentuated catabolism, causing an increase in the amino acid excretion, mainly taurin. Bouillet and Vincent (1967) considered this to be a secondary change, caused by the lowered nutritional status in these patients in connection with their decreased cardio-pulmonary function. Later, Schönenberger et al (1962) confirmed the findings of Ponseti and reported an increased excretion of amino acids in the urine in patients with scoliosis and epiphyseolysis. This was noticed also by Frederich et al (1963) who believed that it could mean that idiopathic scoliosis is caused primarily by a metabolic defect of the tissue. Glauber(1962) found an increase in the glycoprotein concentration in the blood serum from patients with idiopathic scoliosis. Böhmer (1965) and Böhmer and Nolte (1972) reported a decrease of certain globulin fractions in the blood possibly caused by a decreased albumin synthesis in the liver in patients with idiopathic scoliosis.

Hydroxyproline, which is an essential amino acid, contained solely in the collagen molecule, is excreted in increased amounts in patients with idiopathic scoliosis (Benson 1965, Zorab et al 1971). The increased excretion of hydroxyproline is related to the rate of growth, and it also seems to be related to the progression of scoliosis (Zorab et al 1971).

Investigations have been carried out to determine the metabolism of the mucopolysaccharides (MPS) in the ground substance of the tissue in these patients. Ponseti (1959) found in lathyritic animals metabolic changes in the connective tissue with a decrease in the synthesis of hexosamine and a breakdown of chondroitinsulphate in the epiphyseal plates. These changes were accompanied by changes in the bone-joint apparatus, and the disturbance of the MPS metabolism was considered to change the viscosity of the ground substance in the connective tissue and cause a rupture of the ligamentous attachment to bone (Ponseti and Shepard 1954). These authors found changes similar to those in lathyritic animals also in patients with idiopathic scoliosis and in patients with slipped epiphyseal plates. Ponseti (1968) reported an increase in total hexosamine concentration in the cartilage of the iliac epiphysis. He considered this also to be the case in the vertebral epiphysis. In 1972 Ponseti et al found the mucopolysaccharide content of the nucleus pulposus in patients with idiopathic scoliosis to be considerably decreased, while the collagen content was increased. They speculated, that idiopathic scoliosis could be one of the genetic chondrodystrophies, and therefore should be labelled "discogenic" scoliosis. Kazmin and Merkurieva(1969, 1971), found crease of the mucopolysaccharide content in the spinous processes of patients with scoliosis. On the basis of changes in the electrophoretic pattern of glycosaminoglycans and on changes of mucopolysaccha ride excretion in the urine of patients with idiopathic scoliosis Kyselka (1965) and Kyselka et al (1969) discussed the question of a change in the metabolism of MPS in these patients.

Balaba (1972) found a disturbance of the MPS concentrations in ligamentous tissue as well as in bone tissue from patients with idiopathic scoliosis. He beleived that these changes could lead to a change of viscosity and permeability of the tissue causing weakening of its functional stability. Balaba also claimed disturbances of the collagen metabolism with a reduction of the oxyproline-hexosamine ratio in spinous processes and ligaments, and he considered an increased excretion of oxyproline to be indicative of an increased collagen catabolism. This would cause a decrease in the mechanical resistance of the connective tissue.

Franchi et al (1963) directly pointed to the ligament tissue, reporting an increased activity of an inhibitory factor on elastin, which would make connective tissue more sensitive to mechanical forces.

A more direct influence on the connective tissue producing fibroblasts in patients with idiopathic scoliosis was reported by Conen

(1971). He found that cultured fibroblasts from these patients had metachromatic staining properties and an increased cell size, changes comparable to those found in patients with mucopolysaccharide diseases.

The metabolic disturbances found in patients with idiopathic scoliosis lead several authors to propose, that these may be of a primary order in these patients (Kyselka, Balaba, Ponseti). Unlike Ponseti, who emphasized the importance of the intervertebral disc for the pathogenesis, most other authors have not stated which mechanism they believe would cause scoliosis owing to influence of the metabolic changes. However, the metabolic changes observed in most cases, seem to be connected to the connective tissue, the ligaments and the tendons. Kyselka et al(1969)considered the mucopolysaccharides to be of importance for such mechanical properties of the tissue as elasticity, viscosity and plasticity of the ligaments. Viidik (1973) believed the role of the mucopolysaccharides at their usual rate of distribution and presence in tendons and ligaments to be uncertain for the mechanical properties. It is possible, that the MPS play an important role with a stabilizing function for the collagenous skeleton of the connective tissue. In some of the mucopolysaccharide diseases there is reported a looseness of collagenous fibres where the tendons and ligaments insert into bone. A general laxity of the joints and ligaments has also been reported.

## Connective tissue factors

Ligaments and tendons are of importance for the stability of the spine (Lucas 1970) and most likely also for the configuration of idiopathic scoliosis. As these structures in patients with certain inborn errors of metabolism have been shown to have a decreased mechanical resistance, and as also metabolic changes have been reported in patients with idiopathic scoliosis, it is of prime interest to investigate further the functions of the connective tissue. This especially applies to the mechanical properties of the ligaments and tendons in patients with idiopathic scoliosis. It is probable, that a ligamentous relaxation would make the spine unable to endure mechanical stress, such as influence of weight, particularly at increased growth rates.

As yet there has only been published one brief work on the biomechanical properties of tissues from these patients (Waters and Morris 1973). These authors did not find any change of the biomechanical properties of the supraspinous ligaments in patients with idiopathic scoliosis.

CHAPTER TWO

# STRUCTURE AND FUNCTION OF CONNECTIVE TISSUE

## FORMATION AND STRUCTURE OF COLLAGEN

Connective tissue consists of cells (fibroblasts) and the extra cellular matrix: fibres of collagen, elastin and reticulin, the ground substance and water. The specific structure and function of connective tissue depends on the varying proportions of these components. Thus, in the ligamentum flavum there is a large component of elastin, making this ligament highly extensible (Nachemson and Evans 1968). In dense connective tissue, such as tendons, the ground substance is very scarse, and the amount of elastin and reticulin fibres in regular muscle tendons is practically nil. Tendons contain about 30 per cent of collagen on a wet weight basis, but about 70-80 per cent of the dry weight is collagen. Collagen in tendons and ligaments is of a mainly parallel fibre arrangement.

Collagen is synthesized rapidly only during growth and in healing of wounds (Woessner 1968). It is produced by the fibroblast in its endoplasmic reticulum from amino acids. These are arranged in a regular, repetitive manner, where approximately every third amino acid is glycin. Hydroxyproline and proline are found in about 14 per cent each (Neuberger 1965, Ramachandran 1967). Proline and hydroxyproline are considered to augment the number of bonds that make the collagen molecule rigid and stable(Gross et al 1954), and may thus be of special importance to the mechanical properties.

The collagen molecule consists of three polypeptide chains, which are twisted into a left hand spiral. These three spirals make up the collagen molecule, the tropocollagen, by twisting (to the right) around a common axis into a super helix. The main physical characteristics of the tropocollagen macromolecule have been deduced from physicochemical studies, X-ray diffraction analysis and electron microscopy. The tropocollagen molecule is about 2800 Å long and 15 Å in diameter, the molecular weight is about 300000 (Ramachandran and Kartha 1954, Rich and Crick 1961). The tropocollagen molecules are secreted from the cells into the extracellular space, where they aggregate into fibrils. These are held together by H-bonds, but with increasing age a larger number of covalent cross-links are formed, both inter- and intramolecular. Different models for the tropocollagen macromolecules have been constructed, and it is believed that the tropocollagen molecules are combined with overlaps and spaces which could explain the regular periodicity of 640 Å that is demonstrated in the electron microscope (Gallop 1966, Cox and Grant 1969).

The ground substance is also believed to be formed in the fibroblasts prior to the secretion into the extracellular space. It is a metachromatically staining substance made up of mainly different acid glycosaminoglycans (GAG:s) which all contain either galactosamine or glycosamine. The GAG:s are bound in covalent bonds to protein. The importance of GAG:s for various physical properties is not fully known. It is probable, that mucoprotein and mucopolysaccharide-protein take an integral part in stabilizing the collagen fibres by being firmly bound to the fibrils (Mathews 1965).

Another main component of ground substance is water, which is bound ("trapped") to the ground substance and to the collagen. The water content is considered to be of importance for the physical properties of connective tissue (Galante 1967, Tkaczuk 1968).

## METABOLISM OF CONNECTIVE TISSUE

The metabolic turnover of collagen is very slow, especially in older subjects (Thompson and Ballou 1956). The liver and bone collagen have the fastest turnover in the body, while collagen in tendons have a very long half-life (Neuberger 1965, Woessner 1968). Woessner has extensively reviewed the mechanisms of collagen resorption. In degradation of collagen, a fraction of the hydroxyproline of the collagen appears in the urine. However, the urinary hydroxyproline level does not only depend on the break-down of the stable collagen, but also on the amount of the young, newly formed soluble collagen in the body. No particular deficiency of collagen making it more susceptible to degradation has been shown, but the break-down in itself is an effect of rapid turnover of the collagen. An increased break-down usually occurs in connection with an increased collagen synthesis, when the tissue remodels and rearranges its framework in order to meet changing requirements for support and tensile strength. The final balance of the collagen turnover is decided by the proportion of breakdown to the synthesis of new collagen (Woessner 1968). There is a good correlation between the amount of soluble collagen in the body and the total excretion of hydroxyproline, this being a measure of the collagen metabolism (Prockop and Kivirikko 1967). The increased hydroxyproline excretion which is noted in idiopathic scoliosis (Benson 1965, Zorab et al 1971) is mostly dependent upon the collagen turnover in bone, as this exists in larger amounts and has a higher turnover rate. It is also note in a number of other conditions engaging the collagen of bone in particular. The increased hydroxyproline excretion in lathyrism has been attributed to an increased breakdown as well as an increased synthesis of collagen. On the other hand the increased hydroxyproline exretion seen in scurvy depends on a net loss of collagen in spite of a decreased break-down. Other diseases with increased hydroxyproline excretion are Marfan's syndrome and Ehlers-Danlos syndrome also Paget's disease, osteogenesis imperfecta, rheumatoid arthritis, thyroid disturbances and pregnancy(Sjoerdsma 1958, Woessner 1968).

## PHYSICAL PROPERTIES OF COLLAGEN

The physical properties of collagen are dependent on the arrangement of the fibres and in particular on the bonding between the components. Piez (1968) reviewed the subject of cross-links, which are thought to explain many of the physical properties of the collagen. Thus, newly formed young collagen, so far lacking a reasonable amount of cross-links, is to a certain degree soluble in neutral salt solution as well as in acid solutions. It is more easily denaturated by heat i.e. at a low temperature, at which it also reacts to denaturation by a weaker isometric contraction of its substance. With increasing age, there is an increasing number of cross-links, and the collagen becomes "mature". This type of collagen is characterized by being insoluble in salt solutions, and having a higher denaturation temperature and a stronger heat denaturation isometric contraction. The mechanical properties are affected parallel to the heat denaturation properties. Young collagen has a lower elastic stiffness and a lower tensile strength than older collagen.

## FACTORS INFLUENCING THE STRUCTURE AND FUNCTION OF COLLAGEN

A number of factors have been shown to influence the collagenous structure and function:

### Diet

Collagen is sensitive to different diets and environments. Deficiency of vitamin C causes scurvy with increased breakdown and diminished synthesis of collagen (Smith and Shuster 1962).

Lack of copper leads to mechanical weakness of the connective tissue (Coulson et al 1965).

Lack of food and in particular lack of protein causes delayed collagen synthesis (Peacock 1963).

Lathyrism can be produced in experimental animals by feeding them different toxic agents such as beta-aminoproprionitril and semicarbacid. The striking pathological feature in this condition is absence of both intra- and intermolecular cross-links in collagen, however, the amount of collagen is essentially unaltered. The collagen becomes more easily soluble and the tensile strength of rat tissue is reduced by as much as half(Levene and Gross 1959, Fry et al 1962, Zweymüller and Plenk 1968). There is an increased urinary hydroxyproline excretion in the mature subjects, due to an increased break-down of collagen.

### Hormones

Cortical steroids have been shown to produce a more mature, less soluble collagen, and to cause an increase in the tensile strength in tendons from animals. Only doses which are almost toxic produce a decrease in the tensile strength (Vogel 1969). In Cushing's disease the fibrillar connective tissue is reduced and the skin becomes thin (Asboe-Hansen 1963).

Also thyroid hormones have been shown to interfere with the collagen function. Growth hormones cause an increase in the soluble fraction of the collagen, this is accompanied by an increased hydroxyproline excretion.

### Enzymes

Trypsin and chymotrypsin seem to have an effect on the mechanical properties of the connective tissue. This has been attributed to an action on the interfibrillary material, changing the ability of the components of the collagenous framework to move relative to one another (Harkness and Harkness 1959). Partington and Wood (1963) found that impure preparations of hyaluronidas influence the load-extension curve of tendons probably because of enzymic impurities which attack the interfibrillary matrix.

Age, and genetic, hormonal, enzymatic and nutritional factors as well as different pathological conditions thus may have an effect on the collagenous system and its equilibrium. Also training and trauma seem to affect the collagen tissue (Viidik 1967).

CHAPTER THREE

# BIOMECHANICAL PROPERTIES OF TENDONS AND INTERSPINOUS LIGAMENTS IN PATIENTS WITH IDIOPATHIC SCOLIOSIS

## INTRODUCTION

The primary function of collagen is mechanical, and mechanical tests can be used to investigate the structure of connective tissue. Collagenous structures in different forms have been extensively studied biomechanically by different authors. Wertheim (1847) was the first to investigate the mechanical properties of fresh preparations of human connective tissue. He set an example to many investigators, among others Gratz (1931), Cronkite (1936), Rigby et al (1959), Abrahams (1967), Viidik (1968) and Haut and Little (1972).

## TECHNIQUE OF BIOMECHANICAL TESTING

The technique of testing has varied somewhat. However, all tests have been performed by applying a force to the specimen and recording the force and the resulting deformation. The tensile properties of collagen tissue are commonly described by the relation between load and deformation. In order to permit comparison of results from tests of specimens of different sizes, the load-deformation relation has to be reduced to the relation between stress (load per unit cross sectional area) and strain (deformation per unit original length). A typical stress-strain curve is demonstrated in Fig. 6, page 37.

The load-deformation curve or stress-strain curve begins with a toe part with the concavity towards the stress axis. It has been shown that this toe part corresponds to a straigthening of the initially wavy formation of the collagenous bundles (Rigby et al 1959, Elliot 1965, Abrahams 1967). With increasing stress, the fibres become successively arranged in the direction of the strain until there is a linear relation between stress and strain corresponding to the stretching of the collagen fibres themselves. With still a larger stress, the stress-strain curve deflects towards the strain axis when the fibres begin to rupture.

TENSILE PROPERTIES OF BIOLOGICAL TISSUE

Connective tissue has mechanical properties, which can be described by common mechanical laws, but in biological tissues there is a high degree of non-linearity as described by many authors. Because of this non-linearity, it has not been possible to formulate a perfect equation for the viscoelastic behaviour of connective tissue. Viidik (1968) has worked out a theoretical model for the biomechanical properties of connective tissue, this model was verified mathematically by Frisén et al (1969). Such a theoretical framework for elastic properties of soft tissues was also worked out by Fung (1967). It is generally noted, that collagenous tissue exhibits three different qualities: the time independent properties of elasticity and plasticity, and the time dependent property of viscosity (Frisén et al 1969). The mechanical entity "modulus of elasticity" is based on a linear relationship between load and deformation, to which Hooke's law can be applied:

$$E = \sigma/\varepsilon$$

where

$E$ = modulus of elasticity, determined by the slope of the stress-strain curve

$\sigma$ = stress (load/cross-sectional area)

$\varepsilon$ = strain (deformation/original length)

However, "elastic stiffness" can be measured for the fairly linear part of the curve, and equals the slope of the tangent of the curve in this linear part.

The specimen can be cyclically tested with loading-deloading. With such consecutive testing cycles, the stress-strain curve is displaced to the right along the deformation axis because of the presence of a plastic component. With more loading cycles there is also an increase in elastic stiffness, and an increase in length of the linear part of the curve.

Biological tissue also displays a viscous component that is time dependent. This can be demonstrated by a relaxation test: the tendon is subjected to a certain strain, which is kept constant, the stress measured in this isometric state will then decline towards an asymptote.

## FACTORS AFFECTING THE DETERMINATION OF THE BIOMECHANICAL PROPERTIES OF COLLAGENOUS TISSUE

The shape of the load-deformation curve depends upon several basic factors:

- The length and the cross-sectional area of the specimen.
- The geometrical arrangement of the collagen fibres.
- The testing history of the specimen prior to the actual experiment.
- The rate of strain at which the specimen is tested.
- Environmental factors such as time after removal from the body, post mortem changes, influences of temperature and humidity on the specimen.

These factors have to be evaluated and standardized in order to permit an accurate, reproducible testing procedure. The mechanical properties of biological tissue are thus dependent on the condition of that tissue. Testing of tendons and ligaments in completely intravital conditions is impossible, and considerations has to be taken to possible changes occurring post mortem. Several authors have investigated these changes brought about by different modes of storage. With drying, the stiffness of the tissue increases (Morgan 1960, Galante 1967). It has been shown, that of different methods to keep the tissue from drying, the most effective is to keep the tissue in a humidity of 100 per cent (Hirsch and Galante 1967, Tkaczuk 1968) or to wrap the structure in a saline-moistened gauze (Viidik 1966). This latter technique prevents changes of the mechanical properties for at least 4 hours (Viidik (1973). Immersion in different fluids causes swelling of the collagen fibres, this applies to water as well as to Ringer's solution or plasma substitutes or plasma. The swelling causes subsequent changes in the mechanical properties of the structures (Viidik and Lewin 1966, Galante 1967, Tkaczuk 1968).

Opinions vary, as to how long a time it takes for fresh material to change biomechanically after removal from the body. It was shown by Viidik et al (1965), that rabbit ligaments could be kept intact in the body as long as 4 days without exhibiting any consistent post mortem changes biomechanically. However, others consider changes to occur very fast after the removal from the body (Smith 1954). Viidik has concluded, that collagenous tissue stays "fresh" for at least 24 hours provided it is not removed from its normal surroundings in the dead body.

Deep freezing was studied by Hirsch and Galante (1967), who showed that rapid freezing and thawing did not have any effect on the mechanical properties of the tissue. However, Matthews and Ellis (1968) investigated the difference between frozen and fresh cat tendons and found a decrease in elastic modulus after freezing ($-10^{\circ}$ C for two weeks) and brought into question the validity of the practice of pooling data from fresh and frozen specimens. Deepfreezing, however, generally is made at a lower temperature, allowing a fast freezing. In this investigation of the mechanical properties of collagenous structures only fresh material has been used.

## MATERIAL AND METHODS

### Material

For mechanical testing two different collagenous structures were chosen (Fig. 1).

1. The broad, flat insertion tendon of the erector spinae muscle on the iliac crest. This tendon is strictly parallel-fibred.

From 40 patients with idiopathic scoliosis 108 tendon strips were taken out, these were compared with 57 tendon strips from 20 control patients.

2. Interspinous ligament. This is a ligament with two bone-cartilage attachments. The fibres often have a fan-like distribution in its most distal part, the fiber arrangement can vary somewhat from one ligament to another (Rissanen 1960).

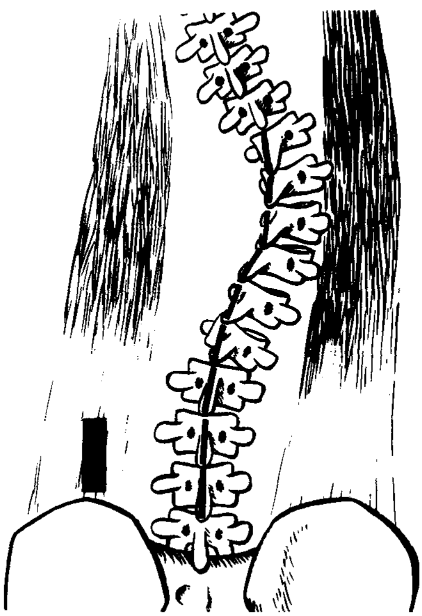

Fig. 1. Sites for the biopsy of the tendon of the erector spinae muscle and the interspinous ligaments.

Altogether 145 ligaments from 40 patients with idiopathic scoliosis and 65 ligaments from 20 control patients were obtained. The structures were removed from the body in connection with surgery of the scoliotic spine ad modum Harrington (1962) performed in two stages. In the first stage, the spinal processes and ligaments were exposed subperiosteally by blunt dissection as part of the routine surgical procedure. After exposure,the spinal processes, with the attached supra- and interspinous ligaments, were osteotomized at the base of the processes,and it was then possible to lift out the whole row of processes and ligaments intact. After removal from the body, the structures were immediately wrapped in a saline-moistened gauze. The operation proceeded with additional exposure of the concave side of the scoliosis, and the scoliosis was corrected with a Harrington rod.

Two weeks later, at the second stage, additional correction of the scoliosis was made, and fusion was performed by decortication of the posterior laminae and by adding autogenous bone from the iliac crest. Near the iliac crest the tendon of the erector spinae muscle was exposed, and a rectangularly shaped part of the tendon was taken out, approximately 2-4 cm wide and 6-8 cm long (Fig. 1). Immediately after removal, the tendons were wrapped in a saline-moistened gauze. The structures were kept at room temperature until preparation, which usually started within one hour.

From the description above it is obvious, that the collection of samples from patients did not subject them to any additional trauma and only slight additional time of surgery (5-10 minutes).

As control material the same kind of structures were taken from patients operated in the same way for scoliosis, but in whom the scoliosis was of a known etiology. Five of these control patients with non-idiopathic scoliosis were operated on in only one stage, where at this stage both correction and fusion of the scoliosis were performed. Both structures to be tested were removed in that same operation. Two control patients did not have scoliosis, but were operated for a lumbar spondylolisthesis. From these patients no interspinous ligaments were obtained. The diagnosis of the control patients is shown in Table 1.

The interval between the removal of the structures from the body and the testing was within three hours for the first structure tested, and within seven hours for the last structure. Only fresh material was used. The specimens compared in both groups were handled and stored in the same way.

The mean chronological age of the patients at operation was for the idiopathic group 15 years and 10 months ranging between 10 years and 25 years and 2 months. For controls, the mean age was 15 years and 1 month, ranging between 10 years and 6 months, and 24 years and 10 months. The skeletal age of each patient was determined from the Greulich and Pyle Atlas (1959) and averaged for the idiopathic group 14 years and 6 months and for the controls 14 years and 9 months.

Preparation of the samples

Preparation of the specimens was commenced within one hour after removal at operation. The tendons and ligaments were prepared into a shape suitable for the testing procedure.

Tendons. The broad tendon strip was fitted between a pair of clamps, and a constant force of 2.45 N*) (250 ponds) was applied to the specimen in the direction of its fibres. With a double-edged knife, the width between the blades being 2.5 mm, strips of that width were cut out along the somewhat stretched tendon. The knife blades easily followed the fibres of the tendon, as far as could be checked macro-

*) N = newton, the force unit in the International System of Units
1 N = 0.102 kp (kilogram force) = 0.225 lbf

TABLE 1 Control patients

| Patient | Chron. age years | months | Apex | Convexity | Diagnosis |
|---|---|---|---|---|---|
| S.N. (Only ligaments) | 12 | 11 | L 2 | Left | Scoliosis Muscle dystrophy |
| B.K. | 15 | 5 | L 2 | Left | Scoliosis Congen. hemivertebra |
| A.S. | 13 | 11 | D 8 | Right | Scoliosis Congen., fusion D7-D8 |
| I.J. | 14 | 6 | D 7 | Right | Scoliosis Congen., fusion D7-D8 |
| M.J. | 24 | 10 | D 11 | Right | Scoliosis Poliomyelitis |
| H.V. | 15 | 8 | D 7 | Right | Scoliosis Congen., fusion D6-D8 |
| P.C. (Only tendon) | 10 | 7 | - | - | Spondylolisthesis |
| T.H. | 18 | 1 | D 10 | Right | Scoliosis Sec. to thoracotomy |
| A.G. | 12 | 10 | D 7 | Right | Scoliosis Congen.,hemivertebra |
| S.B. | 19 | 3 | D 8 | Right | Scoliosis Hemiplegia |
| K.L. | 20 | 10 | D 10 | Right | Scoliosis Poliomyelitis |
| C.R. | 10 | 6 | D 12 | Right | Scoliosis Congen., hemivertebra |
| J.D. | 14 | 5 | D 11 | Left | Scoliosis Poliomyelitis |
| J.T. (Only tendon) | 12 | 10 | - | - | Spondylolisthesis |
| L.W. | 16 | 7 | L 1 | Right | Scoliosis Spastic hemiplegia |
| I.M. | 13 | 7 | D 10 | Right | Scoliosis Congen.,fusions D8-D10 |
| J.N. | 12 | 0 | D 8<br>L 2 | Right<br>Left | Scoliosis Hemiplegia |
| E.B. | 13 | 0 | D 5 | Left | Scoliosis Congen., hemivertebrae |
| U.J. | 15 | 1 | D 12 | Right | Scoliosis Congen., hemivertebra |
| B.J. | 10 | 9 | D 12 | Left | Scoliosis Congen., fusion D11-L1 |
| A.G. | 16 | 5 | D 5 | Right | Scoliosis Congen., fusion D4-D6 |
| A.Å. (Only ligaments) | 19 | 3 | D 9 | Right | Scoliosis Poliomyelitis |

scopically. From 60 patients 2-6 such strips were obtained from each individual in this way and altogether 165 strips were removed. The water content of the specimens was controlled by keeping them covered by a saline-moistened gauze during the preparation.

Ligaments. Each spinal process attached to the ligaments was divided in an upper and a lower part, thereby separating each individual interspinous ligament with its upper and lower bony attachments. Each ligament was fitted between a pair of clamps, identical with those, which are described in connection with the mechanical testing (Fig. 2). A force of 2.45 N was applied to the ligament in the direction of the fibres, and with the knife of two blades - spaced at 2.5 mm - the middle portion of the interspinous ligament was cut out in the frontal plane. Here also, it was possible to examine the direction of the fibres macroscopically. In this middle portion of the ligament, the fibres have a mainly parallel course, however, with a slight fan-like arrangement at the lower bony attachment. For this reason, it has not been possible to avoid cutting off some fibres. It is reasonable to assume, that this occurred equally in both the tested ligament groups. The ligament was then turned at a right angle to the former plane, and was cut in the sagittal plane with the same double-bladed knife. For a few very narrow ligaments a knife with blades spaced at 1.5 mm was used in this plane. Here the fibres have a mainly parallel direction proximo-distally, and cutting

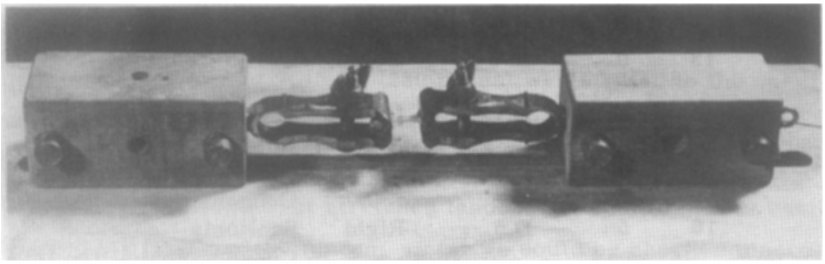

Fig. 2. Equipment with clamps for preparation of the interspinous ligament samples. A ligament is fitted between the clamps.

off fibres did not occur to any appreciable degree in this plane.

## Cross-sectional area mesurement

In order to reduce the load-deformation relation into a stress-strain relation for obtaining a comparable value of elastic stiffness, it is necessary to have an adequate cross-sectional area measuring method. Several methods for determining specimen cross-sectional area have been used by investigators studying the mechanical properties of connective tissue, and the results depend upon the method used. A method modified after Walker et al (1964) has been employed in this study. They developed an instrument for determining the cross-sectional area of tendons by compressing the specimen to a rectangular section of a known width and measuring the height of this section by means of a micrometer. Ellis (1969) compared seven different methods for determination of the cross-sectional area in tendons, and found this method to have the smallest systematic change between the measurements. He concluded that this is the best method in cases, where measurements of the cross-sectional area of fresh, moist specimens are required, as it is accurate and insensitive to slight changes of the specimens such as drying, and extremely easy to apply.

The specimen to be measured is placed between a pair of steel blocks positioned 2.5 mm apart on a flat plexiglass surface (Fig. 3). A cylindrical steel plug,at one end equipped with a rectangular blade, which exactly fits between the steel blocks,is placed between these and allowed to rest on the specimen. The height of the steel plug over the plexiglass plate is recorded with a micrometer, and read to the nearest 0.005 mm. The dimension of the rectangular blade of the plug is 2.49 x 4.50 mm, and the weight of the plug and the micrometer head is 34.60 grams, and the compression force exerted on the specimen is 0.030 $N/mm^2$. Because of its viscoelastic properties, the volume of the specimen decreases towards an asymptotic value. After 5 min. the volume has decreased approximately 20 per cent, and the asymptotic value is considered to be nearly

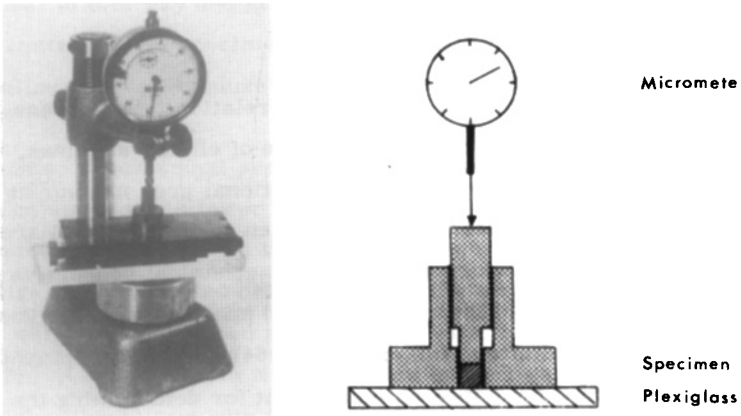


Fig. 3. The cross sectional area measuring device. The specimen is placed between the metal blocks, spaced 2.5 mm. The device with the specimen is then placed on the plexiglass plate, and the height of the specimen is read on a micrometer.

reached. Registration is then made. Through the bottom of the plexiglass plate it is checked that the specimen fits properly between the steel blocks. Measurement of cross-sectional area is made on two locations of the specimen. Between each measurement, the specimen is allowed to recover 5 min. and is then turned "upside down" in the instrument. The mean value of these determinations is calculated and used. Mean error for these measurements were ±0.017 mm (±2.0 per cent) for tendons, and for ligaments ±0.026 mm or ±1.9 per cent. The whole measurement takes place in a humidity chamber with 95-100 per cent humidity, controlled by a hygrometer (Fischer's hair-hygrometer). The cross-sectional area is calculated as the product of the height and the width of the specimen.

Testing equipment

The mechanical testing of the samples was performed in a mechanical testing machine, the Alwetron Universal Testing Machine T-2000. This machine can perform cyclic loading and deloading at constant rates of specimen deformation, and can perform relaxation tests. In the present testing series, the force was measured using a 50 kilopond (490 N) load cell transducer, with an accuracy of ± 1 per cent according to the manufacturer (Lorentzon and Wettre). The machine was also fitted with a displacement transducer of a differential transformer type (Bofors RLK-1) for an accurate deformation measurement. Its range of measurement was ±12 mm, with an accuracy of ±0.5 per cent according to the manufacturer (Bofors). It was fairly insensitive to changes in temperature, less than 0.02 per cent/ $^{o}$C. The machine was positioned in a fairly constant room temperature.

The testing machine was coupled with an amplifier to two X-Y recorder units, one a Mosely type 7005 AM (Hewlett-Packard) with an accuracy of ±0.20 per cent of full scale in the measurement range used. The other was a Houston Instruments Omnigraphic TM X-Y recorder with an accuracy of ± 0.25 per cent of full scale measurement. These recorders were used simultaneously (Fig. 4). On the latter, the deformation axis could be changed into a time axis for the relaxation tests. The time basis accuracy was ±1.5 per cent of maximal measuring range. Both load and deformation measuring systems were calibrated each day before the series of experiments. There was observed a slight deviation of the load measurement re-

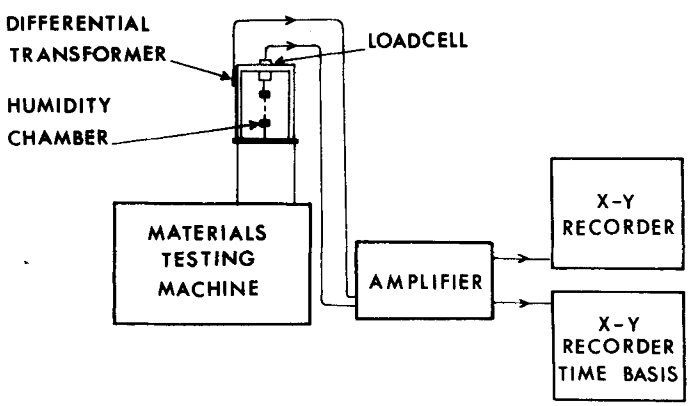


Fig. 4. Block diagram of the testing equipment.

gistrations during one testing day equaling four hours of testing. This deviation amounted to less than 0.7 per cent after four hours of testing. The recorder showed too heavy a load. On the deformation axis less than 0.9 per cent deviation was observed from the first to the last test during the testing day. The tendency was towards recording too long a deformation.

In order to preserve the specimens during the testing procedure, the Alwetron testing machine was fitted with a special humidity chamber (Fig. 5), constructed of plexiglass surrounding the part of the testing machine where the test sample was placed. This chamber was connected to a humidifier, which maintained the humidity in the chamber at 95-100 per cent. The humidity was controlled by a


Fig. 5. Close up view of the Alwetron materials testing machine with the humidity chamber surrounding the test clamps. A tendon specimen is fitted between the clamps.

hygrometer, which was placed at the level of the test sample. During testing, the specimen was also covered with a continuous stream of physiological saline, which dripped on it from an infusion set installed above the specimen. The temperature in the humidity chamber was kept constant at 24$^{\circ}$C and controlled by a thermometer. The total time for testing one specimen was 1/2 hour.

Clamps

For tendons a type of clamps was used, consisting of two parallel aluminium blocks between which the tendon was clamped. A piece of waterproof abrasive paper was glued to the inner surface of the blocks. The blocks were tightened together with a butterfly-screw, using a wrench to get the final torque. More than 10 mm of the tendon strip was fitted within each clamp, and the clamps were adjusted with a distance between them of 20 mm. The exact zero loading point was obtained from the recorder. With this type of clamps there were, among 162 tendons tested, 20 cases of slipping in the clamps. Slipping of a tendon in the clamps was discovered by an abnormal deflection (to the right) of the recorded curve. Mostly, however, these clamps were efficient in holding the tendons. Hirsch (1973), who tested the short peroneal tendon of rabbits, determined experimentally the slipping in these clamps up to loads of 5 kp, and found less than 0.01 mm of slipping. When testing for tensile strength 85 per cent of the tendons broke in the free space between the clamps, and 15 per cent of the tendons broke in the vicinity of one of the two clamp jaws. There was no significant difference in tensile strength between the tendons breaking near the jaws, and those breaking in the middle of the tendon.

The bony attachments of the interspinous ligaments were fitted into a contour-shaped clamp, where the jaws gripped around the spinous process, while the interspinous ligament passed out between the jaws. The clamp was screwed together with a butterfly-screw with enough force to prevent slipping of the spinous process between the jaws, but without causing impression in the ligament. With this type of clamps, there did not occur any complete slipping in 204 tested ligaments. Three ligaments fractured in the bone insertion before reaching the predetermined strain, and another 24 ligaments were discarded because of dips in the loading curve. Such dips occurred when the bony attachment of the ligament was not fitted in close contact with the jaw of the clamp, thus allowing the ligament to slip until the bone came in exact contact with the clamp. Failure of overstrained fibres that are successively strained cannot be ruled out as a cause of the fractures of the ligamentous insertion and possibly also of the dips in the curve noticed in a few tests. For the ligaments, the distance between the clamps was adjusted according to the length of the ligament, and the exact zero loading point was obtained from the recorded graph.

Definitions and determinations of the parameters studied.

The following parameters were estimated:

Elastic stiffness at a certain strain level at two different strain rates.

Plastic deformation between first and third load cycle.

Tensile strength for tendons.

Elongation at failure for tendons.

Stress relaxation (at a certain strain level).

In ligaments, the tensile strength was not tested, as rupture was shown in a preliminary experiment to occur not in the ligament, but in the attachment of the ligament to bone (which has earlier been shown also by a number of authors : Stucke 1951, Davidsson 1954, Viidik 1968). This rupture also seems to depend upon the direction of the attachment of the fibres, the fibres usually breaking successively from the bone along the attachment line.

The recorded load-deformation curve was read from the graph to the nearest half mm, i. e. for ligaments to the nearest 0. 01 mm elongation and 0. 05 N load, and for tendons 0. 01 mm elongation and 0. 20 N load. The values were reduced to the original dimensions of the specimen.

The original length was defined as the length of the specimen at the loading point of 0.49 N. The original cross-sectional area was determined by the method described above.

Strain rate was calculated as percentage elongation of the specimen in one minute. It depends on the rate of deformation and on the length of the specimen.

Elastic stiffness ($N/mm^2$). For estimation of the elastic stiffness ($N/mm^2$) of the specimen subjected to strain, the straight line between two points, A and B, situated on the linear part of the curve, was defined to approximate the tangent to the curve in a point between the two points A and B, (Fig. 6). The elastic stiffness was determined as the coefficient of inclination, tan $\alpha$ , of this straight line, the value was reduced to stress-strain conditions. The elastic stiffness of the sample was determined for each of three loading cycles.

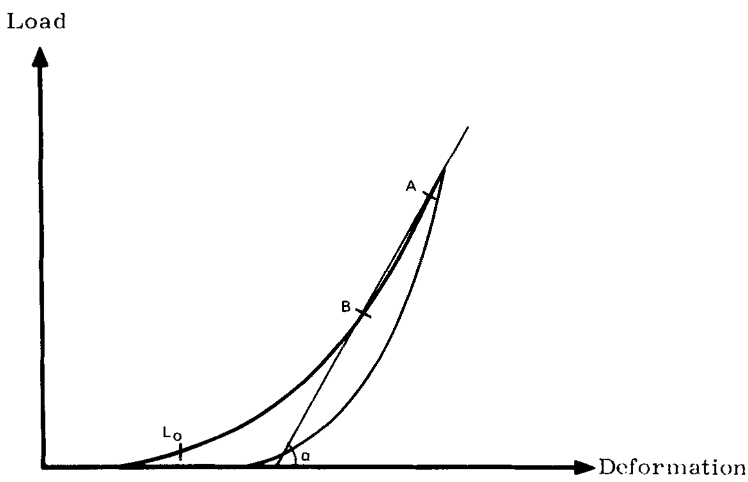


Fig. 6. Load-deformation diagram. The elastic stiffness was determined as the coefficient of inclination, tan $\alpha$ , of the straight line between the points A and B on the linear part of the curve.

Plastic deformation (per cent). The difference (mm) between the length of the specimen at the beginning of the third load cycle and at the beginning of the first load cycle was measured, this elongation (mm) in percentage of original length (mm) was taken as a measure of the plastic deformation of the specimen. The zero load level was defined to be at 0.49 N.

Tensile strength (N/mm$^2$). The tensile strength (N/mm$^2$) was determined from the graph at the point of maximum load in the tensile test to failure. The maximum load value (N) was reduced to units of original cross-sectional area (mm$^2$).

Elongation at failure (per cent). The elongation at failure (per cent) was the elongation (mm) reached at the point of maximum load in the tensile test to failure. It was calculated in percentage of original length (mm) of the tested specimen.

Relaxation test. In the relaxation test, the specimen was strained to a predetermined strain (Table 2) and the time measurement started when the intended strain level was reached. The time needed to reach this level was less than 20 sec. Relaxation was registered during 4 min. Relaxation occurs asymptotically, and after 4 min. approximately 85 per cent for ligaments and 75 per cent for tendons of the asymptotic relaxation have occurred. This allows a fairly exact calculation of the asymptotic value, which is approximately 40 per cent of the original length of ligaments, and 80 per cent of the original length of tendons. The relaxation phenomenon was evaluated from the recorded graph with a time axis. The graph was read at the following intervals of time: 1, 2, 4, 8, 16, 30, 60, 120, 180 and 240 seconds and the relaxation noted at each interval of time. The data were processed in a computer according to a mathematical formula described by Viidik (1968):

$$F(t) = F_o \exp(-\beta t) + F_a [1 - \exp(-\beta t)]$$

where

$F_o$ = initial load,
$F_a$ = asymptotic value of $F(t)$ when time tends to infinity,
$\beta$ = the shape parameter indicating the speed by which the asymptote is reached, calculated with the method of least squares.

The correlation coefficient of the experimental data to the equation was also calculated and found to be very good, above 0.97.

## Methods of testing

Preliminary tests indicated that maximal load after several load cycles would occur for tendons at a deformation of about 17 per cent deformation, and for ligaments at 20 per cent. Deflection from the fairly linear part of the first load-deformation curve would be expected at approximately 11 per cent for tendons and at 16 per cent for ligaments. In order to keep within the linear part of the curve, the first test cycle therefore had to be limited to a deformation of 9 per cent for the tendons, and 14 per cent for the ligaments. Each specimen went through three test cycles of loading-deloading, then a relaxation test, and finally the tendons were strained to rupture (Table 2). Before each testing series, the specimen was preloaded in order to allow it to fit properly in the clamps. In this preloading, the tendon was strained to approximately 3 per cent, and the ligament to approximately 7 per cent, with the same strain rate as in the first loading cycle. Table 2 also shows a specification of the test program performed with each specimen.

## Statistical methods

The elastic stiffness, the plastic deformation, the tensile strength, and the elongation at failure were calculated with the aid of a digital electronic calculating machine. From the experimental data, the mean of each parameter for each patient was estimated. From these means, the mean of each of the two test groups was calculated. The groups of patients were compared by a Student's t-test. This was performed by a table computer. The difference between the groups

TABLE 2. Test program for tendons and ligaments

| | Test Program | | Tendons | Ligaments |
|---|---|---|---|---|
| I | Load-deload | Strain rate %/min | 24.5±0.4 | 46.8 ± 6.0 |
| | | Strain % | 9±0.5 5 | 14±1 |
| | | "Linear" part of the load curve % | 8 - 9 | 10 - 14 |
| | Recovery period 5 min | | | |
| II | Load-deload | Strain rate %/min | 245 ±4 | 468 ±60 |
| | | Strain % (same load as I) | approx. 7 | approx. 10 |
| | | "Linear" part of the load curve % | 6 - 7 | 8 - 10 |
| | Recovery period 5 min | | | |
| III | Load | Strain rate %/min | 24.5±0.4 | 46.8 ± 6.0 |
| | | Strain % | 7.5±0.5 | 10.5 ± 0.5 |
| | | "Linear" part of the load curve % | 6 - 7 | 8 - 10 |
| IV | Isometric relaxation 4 min | Strain % | 7.5±0.5 | 10.5 ± 0.5 |
| | Recovery period 5 min | | | |
| V | Rupture (tendons only) | Strain rate %/min | 24.5±0.4 | |

was regarded as statistically significant if 2p  0.05 (2-sided test). This will in the following be mentioned as "significant". Correlations were tested and regression coefficients were calculated usinf a computer.

In determining the elastic stiffness and the tensile strength of the tendons and ligaments, the age of the subject is of importance as reported by several authors (Rollhäuser 1950, Yamada 1970). The material therefore was subdivided into age groups, and for each age group a variable of elastic stiffness and tensile strength was calculated according to Fisher's test. These test variables were "pooled" into another variable, which was used for a total comparison between the groups. In this last mentioned test as in the Student's t-test, the 5% level was chosen as the criterion of statistical significance in a 2-sided test.

Sources of error

The sources of error in the present study were:

1. Interval between biopsy and testing of the samples. The first sample from each patient was tested within three hours after the biopsy and the last sample two to four hours later. The time difference before testing the different samples is a source of error, as connective tissue is known to change its biomechanical properties after removal from the body (cf page 25 ). Furthermore a deviation of the recording of load and deformation was observed during the testing day, amounting to less than 1 per cent.

The samples, however, were handled with the greatest possible care, the humidity was preserved with saline-moistened gauzes and by preparation in a chamber at a humidity of 100 per cent. When not handled, the samples were stored in a refrigerator at +4$^{o}$C. This minimized the postmortem changes, and no consistent changes in elastic stiffness were observed between the first and the last structure tested over the day. Any change brought about by the difference in time of testing does not seem significant as compared with the biological variations displayed in the tested material. Samples from the idiopathic scoliosis group and the control group were handled in exactly the same way.

2. Preparation of the samples. Macroscopically it was checked, that the knife blades followed the course of the collagen fibres without cutting them across. In tendons, this preparation proceeded with no difficulties, and it was easily checked, that no collagen bundles were cut. In the interspinous ligaments this could not be totally prevented because of the irregular arrangement of the fibres. The number or

volume of cut fibres cannot be assessed, but it is reasonable to believe, that cutting occurred in approximately the same degree in all interspinous samples. Therefore it would not be a source of errror disturbing the comparison between the two groups to any large degree.

3. The mean error of the cross-sectional area measurement was almost 2%. These measurements were performed under standardized conditions with the sample under a constant load for a constant period of time and the mean of two determinations was calculated and used. Those samples, in which the difference between the two measurements exceeded five per cent were discarded. They were, however, included in the above calculation of the mean error.

4.Slipping in the clamps .This was detected by abnormal deflections from the regular curve pattern in the recorded load-deformation curve and,in the test of tensile strength, a complete slip in the clamp. Slipping was not assessed in the present study,however, Hirsch(1973) reports a slip of less than 0.01 mm for rabbit tendons tested in the same type of clamps.

5. The accuracy of the testing and recording equipment. This was stated above, and included:

Accuracy of the loading cell of the Alwetron testing machine: ±1%.
Accuracy of the deformation differential transducer: ± 0. 5%.
(The depence on temperature in the environment can be neglected in the standardized conditions under which the tests were performed.)
Accuracy of the recorders: approximately ±0. 2%.

Both load and deformation systems were calibrated each day before the series of experiments were carried out.

6. The recorded graphs were read to the nearest half mm, or approximately to ±0. 2 per cent load and ±0. 2 per cent deformation of full scale recording.

In relation to the variations observed in this material, 10-20 per cent, these sources of error are small. In the statistical evaluation the sources of error have been considered, and from a statistical point of view, the sources of error described cannot influence the result.

## RESULTS

### Tendons

Altogether, 165 tendon strips from 60 patients (40 patients with idiopathic scoliosis and 20 controls) were removed for examination. Three were discarded because of unacceptable variance (more than 5 per cent) in the cross-sectional area measurement. Of the 162 strips of tendon tested (105 idiopathic, 57 controls), 4 were discarded because of complete slipping in the test clamps, and for another 16, the first loading cycle was discarded because of slipping before the final elongation was reached. All load-deformation curves followed the known pattern of connective tissue in mechanical testing, with first a toe part concave to the stress axis, and then a relatively linear part. At deloading, there was a return with a hysteresis of the curve, and a certain remaining elongation, which was not recoverable(Fig. 6). With an increasing number of loading cycles, the elastic stiffness increased, and also it increased with increasing strain rate. The plastic deformation caused by a load cycle was decreased with increasing number of load cycles. There were no differences between the idiopathic group and the control group regarding the general shape of the load-deformation curve.

Elastic stiffness. The elastic stiffness of different age groups for the different load deformation cycles are listed in Table 3. The elastic stiffness of the third load cycle is demonstrated in Fig. 7. In the idiopathic group, the mean value of the elastic stiffness is slightly lower than in the control group. This is not statistically significant: the pooled statistical analysis for all age groups shows a value of 2 p = 0.20. There was a slight increase of elastic stiffness with increasing age, however, this was not significant.

TABLE 3. Elastic stiffness of the tendons in patients with idiopathic scoliosis.

| Age years | Load cycle | No. of patients | Idiopathic. Mean ± S. D. $N/mm^2$ | Controls Mean ± S. D. $N/mm^2$ | No. of patients |
|---|---|---|---|---|---|
| | I | 5 | 451.6±60.5 | 501.0±10.1 | 7 |
| 10-13 | II | 5 | 564.4±60.5 | 631.3±49.3 | 7 |
| | III | 5 | 586.4±48.0 | 633.4±41.7 | 7 |
| | I | 18 | 456.4±38.7 | 492.6±53.1 | 7 |
| 13-16 | II | 18 | 605.7±25.3 | 604.3±85.6 | 7 |
| | III | 18 | 588.5±29.1 | 593.6±69.4 | 7 |
| | I | 11 | 483.4±24.1 | 490.7±40.3 | 3 |
| 16-19 | II | 12 | 631.4±32.5 | 618.8±21.3 | 3 |
| | III | 12 | 613.6±34.2 | 602.3±22.7 | 3 |
| | I | 5 | 458.0±26.8 | 461.5±10.4 | 2 |
| >19 | II | 5 | 593.7±26.4 | 608.2±23.7 | 3 |
| | III | 5 | 577.9±39.9 | 608.8±33.0 | 3 |

"pooled" statistics: n - 2 = 58
2 p = 0.20, not significant.

Tensile strength. The result of tensile strength measurements of tendons in the two different groups are shown in Table 4. There was no significant difference between the groups, although a small decrease in tensile strength is noticed in the idiopathic group, compared to the controls (Fig. 8). A slight increase of tensile strength is noticed with increasing age. This is not significant (2 p > 0.5).

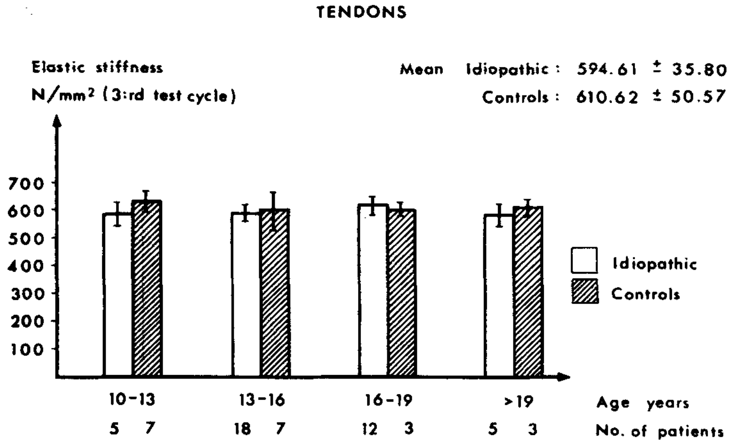


Fig. 7. Mean vaules and standard deviations of the elastic stiffness of tendon samples from patients with idiopathic scoliosis and controls in different age groups.

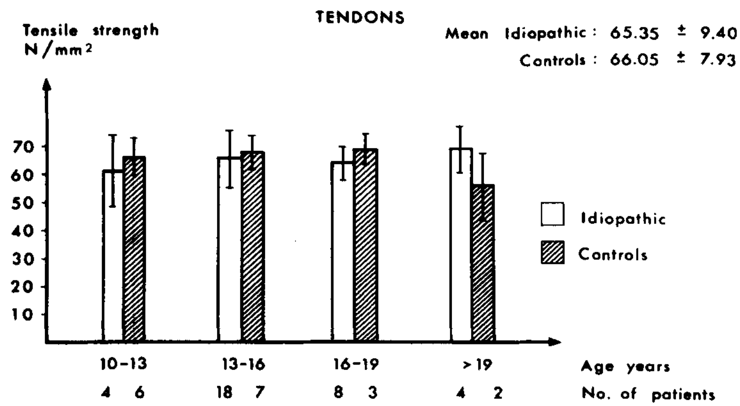


Fig. 8. Mean values and standard deviations for tensile strength of tendons from patients with idiopathic scoliosis and controls in different age groups.

In 8 patients, the tendons were not tested to rupture as the samples were used for chemical analysis.

Plastic deformation. The results are shown in Table 5. No significant difference could be found between the two groups ($2p > 0.05$).

Elongation at failure. No significant difference existed between the

TABLE 4. Tensile strength of the tendons in patients with idiopathic scoliosis (Mean ±S. D.)

| Age years | Idiopathic | No. of patients | Controls | No. of patients |
|---|---|---|---|---|
| 10-13 | 61.1±12.9 | 4 | 66.2 ± 6.8 | 6 |
| 13-16 | 65.6±10.5 | 18 | 68.2 ± 6.6 | 7 |
| 16-19 | 64.5 ± 5.9 | 8 | 69.2 ± 5.6 | 3 |
| 19 | 69.4 ± 8.7 | 4 | 55.9±12.3 | 2 |

TABLE 5. Plastic deformation, elongation at failure and results of the relaxation test in tendons and ligaments from patients with idiopathic scoliosis and controls.

| | Nomination | Idiopathic Mean±S. D. | No. of pat. | Controls Mean±S. D. | No.of pat. | t | 2p |
|---|---|---|---|---|---|---|---|
| Plastic deformation | | | | | | | |
| Tendons | % | 1.64±0.44 | 40 | 1.60±0.35 | 20 | 0.50 | >0.05 |
| Ligaments | % | 4.96±1.44 | 40 | 4.80±1.36 | 20 | 0.43 | >0.05 |
| Elongation at failure | | | | | | | |
| Tendons | % | 17.9 ± 4.6 | 34 | 17.0 ± 3.5 | 18 | 0.69 | >0.05 |
| Relaxation test | | | | | | | |
| Tendons: | | | | | | | |
| Shape parameter (x 100) | | 1.10±0.19 | 38 | 1.15±0.11 | 18 | -0.97 | >0.05 |
| Asymptotic load value | % | 76.2 ± 4.5 | 38 | 79.6 ± 2.2 | 18 | -1.96 | >0.05 |
| Ligaments: | | | | | | | |
| Shape parameter | | 1.19±0.07 | 37 | 1.23±0.08 | 16 | -1.92 | >0.05 |
| Asymptotic load value | % | 32.2 ±7.7 | 37 | 28.7 ±9.0 | 16 | 1.51 | >0.05 |

two groups tested (Table 5).

Load relaxation test. No significant difference between patients with idiopathic scoliosis and control patients was found for either the asymptotic load value or the shape parameter ($2p > 0.05$)(Table 5).

Correlation with surgical correctablity of the scoliosis deformity. For the tendons from patients with idiopathic scoliosis, the parameters of elastic stiffness and tensile strength were correlated with the results of the operation, i.e. the correction in percentage obtained at operation, and with the severity of the original preoperative curve, measured in degrees according to Cobb (1948) (Table 6).

TABLE 6. Original curvature and results after operative correction of the scoliosis.

| | Idiopathic | Non-idiopathic |
|---|---|---|
| Original curvature. Degrees (Cobb) | 74.4±20.0 | 76.9 ±19.0 |
| Average correction | | |
| % of preop. curvature (erect) | 56.5±11.4 | 45.45±14.1 |
| % of preop. curvature (supine) | 49.6±10.5 | 39.2 ±12.5 |

The elastic stiffness of the tendons was not significantly correlated with the original curvature, which ranged between $50^{o}$ and $85^{o}$ in the present material. However, the elastic stiffness of the tendons was significantly correlated ($r = -0.53$, $2p < 0.01$) with the percentage correction of the scoliosis curve after the second stage operation, measured from the preoperative roentgenogram of the spine in the supine position. With increasing stiffness of the connective tissue, there was a decrease in correction (Fig. 9). In a regression analysis of this correlation, the correlation coefficient between these variables was -0.53 and the fraction of explained variance $r^2 = 0.29$, indicating, that almost 30 per cent of the variations of the correction in percentage at operation could be explained by variations in the elastic stiffness of the tendon tissue. When the correction in percentage of the original erect curve was correlated with the elastic stiffness of the tendons, the correlation coefficient was $r = -0.47$.

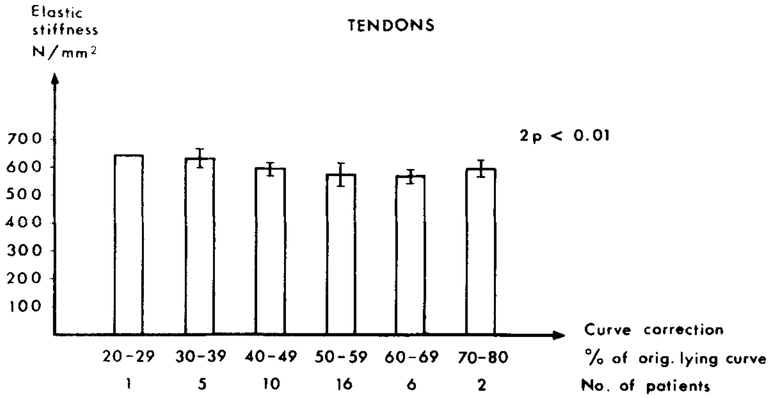


Fig. 9. Mean values and standard deviations for the elastic stiffness of tendons in patient with idiopathic scoliosis correlated with the correction of the curvature by the operations.

There was no significant correlation between the tensile strength and the percentage correction. With increasing age there was a non-significant increase in elastic stiffness.

Ligaments

Altogether 210 ligaments from 40 patients with idiopathic scoliosis and from 20 controls were prepared for testing. Six were discarded because of unacceptable variance in the cross-sectional area measurement (more than 5 per cent). Of the 204 ligaments tested, 3 were discarded because of fracture before the predetermined strain level was reached, and 6 were discarded because of slipping in the clamps. Of the remaining 195 ligaments (135 idiopathic, 60 controls), another 18 had dips in the first load cycle curve, which was discarded, however, the following load cycle curves were evaluated.

When the elastic stiffness of the ligaments from all levels in the idiopathic scoliosis group was compared with that of the ligaments in the control group, there was no significant difference in the different age groups (Table 7). When the different levels of the interspinous ligaments were compared, no consistent correlation could be found between the level of the ligament and its elastic stiffness. Nor was there any significant difference between the mean value of elastic stiffness for ligaments of the idiopathic group when compared at each level with those of the control patients (Table 8).

TABLE 7. Elastic stiffness in 3:rd load cycle of the interspinous ligaments from patients of different age.

| Chron. age years | Idiopathic Mean±S. D. N/mm$^2$ | No. of patients | Controls Mean±S. D. N/mm$^2$ | No. of patients |
|---|---|---|---|---|
| 10-13 | 47.66±4.58 | 6 | 44.27±2.84 | 6 |
| 13-16 | 45.95±4.93 | 17 | 48.61±4.99 | 7 |
| 16-19 | 46.98±4.83 | 11 | 45.01±5.75 | 3 |
| >19 | 46.17±4.34 | 6 | 45.25±5.16 | 4 |

TABLE 8. Elastic stiffness in 3:rd load cycle of the interspinous ligaments from different levels of the spine.

| Interspinous ligament level | Idiopathic Mean±S. D. N/mm$^2$ | No.of samples | Controls Mean±S. D. N/mm$^2$ | No.of samples |
|---|---|---|---|---|
| D 6 - 7 | 43.7±8.7 | 6 | 45.7±3.2 | 2 |
| 7 - 8 | 48.5±8.3 | 13 | 51.9 | 1 |
| 8 - 9 | 46.3±6.6 | 20 | 47.8±4.1 | 8 |
| 9-10 | 44.4±6.5 | 24 | 43.6±6.2 | 10 |
| 10-11 | 46.1±5.7 | 16 | 46.8±4.0 | 10 |
| 11-12 | 45.4±8.5 | 22 | 52.1±8.1 | 9 |
| 12-L1 | 48.2±8.8 | 17 | 46.3±8.0 | 13 |
| L1 - 2 | 49.0±5.6 | 15 | 47.9±8.3 | 4 |
| 2 - 3 | 44.8±8.3 | 2 | 40.2±4.7 | 3 |

The ligaments were subdivided into three groups, according to the localization of the apex of the scoliosis curve. In each of these three groups, the mean elastic stiffness of the ligaments from the idiopathic group and the control group was determined at each ligament level, and compared. There was not any difference between the levels of the ligaments of the spine which could be related to localization of the apex of the curve (Figs. 10, 11, 12).

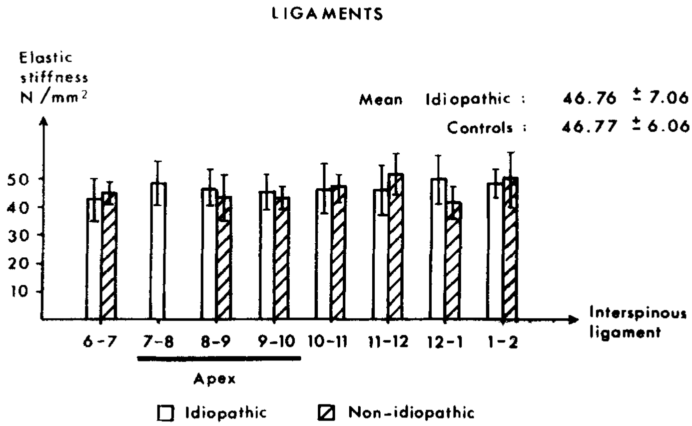


Fig. 10

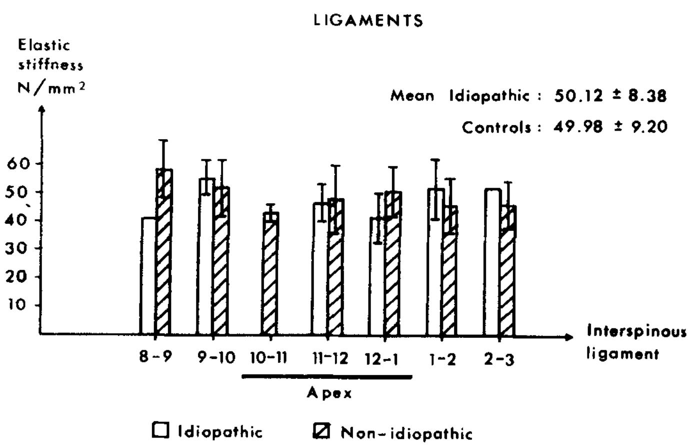


Fig. 11

Figs. 10 and 11. Mean elastic stiffness of interspinous ligaments in patients with idiopathic scoliosis and controls correlated with the ligament level for curves with apex at D 7-9 and at D 10-12 respectively.

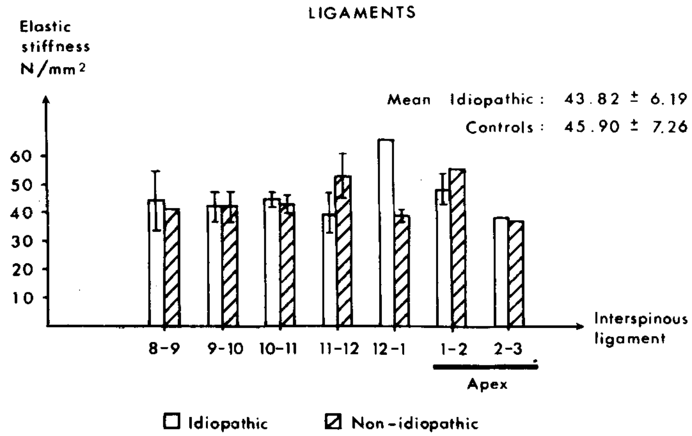


Fig. 12. Mean elastic stiffness of interspinous ligaments in patients with idiopathic scoliosis and controls correlated with the ligament level for curves with apex at L 1-3.

When the other parameters tested in ligaments from patients with idiopathic scoliosis were compared with those in ligaments from controls no significant difference could be found (Table 5, page 46).

CHAPTER FOUR

# JOINT ELASTICITY IN PATIENTS WITH IDIOPATHIC SCOLIOSIS

General joint laxity is a feature of hereditary connective tissue disorders according to McKusick (1966) and others. Patients with Marfan's syndrome and Ehlers-Danlos disease often have scoliosis, and they also have a general laxity of their joints (Beighton and Horan 1969). Wynne-Davies (1970,1971), in an investigation of 589 patients with congenital dislocation of the hip, found one group with this disease which also had a laxity of the joints, and within this group there was an increased frequency of idiopathic scoliosis. She believed, that idiopathic scoliosis might originate from a minor connective tissue defect. Veliskakis (1973) noted in 154 patients with idiopathic scoliosis, that there was a high incidence of ligamentous laxity also in the families of these patients, and maintained, that there is a genetically determined ligament laxity in patients with idiopathic scoliosis.

## MATERIAL AND METHOD

The laxity of the joints was studied in those 48 patients with idiopathic scoliosis in whom biomechanical testing of tendons and ligaments had earlier been performed.

Six different joint groups were studied with respect to mobility and flexibility: Ankle, knee, elbow, interphalangeal, metacarpophalangeal and the wrist joints were examined.

The method was described by Carter and Wilkinson (1964), and has been used by several other authors (Kirk et al 1967, Beighton and Horan 1969, Wynne-Davies 1970). The passive range of motion of the joints was measured, using a goniometer. The test was considered positive for generalized joint laxity, when three or more of the following tests were positive:

Passive dorsal extension of the ankle joint more than 20 degrees, (angle measured between the axis of the leg and the plantar surface of the foot).

Passive hyperextension of the knee joint more than 10 degrees.

Passive hyperextension of the elbow joint more than 10 degrees.

Passive hyperextension of the fingers so that they lie parallel with the extensor aspect of the forearm.

Passive hyperextension of the interphalangeal and metacarpophalangeal joints of the fingers.

Apposition of the thumb against the flexor aspect of the forearm.

Both right and left joints were evaluated, and the mean value was determined for each patient.

## RESULTS

48 patients with idiopathic scoliosis were examined. According to the definition of Carter and Wilkinson, 3 patients (6 per cent) had an increased laxity of the joints, i. e. 3 or more positive parameters. 39 patients (78 per cent) had none or only one joint with increased laxity. The three patients with increased joint laxity did not exhibit any significant decrease of elastic stiffness in their biomechanically tested tissue.

CHAPTER FIVE

# COLLAGEN MATURITY IN PATIENTS WITH IDIOPATHIC SCOLIOSIS

The physical properties of connective tissue change with the "maturity" or age of the tissue. This "maturity" seems to depend upon the number of cross-links existing between the collagen molecules (Sinex 1964, Bjorksten 1968). It is known that progression of scoliosis is closely connected with spinal growth, and it has been shown that adolescent idiopathic scoliosis girls are taller than normal girls (Willner 1972). It would therefore be of interest to study the age properties of the collagen in patients with idopathic scoliosis.

It is known that collagen is changed into gelatin in concentrated electrolyte solutions and when subjected to heat. These processes were shown by Verzàr (1955, 1969) to increase in magnitude with increasing age, so that isotonic shrinkage is larger, and occurs at a higher temperature with older age. This phenomenon is attributed to breaking of the inter- and intramolecular cross-links and is thus dependent on the number of these links.

## MATERIAL AND METHOD

The heat denaturation process was studied in collagen in tendons of the erector spinae muscle from 12 patients with idiopathic scoliosis and 14 controls.

The tendon samples were biopsied at the time of surgery of the scoliosis as described above (page 27 ) and were immediately wrapped in a saline-moistened gauze. Within one hour, the samples were deep frozen (-20$^{O}$C) and the experiment was performed six months to one and a half years later. The specimens were then thawed and a small piece of the tendon was cut out at right angle to the direction of the collagen fibres. In a freezing microtome, with rapid freezing and thawing, sections 30μ thick were cut. Such a sample was placed in buffered Ringer solution (pH 7.4) on the bottom of a chamber in a brass metal plate (chamber diameter 9 mm), where the sample was heated. The bottom of this metal chamber was a translucent glass plate, which allowed examination in a microscope as well as microphotography of the sample. The denaturating heat was provided by a Leitz heating microscope stage 80, in conjunction with a variable transformer to allow for proper heating speed. A calibrated thermisto (Philips type B 8 - 320 0 2 P/4k7) measured the temperature in the Ringer solution just above the specimen. A 2.5/0.10 objective was used for microscopy. The metal chamber was heated, and microphotographs were taken of the specimen at the start of each experiment at 55° C, at 58° C and then at each degree of increased temperature to 65° C. In the photographs, the length of a chosen fibre could be seen to shrink with increasing temperature, the length of the fibre was measured in mm with a ruler to the nearest 0.5 mm. The shrink-

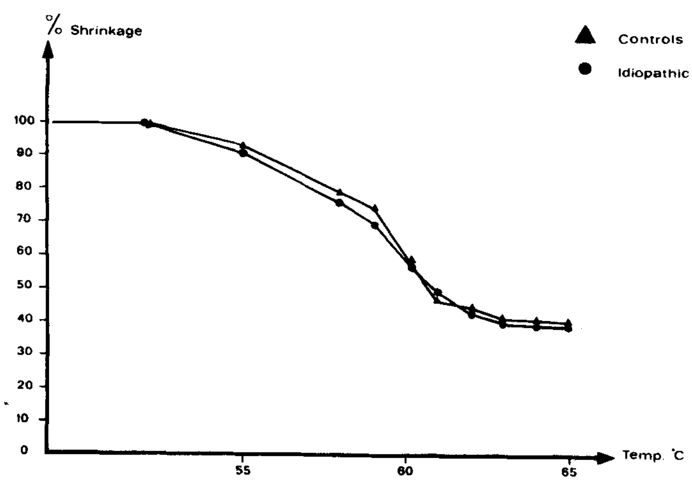


Fig. 13. Heat denaturation. Mean per cent of shrinkage for tendons from patients with idopathic scoliosis and controls.

age in per cent was calculated. Two samples were examined from each individual, the mean percentage of shrinkage was determined. The mean of these means from the group with idiopathic scoliosis was calculated for each temperature degree and compared with the corresponding values of the control group.

## RESULTS

There was no consistent difference between the groups neither in the shrinkage in per cent nor in the speed of shrinkage, nor in temperature where the speed of shrinkage was at its highest ($60^{o}$ C) (Fig. 13).

CHAPTER SIX

## CHEMICAL COMPOSITION OF TENDONS AND LIGAMENTS IN PATIENTS WITH IDIOPATHIC SCOLIOSIS

The mechanical properties of connective tissue depend upon the relative amounts of the different components of the tissue. As described earlier in this paper (page 13) other authors have reported an altered chemical composition of tissues from patients with idiopathic scoliosis. A chemical analysis has therefore been made on the ligaments and tendons.

### MATERIAL

The samples studied chemically were taken from the same kind of structures that were tested biomechanically. They were biopsied from those ligaments of the spine, which were too narrow to allow biomechanical testing, or from residues from the preparation of the ligaments before testing. Tendon samples were removed from the same tendons as the strips for biomechanical testing. 21 tendon samples from 21 patients with idiopathic scoliosis were analysed and compared with 17 tendon samples from 17 control patients. 16 ligaments were sampled from 8 patients with idiopathic scoliosis, and compared with 18 ligaments from 10 control patients. The specimens were wrapped in a saline-moistened gauze immediately after biopsy and the wet weight of the specimen was determined within 1 hour after removal from the body. It was then placed in a plastic container and deep frozen ($-20^{o}$ C) awaiting the chemical analysis. This was generally performed 6 months to 1 1/2 years after the actual biopsy.

## METHODS

For determination of water content 20-50 mg of the sample was vacuum-dried for 24 hours, and the dry weight determined.

In two different samples from the same individual structure, the collagen content and the mucopolysaccharide content were analysed. For the analysis of collagen, the determination of hydroxyproline in the tissue was performed according to the modification of Stegemann's (1958) method as described by Bergman and Loxley (1963). The amount of hydroxyproline recovered from the specimen was assumed to be in a proportion to the collagen content of the tissue as 1/7.46 (Neuman and Logan 1950).

The mucopolysaccharide content was analysed according to the method of fixed negative charge density determination (Maroudas 1968). This method determines the fixed negative charges due to the sulphate and carboxylate groups of condroitinsulphate, hyaluronic acid and keratansulphate.

The method, used by Maroudas on cartilage analysis, has been shown to be equally exact as the determination of hexosamines (Maroudas et al 1969). No controlled analysis on soft tissue has been performed, but there is no reason to believe that the method would be less exact here (Maroudas 1973).

It would have been desirable to make a complete analysis of the total mucopolysaccharide content of the tissue, as well as a specification of each special saccharide involved, however, this has not at the present time been possible because of limited facilities. Thus there remains to be analysed the total amount of glycoproteins in the tissue.

## RESULTS

The results of the chemical analysis is shown in Table 9 with the statistical analysis of the comparison between the groups. There was no significant difference between the groups for any of the substances that were analysed.

In the idiopathic group the elastic stiffness of the tendons was compared with the concentration of collagen in the samples from the same tendons. With those comparatively small variations that existed in the present material, there could not be found any consistent correlation between the collagen concentration and the elastic stiffness expressed in terms of $mm^2$ cross-sectional area.

TABLE 9. Results of the chemical analysis of tendons and ligaments. (Mean±S. D.)

| | Idiopathic | Controls | 2 p |
|---|---|---|---|
| Tendons | n=21 | n=17 | |
| Water content % | 67.3±2.9 | 65.5±4.2 | >0.05 |
| Collagen content (% of dry weight) | 74.8±23.4 | 75.6±18.4 | >0.05 |
| Neg. charged glycosaminoglycans (% of dry weight) | <0.1 | <0.1 | |
| Ligaments | n=16 | n=18 | |
| Water content % | 71.7±8.0 | 70.8±6.2 | >0.05 |
| Collagen content (% of dry weight) | 83.2±17.4 | 85.2±12.9 | >0.05 |
| Neg. charged glycosaminoglycans (% of dry weight) | <0.1 | <0.1 | |

CHAPTER SEVEN

## DISCUSSION

The search for an etiology of idiopathic scoliosis has lead to the study of metabolic conditions in patients with this disease and several authors have reported changes of the metabolism of the connective tissue in these patients. As was described in the introduction both collagen and the ground substance have been reported to be involved. The interest for metabolic causes to idiopathic scoliosis has been aroused by the fact, that several diseases with inborn errors of metabolism have scoliosis as a part of the general disease picture. Also, it has been possible to provoke scoliosis in laboratory animals by a change in their diet, leading to defects of connective tissue in these animals.

Some investigators have stressed the importance of the forces acting on the spine. There has been great interest in the different types of structures, which could possibly interfere with the equilibrium of the spine. A clinical proof of this is the diseases with inborn errors of metabolism with a deficient connective tissue leading to scoliosis and joint disturbances.

The primary function of connective tissue is mechanical, and collagen is the primary force-resisting structure of connective tissue. The mechanical properties depend on the material as such, i.e. the collagen fibre, but also on the geometrical arrangement of these fibres. Probably also other substances in close contact with the collagen, as the ground substance and elastin, may have an influence. On a molecular level, the mechanical properties of collagen are probably the same in all tissues of the same individual. Therefore it is possible, that general metabolic changes of connective tissue and even ground substance, as well as genetically induced variations in the specific collagenous structure, would lead to changes in the resistance of collagen to mechanical forces. This would in turn be a possible cause of scoliosis.

A change of the mechanical properties in the tissue could be brought about by the following changes:

A change of the collagen content of the tissue.

A change of the collagen environment of the tissue, such as water, ground substance or other fibrous structures (i. e. elastin).

A change of the collagen fibre arrangement

A change of the collagen fibrils themselves.

Those authors who have reported changes in the metabolism of patients with idiopathic scoliosis usually have not proposed any theories that would explain the pathogenesis of the process. Ponseti suggested that idiopathic scoliosis be called discogenic scoliosis, as he found a severe derangement of the mucopolysaccharides of the intervertebral disc. This, he believes, would cause a change of the volume of the disc, disturbing the equilibrium of the spine by a decrease of the disc pressure. This has not been corroborated with intravital disc pressure measurement in scoliosis patients (Nachemson 1973).

The tendons studied in this work are mainly built up of collagen fibres arranged in parallel and it is reasonable to believe, that mechanical properties of the whole tendons are similar to those of the isolated fibre. The main difference is in the slight unparallellity causing whole tendons to exhibit a lower tensile strength than isolated fibres.

No reports exist on micro- or macroscopic changes in any separate ligaments or tendons of the body, nor were any changes observed in the structures tested here. Thus, as probably any change in the biomechanical properties of collagen would be general it is believed, that the particular tissues studied in this work - the tendons of the erector spinae muscle and the interspinous ligaments - are representative of any tendons or ligaments around the spine with a parallel fibre arrangement in these patients.

A number of reports has been published on the mechanical properties of such collagenous structures as tendons and ligaments from normal persons. In this investigation, the average tensile strength for the whole group of tendons was approximately 65 $N/mm^2$, which is within the normal values reported for tendons by others: Cronkite (1936) reported an average maximal tensile strength for different human tendons to be 125 $N/mm^2$, Gratz (1931) found the strength of fascia lata to be 48 $N/mm^2$, Walker et al (1964) tested plantaris tendons and found their strength to be approximately 100 $N/mm^2$, while Yamada (1970) stated, that the tensile strength is 50-100 $N/mm^2$ for for tendons of different kinds . It is believed that rupture involves a slip relative to one another of the collagenous elements, probably between fibrils when the intermolecular bonds break. The actual tensile strength of the collagen molecules have been calculated to be approximately 3000 $N/mm^2$, the force required to break the weakest covalent bond C--N (Highberger 1947).

The elastic stiffness (by many authors mentioned as "Young's modulus of elasticity") is reported for normal tendons to be between 830 $N/mm^2$ (Wright and Rennels 1964), and 1170 $N/mm^2$ (Walker et al 1964).

Only in one earlier work supra- and interspinous ligaments have been studied (Waters and Morris 1973). The structures were tested in a way similar to that in the present study, and included specimens from patients with idiopathic and other types of scoliosis. The mean elastic modulus of the tested ligaments was found to be 129 $N/mm^2$ in the idiopathic group, and 113 $N/mm^2$ in the non-idiopathic group.

The mean elastic stiffness of the tendons in the present work was about 600 $N/mm^2$, while in the ligaments it was considerably less, in average 45 $N/mm^2$, or less than 1/10 of the stiffness in tendons.

The reasons for this comparatively low elastic stiffness in the ligaments are several. First, the fibres in the ligaments are not as strictly parallel as in tendons, where the direction of the force exerted by the muscle pull is very strictly defined. Secondly, in the preparation of the test samples, the original ligaments have been considerably reshaped and it is certain, that some fibres at the caudal, fan-like end of the ligament have been cut. Thirdly, the insertion of the fibres into the bony spinous process is seldom straight, but oblique, making the force unevenly distributed here.

The reasons for great differences in the values reported by different authors are several: different tendons have been used (different values for extensor and flexor tendons have even been reported, Viidik 1967) and the specimens have been treated differently after removal from the body (tanning, drying), which also occurred at different time intervals before the actual testing. Different authors have used different cross-sectional area measurement methods, and the elastic modulus has been measured at different strain rates and at different strain levels. Also the subjects from which the specimens have been taken have been of different ages. With increasing age, there is a tendency for the tissue to become stiffer (Rollhäuser 1950, Yamada 1970).

In the present study, these differences have been eliminated between the two groups tested. In the age groups the patients were of the same chronological as well as skeletal age. The tendons and ligaments were of the same kind in the two groups. Macroscopically there were no differences between the groups in the general appearance of the samples, nor in the arrangement of the fibres. The structures were treated in exactly the same way in both groups. The measurements of elastic modulus were made at the same strain level, and they were performed at approximately the same strain rate.

The ultimate tensile strength, and probably also the strain at which the elastic stiffness has been determined, lies well beyond the physiological range of function of the tissue. It has been calculated that most of the functiononal activity of a tendon occurs within the toe part of the stress-strain curve, and at times during the early linear part of it. The normal physiological range of activity of tendons probably is below 3 per cent strain(Rigby et al 1959, Elliott 1965, Abrahams 1967). Although a physiological testing with small strains and forces during a long period of time would be desirable, such tests are not possible at present as the problems for preserving the structures in a working condition during a lengthy time have not been solved.

In the present study, the elastic stiffness, the tensile strength and the other biomechanical parameters of tendons and interspinous ligaments from patients with idiopathic scoliosis did not differ from those of patients with scoliosis of known origin. Nor did the interspinous ligaments differ biomechanically between the two groups. This was also the result in a recent study by Waters and Morris (1973). They studied supra- and interspinous ligaments in 16 patients with idiopathic scoliosis, and compared these with ligaments from a group of 10 patients with scoliosis of a known etiology. In this control group, however, was also included a patient with Marfan's syndrome, which might be responsible for the lower elastic modulus in the latter group, 113 ±39 $N/mm^2$ as compared to 129 ±42 $N/mm^2$ in the idiopathic group. The strain level was not specified, and the cross-sectional area of the ligaments was determined after the testing. In the present study, there could be shown neither in the control group nor in the idiopathic group any consistent change in mechanical properties of interspinous ligaments of different levels in the spine. Nor was it possible to demonstrate any significant change of elastic stiffness in the ligaments with different locations of the scoliotic curve. The high frequency of middle and low thoracic levels in idiopathic scoliosis does not seem to depend on any change in the mechanical properties of the interspinous ligaments.

It was found, that the correctability of the scoliosis with the Harrington distraction instrument was highly correlated to the elasticity of the tendons adjacent to the spine in patients with idiopathic scoliosis. In the Harrington operation, a force is used to extend the distance between the end points of the curve, this force is counter-acted by a number of structures along the curve. Such structures are the discs, the muscles, the tendons and the ligaments as well as the structural changes in the bone. It has been obvious to scoliosis surgeons, that a better result of the operation is obtained in patients, who already preoperatively show a less rigid curve as demonstrated by the change in the curvature from an erect to a recumbent position. In this study,the result of the surgical treatment seemed to depend more on the biomechanical properties of the structures in the vicinity of the spine than on other parameters, such as the age of the patient (up to approximately 20 years), (correlation coeff. $r = -0.20$, $2p > 0.05$), or the original severity of the curvature ($r = -0.08$, $2p > 0.05$). The difference in elastic stiffness of the connective tissue of different patients can explain the sometimes different end results in correction of two preoperatively identical curves in patients of the same age.

The clinical study of the joint elasticity shows, that about 6 per cent of the patients with idiopathic scoliosis were affected with such a joint laxity. 75 per cent did not have more than one joint affected. In other studies, normal controls have been reported to have a total prevalence of such increased laxity of joints in 7 per cent of subjects aged 6-11 years (Carter and Wilkinson 1964). In a series of Kirk et al (1967), none of 9 normal controls about 15 years of age had any laxity of the joints, while at the age of 5 to 14 years there were 2 cases out of 7. Wynne-Davies(1970) reported 5 per cent of normal children in the 6 year age group to be affected, while this had decreased to less than 1 per cent in the 12 year age group. Thus, it seems as though the patients with idiopathic scoliosis would be in an upper range of the normal prevalence for such joint laxity. This is in accordance with the experimental findings of the mechanical properties of the tendons and ligaments, where a non-significant tendency towards a decrease of elastic stiffness in the idiopathic group was noted. The tensile properties of those three patients with increased joint laxity in our study did not, however, show any difference, when compared with the mean of the whole idiopathic group.

The biomechanical studies of collagen maturity did not reveal any difference of heat denaturation properties between the idiopathic and the control groups. Although progression of scoliosis is related to the maturity of the tissues, this probably is mostly dependent upon the skeletal growth as noted by Risser (1958) and others, and not the soft tissue. The increase in height that has been observed in these patients (Willner 1972) also probably has to do with bone growth, and is not primarily related to soft tissue.

The chemical analysis of ligaments and tendons of the two groups tested in this investigation shows, that although there is a considerable variation within the groups, there is no difference between the two groups in the percentage of collagen. Mathews (1965) maintained, that a certain minimum amount of water of hydration appears to be essential for the stability of the molecule, for the collagen molecule this comprises about 30 per cent weight of the protein. In the present study there was no difference in water content between the two groups, nor did the concentration of the negatively charged glycoprotein (less than 0.1 per cent on a dry weight basis) differ between the idiopathic and the control groups. The mucopolysaccharide content analysed by this method is only a part of the total MPS content of the tissue. There remains to be made a further analysis of the amounts and types of glycosaminoglucans. Because of limited chemical facilities this could not be performed in the present study.

Thus, in this investigation it has not been possible to show, that any of the different properties of collagen studied in relation to the etiology of idiopathic scoliosis - biomechanical or biochemical - would be of any importance. It seems unlikely that the theories for the pathogenesis of idiopathic scoliosis based on the occurrence of scoliosis in individuals with collagen diseases would be relevant for the etiology of idiopathic scoliosis.

CHAPTER EIGHT

## SUMMARY


The etiology of scoliosis is largely unknown. The prevalent theories concerning the etiology and pathogenesis of idiopathic scoliosis have been briefly reviewed. In later years there have been an increasing interest in studies of the metabolism and chemistry of various tissues in particular ligaments of the spine and the intervertebral disc, in patients with idiopathic scoliosis. Scoliosis is a common feature in diseases with inborn errors of metabolism. Like in lathyritic animals, the connective tissue has been shown to be deficient in these diseases, with a decreased resistance of the collagen against deforming forces. As it also has been proposed that patients with idiopathic scoliosis have an increased laxity of their joints, it seemed that a derangement of the mechanical properties of the tendinous tissue might be a cause to the deformity also in idiopathic scoliosis. Therefore mechanical testing of tendons and ligaments from patients with idiopathic scoliosis was performed.

From 40 patients with idiopathic scoliosis interspinous ligaments and tendons of the erector spinae muscle were studied biomechanically and biochemically, and compared with the same type of structures from 20 control patients. All but two of these had scoliosis of a non-idiopathic, known etiology. The samples were obtained in connection with surgical correction of the scoliotic spine with the Harrington distraction rod.

Within one hour after removal from the body the samples were prepared with a double-edged knife into strips of a uniform size. The cross-sectional area of the samples was measured in $mm^2$.

The structures were tested fresh within 3 to 7 hours after the biopsy. They were subjected to three load-deload experiments and one relaxation test. The tendons were also strained to rupture. The elastic stiffness of each specimen was determined in each load cycle, and was found to increase with successive loading cycles and also with increased strain rate. In the third load cycle the mean elastic stiffness for tendons was approximately 600 $N/mm^2$ (61 $kp/mm^2$), while the ligaments had a considerably lower elastic stiffness, in average 46 $N/mm^2$ (4.7 $kp/mm^2$). In order to eliminate possible age dependence in the evaluation of the results, the patients were subdivided into age groups. There was not noticed any significant difference in elastic stiffness with increasing age up to about 20 years of age.

The elastic stiffness of the tendons and interspinous ligaments from patients with idiopathic scoliosis did not differ significantly from the elastic stiffness of the same type of structures from the control patients.

Other parameters that were studied in both tendons and interspinous ligaments were plastic deformation, and the percentage and speed of relaxation in the isometric relaxation test. In tendons, the tensile strength and the elongation at failure were tested. The means of these parameters did not either differ between the idiopathic and the control groups.

Interspinous ligaments were not tested for tensile strength and elongation at failure, as they do not break in the ligament substance, but in the attachment of the ligament to the bone.

The elastic stiffness of the tendons from the patients with idiopathic scoliosis was highly correlated ($r = -0.53$) to the correctability of the scoliosis deformity with the Harrington instrument.

The elastic stiffness of the interspinous ligament was not correlated to the location of the ligament, nor was the location of the scoliotic curve dependent upon the elastic stiffness of the interspinous ligaments.

In a clinical study of the joint mobility of 48 patients with idiopathic scoliosis with a method based on examination of 6 pairs of joints it was found, that approximately 6 per cent of these patients had an increased joint elasticity. This is in the upper range of what has earlier been found in the normal population. The patients with increased joint laxity did not show any decreased elastic stiffness in the tendons tested biomechanically.

The physical properties of collagen are dependent upon the biological age or the "maturity" of the tissue, which is considered to be determined by the number of cross-links existing between the collagen molecules. This "maturity" was evaluated in tendons of the erector spinae muscle from 12 patients with idiopathic scoliosis by studies of the heat denaturation process. There was no significant difference in shrinkage temperature nor in shrinkage percentage of the idiopathic tissue when this was compared with tendons from 14 control patients. It can be concluded that the biological age of tendinous structures in idiopathic scoliosis patients do not differ from that in normal subjects.

A chemical analysis was made of the content of collagen, water and negatively charged glycosaminoglycans in 21 tendon samples from idiopathic scoliosis and 17 tendon samples from control patients. These parameters did not either differ between the groups.

## CONCLUSION

It has not been possible to demonstrate in this study that idiopathic scoliosis is caused by any derangement of the mechanical properties of ligaments and tendons or by a laxity of the joints. Studies of the biological age of the tendinous tissue revealed normal conditions in tendons of patients with idiopathic scoliosis when these were compared with controls.

Chemical analysis of the content of the collagen, water or glycosaminoglycans did not indicate any cause to idiopathic scoliosis in the structures tested.

It was shown that the result obtained in surgical correction of the deformity in patients with idiopathic scoliosis was correlated to the elastic stiffness of the tendinous tissue of the patient.

# PART II

## RESULTS OF CONSERVATIVE TREATMENT WITH THE MILWAUKEE BRACE AND OPERATIVE TREATMENT WITH A TWO-STAGE HARRINGTON ROD PROCEDURE

CHAPTER NINE

## REVIEW OF TREATMENT METHODS

### INTRODUCTION

Scoliosis has always been a difficult problem in orthopaedics, and for centuries, the orthopaedic surgeon could do nothing but observe the gradual deterioration of the deformity. Scoliosis is a progressive disease, which untreated shortens the patient's life, affecting her physically, mentally and socially.

The deformity causes pulmonary and cardiac disturbances. Already at curves exceeding $60^{o}$ it has been noticed that a significant percentage of patients have a decrease in respiratory vital capacity (Collis and Ponseti 1969). The prognosis for patients with untreated idiopathic scoliosis has been studied in three different reports, which all appeared at about the same time. Nachemson (1968) reported in a 35 year follow-up of 117 scoliotic patients, that nearly 50 per cent had some cardiopulmonary trouble. There was a more than 100 per cent increase in mortality in these patients when compared with the general population. In the more severe curves, this increased mortality was about four times. An increased mortality rate was also reported by Nilsonne and Lundgren (1968) in a 50 year follow-up of 100 patients with idiopathic scoliosis. A significantly great number of women with this deformity did not marry. Collis and Ponseti (1969) showed in 195 patients followed-up for an average period of 24 years that the decrease in vital capacity was proportional to the degree of the scoliosis. This has also been demonstrated by others e.g. Nilsonne and Lundgren (1968) and Westgate (1970).

Progression of the scoliotic curve is related to the growth of the patient (Risser 1964, Calvo 1957, Duval-Beaupère 1971). A fast progression of the deformity is often noticed during the adolescent growth spurt. After the end of vertebral growth, which is indicated by the completion and fusion of the iliac apophysis, the progression rate slows down considerably (Risser 1948, Zaoussis and James 1958). Risser (1964) stated that progression of the scoliosis after growth completion is approximately one degree per year. This was also verified by Collis and Ponseti, who in their follow-up study showed that the scoliosis increased somewhat after end of growth. It was particularly the thoracic curves between $60^{\circ}$ and $80^{\circ}$ which increased, on an average $30^{\circ}$. Double primary curves and lumbar curves did not progress to the same extent as thoracic curves, nor did they influence the pulmonary function as seriously.

Treatment of the deformity is thus for several reasons of great importance for the patient. Milder curves need primarily to be protected from further progression, while more severe curves need active correction as well.

## CONSERVATIVE METHODS OF TREATMENT

Already Hippokrates described, that the deformity could be temporarily corrected by traction. It was not until recently that effective methods of treatment were introduced, however, and the Hippokrates method was still in use in Europe up to the 20th century. In the literature a great number of methods have been described for treatment of scoliosis, several with the purpose of correcting the deformity by means of active exercises. One well-known system was described by Klapp in 1905,and only a few years ago a description of the Klapp method for correction of scoliosis was published in a new edition(Klapp 1966). Such methods were proved to be of no significant value in the treatment of scoliosis already many years ago(Shands et al 1941),and there have been no reports since,on the favourable correcting or preventing effect of physiotherapy for scoliosis. Nor has prolonged bed rest or recumbency in a plaster cast proved to have any permanent effect on the deformity (Arkin 1964). Some external force has to be applied, and of conservative methods of treatment different means of bracing can have a corrective influence on the deformity. Particularly the Milwaukee brace has been shown to give a satisfactory result when used correctly and with the proper indications. It is effective in curves under $40$-$50^{\circ}$ (Moe and Kettleson 1970), and its primary function is then to prevent further progression of the scoliosis.

The Milwaukee brace, which was presented in 1945 by Blount and Schmidt, was first designed as a recumbent postoperative frame for patients undergoing spinal fusion for scoliosis (Blount et al 1958a). Clinical experience in time revealed, that the brace was effective also in the non-surgical, ambulatory treatment of scoliosis (Blount 1963). The brace has undergone many improvements since its first introduction, and is now used in most clinics treating scoliosis. The use of the brace has resulted in a better prognosis for cases of mild and moderate scoliosis. Goldstein (1971) has estimated that the number of patients requiring surgery in his practice has become reduced by 50 per cent by the use of the Milwaukee brace. Blount has described the design and principle of the Milwaukee brace in several works (Blount et al 1958b, Blount and Moe 1973). The brace should have a well-molded pelvic girdle and a chin piece, the function of which is to hold the head back towards the occipital support rather than to exert pressure on the mandible. The chin piece has been manufactured since 1969 by Blount as a small contour-shaped plastic device. The traction force should be supplied mainly by the occipital support. The brace should be fitted with two padded straps, one of which usually is a sling around the shoulder on the concave side. The pressure pads are supposed to give an efficient lateral pressure and their position should be controlled in a roentgenogram. According to Blount, the efficiency of the Milwaukee brace depends on the alternating forces of passive distraction and active derotation, with active distraction and passive derotation. As a consequence of this, Blount (1964) also pointed put, that the activity of the patient in the Milwaukee brace should be as normal as possible, including sports of different types.

Curves of a more severe degree need to be forcibly corrected, and several external methods for a more active correction of scoliosis have been described. There are the surcingle cast (von Lackum and Miller 1949), the localizer cast (Risser 1955), the E. D. F. -cast (Cotrel and Morel 1964),the turnbuckle cast and traction devices such as steady or dynamic Cotrel traction(Hensinger and MacEwen 1973) or skeletal traction with a halo-femoral or halo-pelvic arrangement (DeWald and Ray 1970,O'Brien et al 1973).

## OPERATIVE METHODS OF TREATMENT

It was not until Hibbs in 1911 described the first spinal fusion, that an effective operative method to prevent progression of the deformity was introduced. Hibbs reported on three patients operated with posterior spinal fusion for tuberculosis of the spine. He performed the first fusion in a patient with paralytic scoliosis in 1914, and reported the result of such fusions in 59 patients in 1924. It was first in the 1950:ies, however, that operative treatment of these patients was introduced to a larger extent. The Hibbs technique of fusion has later been modified. Cobb (1948, 1960) emphasized the importance of articular facet fusion, and this technique was also described by

Moe (1958, 1961) and others.

For internal operative correction of severe progressive scoliosis, several methods have been described. In 1951 Nachlas and Borden described a correction of curves in experimental animals by stapling of the vertebral epiphysis. This was also tried by Smith et al (1954), however, with disappointing results. Convex side epiphyseodesis of the apex vertebra has been used by Apley (1961), Roaf (1963) and by Nilsonne (1969), but without success. Others have tried osteotomies of one or several vertebral bodies (Roaf 1955,Kazmin et al 1962, Hodgson 1965), or disc resection (Kazmin 1969). Lindahl (1966) tried resection of the transverse processes, and recently Piggott (1971) reported results after posterior rib resection in scoliosis, but with negligeable and unpredictable effect on the curve

Instruments of different kinds have been used, such as various types of plates, springs, hooks and grafts. Gruca (1958, 1973) described correction of the curve with springs attached to the vertebral pedicles in combination with alloplasty of the muscles. Dwyer recently introduced an effective method of correction with the aid of screws and wires along the convexity of the vertebral bodies (Dwyer et al 1969). In 1962 Harrington introduced a method with the purpose to correct the scoliosis with an instrument, which also stabilizes the spine while the fusion is healing. With the Harrington instruments the curve can be internally straightened by distraction on the concave side, and by compression on the convex side. A fusion is, however, also necessary for the long term maintenance of correction.

The reliability and superiority of the Harrington technique is by now well established both concerning the operative correction of the deformity and the minimal long term postoperative loss of correction.

# CHAPTER TEN

## MATERIAL

Since 1966 scoliosis is treated in Gothenburg at a special department with fixed indications for conservative and operative treatment and with a strict follow-up routine. The majority of the patients was referred from other orthopaedic hospitals in the Western and Southern parts of Sweden, an area covering a population of about 4 million people.

During the period March 1967 to April 1971, treatment for scoliosis was started in 264 patients, which is approximately 2/3 of the total number of patients referred under the diagnosis of scoliosis during that period. All patients were seen by the same physician, Prof. Alf Nachemson.

In the material 24 patients (9%) were boys. 227 patients (86%) had idiopathic scoliosis. 15 patients (6%) had scoliosis of a congenital origin, while poliomyelitis was the cause of scoliosis in 10 patients (4%). Two patients had neurofibromatosis, and two patients had osteogenesis imperfecta. Muscle disease was the cause of scoliosis in two patients, and hemiplegia in two patients. One patient had Marfan's syndrome. The deformity was secondary to medical interventions (iatrogenic) in three cases.

### AGE OF PATIENTS AT DIAGNOSIS

The deformity was diagnosed in 15 patients aged under 3 years, and in 29 patients aged between 3 and 9 years, while the remaining were diagnosed in ages ranging from 9 years and older. The most common age at which the deformity was diagnosed was 12 years.

In 48 per cent of the cases, the parents discovered the deformity, and in 36 per cent it was diagnosed by a physician usually at a routine examination. Only 7 per cent of the patients discovered the deformity themselves.

## SUBJECTIVE SYMPTOMS

Very few patients consulted a doctor because of subjective symptoms. When questioned, 23 per cent of the patients stated that they had back pain, usually of a mild degree - never severe enough to warrant pain medication. The pain was either located distally in the lumbar spine, or along the convexity of the scoliosis, usually near its apex. Back pain was most common among those patients who had scoliosis located proximally in the spine, three out of seven patients with high thoracic curves complained of such pain. For patients with other curve locations the frequency of pain was approximately 23 per cent, and did not differ with the curve locations. Nor did the frequency of pain change with the severity of the curve.

## CLINICAL EXAMINATION

Five patients had an abnormal electrocardiogram, three of these were mild right intraventricular blocks, while two patients with heart malformations exhibited more severe changes in the ECG. One patient with a thoracic idiopathic scoliosis had a coarctation of the aorta, and the other patient - with a thoracic congenital scoliosis - had a V.O.C. with a septal defect. In two patients, both with idiopathic curves, there were anomalies of the urinary tract, one had an aplasia of the right kidney, and the other had a duplication of the renal pelvis and a malformation of the bladder.

One patient with thoracic idiopathic scoliosis had a lumbar spina bifida. Another had a spondylolisthesis grade 1, this patient had an idiopathic thoraco-lumbar curve of $40^{\circ}$.

## RESPIRATORY FUNCTION

The respiratory function was determined in all patients by dynamic spirometry before any treatment was started. In all cases with a decrease of the vital capacity there was a restrictive change of the lung function, while one case also had an obstructive lung disease. The vital capacity was assessed as the percentage of the predicted normal value, using the corrected height of the patient as a reference (Bjure et al 1968). In most patients the vital capacity was lower than the predicted normal value. There was a significant negative correlation between the degree of curvature and the vital capacity. It was in particular in curvatures of more than 60° that the decrease in vital capacity became pronounced (Fig. 14). Patients with double primary curves had a smaller decrease in vital capacity than those with single primary curves.

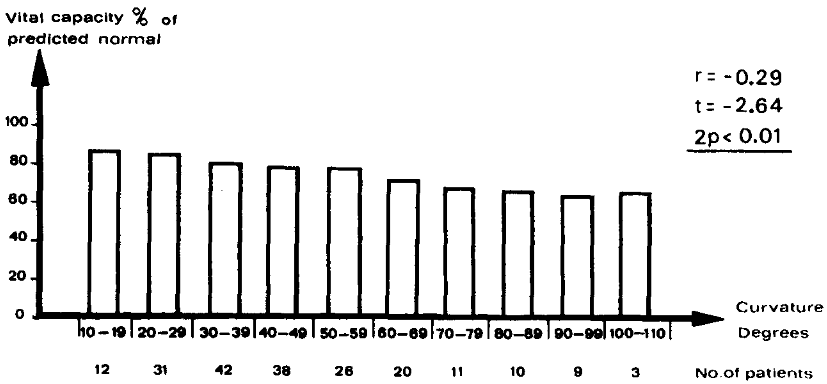


Fig. 14. Mean value of the vital capacity correlated with the degrees of curvature before treatment.

## INDICATIONS FOR TREATMENT

With a knowledge of the progressive nature of scoliosis, the indications for treatment of these patients should be based on an evaluation of the rate of progression and of the degree and location of the scoliosis. The following general rules for treatment have been applied:

Treatment with the Milwaukee brace

- Patients with scoliosis of all types of etiology, who have not yet attained full spinal growth.
- Thoracic curves ranging from $15^{o}$ (Cobb) and up to $50^{o}$.
- Lumbar curves from $20^{o}$ to $60^{o}$.
- Curves in patients near end of growth from $20^{o}$ up to $60^{o}$.

Treatment with the Harrington instrument

- Thoracic curves of $50^{o}$ or more, in patients with 2 years or more of expected additional skeletal growth.
- Thoracic curves of $55^{o}$ or more in patients approaching end of growth.
- Thoraco-lumbar curves of $50^{o}$ or more.
- Lumbar curves exceeding $60^{o}$.
- Double primary curves of $60^{o}$ or more in either one curve or both.

Patients have rarely been operated after end of growth, in these cases the flexibility of the spine together with the severity of the curve were the decisive indications for operation.

## CONSERVATIVELY TREATED MATERIAL

A Milwaukee brace was used for the treatment of 159 patients (60%) (13 boys and 146 girls). These patients were all followed for two years or more. Also those patients who refused to continue with the brace were regularly checked until skeletal maturity. 86 patients had in April 1971 completed the full treatment program one year ago or more, and were all followed-up retrospectively. Of these 86 patients a selective study was made of 77 patients with idiopathic scoliosis. Thus, the patients who did not wear the brace as directed were excluded (14 patients). Those patients who had been treated surgically before end of brace wearing at cessation of growth were also excluded (15 patients). In dealing with these results, it must also be remembered, that most of the patients, in whom the curve progressed to about $60^{o}$ despite treatment with the brace were operated on and thus excluded from this selective study. Also, if the brace proved very successful with a good correction of the curve, weaning of the brace was often started earlier. No doubt, in these cases the curve would have improved more with the full brace program.

Of the 159 conservatively treated cases, 48 (30%) were considered to be double primary curves, i.e. had two curvatures of a major degree, each with structural changes causing cosmetic deformity. In none of the patients were there more than two main structural curves in the thoracic and lumbar areas. The distribution of the curves according to the apex location is shown in Table 10. 90 per cent of the curves in the thoracic region were convex to the right, while the majority of the curves in the lumbar region were convex to the left.

TABLE 10. Curve distribution in 159 patients treated primarily with the Milwaukee brace.

| | Number of curves | | | |
|---|---|---|---|---|
| | Single primary | | Double primary | |
| Apex | left | right | left | right |
| D 4 - 6 | | 1 | 1 | 3 |
| D 7 - 9 | 2 | 58 | | 38 |
| D 10-12 | 10 | 26 | 3 | 7 |
| L 1 - 3 | 11 | 3 | 44 | |

SURGICALLY TREATED MATERIAL

Of the 264 patients with scoliosis, 105 patients (40%) were primarily treated surgically with the Harrington distraction rod, 9 patients with a one stage procedure, all non-idiopathic, and 96 patients with a two-stage method. In addition to these, there were 4 patients initially treated in a Milwaukee brace who were later operated with the Harrington rod in two operations during the period March 1967 to April 1971. Thus, altogether 100 patients were operated with a two-stage procedure in that period. Nine of these were boys. In all patients the same type of operation was performed with the same type of treatment pre- and postoperatively. All patients were operated by the same surgeon, Prof. Alf Nachemson. 86 patients had single primary curves, 14 had double primary curves, and altogether 114 curves were corrected and fused in 203 interventions in these 100 patients. (One patient was reoperated twice after laminar fractures, and one patient was reoperated because of hook dislocation.)

The average age at operation was 15 years and 4 months. On an average the iliac apophysis was then close to its full excursion.

A two-year follow-up was performed in all these 100 patients. All the patients were treated postoperatively in a Milwaukee brace following 3 weeks of postoperative recumbency after the second operation (lumbar curves 5 weeks). 55 patients were followed-up for 3 years, 23 for 4 years.

# CHAPTER ELEVEN

## METHODS

### CONSERVATIVE TREATMENT

The indications for Milwaukee brace treatment are stated above. When decision has been made to treat the patient with a Milwaukee brace, she or he is carefully informed about the disorder and about the importance of the treatment. The patient is told that brace wearing is necessary if the curvature is not to deteriorate. At the first visit to the scoliosis clinic, the patient is seen not only by the physician, but also by a physiotherapist with special interest in these problems. The patient also visits the scoliosis ward to become informed about the program and to meet other patients under treatment with the Milwaukee brace. A social worker is visited and special educational and emotional problems are tackled. In the orthotist shop a positive cast is made for the manufacturing of the brace girdle. The brace is then produced in 4-6 weeks. When the brace is ready for final fitting, the patient is always admitted to the scoliosis ward for approximately one week. In the hospital clinical and roentgenographic examination, orthodontic evaluation, dynamic spirometry, electrocardiography at rest and at work, and height measurements are performed. Roentgenograms of the whole spine in the erect and supine positions, A - P and side views, are taken. The development of the skeleton is assessed from roentgenograms of the iliac apophysis. The Milwaukee brace is carefully fitted, and after one day a frontal view of the spine in brace is taken with the patient standing. The position of the side straps is controlled, and if the curve has not decreased, the brace and the side straps are corrected. In this way, most curves become corrected initially. As will be shown on page 109, the final result at the end of brace treatment years later is highly correlated with this immediate post-fitting correction in the brace.

While in the ward, the patient is taught the physical exercise program according to Blount an Bolinske(1967), which she or he is supposed to continue until the end of the brace treatment. This is further controlled by the physiotherapist at each subsequent visit to the out-patient clinic.

The patient is controlled in an out-patient scoliosis clinic approximately every third to sixth month. The effect and the fitting of the brace are checked in a standing A-P roentgenogram. The skeletal development is assessed from roentgenograms of the iliac apophysis, and standing and sitting heights are measured. The patient is also regularly checked by an orthodontist. On the patient's first visit to the orthodontist, dental and cephalometric roentgenograms are taken, and an orthodontic cast is produced. On regular controls the bite is checked for any deformities, and if such are diagnosed the patient is supplied with a preventive, and in some cases also corrective, dental brace or activator. Such an appliance is also given patients, who already at the start of the treatment have a tendency to protrusion of the teeth or in any other way run a greater risk of complications from the treatment in Milwaukee brace.

During the brace treatment, the patient is encouraged to take part in sports and physical exercises. However, she or he must use the brace 24 hours a day, and can only be allowed out of the brace during short periods for showers or baths. This is in accordance with the directions given by Blount. Furthermore, the brace by measurement has been shown to exert effective distraction force on the spine also in the supine position (Nachemson and Elfström 1971, 1973).

Weaning of the brace is begun when skeletal growth can be considered to be ended, i. e. when the iliac apophysis has started to fuse with the ilium and when there is no further increase in sitting height. The patient is allowed to be without the brace during successively longer periods of time, starting with two additional hours a day each month. After approximately half a year the patient only wears the brace at night, and is finally allowed complete freedom. If the curvature increases during brace weaning, treatment is again adopted and weaning is tried half a year later.

## OPERATIVE TREATMENT

### Preoperative treatment

No preoperative cast or traction device was applied. The patient was admitted to the hospital one week before the operation for preoperative cardio-pulmonary examination with ECG at rest and at work, dynamic spirometry, and respiratory exercises under the supervision of the physiotherapist. Preoperative roentgenographic examination included A-P views of the whole spine in the erect position with a film size of 100 by 33 cm, and supine A-P and lateral views with a regular film size. Standing and sitting height measurements were also taken, and the remaining skeletal growth was assessed from roentgenograms of the iliac crest.

### Operation

#### Anaesthesia

All patients included in this investigation were operated under general intubation anaesthesia, most of them with halothane, which was induced with the patient in the supine position. In the latter part of the series neurolept analgesia with fentanyl and the muscle relaxant tubocurarine was used. Before extubation all patients received diazepam intravenously. This type of anaesthesia facilitated a more gentle awakening of the patient after the operation. The risk of extensive forces on the hook purchase sites during these critical moments were thus reduced.

Operative technique

After induction of the anaesthesia the patient was turned in a prone position and placed in the operation frame, modified according to Relton and Hall (1967). Haemostasis was achieved by 100 - 150 cc of a 1/600 000 solution of adrenaline injected into the paraspinal and subcutaneous tissue before the skin incision, after this with electrocoagulation.

At the first operation, the distraction rod was used in the same manner as was recommended by Harrington (1962). The posterior structures of the spine, i.e. the spinous processes, the laminae, and the transverse processes, were exposed subperiosteally along the concave side of the curve. The segments, which were to be included in the fusion, and the upper and lower hook sites had been selected preoperatively from the roentgenograms, and were identified during the operation by direct palpation and inspection of the ribs and the transverse processes.

The superior hook site was prepared by an osteotomy of a small edge of the most caudal part of the inferior articular process of the most proximal vertebra which was to be included in the fusion. The hook was not driven into the pedicle, but attempts were made to place the hook medial to the pedicle, which will then prevent the hook from slipping laterally. The stability of the hook was tested by lifting it and by trying to rotate it laterally. This placement of the upper hook purchase site is according to the experimental findings reported by Waugh (1966).

The inferior hook site was prepared on the proximal border of the lamina of the most distal vertebra which was to be included in the fusion, usually medially to the intervertebral joint. A small osteotomy was generally necessary here, although it was sometimes sufficient to remove the flavum ligament in the interlaminar space.

After the hooks had been seated, the distraction rod was inserted. In this material, the curve was corrected by an average force of 45.1 kiloponds (range 30-50 kiloponds). The force was measured by a special force-indicating distractor (Nachemson and Elfström 1969). The aim was to prevent laminar fractures, the use of a known force also allows a more correct analysis of the influence of other factors on the result. After securing the distraction rod with a twisted stainless steel wire, the wound was closed in layers with continuous intracutaneous Supramid sutures in the skin. Two Redon drainages were always left in the wound to evacuate the haematomas. These drainages were kept in the wound as long as they functioned properly, i.e. 1 - 2 days.

The patients were all operated in two stages. A two-stage operation is based on the fact, that already during operation, as the tissue relaxes, the force on the rod decreases, allowing additional correction after a short time. The force on the Harrington rod and the force decrease during operation was studied by Waugh (1966) by intravital measurements in three patients, and later by Nachemson and Elfström (1973) who reported on 10 patients followed for two weeks by means of telemetry. The force on the rod successively diminishes in periods, after 10 - 11 days being only about 25-30 per cent of the original force that was used primarily for correction. After this period it is possible to distract the spine further, gaining an additional amount of correction.

A second operation also allows a thorough check of the hook purchase sites. Furthermore, it might have some biological advantage in promoting a rapid bony fusion (Siffert and Barash 1961, Sako and Marchetta 1966).

The second stage operation was performed 14 days later. The wound from the previous operation was re-incised, and the posterior structures of the spine were exposed subperiosteally on both the convex and the concave sides. The Harrington distraction rod was removed in order to permit the preparation of the fusion site on the concave side. The rod was then inserted again and distracted, not exceeding the distraction force used in the first operation. Usually one additional notch was gained on the rod. A meticulous fusion was undertaken with decortication of spinal processes, laminae and transverse processes in the thoracic and in the lumbar area. Each intervertebral joint in the thoracic and lumbar area was destructed, and autogenous bone was taken in large amounts from the iliac crest. Usually one donor side was sufficient. The fresh bone was stored only for 10-15 minutes in saline and was then directly laid down on the convex and concave sides along the whole extent of the curve. Before closure of the wound, a final attempt at distraction was applied to the rod. The skin was closed with continuous intracutaneous Supramid sutures leaving three drainages in the wounds, two at the spine and one at the iliac crest.

In 86 patients with single primary curves one single rod was used in 85 patients and two parallel rods were inserted in one patient. In 14 patients with double primary curves one rod was used in 9 patients with the "dollar-sign" application, while two rods were used in 5 patients, one rod correcting each curve.

Table 11 shows the operation time, blood loss and blood replacement. There is an apparent overtransfusion, as the blood replacement also compensates for the losses into the drainages and into the tissues, while these losses are not included in the blood loss stated in the table. The values include all types of scoliosis, mostly (84%) idiopathic. Congenital and neuropathic cases required a slightly longer mean operation time (1:st stage 2 hours 5 min, and 2:nd stage 2 hours 48 min). The blood loss in these cases was also slightly higher, in the first stage mean 687 cc, in the second stage mean 1625 cc. As a routine with an operating time exceeding 2 hours, penicillin was administered in doses of 5 Mill. I. U. parenterally per day for three days. After this 3 Mill. I. U. per day was given orally for another week.

TABLE 11. Operating time, blood loss and blood replacement in 100 patients operated with the two-stage Harrington method.

| | 1:st stage | 2:nd stage | Total |
|---|---|---|---|
| Single curves | | | |
| Operating time (hours. min) | | | |
| mean | 1.44 | 2.36 | 4.20 |
| range | (0.45-3.45) | (1.20-4.45) | (2.15-7.40) |
| Blood loss at operation (cc) | | | |
| mean | 492 | 1431 | 1923 |
| range | (150-3200) | (400-4500) | (650-5200) |
| Blood replacement (units = 400 cc) | | | |
| mean | | | 9 |
| range | | | (3-17) |
| Double curves | | | |
| Operating time (hours. min) | | | |
| mean | 2.12 | 3.10 | 5.22 |
| range | (1.15-3.25) | (2.30-4.30) | (4.10-6.45) |
| Blood loss at operation (cc) | | | |
| mean | 541 | 1825 | 2366 |
| range | (250-900) | (900-3800) | (1150-4600) |
| Blood replacement (units = 400 cc) | | | |
| mean | | | 9 |
| range | | | (5-15) |

Postoperative treatment

Postoperatively and between the two operations the patient was treated with recumbency mainly in the supine position for three weeks (lumbar curves and double primary curves 5 weeks) after the second operation. The detailed postoperative treatment program during the first few weeks following the operations was described by Nachemson and Elfström (1971, 1973), and is based on intravital measurements of the forces in the Harrington rod, using an implanted telemetry device (Elfström 1972).

At ambulation the patient was fitted with a carefully adjusted Milwaukee brace, and returned home about 4 weeks after the second operation. Average time in hospital was 6-7 weeks. During the next six months, the patient was gradually allowed to take up full activity of daily life, first at home, and later at school. At regular three months intervals the patient was controlled in the out-patient scoliosis clinic. At each visit, the whole spine - including major and compensatory curves - was controlled with one A-P roentgenogram in the standing position. The same roentgenographic technique was used on all occasions.

In none of the operated patients was there any problem with the patient wearing the brace according to routine. The brace was worn full time on an average for seven months, and was gradually weaned over nine additional months.

## METHODS OF RECORDING

Roentgenograms of the spine of each patient were obtained in the same standard manner each time. With the patient in the standing position, a roentgenogram of the whole spine including secondary curves was obtained with a film size of 100 by 33 cm and with a focal distance of 5 m. For the supine roentgenograms a standard film size was used. The focusing of the tube was at the apex of the curve. The patients were radiographically checked before treatment, immediately after each operation, immediately after fitting of the brace for the first time, immediately after the en of brac treatment, and at regular follow-up controls every third to sixth month up to one year and two years, respectively. Furthermore, they were checked three and four years after the operations. While under brace treatment, the spine was always controlled with the patient wearing the brace. Great care was always taken to minimize the number of exposures, usually only one single film was obtained, thereby reducing the hazards of radiation.

## DEFINITIONS AND MEASURING METHODS

A retrospective analysis of the roentgenograms was made on all patients. The information recorded included the initial curve, the curve correction and loss of correction, the vertebral rotation and vertebral and disc wedge. All roentgenograms for each individual were evaluated up to and including the one-year follow-up control in the conservatively treated material, and up to and including the two-year control in the operated series. Curve correction and loss of correction was also evaluated in the operated material three and four years after operation for those patients who could be followed-up for a period of that length. Although measurements of curvature had been performed earlier in connection with the patient's visit to the clinic, these measurements were again performed by the author for the purpose of this study.

### Curvature

The curvature was measured in degrees according to the method described by Cobb (1948).

### Correction

Correction of the curvature was calculated in degrees and in percentage. The curve was compared with the original curve in the erect position before treatment was started.

In patients treated with the Milwaukee brace:

Supine correction is the correction of the curvature which occurs when the patient lies down.

Initial correction in brace is the correction of the curvature obtained when the brace is fitted for the first time. It is determined from roentgenograms obtained in the erect position.

Final correction is the correction of the curvature at the end of brace treatment. It is determined from a roentgenogram obtained with the patient in the erect position not wearing the brace.

Correction maintained is the correction of the curvature measured on the roentgenogram made in the erect position at the follow-up one year after the end of brace treatment.

In patients treated with the Harrington method:

Correction of the curvature is the correction of the scoliosis immediately after the second operation. It is determined from a roentgenogram of the spine in the supine position which is compared with the preoperative roentgenogram in the erect position.

Correction in second operation is the correction of the curvature obtained by the second operation. It is determined from the postoperative roentgenogram of the spine in the supine position which is compared with the roentgenogram after the first stage operation.

Supine correction is the correction of the curvature which occurs when the patient lies down.

Correction maintained is the correction of the curvature in the roentgenogram (erect) taken at the follow-up 2 years after the operations.

Loss of correction

The loss of correction was calculated in degrees and in percentage of the curve with which the comparison was made.

In patients treated with the Milwaukee brace:

Loss of correction is the loss of correction of the curvature at the one-year follow-up compared with the curvature at the end of brace treatment. It is determined from roentgenograms obtained in the erect position.

In patients treated with the Harrington method:

Initial loss of correction is the loss of correction that occurs, when the patient is ambulated in a Milwaukee brace after 5-7 weeks of recumbency. This loss is determined on the roentgenograms as the difference between the degree of curvature in the first postoperative roentgenogram in the erect position and the degree of curvature in the first supine roentgenogram after the second operation. It is also calculated in percentage of the degrees of curvature on that supine roentgenogram.

The late loss of correction is the difference in degrees between the curve on the first postoperative roentgenogram in the erect position and the curve on the roentgenogram recorded in the erect position at the follow-up two years later.

Total loss of correction equals the sum of the above described losses of correction, i.e. the increase in curvature on the roentgenogram obtained in the erect position at the follow-up to two years postoperatively as compared to the curvature on tl first supine roentgenogram after the second operation.It has also been calculated in percentage of the degree of curvature on the first supine roentgenogram after the second operation.

The loss of correction during the second postoperative year in degrees has been determined in order to evaluate late losses as an indication of pseudoarthrosis.

Additional losses during the third and fourth postoperative years have been calculated for the patients, who were followed-up three and four years postoperatively.

## Rotation

Rotation was evaluated from the roentgenograms according to the method described by Nash and Moe (1969). This method is based on the relationship of the pedicle outlines to the lateral margins of the vertebral body on the roentgenogram (Fig. 15). When the two pedicles are equidistant from the vertebral margins of each side, the vertebra is in the neutral position. With rotation, the convex

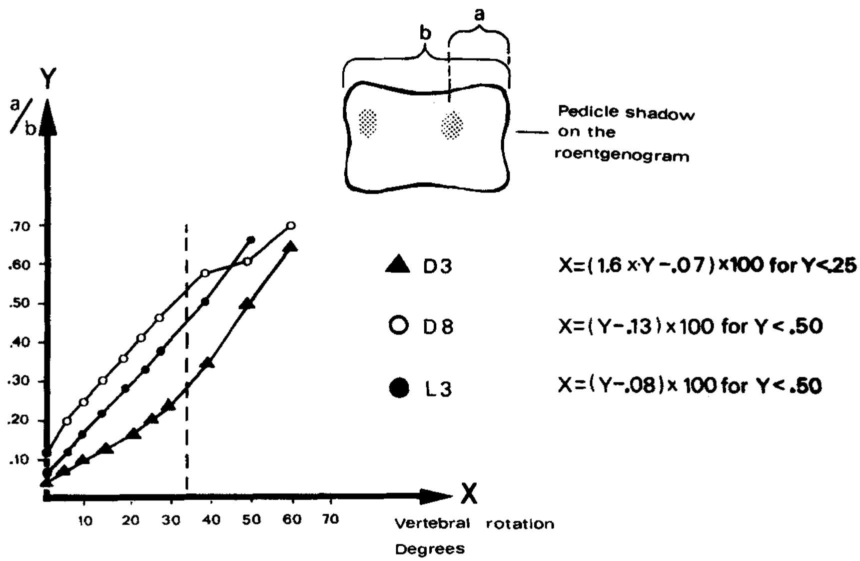


Fig. 15. Vertebral rotation correlated with the pedicle ratio. The pedicle ratio method for determination of rotation (Nash and Moe 1969) is indicated in a schematic drawing.

pedicle moves towards the centre of the vertebral body projection. Nash and Moe studied the actual rotation in a young adult spine. Comparing the actual vertebral rotation with the pedicle outline in the roentgenogram, they found that the rotation could be well estimated by the ratio a/b (Fig. 15). This was also verified by Fait and Janovec (1970). However, Nash and Moe did not specify the pedicle-vertebral ratio in the neutral position of the vertebra, nor did they specify, whether or not there are any differences between the upper and lower part of the spine. Therefore, when rotational deformity was to be determined in the present study, the experiment of Nash and Moe was repeated with a view of these factors.

Three segments of a normal spine obtained at autopsy from a 16-year old girl were used: D 2-4, D 7-9 and L 2-4. The segments were assembled with a felt material simulating the intervertebral disc, and the pedicles were outlined with a steel wire around the base of each pedicle. Each segment was mounted to allow rotation, which

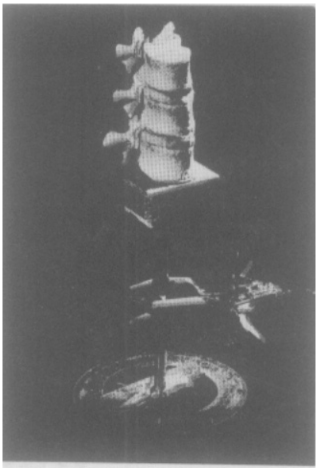

Fig. 16. A segment of the spine mounted to allow rotation, and forward, backward and lateral tilting.

could be read on a graded scale, the specimen could also be tilted laterally, forwards and backwards (Fig. 16). Roentgenograms were taken at every 5th degree of rotation to the right and to the left, and at 10 degrees of lateral, forwards and backward tilting. The vertical axis of rotation was thereby considered to be located in the lumbar segment slightly anterior to the posterior longitudinal ligament, in the thoracic segment in the middle of the posterior half of the vertebral body and in the upper thoracic segment slightly forward to the middle of the vertebral body (Roaf 1958, White 1969, Cossette et al 1971).

There was a direct correlation between the ratio a/b and the degrees of rotation of the segment up to a ratio of approximately 0.50 in the thoracic and lumbar area, and 0.25 in the upper thoracic spine (Fig. 15). This corresponded to a rotation of approximately 35-40$^{o}$. Neutral position of the vertebrae equals a ratio a/b which coincides with the intersection of the curve with the y-axis in Fig. 15. Forward and backward tilting within 10$^{o}$ did not influence the result, and lateral tilting without lateral displacement did not either influence the rotation evaluation.

With this method there was no difficulty in determining the rotation of the vertebrae in the lumbar and middle and lower thoracic area. However, in the upper thoracic spine (mostly the upper compensatory curves) the evaluation of rotation could not be performed with any accuracy because of the narrow conditions in this region, and was therefore not included in the present study.

It is recognized, that this method of determining rotation is based on the observations of a normal, non-deformed spinal segment. In the scoliotic subject, the deformation of the vertebrae probably influences the assessment of the rotation.

### Wedge of the vertebral body

The wedge of the apical vertebral body was determined on the roentgenograms both as the angle in degrees ($\alpha$ in Fig. 17) between the planes of the two end plates of the vertobral body, and as the ratio of the concave side height/convex side height of the vertebral body($c/d$ in Fig. 17). There was a good correlation between the wedge angle and the wedge ratio ($r = 0.89$, $2p < 0.01$).

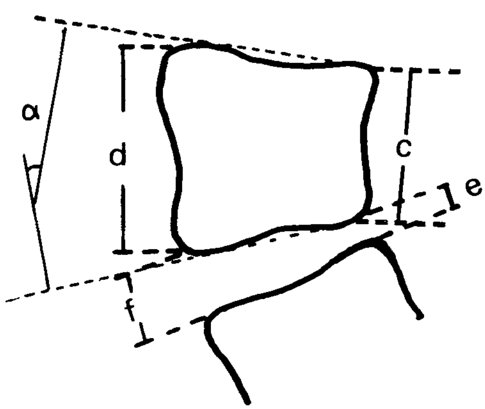


Fig. 17. Methods for measurement of the vertebral and disc wedge. For explanation, see text.

## Wedge of the intervertebral disc

The wedge of the intervertebral disc was determined on the roentgenograms as the angle in degrees between the planes of the two vertebral end plates surrounding the disc, and also as the ratio of concave side height/convex side height of the disc (e/f in Fig. 17).

There did not always exist a single, apical vertebra or disc. The most deformed of two possible structures was then measured. The same individual disc and vertebra was always measured throughout the investigation.

## Compensatory curves

The compensatory upper and lower curves were measured according to the Cobb method (1948) preoperatively, postoperatively and at the one-, two-, three- and four-year follow-up. The rotation and the vertebral wedge of the secondary curves were also recorded. Rotation was not determined in the upper thoracic spine.

## Development of the iliac apophysis

The development of the iliac apophysis was defined on a five-graded scale:

1. The development of the iliac apophysis has not started.
2. The iliac apophysis has started to develop.
3. The iliac apophysis is completely developed.
4. Fusion of the iliac apophysis has started.
5. Fusion to the ilium of the iliac apophysis is completed.

## General remarks

The results in double primary curves have been separately analysed in the operated series. In patients with double primary curves treated with the Milwaukee brace, the most severe of the two curves has been included in the statistical analyses of the results at the end of brace treatment. Complete data were not available for all parameters of all patients in this retrospective study. In a few instances, the quality of the roentgenograms did not allow any measurement of some parameters. The number of patients evaluated for each parameter are demonstrated in the diagrams showing the results of treatment.

## Statistical methods

All data were stored in a computer and the means, standard deviations, ranges and other statistical quantities including correlation and regression coefficients were calculated by the computer. For comparisons between groups of data, the Student's t-test was used. The 5 per cent level was considered to be statistically significant. This will in the following be mentioned as "significant".

## Sources of error

The error of measurement in the present work was determined by the author for each of the following parameters: curvature, rotation, wedge of the apical vertebra and wedge of the apical disc.

This was obtained by measurement of each of these parameters in 40 roentgenograms of scoliosis curves ranging from $24^{o}$ to $80^{o}$. Each parameter was measured twice on two separate occasions, and the mean error of measurement was calculated from the difference between these measurements (error of measurement = $\sqrt{\frac{\Sigma d^2}{2n}}$. where d = difference between the measurements and n = the number of differences). It is recognized that the true deformities could not be measured, but only the projections of the deformities on the roentgenogram.

### Curvature

In the present work the error of measurement was $\pm 2.2^{o}$. The accuracy of the Cobb measurement method has been evaluated by a few authors. Sevastikoglou and Bergquist (1969) measured the curve in a scoliotic skeleton, and found that the error of measurement was $\pm 3.12^{o}$ for the Cobb method. Rotation of the spine by $5^{o}$ to $10^{o}$ and movements of the X-ray tube 5 cm in either direction from the apex of the curve did not result in errors surpassing the margins of error of curve measurement. Neugebauer (1972) reported an error of measurement of less than $3^{o}$ with the Cobb method, basing this on theoretical considerations and spine radiographs of scoliotic patients.

Rotation

No previous study has been performed on the error of measurement of rotation. The error of measurement of rotation in the present work was found to be $\pm 2.0^{o}$.

The evaluation of the rotation is naturally very dependent upon the projection. A small rotation of the patient will cause a derangement of the measurement. However, rotation was always measured in at least two curves of the spine. Any large rotational change on behalf of the patient between two measurements would cause inconsistent changes in the results. One curve would derotate, and the other would increase its rotation projection. Such differences were not encountered.

Wedge of the vertebral body

The mean error of measurement was found to be $\pm 0.04$ and $\pm 1.4^{o}$ for the ratio and the angle method respectively.

These measurements depend on the projection of the roentgenogram, and several roentgenograms had to be omitted from this study because of focusing errors. However, as the vertebral wedge does not change over short periods of time, it was possible to use several roentgenograms for the evaluation of the wedge at a certain time, such as pre- and immediate postoperative recordings for determining the status of the patient at the time of operation.

Generally, after the operation the vertebral bodies grew, which was observed by the narrowing disc spaces. This growth distorts the evaluation of the vertebral wedge ratio in the follow-up. If growth in vertebral height occurs equally on the concave and convex sides, the wedge angle will not change, but the wedge ratio will decrease. However, the increase in height of the vertebrae in the ages 10-16 years is very small, less than 0.1 mm/month (Calvo 1957). In one year this would cause a decrease in the wedge ratio of approximately 0.01, well within the margins of the error of measurement. The growth of the vertebrae therefore need not be considered as a source of error in this material.

Wedge of the intervertebral disc

The error of measurement was $\pm$ 0.05 with the ratio method and $\pm 1.9^{\circ}$ with the angle method. These measurements were subjected to the same kind of errors as were the measurements of the vertebral wedge. The disc measurements depended even more upon the projection, as the disc is narrower. With vertebral growth the disc narrowed considerably in the postoperative period and it was therefore impossible to assess the disc wedge at the follow-up examinations.

CHAPTER TWELVE

## RESULTS

### EARLY RESULTS IN 159 PATIENTS TREATED WITH THE MILWAUKEE BRACE

159 patients were followed-up for 2 years or more after the beginning of brace treatment. In this analysis, patients with single primary and double primary curves were evaluated separately.

#### Curvature

The mean curvature at the beginning of the treatment was $39 \pm 14^{o}$ for the single primary, and $39 \pm 11^{o}$ for the upper double primary curves. Mean curvature of the lower double primary curves was $35 \pm 12^{o}$.

#### Initial correction in brace

The fitting of the brace resulted in an immediate correction of the curvature which was significant, $7.4 \pm 5.8^{o}$ or 20±15 per cent in all curves. This correction was best in the single primary curves where it was $9.1 \pm 5.9^{o}$ or 24±15 per cent. The percentage correction decreased significantly ($2p < 0.01$) with an increased original curvature, while the degrees of correction in the brace increased slightly with an increasing degree of curvature (Table 11). With the apex located more distally in the single primary curves, the initial correction in the brace increased strongly. In double curves, the correction also was most satisfactory in the lumbar region, approximately 23±13 per cent while it was only 11±8 percent in the thoracic region.

This strong increase of the correction in the brace with the scoliosis located more distally can partly be explained by a smaller curvature

TABLE 11. Initial correction in degrees (Mean±S. D.) when the Milwaukee brace is fitted.(n=number of patients).

| Curvature Degrees | Single curves | n | Double curves upper | n | lower | n |
|---|---|---|---|---|---|---|
| 10-19 | 7.4 ±4.1 | 10 | 2.3 ±3.6 | 5 | 3.7 ±9.1 | 6 |
| 20-29 | 7.8 ±4.9 | 27 | 3.0 ±4.5 | 8 | 6.2 ±4.5 | 17 |
| 30-39 | 8.7 ±6.6 | 36 | 5.6 ±5.8 | 13 | 6.0 ±6.5 | 15 |
| 40-49 | 8.9 ±6.7 | 26 | 4.2 ±3.5 | 16 | 4.2 ±3.5 | 4 |
| 50-59 | 12.0 ±6.7 | 8 | 8.5 ±8.0 | 2 | 4.0 ±1.4 | 2 |
| ≥60 | 10.3 ±8.4 | 2 | 10.0 | 1 | 10.0 | 1 |

in the distal parts of the spine. Despite this, there was an increase in degrees of correction also in the lower part of the spine, and it is apparent that the brace is more effective for correction of thoraco-lumbar and lumbar curves.

### Rotation

The mean rotation was $15 \pm 4^{\circ}$ (double primary curves $13 \pm 4^{\circ}$). Rotation increased significantly ($2p < 0.01$) with increasing curvature. The brace had an immediate significant derotating effect on the apical vertebra of approximately $4^{\circ}$ or 28 per cent.

### Vital capacity

The vital capacity was decreased in most patients, the mean percentage of predicted normal was $79 \pm 11$ per cent. There was a significant decrease in the vital capacity with increasing curvature ($0.01 < 2p < 0.05$). However, the vital capacity was even stronger correlated to the rotation of the apical vertebra, with $2p < 0.01$. In a partial regression analysis it was verified that the vital capacity depended more on the rotation of the vertebrae than on the degrees of the curve. The partial regression coefficients were -0.26 for the vital capacity correlated with rotation and -0.05 for the vital capacity correlated with degrees curvature (with constant rotation).

### Treatment failures

Conservative treatment failed in 29 of the 159 patients. In spite of proper brace treatment the scoliosis curvature progressed in 14 patients to such a degree that operation became indicated. In all but three of these patients, the curves were $40^{\circ}$ or more at the start of brace treatment. One was a $35^{\circ}$ idiopathic thoraco-lumbar curve in a 10 year old girl. She wore a brace for four years. During treatment the patient increased 14.5 cm in sitting height but the curve progressed to $45^{\circ}$ and was then operated. One was a $35^{\circ}$ idiopathic thoracic curve in a 13 year old girl During three years of brace treatment it increased to $15^{\circ}$. The patient increased 4 cm in sitting height during treatment. The third patient was a boy with a congenital thoracic scoliosis in whom treatment was started at the age of eight when the curvature was $30^{\circ}$. The curve progressed to $45^{\circ}$. During three years of treatment the patient increased 11 cm in sitting height. The curve progressed to $45^{\circ}$ and was then instrumented and fused. It was significant, that 10 of those 14 curves that later had to be operated on improved 10 per cent or less initially when the brace was first fitted .

One patient had psychiatric problems, and did not use the brace as directed. It was a 14-year old girl with a $50^{\circ}$ thoracic idiopathic curve. The scoliosis progressed without treatment to $70^{\circ}$, and she was operated on at the age of 17 years.

In another 14 patients, all idiopathic, the brace treatment was interrupted because the patients failed to wear the brace as directed. Twelve of these patients showed poor cooperation within one year after the start of brace treatment. Five patients stopped wearing the brace completely. No patient in this group was operated on.

## FINAL RESULTS IN 86 PATIENTS WHO COMPLETED BRACE TREATMENT

86 patients completed the brace treatment and were all followed-up one year after its termination. 77 patients (90 %) were idiopathic, 5 patients (6%) had a congenital scoliosis, and one patient had in early years a Wilm's tumour and developed a structural scoliosis caused by irradiation treatment. One patient had a scoliosis probably of sciatic origin, one patient had scoliosis caused by poliomyelitis, and one patient had spastic hemiplegia.

The development of the iliac apophysis in these patients at the start of the treatment, at the end of the treatment and at the follow-up one year later, is shown in Table 12.

TABLE 12. Development of the iliac apophysis during treatment.

| Development of iliac apophysis | At start of treatment | At end of treatment | At follow-up 1 year after end of treatment |
|---|---|---|---|
| | | per cent of patients | |
| Not developed | 13 | 0 | 0 |
| Developing | 51 | 0 | 0 |
| Development complete | 34 | 21 | 1 |
| Fusion started | 1 | 75 | 51 |
| Fusion complete | 0 | 4 | 48 |

The results for the patients with structural scoliosis of different etiology in this one-year follow-up group are shown in Table 13. It can be noted that in patients with scoliosis of congenital origin and in patients with neurological or muscular disorders, there was generally less correction. The result of the interrupted treatment in those patients who did not wear the brace as directed is shown in Table 14. The result of the interrupted treatment in these patients is less satisfying, the curve correction is significantly smaller than

TABLE 13. Results (Mean ±S. D.) of treatment with the Milwaukee brace in patients who completed the treatment program.

| Etiology | No. of pat. | Orig. curvature | Initial correction in brace | Corr. at end of treatment | | Corr. loss at follow-up | | Corr. maintained |
|---|---|---|---|---|---|---|---|---|
| | | Degrees | % | Degrees | % | Degrees | % | % |
| Idiopathic | 77 | 36.3 ±10.2 | 20.4 ±17.5 | +3.5 ±9.4 | +9.7 ±26.5 | 2.0 ±3.6 | 6.1 ±22.4 | +4.2 ±8.2 |
| Poliomyelitis | 1 | 33 | 21 | -2 | -6 | 4 | 11 | -17 |
| Spasticity | 1 | 40 | 25 | -15 | -38 | 15 | 27 | -75 |
| Congenital | 5 | 41.4 ±12.8 | 13.8 ±12.0 | -2.6 ±9.2 | -6.4 ±18.6 | 1.9 ±2.4 | 4.3 ±9.3 | -10.9 ±14.6 |
| Radiation | 1 | 50 | 0 | +2 | +4 | 10 | 21 | -16 |
| Sciatica? | 1 | 40 | 38 | +15 | +38 | 5 | 20 | +25 |
| All | 86 | 36.4 ±10.0 | 19.6 ±17.0 | +2.4 ±9.3 | +6.8 ±26.1 | 2.6 ±3.9 | 7.7 ±21.5 | -0.6 ± 8.6 |

TABLE 14. Results of brace treatment in patients with idiopathic scoliosis not cooperating (Mean ± S. D.).

| | | No. of patients |
|---|---|---|
| Curvature | 37.6 ±9.6 degrees | 14 |
| Correction when brace was discarded | -2.2 ±6.3 degrees<br>-5.9 ±17.0 per cent | 14 |
| Additional loss of corr. until end of growth | 4.4 ±3.0 degrees<br>11.5 ±7.6 per cent | 10 |
| Corr. maintained at end of growth | -6.5 ±6.7 degrees<br>-13.0 ±22.5 per cent | 10 |

in those patients with idiopathic scoliosis who fulfilled the treatment. Twelve of these 14 patients declined the use of the brace according to the full program within one year after the start of the treatment. All patients, however, were checked at the scoliosis clinic although they were not wearing the brace. Ten of these 14 patients had reached skeletal maturity at the follow-up.

## FINAL RESULTS IN 77 PATIENTS WITH IDIOPATHIC SCOLIOSIS

The result of treatment in the 77 patients with idiopathic scoliosis is shown in Table 15. Mean time of brace wearing was 23.3 months (range 12-60). The mean over all final correction was $3.5\pm9.4^{\circ}$ and significant. The mean loss recorded at the one-year follow-up was significant, $2.0\pm3.6^{\circ}$, making the total result of correction only about $1.5\pm8.4^{\circ}$, or 4 per cent, a non-significant improvement of the original curve.

### Age at onset of the scoliosis

The age at onset of the deformity was of importance for the final result of treatment. This naturally was connected to the age at which treatment was started. In those patients, where the scoliosis

TABLE 15. Results of treatment with the Milwaukee brace in patients with idiopathic scoliosis who completed the treatment program (Mean±S.D.).

| Apex | No. of pat. | Orig. curve | Initial corr. in brace | Corr. at end of treatment | Loss of corr. at follow-up | Corr. maintained at follow-up |
|---|---|---|---|---|---|---|
| | | Degrees | % | % | % | % |
| D 4-6 | 1 | 25 | 4 | 16 | 29 | -8.5 |
| D 7-9 | 47 | 38.5<br>±9.4 | 14.2<br>±11.5 | 1.2<br>±25.7 | 6.6<br>±9.5 | -5.2<br>±22.8 |
| D 10-12 | 24 | 34.0<br>±11.1 | 29.0<br>±19.0 | 20.6<br>±22.0 | 11.5<br>±16.5 | +11.2<br>±18.1 |
| L 1-3 | 5 | 30.0<br>±10.8 | 46.8<br>±28.2 | 36.3<br>±36.6 | 19.0<br>±92.5 | +23.4<br>±28.0 |

had an early onset, at the age of 10 years or younger, the correction was not as good as in those patients in whom the scoliosis had a late onset. However, for scoliosis cases where discovery and start of treatment was very late - in the 15:th and 16:th year - when growth was about to cease, the result with brace treatment was less good too (Fig. 18).

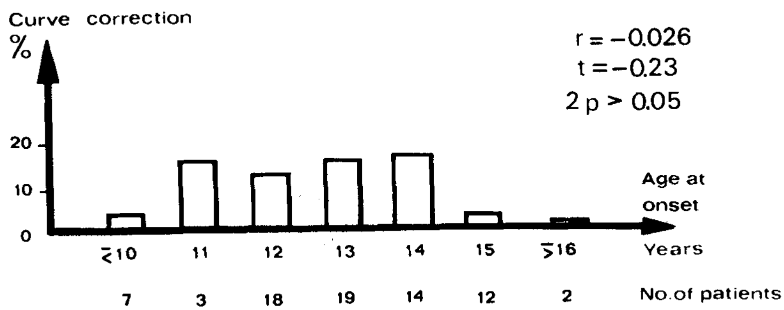


Fig. 18. Mean percentage correction at the end of brace treatment correlated with the age at onset of deformity.

Age at start of treatment

The mean age of the patients at the start of treatment was 14 years and 7 months (11-17 years).

The age of patients at the start of treatment, chronological or measured as iliac apophysis development, did not show any consistent correlation with the initial percentage correction or the correction at the end of brace treatment (Fig. 19). Those patients, in whom brace treatment was started at an early age, in this series at 11 years, corrected less satisfactory with the brace (fig. 20).

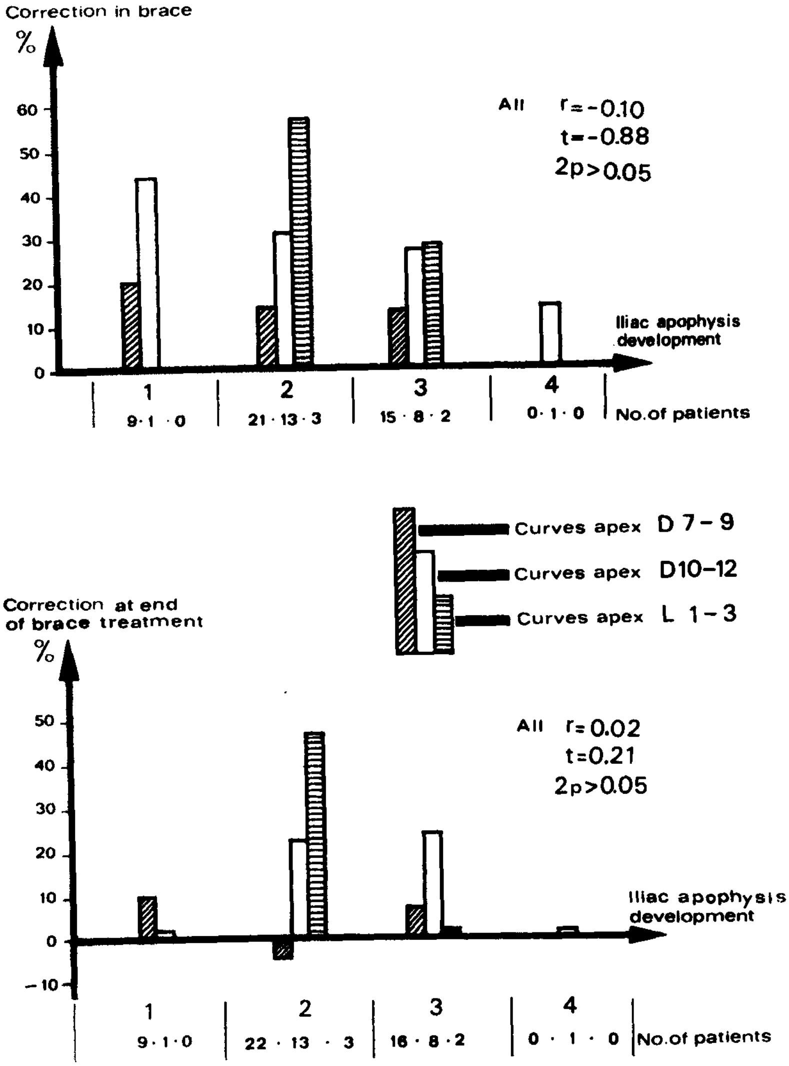


Fig. 19. Mean percentage correction in the brace, and at the end of brace treatment for curves in different locations correlated to the development of the iliac apophysis.

## Original curvature

The mean original curve was 36.3±10.2°, the medium was 35°. With an increasing curve there was a significantly increasing rotation and an increasing wedge of the vertebral body and intervertebral disc at the apex of the curve.

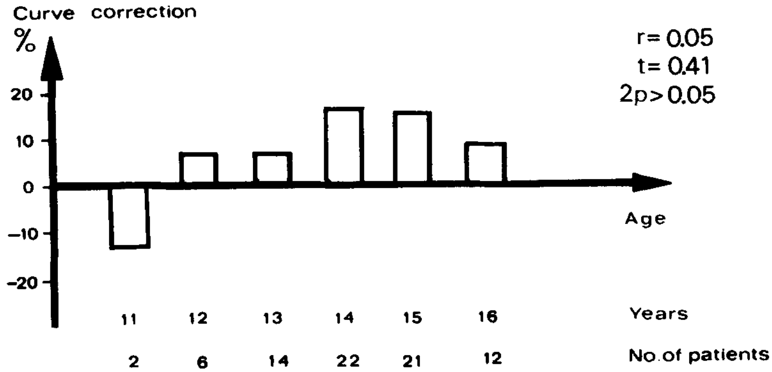


Fig. 20. Mean percentage of correction at the end of brace treatment correlated with the age of the patient at start of treatment.

There was a small non-significant overall decrease of correction in percentage, and a small non-significant increase in correction in degrees with an increasing original curvature. Fig. 21 shows the

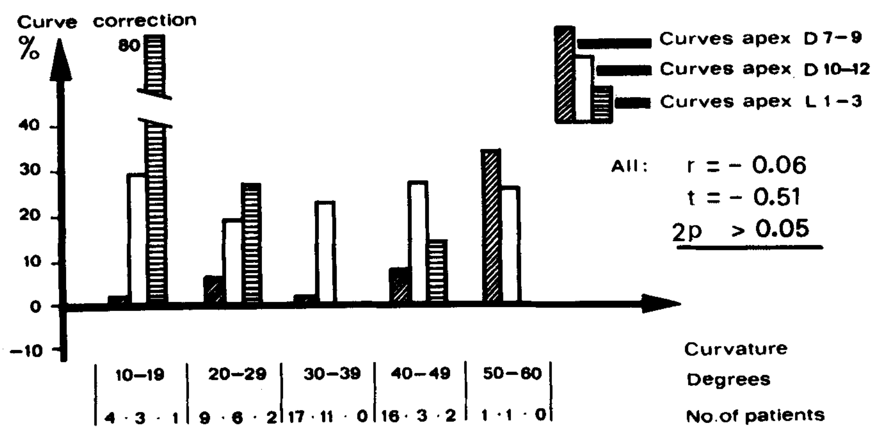


Fig. 21. Mean percentage correction at the end of brace treatment correlated with the original curvature.

percentage correction of the curve at the end of brace treatment correlated with the original curvature for different curve locations and Fig. 22 gives the initial correction and the correction at the end of brace treatment in all curve locations. The original curvature within those relatively small variations which are present does not seem to have any large influence on the result.

Number of vertebrae

The average number of vertebrae in the primary curve was 6.7 (5-9). In this investigation the percentage correction was less in the longer curves as compared to the shorter ones (Fig. 23). The correlation of the percentage correction with the curve length was not significant. Tall patients had a tendency to have curves with a larger number of vertebrae involved ($2p<0.05$).

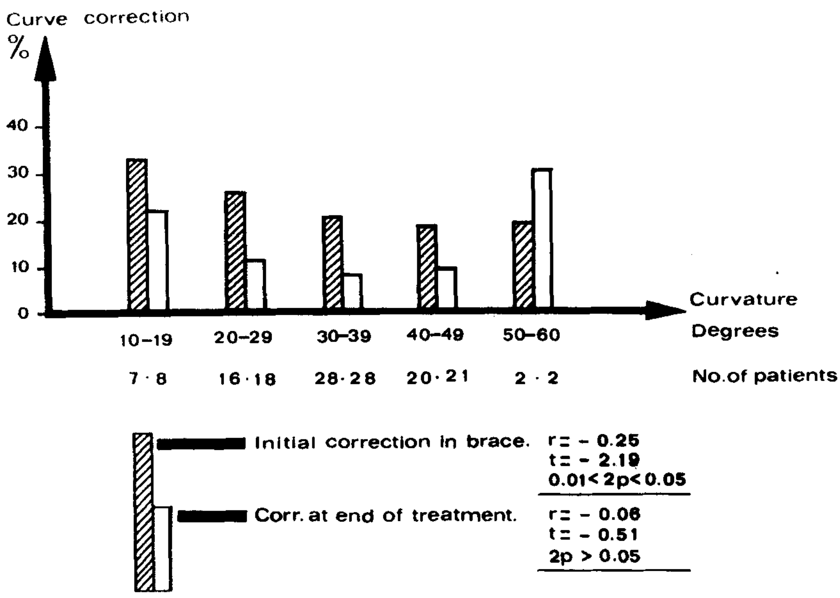


Fig. 22. Mean percentage correction initially in the brace and at the end of brace treatment correlated with the original curvature.

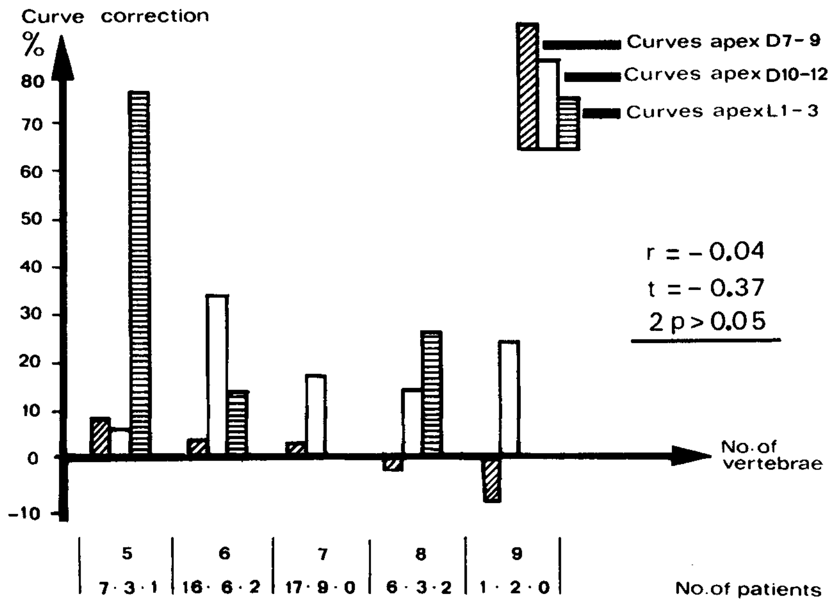


Fig. 23. Mean percentage correction at the end of brace treatment of scoliosis in different locations correlated with the number of vertebrae in the curve.

## Initial correction in brace

The average correction obtained in the brace immediately after its fitting was significant, $7.0 \pm 5.7^{\circ}$ or $20 \pm 17$ per cent. This correction increased during the brace wearing, and reached its maximum at the control six months later ($7.6^{\circ}$) after which the correction decreased. The initial correction in percentage increased significantly in the more distal curves, as is shown in Fig. 24.

There was a strong significant correlation between the initial correction in the brace, and the final result at the end of brace treatment, ($r = 0.58$, $2p < 0.01$), Fig. 25.

## Supine correction

The average correction of the curvature from an erect to a recumbent position was significant, $8.2 \pm 6.9^{\circ}$ or $23 \pm 15$ per cent. The supine

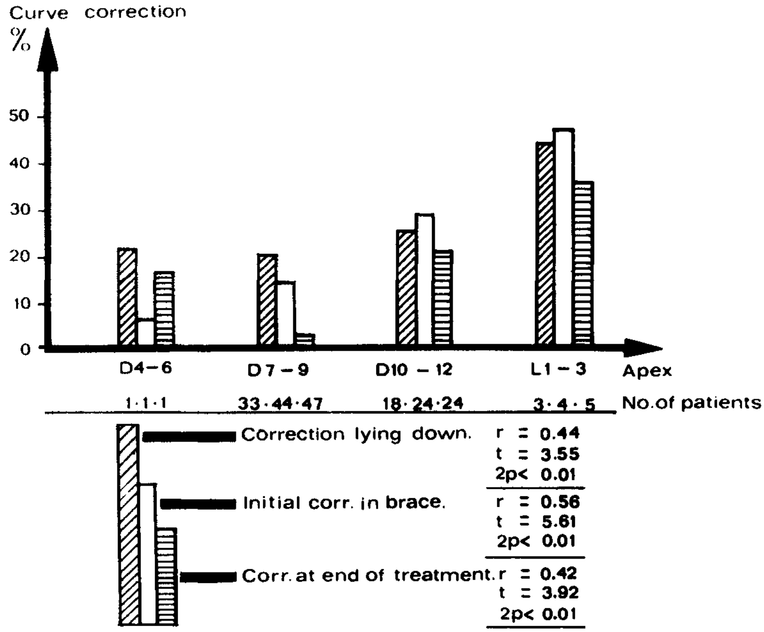


Fig. 24. Mean percentage correction in the supine position, initially in the brace and at the end of brace treatment correlated with the location of the curve.

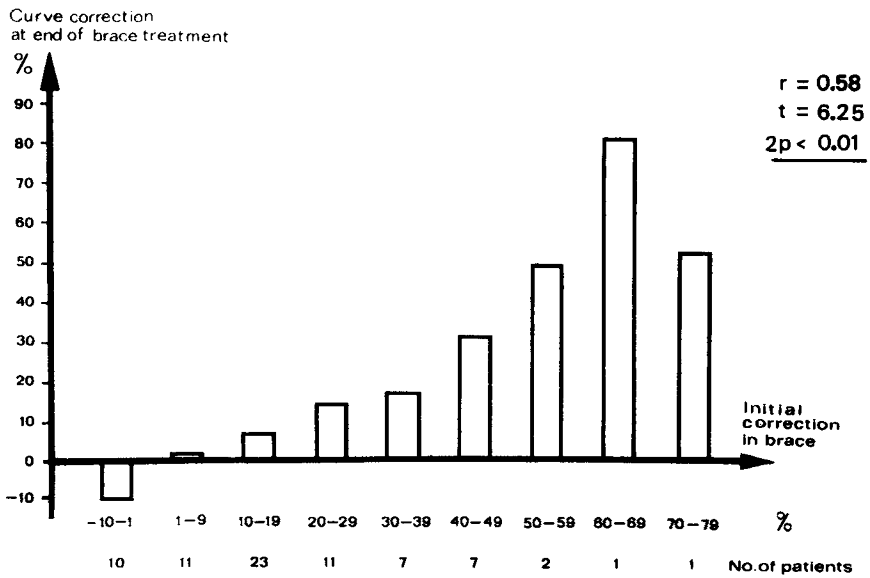


Fig. 25. Mean percentage correction at the end of brace treatment correlated with the initial percentage correction in the brace.

correction increased distally in the spine (Fig. 24). The supine correction was significantly correlated with the correction in percentage at the end of brace treatment with a correlation coefficient of $r = 0.44$. However, the initial brace correction was an even better prognostic sign than the supine correction.

### Loss of correction

The average loss of correction one year after the end of brace treatment was $2.0 \pm 3.6^{\circ}$ or $6.1 \pm 22.4$ per cent. The correction maintained at the one-year follow-up was a non-significant correction of 4.2 per cent. The loss of correction in percentage depended on the location of the scoliosis, however, not significantly (Fig. 26). The loss in degrees was fairly uniform for different curve localizations, however. The loss was independent of the age of the patient at the beginning of treatment, and neither was it significantly correlated to the original curvature or its structural changes.

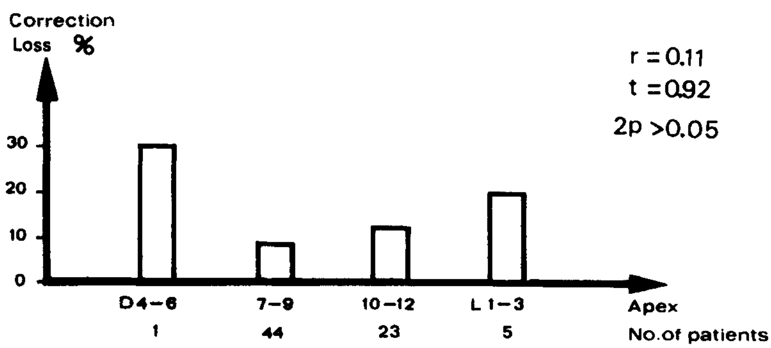


Fig. 26. Mean percentage of correction loss correlated with the location of the curve.

### Location of curve

A most significant factor for the result of brace treatment was the location of the scoliosis. There was a significant increase in percentage correction (Fig. 24), as well as in degrees of correction in the distal part of the spine (Fig. 27). There was also an increased

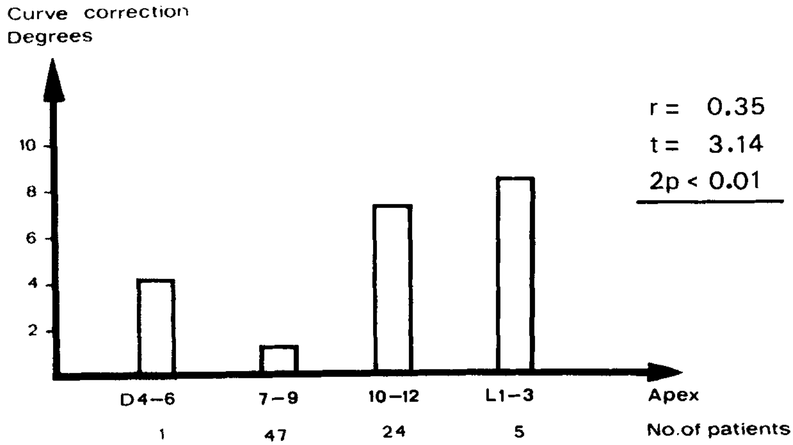


Fig. 27. Mean percentage of curve correction at the end of the brace treatment correlated with the location of the curve.

loss of correction in percentage and in degrees in the distal part of the spine. This was not significant, however, and the correction maintained at the one-year follow-up was still the best in the lumbar spine as was shown in Table 15(page 104). There was a significant increase in rotation of the vertebrae more distally in the spine.

## Rotation

Mean rotation of the apical vertebra was $13.8 \pm 4.8^{\circ}$. Rotation of the apical vertebrae increased with increasing original curvature (Fig. 28). The final result of treatment was not significantly correlated to the initial rotation deformity.

With treatment a slight decrease of rotation of the apical vertebra was noticed, on the average $2.0^{\circ}$, which was significant. However, at the one-year follow-up, again an increase of rotation of approximately one degree was found, and although the brace seemed to have a derotational effect on the curve this did not persist through the follow-up period.

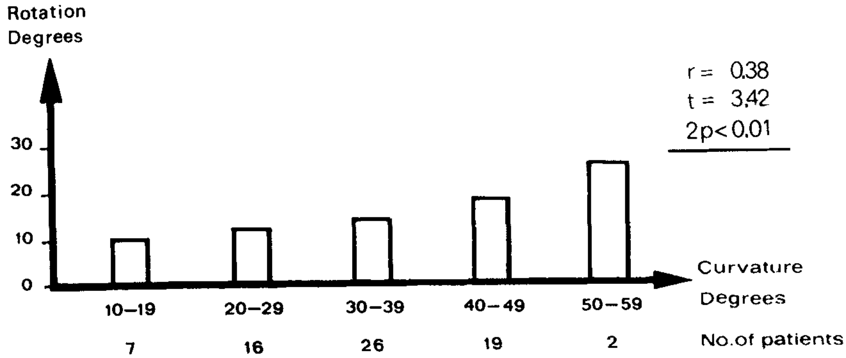


Fig. 28. Mean degree of rotation of the apical vertebra at the start of treatment correlated with the degree of the scoliosis.

### Wedge of the vertebral body

Mean vertebral body wedge ratio was $0.90 \pm 0.14$ when the treatment was started. As expected, the vertebral wedge increased with increasing curvature ($0.01 < 2p < 0.05$). The original wedge of the vertebrae did not show any significant correlation to the final result of treatment. In spite of treatment there was no change of the mean vertebral wedge ratio, which at the end of treatment still was $0.90 \pm 0.15$, and at the follow-up also $0.90 \pm 0.16$. However, it was noticed that a change of the vertebral wedge depended on the age of the patient. There was a non-significant increase in the vertebral wedge in those patients, who were young (11-12 years of age) at the start of treatment. In older patients, on the other hand, there was a reversal – also non-significant – of the vertebral body wedge (Fig. 29).

### Wedge of the apical disc

The mean disc wedge ratio was $0.66 \pm 0.14$ at the start of the treatment. The wedge of the apical intervertebral disc increased with an increasing curvature. The wedge shape of the apical disc did not change significantly during the treatment and it was not significantly correlated to the correction result.

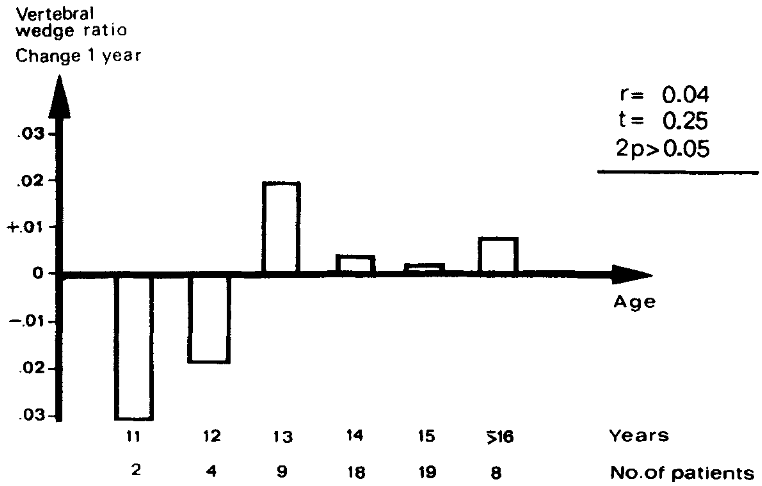


Fig. 29. Mean vertebral wedge change at the one-year follow-up correlated with the age of the patient at the start of brace treatment.

Compensatory curves

There did not always exist a measurable lower and upper compensatory curve with the major curve. The sum of the upper and lower compensatory curves amounted approximately to the same degree as the major curve degree in each patient (Fig. 30). The upper curve sometimes continued into the cervical spine. This part of the curve could not be included in the measurement because of technical difficulties in measuring that curve objectively. Fig. 31 shows the correction of the compensatory and major curves. Of the compensatory curves, the lumbar curve corrected most satisfactory, but usually 10 per cent less than the major curve. The upper compensatory curve usually deteriorated during brace wearing, and even in cases where the major curve corrected 40-50 per cent, the upper compensatory curve could increase by as much as 30 per cent. The change of the upper compensatory curve did not significantly correlate to the correction of the major curve in percentage. The correction of the lower secondary curve in percentage increased significantly with that of the major curve ($r=0.59$, $2p<0.01$). The sum of the correction in degrees of the upper and lower compensatory curves

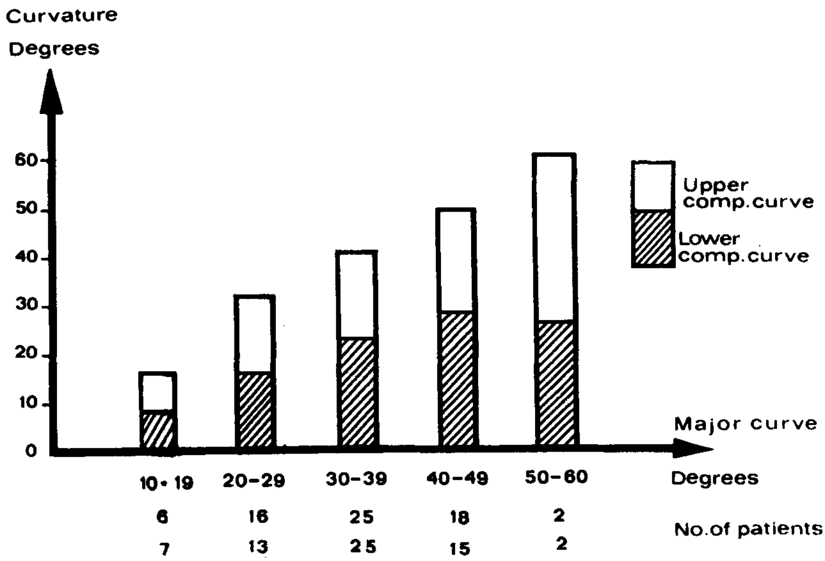


Fig. 30. The degree of the upper and lower compensatory curves correlated with the major curve degree.

was in a relation to the correction in degrees of the major curve as approximately 1/2(Fig. 32).At the one-year follow-up,the upper compensatory curve had deteriorated $1.7 \pm 3.3^{\circ}$ and the lower compensatory curve $1.7 \pm 3.1^{\circ}$ when compared with the curvatures at the end of brace treatment, in both curves a significant increase of curvature.

## Height of the patients

The patients grew considerably while under brace treatment (Table 16), and the brace was thus effective in controlling the scoliosis during growth, when most curves are known to increase (Risser 1964, Duval-Beaupère 1971).

## Complications

The greatest problem in conservative treatment with a Milwaukee brace is to make the patient wear the brace as directed. In the presentation of the 86 patients who completed the treatment as directed,

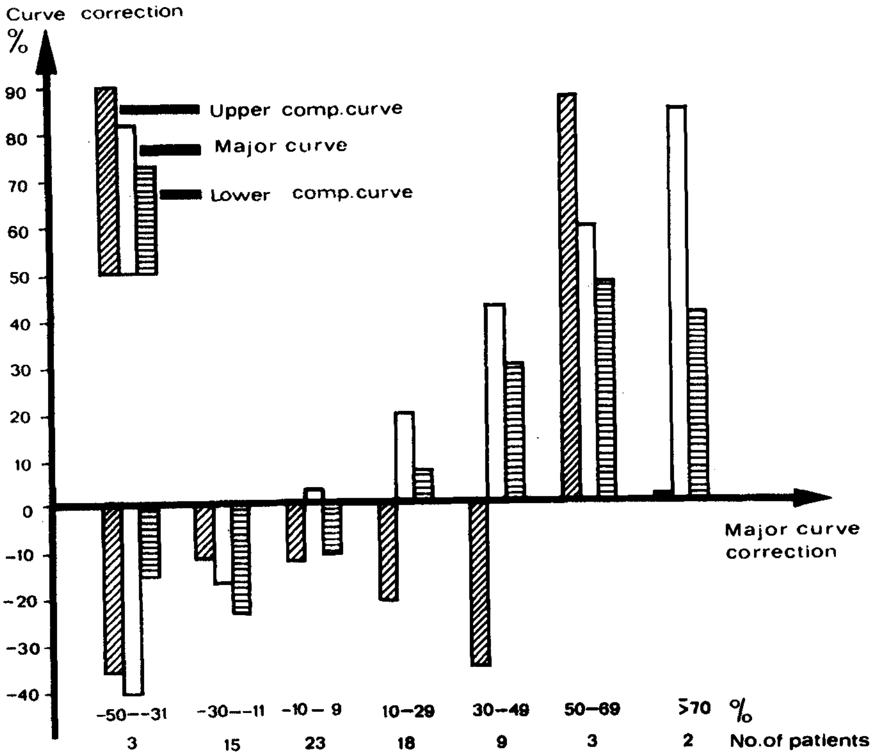


Fig. 31. Mean percentage correction of the major and compensatory curves correlated with the percentage correction of the major curve at the end of brace treatment.

TABLE 16. Growth of the patients treated with the Milwaukee brace

| Age at start of treatment | No. of pat. | Average increase in height (cm) during treatment | | Average height (cm) change during the follow-up period |
|---|---|---|---|---|
| | | standing | sitting | sitting |
| 11 | 2 | 18.5(14-23) | 10.5(7-14) | +0.5 |
| 12 | 6 | 8.7±7.6 | 5.1±3.0 | -0.3 |
| 13 | 14 | 4.0±2.6 | 2.3±2.0 | +0.1 |
| 14 | 22 | 1.8±1.5 | 1.0±1.4 | +0.1 |
| 15 | 21 | 1.1±1.5 | 0.7±1.1 | 0 |
| 16 | 12 | 0.5±1.0 | 0.2±1.2 | +0.6 |

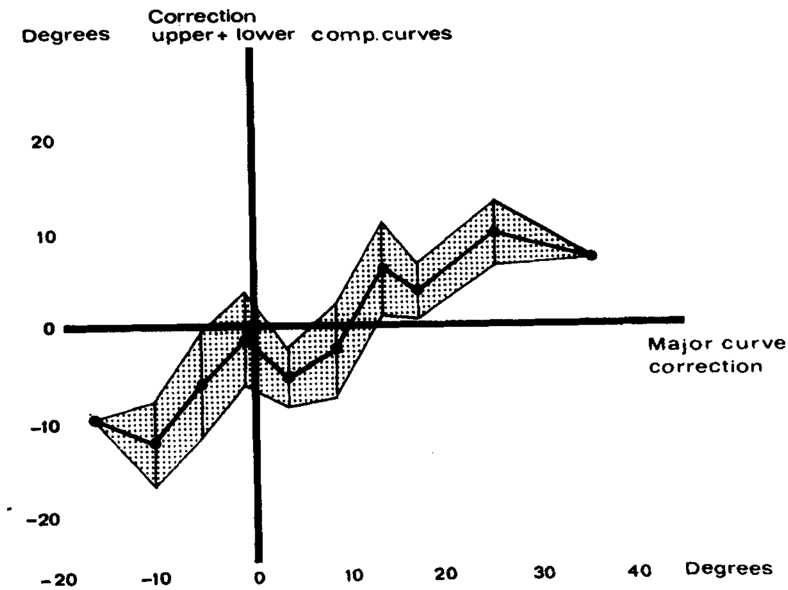


Fig. 32. Sum of correction of the upper and lower compensatory curves correlated with the correction of the major curve at the end of brace treatment.

this problem was not included, although several of these patients during the course of treatment indicated tiredness of wearing the brace. This was overcome by discussions with the physiotherapist, the social worker and the treating physician.

In Table 17 are listed the complications that were encountered in these 86 patients. In three patients, there were complaints of

TABLE 17. No. of cases with complications in 86 patients completing the full Milwaukee brace program.

| | |
|---|---|
| Skin pressure | 3 |
| Meralgia paraesthetica | 2 |
| Allergy | 2 |
| Orthodontic complications | 2 |

soreness in the skin. No decubitus pressures were encountered, and the condition was easily relieved by changes of the brace girdle. Meralgia paraesthetica occurred in two cases and was also relieved by a change of the brace. Two patients were allergic to the chromium-treated leather in the brace and a new girdle of Orthoplast was manufactured for these patients. In this material the regular Milwaukee brace chin support was used. It was, however, worn with a 2 cm distance to the chin. In spite of this,orthodontic complications occurred in two cases. In one patient, aged 14 years with a 35$^{o}$ thoracic curve, there occurred a protrusion of the incisors after 8 months of brace wearing. Another 12-year old patient with a 33$^{o}$ thoracic idiopathic scoliosis after one year developed the typical deformity described in connection with treatment in the Milwaukee brace, i. e. bite distortion of the teeth, and a decrease in facial and vertical mandibular height. Both conditions have resolved after treatment with occlusion stabilizing appliances. It should also be mentioned that no additional complications have been noticed up to the present date in the group of 55 patients who have not ended brace treatment.

## DISCUSSION OF CONSERVATIVE TREATMENT WITH THE MILWAUKEE BRACE

Although widely accepted, the favourable effect of the Milwaukee brace in conservative treatment of scoliosis has been documented in relatively few reports. In a series of 83 patients with idiopathic scoliosis followed-up on an average for 18 months, Moe and Kettleson (1970) reported a correction at the end of brace treatment of 10-24 per cent which was best in the thoraco-lumbar curves. The loss of correction was small except in the lumbar curves where it was reported to be approximately 5 per cent of the original curvature. MacEwen and Kirsch (1971) obtained an average correction of 17 per cent in 48 patients with idiopathic scoliosis. Barash and deWald (1971) reported of 15 patients with 20 idiopathic curves followed-up on an average for 18 months after brace treatment that there was an improvement of 10-30$^{o}$ in 12 curves, while 8 curves remained unchanged. There was no loss after brace removal. Edmonson in 1969 reported an average improvement of $11^{o}$ in 39 patients with idiopathic scoliosis. In a follow-up on an average for 22 months there was a retained improvement in 52 patients, which was the best in the thoracic region with $6.9^{o}$ or 18 per cent. In the high thoracic curves the improvement was only $0.3^{o}$ or 1 per cent (Edmonson and Morris 1973).

In the present study it was confirmed that the Milwaukee brace, if worn correctly, is effective not only in preventing progression of the curvature, but also in correcting the curve. The mean correction percentage of the idiopathic scoliosis group in this investigation was approximately 10 per cent, but one year after end of treatment at the follow-up, this had decreased to 4 per cent. In the lumbar region, the correction was as much as 30 per cent, while in the thoracic region, there was practically none.

In the series reported by Moe,the lumbar and thoracic curves corrected to about the same, while in MacEwen's material the best correction was obtained in the thoracic curves. From the present work it seems that the brace exerts pressure against the transverse processes in the lumbar spine as effectively as it does against the ribs in the thoracic region. Also the lumbar spine normally is more flexible because of the arrangement of the intervertebral joints, and in the lumbar region the brace works mechanically with a long lever, while the lower part of the spine is held firmly by the pelvic girdle.

The loss in the present series was about 2 degrees and was approximately the same in the different curve patterns. As it has been shown that scoliosis progresses even after fusion of the iliac crest (Risser.1964, Urbaniak and Stelling 1969, Collis and Ponseti 1969), there will most certainly occur some slight additional loss of correction in this material during the years to come. Thus, a new follow-up of these patients is indicated at a later date. The further loss of correction will in all probability be very limited since more than 95 per cent of these patients who were followed-up had reached the end of growth. Furthermore, most of them had a curvature below $50^{\circ}$ which is known to progress only little after end of growth.

Thus, the final average result of treatment in the present series is a curvature of approximately the same degree as that at the start of treatment. This, however, is a great improvement compared with the result which could be expected without treatment. According to James (1967) 63 per cent of the thoracic curves, 31 per cent of the thoraco-lumbar curves, and 9 per cent of the lumbar curves would progress to more than $70^{\circ}$ without treatment.

Several factors were shown to be of importance for the result at the end of treatment. It must first be recognized that the result of brace treatment on the curvature, and also on the frequency of brace failures and complications, to a large extent depend on a skilled craftsman for the manufacture of the braces. In Göteborg we have been fortunate to have an orthotist interested in these matters. Since we started with the Milwaukee brace, he has supervised the construction of these devices with great care and skill. A prerequisite for a satisfying result with the Milwaukee brace is full cooperation of the patient. The brace must be worn all day and night. In several reports it has been shown that the brace exerts a considerable tractile force on the spine. Cochran and Waugh (1969) reported results with strain-gauges mounted on Milwaukee braces. In 10 patients the total tractile force amounted to an average of 2.5 kp, and the forces acting on the side straps were approximately also 2.5 kp, more on the right side. This experiment was also performed by Galante et al (1970), who reported an average tractile force acting on the brace to be about 2 kp. In the supine position the tractile force of the brace increased. These forces also increased by breathing and physical exercises. Nachemson and Elfström (1971) showed with telemetry technique in intravital measurements in a Harrington rod that a total tractile force of more than 2-4 kiloponds was exerted by the brace. The side straps were shown to be of great importance for the correction of the deformity. The relatively greater mechanical efficiency of the brace in the supine position was also demonstrated.

Therefore, the brace should be worn all day and night. This is con firmed in the present study by the result in those patients who did not follow the routine of brace wearing. Their deformity increased, whereas the scoliosis improved in those patients wearing the brace as directed.

Other factors of importance for the correction have been mentioned above. In a stepwise regression analysis, these factors below were found to be in order of importance:

| | Mult. regr. coeff. |
|---|---|
| The apex of the curve | 0.40 |
| The wedge of the apical intervertebral disc | 0.40 |
| The wedge of the apical vertebra | 0.40 |
| The original curvature | 0.41 |
| The rotation deformity of the apical vertebra | 0.41 |

Thus, the location of the apex of the curve is most important for the result. All other factors, being a total measure of the severity of the curve and its structural changes, have a small influence on the result compared with this. In this material few curves were more than $50^{o}$, and the conclusions therefore are not valid for curves above $50^{o}$.

The prognosis for scoliosis with an early onset is known to be more severe (Moe 1957). This was noticed in the present study, where in early diagnosed scoliosis at 10 years of age or below, the correction was less than in later diagnosed scoliosis cases, with the exception of those diagnosed near cessation of growth.

Moe and Kettleson (1970) reported that the correction in percentage was better in longer curves (i.e. curves involving a larger number of vertebrae) than in shorter curves. This could not be verified in the present investigation, where the correction was better in the short curves. It seems that these shorter curves are more easily controlled by the pads of the brace.

It has been stated that the Milwaukee brace through its active and passive forces has a distracting and a straigthening as well as a derotating effect on the spine. To the author's knowledge, however, no studies have been published on the results of brace treatment on the rotation of the vertebrae. There is no doubt in clinical experience about the improvement of the rib hump and balance of these patients undergoing Milwaukee brace treatment. This is difficult to document and describe objectively, but it does not seem likely that this favourable cosmetic effect of the brace depends on a derotation of the vertebrae in the scoliosis as it is shown in this investigation that the rotation of the apical vertebra is not significantly influenced in the end by the brace treatment. The improvement in cosmetic appearance by the Milwaukee brace treatment probably more depends on an influence on the rib hump and over-all body balance than on the underlying rotational deformity.

It is to be noted that the respiratory function as evaluated by the vital capacity did not show any large change with the localization of the scoliosis. The vital capacity of the patients in this study depended more on the rotational deformity of the curve than on the curve angle. The mild curves in this conservatively treated material did not to any large extent influence the vital capacity. The ventilatory function was in no case a limiting factor for the physical performance of the patients.

In the present investigation there was a small decrease of the wedge of the vertebral body in patients, who were more than 13 years old at the beginning of treatment. On the other hand, there was an increase of the vertebral wedge of those patients who were younger. On an average, in the whole group, there was no change in the vertebral wedge. Breitenfelder (1971), examining the vertebral shape in 23 patients of different ages who had completed the treatment in the Milwaukee brace, noticed a decrease in the vertebral wedge in 19 patients. However, no statistical analysis was presented. MacEwen (1970) and MacEwen and Kirsch (1971) also discussed the reversal of the vertebral wedge shape by the Milwaukee brace treatment.

Experience with other types of braces have shown that there has been a tendency for the compensatory curves to increase under brace treatment of the major curve (Cobb 1948, Smith 1958). For the upper compensatory curve this was confirmed in this study to apply also to the spine treated in the Milwaukee brace. The lower compensatory curve, on the other hand, corrected almost as much as the primary curve. It is probable, that this is a result of the inability of the Milwaukee brace to have any positive influence on the scoliosis in the upper part of the spine.

The initial correction immediately after the brace was fitted was highly correlated with the final correction result at the end of brace treatment. This initial correction was the most valuable prognostic sign of all those parameters studied. The scoliotic curves which detoriated and therefore had to be operated on, corrected very little usually less than 10 per cent, initially when the brace was fitted. In borderline cases, where doubt exists whether conservative or operative treatment should be used, this initial correction in the brace could serve as a valuable guide to the further treatment program.

Complications with this type of treatment are fairly few and mild. The most difficult problem is to make the patient wear the brace correctly. The comparatively low drop-out frequency in this material, 15 patients in 159 patients observed for two years or more, is less than what has been reported in other materials. In the series of Moe and Kettleson (1970) 33 out of 169 patients or approximately 20 per cent were considered to cooperate poorly. Only two of the 15 drop-out patients in the present investigation interrupted the routine treatment after more than one year of brace wearing. In order to persuade the patients to continue the treatment, it is essential that the physician and the personnel involved establish a good emotional contact with the patient. This is best accomplished by having the patient spend a shorter time in the scoliosis ward in connection with the fitting of the brace and the immediate control of the scoliosis curve after the brace fitting. The patient should also be seen fairly often at regular visits in a special scoliosis clinic, where she will feel the emotional support of all those involved in her treatment. Here the patient also can meet other patients with scoliosis and personnel who are accustomed to the special problems involved. It will most certainly be easier for the patient to maintain the brace wearing routine, if told that with great certainty the scoliosis will not progress and possibly improve a little, if the brace is worn according to the routine.

## RESULTS OF OPERATIVE TREATMENT IN 100 PATIENTS

### Correction

The results of operative treatment of scoliosis of different etiologies are shown in Table 18. Here the correction obtained by a second stage procedure, the total curve correction, and the correction maintained at the two-year follow-up are given . In the idiopathic cases the average original curvature was a little less severe than in the congenital and the polio cases. The correction in percentage was higher in the idiopathic cases.

Table 19 demonstrates the results of 14 patients in whom double primary curves were instrumented and fused. In patients operated with one rod with the "dollar-sign" application the correction was better and the loss smaller than in patients operated with two rods. The material is too small to allow any statistical analysis, however.

TABLE 18. Results of operative treatment (Mean±S.D.)

| | Etiology | No. of pat. | Preop. Curvature Degrees | Corr. in 2:nd stage Degrees | Correction after 2:nd stage Degrees | Correction after 2:nd stage % | Correction maintained at 2-year follow-up Degrees | Correction maintained at 2-year follow-up % |
|---|---|---|---|---|---|---|---|---|
| Single curves | Idiopathic | 72 | 71.5 ±16.6 | 5.8 ±3.0 | 40.6 ±9.3 | 56.8 ±10.8 | 35.6 ±9.3 | 49.8 ±10.7 |
| | Congenital | 4 | 75.0 ±17.3 | 7.0 ±5.4 | 38.8 ±11.1 | 51.3 ±2.5 | 33.8 ±11.8 | 44.5 ±6.8 |
| | Neuropathic | 5 | 90.0 ±18.4 | 12.8 ±7.8 | 46.6 ±13.8 | 51.8 ±17.7 | 41.6 ±13.1 | 46.2 ±16.0 |
| | Myopathic | 1 | 80 | 10 | 35 | 44 | 23 | 29 |
| | Marfan | 1 | 100 | 10 | 50 | 50 | 30 | 30 |
| | Neurofibro-matosis | 2 | 85.0 (85-85) | 7.5 (0-15) | 37.5 (35-40) | 44.0 (41-47) | 35.0 (30-40) | 41.0 (35-47) |
| | Thoracoplasty | 1 | 85 | 7 | 42 | 49 | 35 | 41 |
| | All | 86 | 73.4 ±17.0 | 6.5 ±3.9 | 41.3 ±9.4 | 56.3 ±11.0 | 35.7 ±9.5 | 49.4 ±11.1 |
| Double curves upper/lower | Idiopathic | 12 | 68.9/66.2 ±15.5/±14.5 | 4.5/3.7 ±5.7/±4.6 | 32.1/39.1 ±8.6/±9.4 | 45.9/59.0 ±12.4/±14.2 | 27.6/31.8 ±11.6/±7.3 | 40.0/48.6 ±14.7/±13.9 |
| | Congenital | 1 | 75/70 | 5/5 | 25/45 | 33/64 | 20/40 | 27/57 |
| | Neuropathic | 1 | 65/75 | 0/3 | 20/45 | 31/60 | 15/25 | 23/33 |

TABLE 19. Result in double primary curves. (Mean)

| Double curves | No. of patients | Preop. curve Degrees | Correction Degrees | Correction % | Total loss Degrees | Total loss % |
|---|---|---|---|---|---|---|
| Double rods: | 5 | 69/66 | 28/39 | 41/60 | 7/14 | 17/52 |
| "Dollar sign" | 9 | 64/62 | 32/45 | 50/73 | 5/5 | 16/30 |

Loss of correction

Table 20 shows the initial loss of correction when the patient after three weeks of recumbency (after the fusion operation) is mobilized in a Milwaukee brace, and the late loss of correction from then on up to the 2-year follow-up. It also shows the total loss in the follow-up examinations. In Table 21 the losses of correction over different periods of time are specified.

An initial loss of $11^{\circ}$ occurred in a patient in whom the vertebral lamina fractured after the second operation (see page 131). In one patient with double primary curves there was a late loss of $27^{\circ}$. The patient had an idiopathic, double primary curve of $45/65^{\circ}$, the curve was operated with 2 rods and was reduced to $23/15^{\circ}$, but during ambulation the next year increased to $30/35^{\circ}$. No sure hook complication could be noticed. The curve was $30/35^{\circ}$ on the 3-year follow-up roentgenograms.

The other patient with a large loss was a girl with a double primary curve caused by a disorder of the central nervous system after an extirpation of a dermoid cyst of the cerebellum. This caused amaurosis, and difficulties with the balance. The curve which was operated with two rods increased 20 degrees during the first postoperative year, but has not deteriorated since then in the 3-year follow-up.

A probable cause of loss of correction in these cases operated with two rods is a change of the hook positions even though this was not clearly detected on the roentgenograms.

TABLE 20. Loss of correction. (Mean ± S. D.)

| Etiology | Immediate loss | Late loss | Total loss at the 2-year follow-up | | at the 3-year follow-up | | at the 4-year follow-up | |
|---|---|---|---|---|---|---|---|---|
| | Degrees | Degrees | Degrees | n | Degrees | n | Degrees | n |
| **Single curves** | | | | | | | | |
| Idiopathic | 3.4 ±3.6 | 2.5 ±2.8 | 5.8 ±5.0 | 72 | 6.5 ±5.0 | 37 | 7.0 ±7.7 | 14 |
| Congenital | 3.8 ±2.5 | 3.8 ±2.5 | 7.6 ±4.3 | 4 | 2.5 (0-5) | 2 | 5.0 | 1 |
| Neuropathic | 3.8 ±4.0 | 2.2 ±2.3 | 6.0 ±5.2 | 5 | 6.0 ±5.4 | 3 | 7.2 ±5.9 | 3 |
| Myopathic | 5 | 7 | 12 | 1 | 12 | 1 | - | - |
| Marfan | 10 | 10 | 20 | 1 | 20 | 1 | - | - |
| Neuro-fibromatosis | 0 (0-0) | 2.5 (0-5) | 2.5 (0-5) | 2 | 2.5 (0-5) | 2 | 2.5 (0-5) | 2 |
| Thoraco-plasty | 7 | 0 | 7 | 1 | - | - | - | - |
| All | 3.5 ±3.6 | 2.7 ±3.0 | 6.2 ±5.2 | 86 | 6.3 ±5.4 | 46 | 6.9 ±7.6 | 20 |
| **Double curves** | | | | | | | | |
| Idiopathic | 1.6/3.0 ±2.9/±2.3 | 4.0/4.4 ±3.9/±6.4 | 5.6/7.4 ±5.4/±8.0 | 12 | 6.3/9.4 ±5.2/±6.4 | 7 | 8.5/5.0 (7-10)/(5-5) | 2 |
| Congenital | 0/0 | 5/5 | 5/5 | 1 | 5/5 | 1 | - | - |
| Neuropathic | 0/5 | 5/15 | 5/20 | 1 | 5/20 | 1 | 5/20 | 1 |

TABLE 21. Loss of correction.

| Degrees | Initial postop. loss | Late loss at 2-year follow-up | Loss during 2:nd postop. year | Total loss at the 2-year follow-up | Loss during 3:rd postop. year | Loss during 4:th postop. year |
|---|---|---|---|---|---|---|
| | No. of patients | | | | | |
| Single curves | | | | | | |
| 0 - 5 | 78 | 79 | 86 | 52 | 46 | 20 |
| 6-10 | 7 | 7 | - | 25 | | |
| 11-15 | 1 (laminar fracture) | - | - | 9 | | |
| Double curves (Sum of curves) | | | | | | |
| 0 - 5 | 10 | 7 | 13 | 3 | 9 | 3 |
| 6-10 | 4 | 5 | 1 | 6 | | |
| 11-15 | - | - | - | 3 | | |
| 20 | - | 1 | - | 1 | | |
| 27 | - | 1 | - | 1 | | |

### Length of fusion

The average number of fused vertebrae in the single primary curves was 11.1 (range 8-13) and in the double primary curves 13.1 (range 12-15). The number of vertebrae which was included in the fusion above the upper neutral non-rotated vertebra in the major curve was on an average 1.4 vertebrae, while in average 2.0 vertebrae were included in the fusion below the lower neutral vertebra of the major curve.

In three patients, the original curve extended one vertebra above the fusion, and in one patient it extended one vertebra below the fusion. In this last-mentioned case an increase in the length of the curve was noticed, associated with an increased loss of correction. It was a girl, 11 years old, with an idiopathic thoracic 75$^{o}$ curve, which was reduced to 27$^{o}$ at the operations. A loss of approximately 5$^{o}$ a year after the operations occurred, and at the follow-up 4 years postoperatively the curve was 49$^{o}$. This loss of correction was associated with a successive lengthening of the lower part of the curve, where the fusion included the last rotated but not the neutral vertebra. The fused part of the spine remained unchanged at 35$^{o}$, however.

## Complications

The complications in 203 interventions in 100 patients operated for scoliosis of different etiologies are shown in Table 22. There were no mortal complications and no deep infections. One gastro-intestinal complication was a retention for four days after both the first and the second operation on the same patient. This was a 14-year old girl with a thoracic idiopathic curve of 67$^{o}$ which was corrected in the first stage to 40$^{o}$ and in the second stage to 35$^{o}$. The patient was treated with parenteral nutrition and the condition resolved after four days without any further signs of duodenal stenosis or paralysis.

The neurological complications include one boy with a 75/70$^{o}$ congenital double primary scoliosis curve, which was reduced to 55/30$^{o}$ in the first stage operation, after which a positive Babinsky sign appeared for three days. At the second stage operation the curve was further reduced to 50/25$^{o}$ with no postoperative complications this time. The second case with neurological complications was a more severe one, which showed signs of lower motor neuron damage to the 2:nd-4:th sacral nerve roots. It was a girl, 12 years of age, with an 80/75$^{o}$ idiopathic double primary curve, which was corrected to 45/38$^{o}$ in the first operation. Postoperatively there was incontinence of both urinary and fecal functions. The girl was operated in a second stage with less distraction, and corrected to 50/45$^{o}$. The condition successively resolved during the following months, and the patient is

TABLE 22. Complications in 100 patients operated with the Harrington distraction rod in two stages.

| Complications | No. of patients 1:st stage | 2:nd stage | |
|---|---|---|---|
| Wound infection, | | | |
| cutaneous | 1 | 1 | |
| deep | - | - | |
| Haematoma | 1 | 1 | |
| Pneumonia | 1 | - | (atelectasis) |
| Pneumothorax | 1 | - | (after rib resection) |
| Urinary infection, | | | |
| lower tract only | 9 | 2 | |
| upper tract | 1 | - | |
| Gastro-intestinal (retention) | 1 | 1 | (same patient, resolved after 4 days) |
| Neurological | 2 | - | |
| Hook displacement | 1 | 2 | |
| Rod displacement in hooks | - | 2 | (1 notch) |
| Instrument fracture | - | 1 | (after 14 months, fusion intact, correction maintained) |
| Laminar fracture | | | |
| during operation | 2 | 1 | |
| after operation | 3 | 1x2 | (same patient, fractured twice) |
| Pseudoarthrosis | 0 | | |

now well recovered without any symptoms and no residual urine. In 3 patients there were hook displacements, which all occurred at the thoracic hook purchase site. One of these cases occurred after the first stage operation, the hook displacement was corrected by reseating the hook on the next proximal vertebra in the second intervention. In one patient the hook displacement with a lateral shift to the transverse process at the thoracic purchase site was discovered two weeks after the second stage operation. A loss of correction of $8^{\circ}$ was revealed in these two weeks, the scoliosis progressing from $12^{\circ}$ to $20^{\circ}$ (original curve $60^{\circ}$). However, from two weeks postoperatively, there were no losses at all, and the two year result is still a curve of $20^{o}$. In a third patient,the hook displaced completely from the vertebral lamina, causing a considerable increase in curvature and the patient was reoperated ten days after the second intervention. The hook was then placed on one vertebra above the first chosen vertebra. This patient, a boy aged 16 years, had the most severe curve in this series, $130^{o}$, which was corrected in the second intervention to $95^{o}$. In the third operation, the curve was only corrected to $100^{o}$, and has not changed 3 years after operation.

Despite the fact that care was taken not to exceed about 45 kp of distraction force, there occurred 8 laminar fractures in 203 interventions, all at the upper hook purchase sites. Three fractures occurred during operation, all due to improper seating of the hook in the lamina instead of under it. In these cases, the hook was immediately reseated on the next vertebra above or below the original purchase site. Postoperatively, there occurred four laminar fractures, three of which occurred before the second stage operation, also due to an improper seating of the hook. The hook could be reseated in this second intervention. In one patient, a 17-year old girl with an idiopathic scoliosis of $74^{o}$, a laminar fracture at the upper hook purchase site occurred after the second stage operation. The patient was reoperated. Again there occurred a laminar fracture at the proximal hook. The rod was then extracted in a fourth intervention 3 months after the first operation, and the patient was treated in a Milwaukee brace. In the two-year follow-up she had a stable curve of $50^{o}$, which also was unchanged in the three-year follow-up.

In one patient, a 14-year old girl with a $100^{o}$ congential scoliosis and a $70^{o}$ kyphosis, a fracture of the Harrington rod occurred. 14 months after the operation the patient felt a short sudden pain in her back as she lifted a heavy suitcase. Roentgenograms revealed a fracture of the rod at the first notch, the ends of the rod did not overlap. When the patient was operated with extirpation of the fractured rod the whole fusion area was carefully inspected, but no sign of pseudoarthrosis could be found. The curve, which was corrected to 45° still was 50° in the three year follow-up. The kyphosis increased, however, from 50° to 70° following the fracture of the rod, but has now remained stable for 2 years.

### Pseudoarthrosis

All patients were carefully examined for any signs of pseudoarthrosis. Roentgenograms including oblique views of the fusion and the intervertebral joints have not revealed any signs of pseudoarthrosis in any patients, nor has the clinical status of the patients caused any suspicion of this. There was thus very little loss of correction after the support of the Milwaukee brace had been abandoned.

## RESULTS IN 70 PATIENTS WITH IDIOPATHIC SCOLIOSIS

A selective analysis of the results and the factors influencing these results was performed in the patients with idiopathic scoliosis in the material. There were 72 patients with single idiopathic scoliosis, among these were two patients with extraordinary complications, both mentioned above. One patient twice had a fracture of the upper hook purchase site, and the rod was removed 3 months postoperatively. The second patient had a 130° curve and when the upper hook had displaced after the second stage operation, the patient was reoperated. These two patients were omitted from this particular study, which thus comprises 70 patients with single primary idiopathic scoliosis.

### Respiratory function

The respiratory function was lowered in all these patients, the mean vital capacity was 69 per cent of the predicted normal value. Vital capacity decreased significantly ($2p<0.01$) with an increasing curve degree, and also significantly ($0.01<2p<0.05$) with an increasing rotation of the curve.

A regression analysis of the correlation of vital capacity to the original degree of curvature and to the rotation showed, that in this material with more severe curvatures, practically all of the decrease of the vital capacity depended upon the lateral angulation of the scoliosis, while only a small part depended upon the rotation of the apical vertebra. This result is quite contrary to what was found in the smaller curves in the conservatively treated cases, where the vital capacity primarily depended on the rotation of the vertebrae.

In this material the vital capacity was not significantly correlated to the localization of the curve, but was only measured in 2 upper thoracic and 2 lumbar cases of scoliosis.

### Age of patient

The mean age of the patients at the operation was 15 years and 2 months (10-26 years). Most patients - 45 per cent - were operated in their 13:th and 14:th year. The chronological age at the operation, or the maturity of the skeleton (estimated by the development of the iliac apophysis) did not show any significant correlation to the correction in percentage or degrees, nor to the subsequent loss of correction.

Those patients in whom the scoliosis had an early onset (was diagnosed early) were younger at operation than patients in whom the deformity was diagnosed later ($r=0.52$, $2p<0.01$).

### Original curvature

The mean curvature was $70.6 \pm 15.2^{\circ}$. There was a significant ($2p< 0.01$) negative correlation between the degree of curvature and the percentage correction of the curve (Fig. 33). Curves below $80^{\circ}$ corrected approximately 60 per cent while curves above $80^{\circ}$ were corrected only about 50 per cent. There was a strong correlation between the degree of curvature and the structural changes in the apical segment, such as vertebral rotation and vertebral and disc wedge.

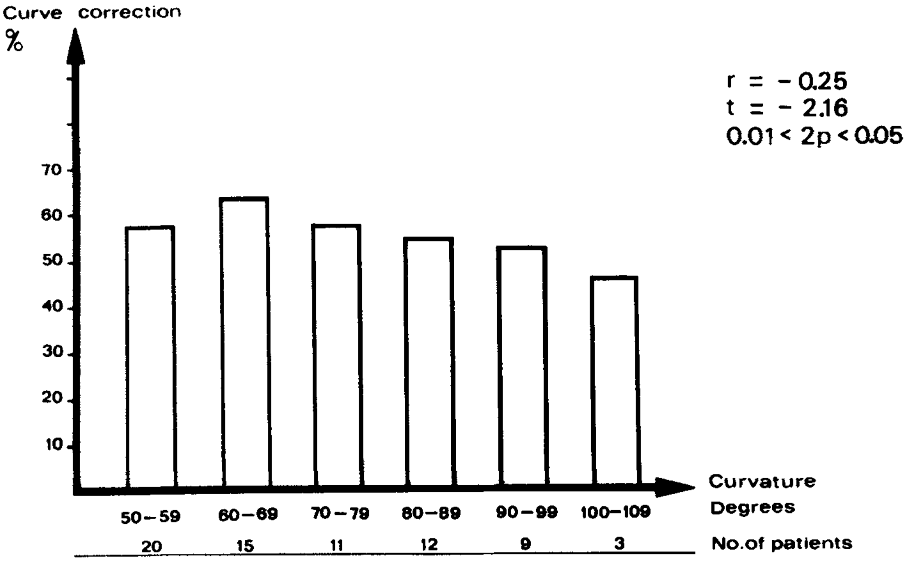


Fig. 33. Mean percentage correction correlated with the original curvature.

Number of vertebrae

The mean number of vertebrae in the curve was 7.4 ±1.0. An increased number of vertebrae was significantly associated with an increased curve degree ($2p<0.01$). It was also associated with an increased correction in degrees ($2p<0.01$) and a slight, non-significant increased correction in percentage. In equal degree curves, a shorter curve had more severe structural changes as evidenced by the wedge of the disc and vertebrae.

Degrees/number of vertebrae

Harrington already in 1960 described the Harrington factor (degrees/ number of vertebrae) as a criterion for treatment of scoliosis. He stated that with a factor above 5, instrumentation is indicated. The prognosis of scoliosis can be evaluated with the aid of this factor (Harrington 1972). In the present study, the correction in percentage was better correlated to the Harrington factor, than either to original curvature or the number of vertebrae in the curve. With the Harrington factor below 8, the correction was 70 per cent or more, but with a factor of more than 10, the correction was below 60 per cent (Fig. 34).

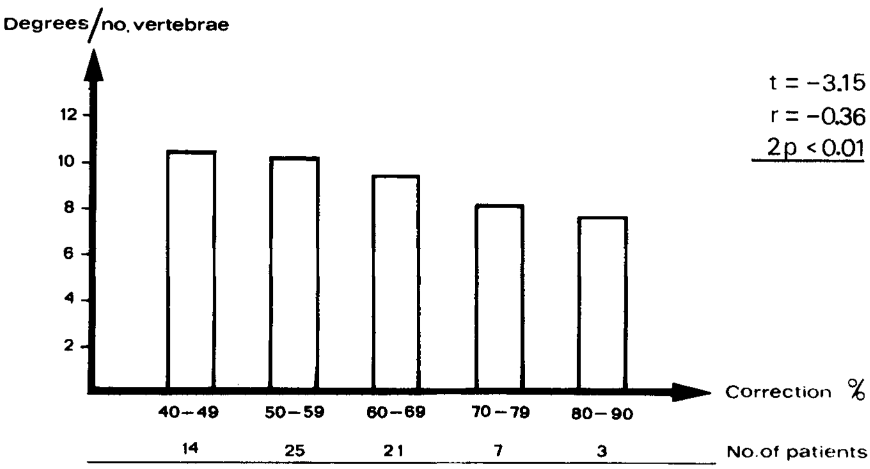


Fig. 34. The "Harrington factor" - degrees/number of vertebrae - correlated with the percentage correction.

For an estimation of the result with an operation, an evaluation of the Harrington factor is more reliable than an evaluation of the curve degree with the limits for operation used in this series.

### Correction obtained by the second stage operation

The correction obtained by the second stage procedure averaged 5.8 $\pm 3.0^{\circ}$, i.e. compensated approximately for the subsequent total loss. The degrees of correction by the second procedure were closely correlated with the degrees of total correction ($2p<0.01$) (Fig. 35). With an increasing original curve degree, there was a significant increase in the degree of correction at the second stage operation.

### Supine correction

In this material, the supine correction (mean $16.2 \pm 7.9$ per cent) was used as an estimation of the flexibility of the curve. This correction

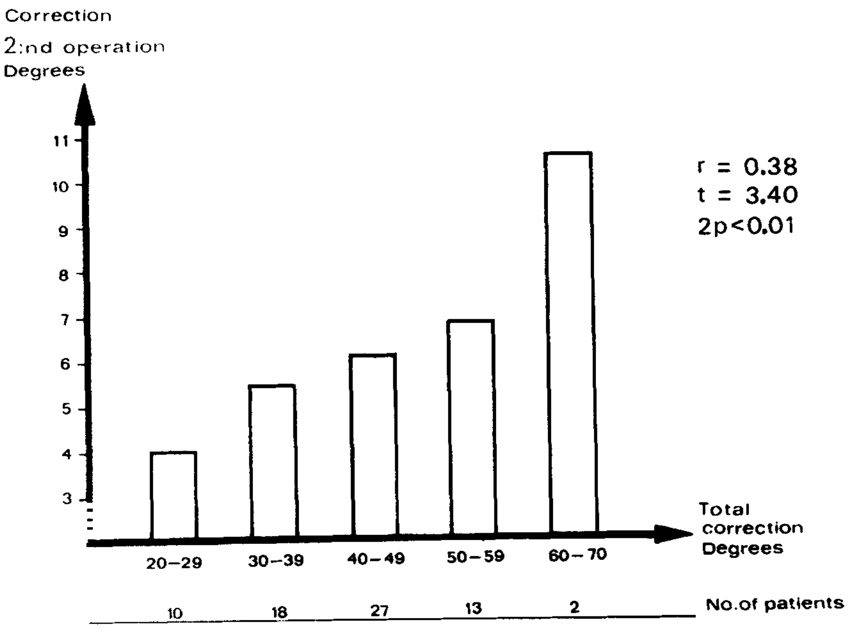


Fig. 35. Correction obtained in the second operation correlated with the total correction (in degrees).

in percentage correlated very well ($2p<0.01$, $r=0.54$) with the final postoperative correction in percentage (Fig. 36). With increased correction in degrees in the supine position, there was also an increased correction in degrees postoperatively.

## Loss of correction

The mean initial loss of correction was $3.3 \pm 3.6^{\circ}$ (significant), and the mean late loss was $2.5 \pm 2.8$ (significant). The mean total loss was $5.8 \pm 5.0$.

There was a non-significant tendency for the initial loss in degrees at ambulation to decrease in the more distal curves. This tendency was reversed when the total loss was evaluated. It seemed that an immediate small loss later was followed by a larger loss and vice versa (Fig. 37). The total loss in degrees was approximately the same in all curves and was not significantly correlated to the cor-

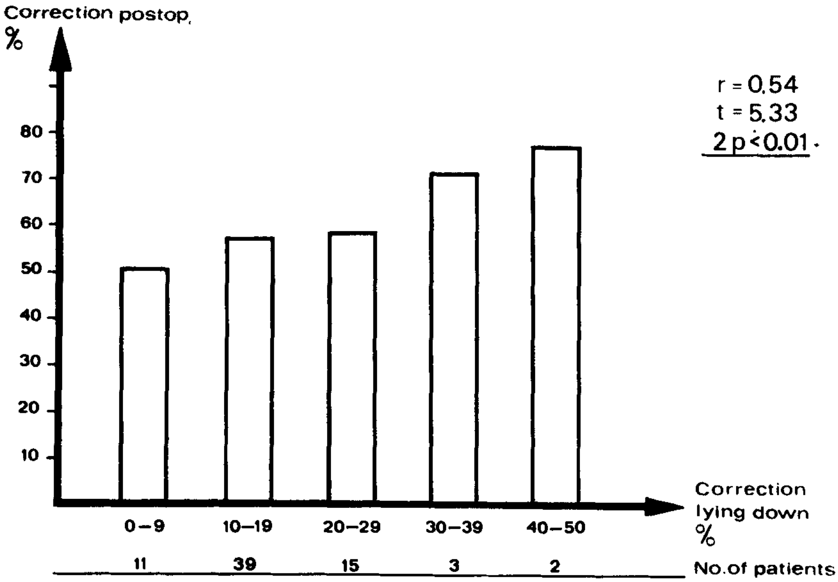


Fig. 36. Mean percentage correction after operation correlated with percentage correction in the supine position preoperatively.

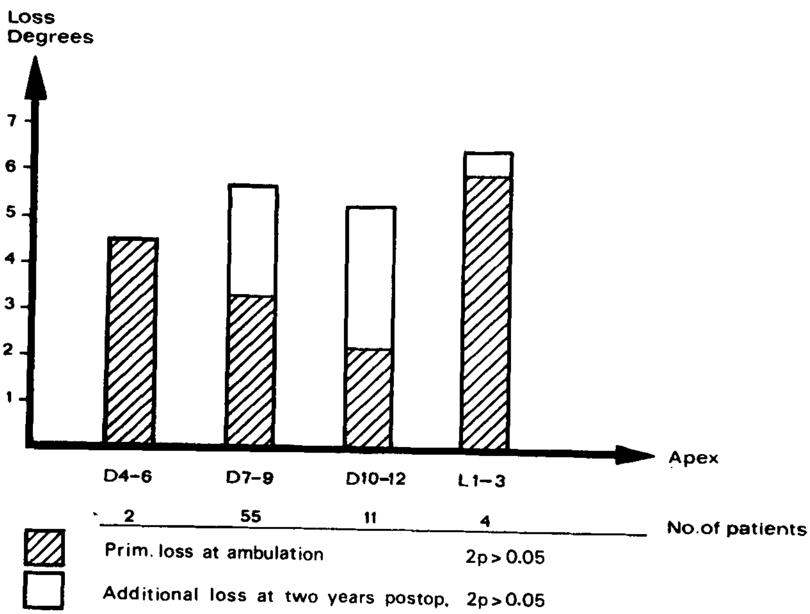


Fig. 37. Mean total loss of correction divided into primary loss and additional loss and correlated with the location of the curve.

rection. The loss in percentage was significantly higher both initially and late in those curves which were more corrected (Fig. 38). In curves which corrected more than 70 per cent the loss was **40** per cent and more, calculated from the small postoperative curve.

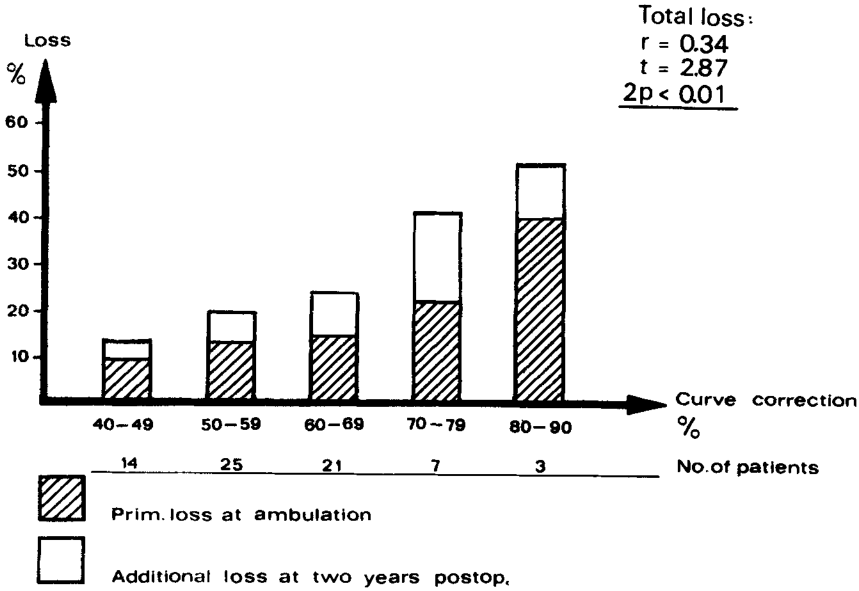


Fig. 38. Mean total percentage loss divided into primary and additional loss correlated with percentage correction.

The age of the patient at operation was not significantly correlated to the postoperative loss, nor was the weight of the patient.

## Apex of the curve

Curves located more distally were significantly better corrected. The best correction in percentage was obtained in the thoraco-lumbar curves, about 60 per cent (Fig. 39).

## Rotation

The mean rotation of the apical vertebrae was $25.8 \pm 8.3^{\circ}$. The

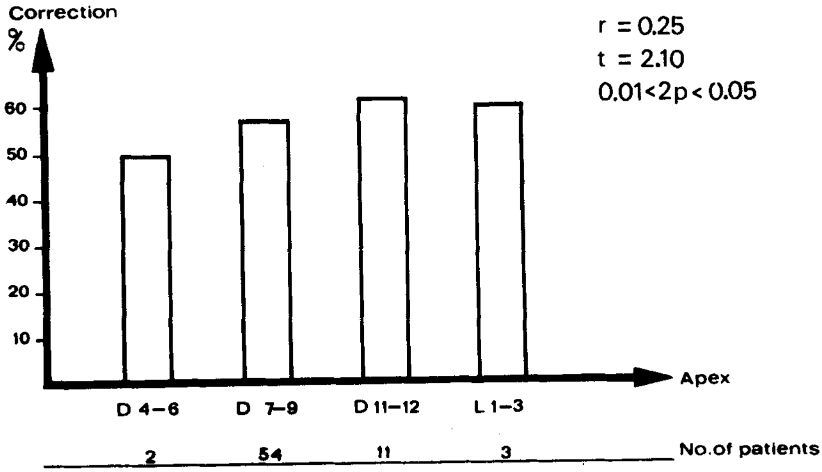


Fig. 39. Mean percentage correction in curves of different locations.

rotation closely accompanied the curve degree (r=0.64). As Fig. 40 shows, there was a gradual, significant, decrease in curve correction with increasing vertebral rotation. The correction was better correlated with the rotation than with the original curve degree, which

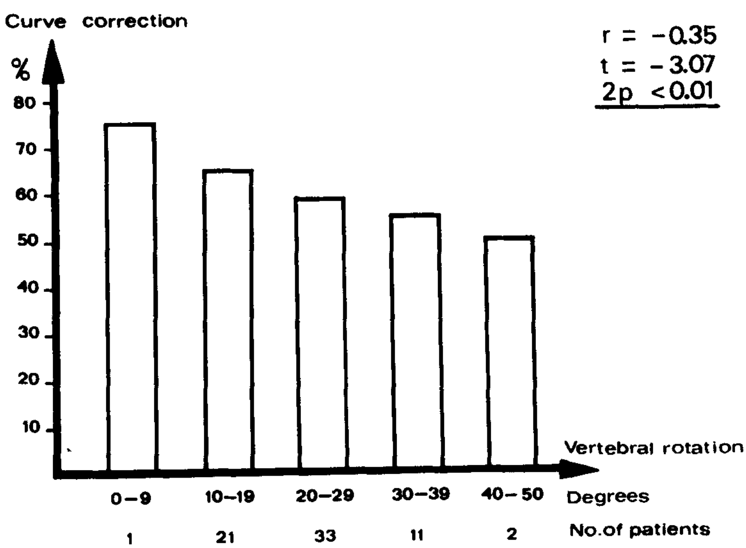


Fig. 40. Mean percentage correction correlated with degrees of rotation of the apical vertebra.

was also proved by a partial correlation test. The rotation increased particularly in curves above $80^{\circ}$. In the lumbar region, the vertebrae were more rotated than in the thoracic region ($2p<0.01$). The operation had a derotating effect on the spine, on an average 4.8 degrees, or approximately 20 per cent ($2p<0.01$). The decrease in rotation deformity in degrees was largest for those with the largest rotation deformity. In the two-year postoperative follow-up there was again an increase of vertebral rotation, about $2^{\circ}$, which was significant.

Wedge of the vertebral body

The mean vertebral body wedge angle was $9.2 \pm 2.7^{\circ}$. The mean wedge ratio was $0.80 \pm 0.06$. The vertebral wedge increased significantly with an increasing curve. With an increasing wedge, there was a decreased correction in percentage ($0.01 < 2p < 0.05$). In those patients, who were operated at a younger chronological age or in whom the iliac apophysis had not developed, the vertebral wedge was more pronounced than in older patients ($2p<0.01$).

When the vertebral wedge was measured on the roentgenograms obtained one year after the operations, a significant improvement of the wedge ratio was noticed. The wedge ratio increased $0.06 \pm 0.05$ compared with the preoperative vertebral wedge. The average decrease in the wedge angle was $2.9 \pm 2.6^{\circ}$ or about 20 per cent. The wedge improvement was greatest for the curves which corrected best ($0.01 < 2p < 0.05$) (Fig. 41). In the roentgenograms at the follow-up 2 years after the operations the vertebral wedge was not significantly changed as compared with the status in the one-year follow-up roentgenogram. The improvement of the vertebral wedge shape was significantly correlated to the development of the iliac apophysis (Fig. 42).

Wedge of the intervertebral disc.

The mean wedge angle of the intervertebral discs was $8.5 \pm 2.2^{\circ}$.

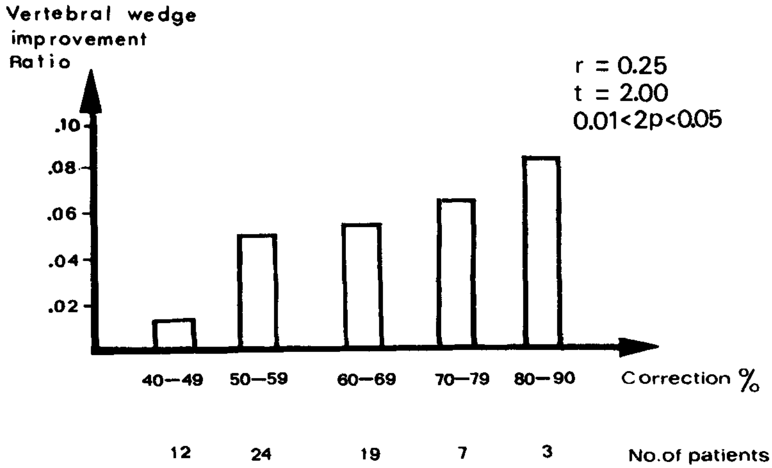


Fig. 41. Improvement of vertebral wedge ratio one year after the operation correlated with the percentage correction after operation.

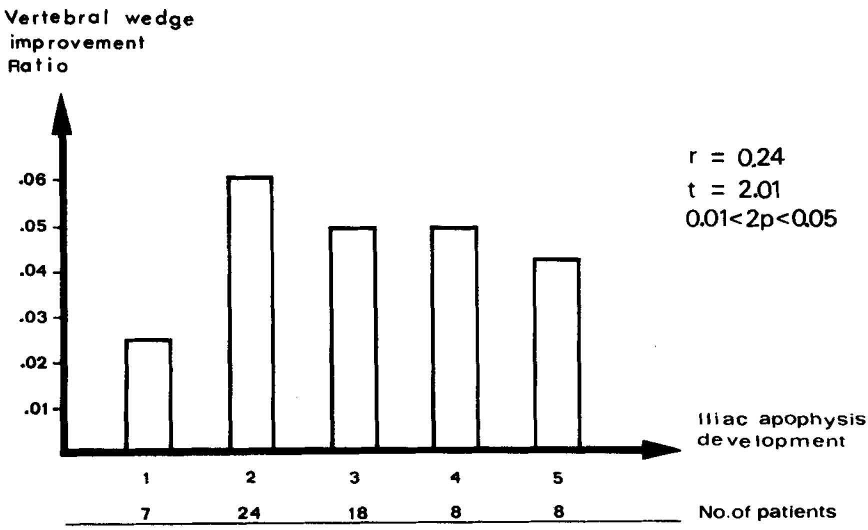


Fig. 42. Improvement of vertebral wedge one year after the operations correlated with the development of the iliac apophysis at the time of operation.

The mean wedge ratio was 0.47±11.5. The disc wedge was closely correlated to the curve degree. With an increased disc wedge there was a significantly decreased correction in percentage($2p < 0.01$) and it was particularly in patients with a disc wedge ratio below 0.4 that correction of the curve was small (Fig. 43). In the first postoperative roentgenograms the disc wedge was reversed to a mean wedge ratio of 1.13±0.22. This postoperative correction of the disc wedge increased with increasing correction of the curve in degrees, but was soon reduced to below 1.0 again, already in the next roentgenogram two weeks later . This return of the disc wedge at the apex segment postoperatively seemed to occur at the expence of the adjacent discs, as the curve angle did not change to such a degree as to explain this return of wedge shape in the apical intervertebral disc.

Compensatory curves

As in the Milwaukee brace material, not all major curves had both an upper and a lower compensatory curve that was measurable. Of existing curves the upper compensatory curve had a mean curvature of $34.0 \pm 10.7^{\circ}$ and the lower compensatory curvature was $37.4 \pm 10.6^{\circ}$. The correction in the compensatory curves was significantly ($2p<0.01$)

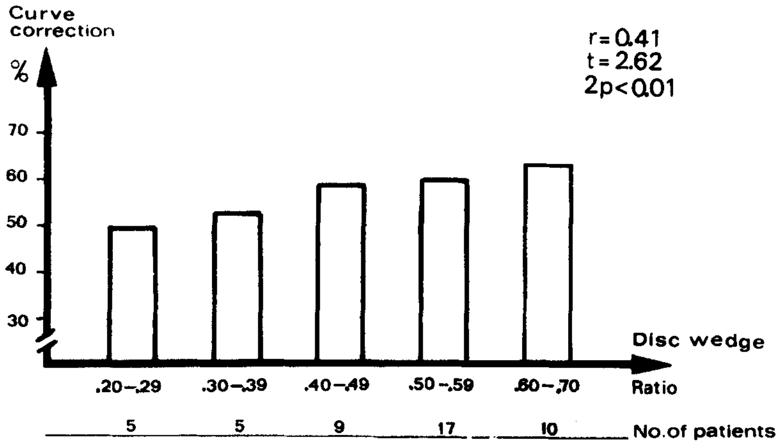


Fig. 43. Mean curve correction in percentage correlated with the preoperative disc wedge ratio.

correlated with the correction of the major curve . The lower non-operated compensatory curve was corrected approximately 10 per cent less than the major curve, while the upper compensatory curve corrected about 20-30 per cent less than the major curve. (Fig. 44). The sum of the correction in degrees of the upper and lower compensatory curves was generally lower than the major curve correction (Fig. 45).

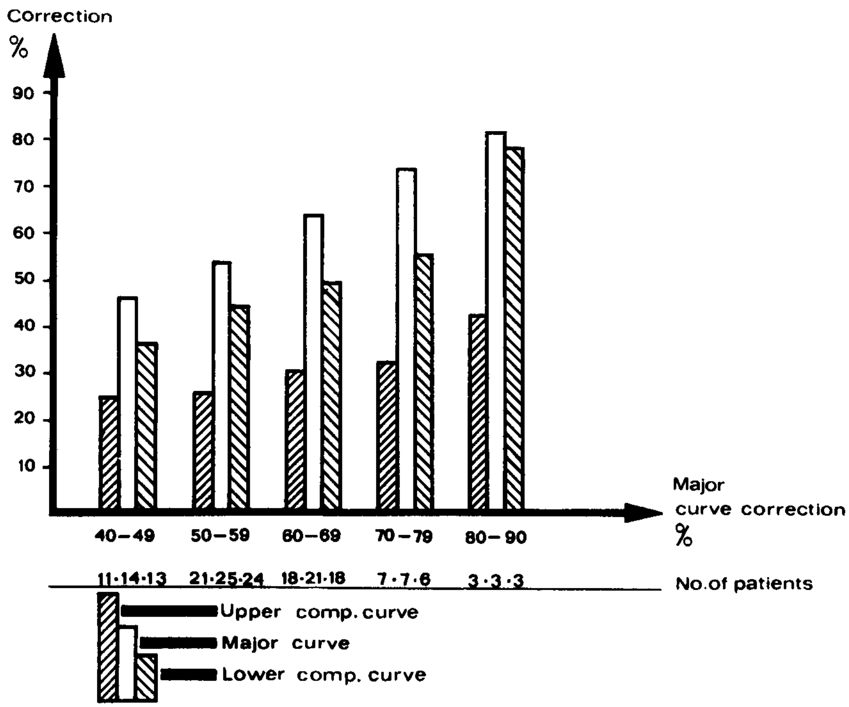


Fig. 44. Mean percentage correction of the major curve and the upper and lower compensatory curves correlated with the correction of the major curve.

There was a significant ($0.01 < 2p < 0.05$) negative correlation between the percentage correction of the lower non-operated compensatory curve and the rotation in that curve (Fig. 46).

The lower double primary operated curves had a mean original curve of $66^{o}$, mean rotation of $28^{o}$ and were corrected 60 per cent.

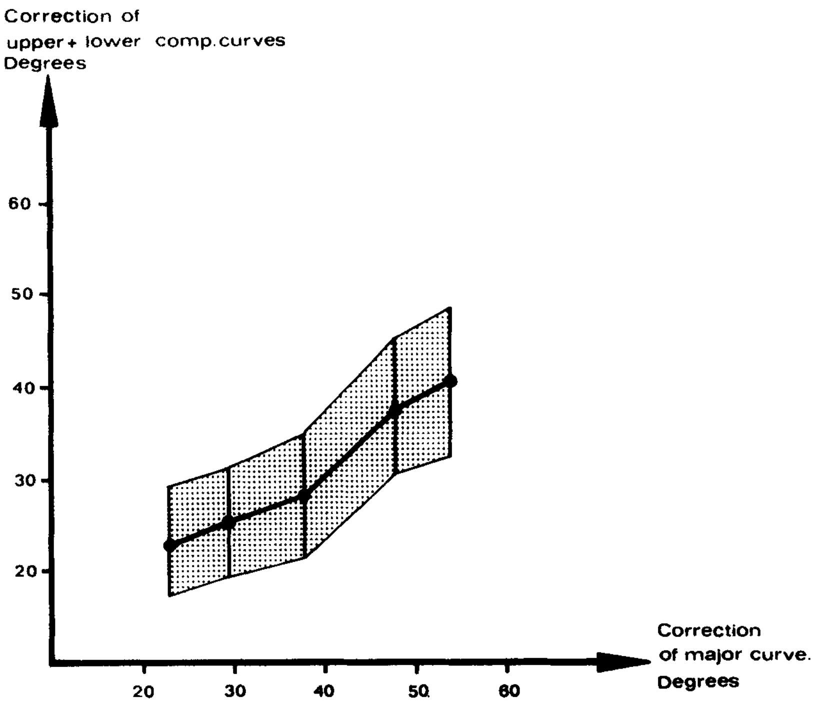


Fig. 45. Mean correction with standard deviations of upper and lower compensatory curves correlated with the degrees correction of the major curve.

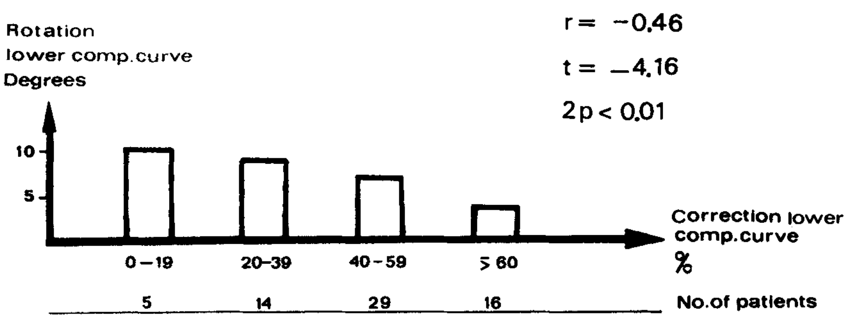


Fig. 46. Mean rotation of the lower compensatory curve correlated with the mean percentage correction of that curve.

If the correction of the lower compensatory curves with no or slight rotation was compared with that of the curves with a rotation of up to 15-20°there was not any large difference, provided the curvatures were below 50°. The correction of the compensatory curves seems to depend more on other factors, such as the severity and correction of the major curve. It is questionable, whether such curves need to be included in the correction and fusion.

The mean loss of the upper and lower compensatory curves at the second-year follow-up examination was 0.3° and 0.5° respectively and not significant, nor was there any significant loss of correction of these curves in the third and fourth follow-up years.

Height measurements

Willner (1972) has noted that girls with idiopathic scoliosis had a significant increase in sitting height as compared to normal girls. In the present material, with comparatively severe curves above 50° the sitting height of the patients with scoliosis usually was more than 5 cm shorter than that of the controls (Fig. 47). The operation caused a considerable increase in the sitting height, in all patients averaging 4.0 ±1.4 cm. After the operations, the change in the sitting height has been followed-up for two years. A complete investigation of the total increase in height of all patients until the end of growth would have been desirable, but was impossible as several patients who were young at operation had not yet completed their growth. In order to evaluate the effect of the operation on the total remaining growth also in younger patients, these young patients have been estimated to grow after the second postoperative year, each year, at the same rate as the older patients in the follow-up study had grown in the second postoperative year in the corresponding age. Fig. 47 shows for each age group the different phases of the increase in sitting height, which is the result of the operation:increase by correotion of the deformity, growth during the next two years and in the younger age-groups, expected additional growth until the age of 17 years. The diagram also reveals the expected total increase in sitting height for each age group of normal controls (controls: 10-16 years, Karlberg 1972, 16-17 years, Heimendinger 1964).

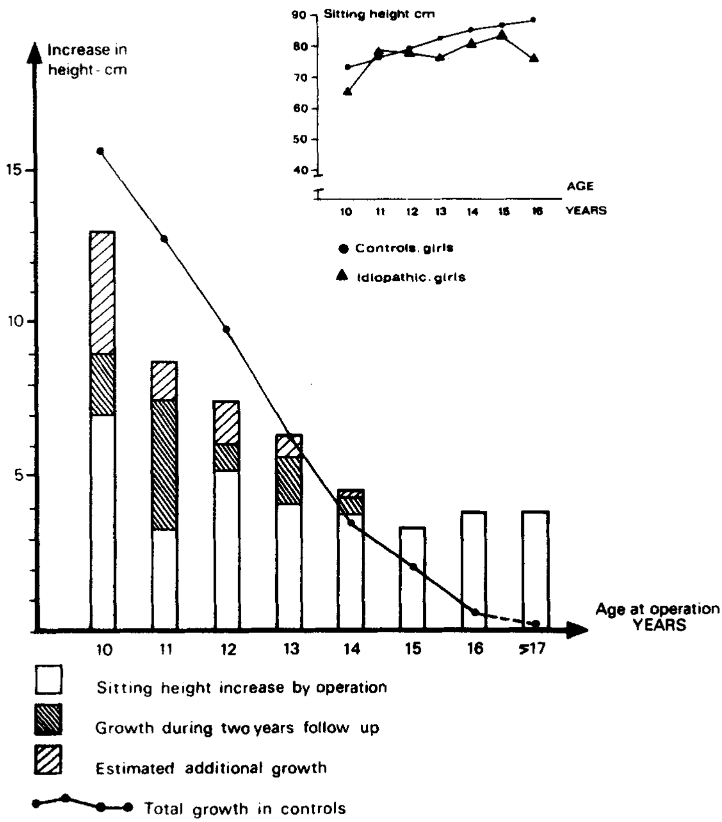


Fig. 47. Increase of sitting height in patients operated with correction of the scoliosis and fusion in different age groups at operation compared with the normal growth of the corresponding ages. The inset shows the sitting height of girls with idiopathic scoliosis and controls.

## DISCUSSION OF OPERATIVE TREATMENT

The technique for the correction of scoliosis with the Harrington method varies considerably among different surgeons both in operative procedure and in pre- and postoperative treatment.

Some use a preoperative localizer cast or a Cotrel type cast (Goldstein 1969, Leider et al 1972). Traction of different types - dynamic or steady Cotrel traction, Stagnara type traction or traction with a halo and femoral or pelvic pins - are used by others, particularly in curves exceeding 75-80$^{o}$ (Scheier 1969, Hall 1970, Michel and

Onimus 1970, Goldstein 1971, Götze 1971, Wilson et al 1971, Stagnara 1973, Hensinger and MacEwen 1973). Preoperative traction is an effective means of correction, and can often correct the deformity to about the same degree as can be achieved with the Harrington distraction rod. It has also been said to be of advantage in diminishing the risk of neurologic complications in severe curves by successive traction-extension.

Others have not used any preoperative correction of the scoliotic curve at all, relying solely upon the Harrington instrument to correct the deformity (Tambornino et al 1964, Gaubert et al 1967, Dolan and MacEwen 1971, Morscher 1971, Krol et al 1972). Several descriptions of the operative technique for the Harrington distraction rod and the bilateral-lateral posterior fusion have been published (Harrington 1962, 1967, 1972, 1973, Goldstein 1966). The original Harrington (1962) method includes the use of a distraction rod on the concave side in conjunction with a compression instrument on the convex side. Opinions vary as to the necessity of using this compression instrument. Harrington (1967, 1973) obtained an improved initial correction of about $10^{\circ}$ with this compression instrument as compared to using only the distraction rod on the concave side. In an investigation of 210 cases, Hall (1970) reported a correction of 85 per cent in moderate curves of less than $50^{\circ}$ with the complete set of instruments as compared with 60 per cent correction with only the distraction rod. No such beneficial effect of the compression set was seen in patients with more severe curvatures. Harrington is of the opinion that the correction loss, due to erosion in the hook sites, will decrease with the use of the compression system. Others, who have used the compression instrument are Goldstein (1969) and Gaubert et al (1971). However, most authors have refrained from using compression, claiming that this does not increase the correction (Stagnara 1967, Moe 1972). The instrument also makes an adequate decortication and fusion on the convex side difficult (Gaubert et al 1967). Waugh (1966) and Nachemson (1972) showed in measurements during operations, that the application of the compression rod did not have any large deloading effect on the distraction rod when measured during the operation in patients with curves exceeding $55^{o}$.

The two-stage operations have earlier been performed only by a few surgeons(Gaubert et al 1971, Götze 1971, Stagnara 1973) and then only in selected cases in longer curves or double primary curves. The present work is the only material published with results from a routinely performed two-stage procedure.

In numerous works the results with the Harrington operation have been reported. Usually an approximate correction of 50 per cent has been obtained by the operation.

Stagnara(1967)obtained in 88 idiopathic cases an average correction of 40 per cent, Picard et al(1966) reported in 14 patients a correction of about 40 per cent, Michel and Jouvinroux(1967)reported a correction of about 50 per cent in 44 patients with idiopathic scoliosis. 50 per cent correction was also obtained by Scheier(1969)-using a preoperative cast, in 27 cases, by Gaubert(1971)in 92 patients with idiopathic scoliosis, and by Götze (1971) in 60 cases. Hall (1970) reported of 210 patients with idiopathic scoliosis, and obtained an average correction of 56 per cent in the thoracic curves, 54 per cent in the lumbar curves. Wilson and Levine (1971), in a series of 67 patients with idiopathic scoliosis, received the best correction (60%) in the thoraco-lumbar curves. Goldstein (1969, 1973), using preoperative localizer cast correction, reported a correction of 63 per cent in 80 patients with idiopathic scoliosis. Harrington and Dickson (1973) recently published a large series of patients in eight groups of treatment modifications. The correction in most groups was about 65 per cent, but only 53 per cent if the compression instrument was not used, the correction after 3 yars in this group was only 40 per cent.

The result obtained in the present material with an initial average correction of 58 per cent in the cases of idiopathic scoliosis is better than has been reported in most published materials. It was shown here, as in other works, that the smaller curve had a better correction in percentage. Judging from these reports, and from clinical experience, there seems to be a natural resistance against correction of the curvature of more than 50-60 per cent with those forces, about 40 kp, which can safely be used without laminar fractures. It is also natural that with straightening of the spine there is a gradually increasing distraction force required for each additional degree of correction. This was recently verified in an experimental model by Schultz and Hirsch(1973).

Most surgeons only use one distraction rod. In more severe curves Krol et al(1972)have used two parallel rods, and this was also described by Stagnara (1973). Wilson and Levine (1971) in the first part of their series used two rods, however, later they abandoned this technique.

In the present series, in 14 patients with double primary curves, 5 patients were operated with two distraction rods, one rod placed in each curve. The method of using two rods has been considered to increase the total force on the scoliosis and thereby the correction by distributing the force on four instead of two laminae. However, in this series, there was a smaller correction and a greater loss in those patients operated on with double rods compared to those operated with the "dollar-sign" method.

Harrington (1967), and also Cotrel (1966), stated that the fusion does not attain its full strength until about 18 months after the operation. However, most surgeons allow ambulation without a support before that time. The postoperative treatment most commonly used is a plaster cast (Risser type or a plaster jacket or localizer cast) for about 10 months, and ambulation is allowed after approximately 3 months (2 weeks-6 months) of recumbency. The correction loss during 1-2 years postoperatively with this type of postoperative treatment has usually been reported to average $5\text{-}10^{\circ}$: (Michel and Jouvinroux 1967: $7.5^{\circ}$ three years after operation, Stagnara 1967: $6^{\circ}$ one year after operation, Gaubert et al 1971: $6^{\circ}$ one year after operation, Wilson and Levine 1971: $5\text{-}10^{\circ}$ 2 years after operation).

Goldstein (1969), who treated patients with a localizer cast postoperatively, however, reported a loss of only approximately $3^{\circ}$ in 86 curves, not counting 4 cases with pseudoarthrosis in a follow-up of 1 1/2 to 5 years. Recently several reports have been published with favourable results of early ambulation (after 10-16 days) (Leider et al 1972, Hardy et al 1973), while Moe (1972) described early ambulation with a Milwaukee brace, but claimed a considerable loss of correction at ambulation. Harrington (1973) concluded that a lateral gutter fusion and an early mobilization in a plaster cast (after 1 month) seemed to give a very good result with little total loss.

In the present series, the Milwaukee brace was used postoperatively, and the patients were ambulated in this after three to five weeks of recumbency after the last operation (five to seven weeks after the first operation). The total loss (mean $5.8 \pm 5.0^{\circ}$) over two years in this material is in the lower range of the loss reported by other authors. This also applies to the total loss of $6.5^{\circ}$ in 37 patients with single idiopathic scoliosis followed-up for three years and $7.0^{\circ}$ in 14 patients followed-up for four years. The Milwaukee brace seems to be effective in holding the postoperative curve until the fusion attains strength.

Most of the loss is probably due to erosion at the hook sites. Erosion at the hook site is inevitable, but naturally depends upon the force of the instrument on the vertebrae. The Milwaukee brace has been shown by Nachemson and Elfström (1971) to be an effective load relieving device. In experiments with intravital force measurements in a Harrington rod, a lower value for the load on the rod was recorded with the patient wearing this brace than without the brace in all types of positions and activities. It was important that the brace was fitted with two side straps. It was also noted that about 10-11 days after the first operation the longitudinal forces on the distraction rod decreased to about 30 per cent, which therefore ought to be the minimum time of recumbency. Also Waugh (1966) noted a decrease in load with time.

Erosion at the hook sites was estimated by Harrington (1972) to give a loss of correction of 6-8$^{o}$ for every 1/16 inch of erosion. Harrington also emphasized, that an early loss of correction of 5-10$^{o}$ usually is due to rotation of the upper hook. Fielding and Waugh (1962) considered losses exceeding 8$^{o}$ to be due to pseudoarthrosis in patients treated after spinal fusion in a surcingle cast.

In the present material, there were three patients with large losses within the first postoperative year (Table 21 page 128). In one of these patients a loss of 11$^{o}$ occurred before ambulation, and was due to a laminar fracture. The two other patients had double primary curves with a considerable loss of correction during the first months after ambulation. Both these patients were operated with double rods and it is probable, that there occurred hook rotations causing the loss of correction. In these two patients there were no additional losses during the second and third follow-up years. In a fourth patient a loss of correction during the second postoperative year was due to a lengthening of the curve because of an inadequate fusion length.

There were no signs of pseudoarthrosis in these or any other patients in the series. As mentioned above, there were no patients with a late loss, which could be attributed to pseudoarthrosis.

Pseudoarthrosis always has been a great problem in patients operated with spine fusions. The rate of pseudoarthrosis has been reported to be as high as 68 per cent (Ponseti and Friedman 1950). With the Harrington rod the spine can be stabilized thereby decreasing the rate of pseudoarthrosis. Harrington (1967) thus reported of a 5 per cent rate of pseudoarthrosis in 500 cases. Goldstein (1969) also had a 5 per cent frequency of pseudoarthrosis but in a later report (1973) only one case of pseudoarthrosis in 129 patients. Götze (1971) had two cases of pseudoarthrosis in 60 patients operated upon. Leider et al (1972) reported a frequency of 4.7 per cent of pseudoarthrosis, and Hall (1970) had 3 cases of pseudoarthrosis in 210 cases, all in the lumbo-sacral region.

Different investigations have shown that for a good fusion it is important that the bone for grafting is autogenous and fresh. It should not have been exposed to air (Riska 1967). Moe and Gustilo (1964) found that the pseudoarthrosis frequency was higher if the patient was operated prior to the development of the iliac crest. For the best results, the fusion should include the transverse processes in the thoracic as well as in the lumbar region (Harrington 1973). In the present series, the iliac bone was immediately stored in saline before being laid down on the decorticated spine, which could be done only 10-15 minutes after it was removed from the iliac crest. The fusion included the transverse processes along the whole curve on both sides.

It is possible that the two-stage procedure offers a biological advantage with regard to healing of the fusion and hence avoidance of pseudoarthrosis. Siffert and Barash (1961) and Sako and Marchetta (1966) demonstrated the advantage in delayed transplantation technique in obtaining fracture healing.

The small correction loss in this material can probably also mechanically be attributed to the second stage operation. By a second intervention it is possible to correct changes of the hook positions, thereby improving the final result. Additional correction, usually 1 notch (6.3 mm) on the rod, can be obtained after allowing two weeks for relaxation of the tissue, which after this may not relax further at the same rate. Thus, it is probable that the final result has gained more than those $6^{o}$, which were obtained directly by the second stage operation. The correction in degrees obtained by the second stage operation was higher for the more severe curves above $70\text{-}80^{o}$. These patients, therefore, will gain the most with such a second intervention, while in the less severe curves a one-stage procedure will be sufficient to reduce the deformity to an acceptable degree. However, two operations necessarily lead to a prolonged operation time, increased blood loss and need for increased blood transfusion. In this series, the average time of operation for the two stages added together, 4 hours and 20 min. is more than what is generally reported in the literature. Furthermore, the total blood loss, 1920 cc, and the blood transfusions, 9 units or 3600 cc, were more than is reported in other works(Gaubert et al 1966, Goldstein 1966, 1969, Dolan and MacEwen 1971, Wilson and Levine 1971, Harrington 1973). The blood transfusion compensates the blood loss at operation as well as the bleeding into the drainages 24-48 hours postoperatively.

In spite of two operations the frequency of complications in the present material was lower than in any previously published material of surgically treated scoliosis. Wound infection rates have in several works been reported to be about 10 per cent (Götze 1971, Wilson and Levine 1971) while Goldstein reported 8 cases of deep infection in 295 patients. In the present material there were no deep wound infections in spite of a two-stage procedure. The high frequency of urinary infections can be attributed to a routine of in-dwelling catheterization in all patients, who cannot micturate 6 hours after the first or second stage operation. Routine urine cultures were made on all patients on several occasions during the postoperative course. These infections were treated according to the resistance pattern demonstrated by cultures, and there have been no late complications up to the follow-up date. All patients were free of infection when leaving the hospital.

In eight cases there were laminar fractures, most of these due to erroneous seating of the upper hook . Only in one case was this the cause of re-operation when it occurred after the second operation and only in this case was it the cause of loss of correction. This low incidence of laminar fracture is due to the use of a force-indicating distraction instrument (Nachemson and Elfström 1969). The average force used in the present series was 45.1 kp (30-50 kp).

The result of operation in 70 idiopathic patients expressed as correction in percentage, was shown to depend on a number of factors. The best prognostic sign was the "flexibility test", the correction in percentage of the original erect curvature when the patient lies down. This was very well correlated to the final result. With more than 30 per cent preoperative correction in the supine position, it is probable that the final curve correction will be more than 70 per cent. Compared to the bending test, which is as much a test of the energy of the examiner and of the ability of the patient to cooperate, this test under standardized conditions is objective.

The final correction also depended upon the number of vertebrae in the curve. As shorter curves were more difficult to correct, they should be operated comparatively early.

The Harrington factor - degrees/number of vertebrae - proved to be a very useful sign for the prognosis of the operation, as it was well correlated with the correction result.

As in the patients treated conservatively in a Milwaukee brace, the location of the scoliosis in these operated patients was of importance for the result of treatment. Distally located curves corrected better. The best correction, approximately 60 per cent, was obtained in the thoraco-lumbar curves.

The correction result did not depend on the age of the patient at the time of operation in this material, where no patients were operated under the age of 10 and very few were operated above the age of 20 years, and then only those with a flexible curve.

Rotation of the apical vertebra was highly correlated with the original curve degree. The rotation correlated better with the correction of the curvature after the operations than did the original curvature. It is important to take the rotation of the vertebrae into consideration, when the indication for operation and a prognosis are to be made.

A decrease of the wedge deformation of the apical vertebral body was noticed. The relationship between vertebral growth and scoliotic deformity can be explained by the Hueter-Volkmann's epiphyseal pressure rule. It states that the bone growth is retarded when pressure is diminished. When the scoliosis is distracted on the concave side, the pressure is inversed or at least equalized on the concave and convex sides of the vertebral end plates, as demonstrated by the improvement of the wedge shape of the disc. The growing bone then corrects the deformity.

In the present material there was a considerable increase in height, on the average 4 cm, by the operation. In the patients aged 10 to 14 years, there was also an additional postoperative growth, which was smaller than the growth of normal individuals in these age groups. There was no large difference between the total increase in height of the operated patients and the height increase in normal controls. It can be concluded from this study, that even in young individuals, 10 to 12 years of age, the correction and fusion of the scoliotic curve causes only a loss of a few centimeters in height when compared to normal growth. The operation needs not be postponed for such reasons as loss of height. Retarded postoperative growth of the fused segments is no contra-indication for surgery.

Correction of the compensatory curves, and particularly the lower compensatory curve, follow quite closely the correction of the major curve, even with quite notable rotation in the low compensatory curve. Hence this curve seems to have a considerable corrective ability even if it is structurally changed. Lumbar curves, with rotation below $15\text{-}20^{o}$ (corresponding to a pedicle-vertebral ratio of approximately 0.25-0.30) should not require a fusion. In doubtful cases a flexibility test should be performed. These findings are in accordance with the opinion of Moe (1958), who described an often surprising degree of flexibility in the lower compensatory curve in bending tests, even if these curves show clinical evidence of being structural.

A stepwise multiple regression analysis of six different factors of importance for the correction was performed. The "steps" are shown in Table 23, where for each additional factor, the final result can be predicted with greater accuracy.

TABLE 23. Stepwise multiple regression analysis (six factors studied).

| Factors in order of importance for the percentage correction | Explained % of variations in percentage correction | Mult. regr. coeff. |
|---|---|---|
| 1. Disc wedge ratio | 16 | 0.40 |
| 2. Rotation degrees | 20 | 0.45 |
| 3. Apex level | 22 | 0.47 |
| 4. Degrees/no of vertebrae | 32 | 0.57 |
| 5. Vertebral wedge | 34 | 0.58 |
| 6. Skeletal age | 35 | 0.59 |

Thus, a considerable part of the restriction against correction seems to be located in the disc, the more severe the disc wedge, the less correction obtained. A trend towards this was also seen in the conservatively treated material. When the disc wedge and the vertebral wedge were measured in degrees, they corresponded very well to each other indicating that the curve angle was equally distributed in the discs and the vertebral bodies.

Perey (1963) and later Kazmin et al (1969) demonstrated by discography, that the nucleus pulposus in severe scoliosis is in an eccentric position in the intervertebral disc. It is possible that in scoliosis with a severe disc wedge, this eccentric position of the nucleus prevents an effective correction of the scoliosis, as Kazmin et al (1969) also suggested.

If also the correction in percentage in the supine position is included with the factors in Table 23, the variations in postoperative correction can be explained in nearly 50 per cent. Thus, it is theoretically possible to predict the final result with an accuracy of almost 50 per cent by studying clinical factors preoperatively. The additional variations can partly be explained by the rigidity of the tissues as these structures resist the distraction. In the experimental work reported by the author, it was shown that the tendinous structures were of importance for the result at operation. Variations in the tensile stiffness of the tendons could explain about 30 per cent of the variations in the result of treatment.

CHAPTER THIRTEEN

## SUMMARY

Many different methods have been applied in the treatment of scoliosis. The present study is a retrospective analysis of the results of conservative and operative treatment.

During the period March 1967 to April 1971 treatment with the Milwaukee brace was started in 159 patients with scoliosis, all followed for two years or more. 86 of these have ended the full treatment program and have been followed-up for one year. During the same period 100 patients were operated with the Harrington distraction rod and fusion in two stages. These patients were treated post-operatively without external support in the recumbent position for 3-5 weeks, ambulated with the Milwaukee brace worn for 6 months, and then gradually weaned from the brace for another 6-9 months. The patients were all followed-up for two years after the operation, 55 of them were followed-up for three years and 23 for four years.

For the patients with idiopathic scoliosis a multifactorial analysis of the factors of importance for the conservative and operative treatment result was performed. The following factors were studied:

Age at the diagnosis of the deformity.
Age at the start of treatment.
Development of the iliac apophysis at the start of treatment.
Original curvature.
Number of vertebrae in the curve.
Degrees/number of vertebrae in the curve.
Curve location.
Rotation of the apical vertebra.
Wedge of the apical vertebral body.
Wedge of the apical intervertebral disc.
Correction of the compensatory curves.
Height of the patients.

All patients with curves of 15-50$^{o}$ irrespective of etiology and location were fitted with the Milwaukee brace on an in-patient basis and were subsequently seen on regular controls every third to sixth month. The mean age of the patients was 14 years and 7 months at the start of treatment. The mean brace wearing time was 23.4 months, during which time the average sitting height increased 2.9 ±2.0 cm. The mean curvature at the start of treatment was 39 ±9$^{o}$ in the thoracic region and 33 ±11$^{o}$ in the thoraco-lumbar and lumbar regions. In the idiopathic cases the average correction of all curves at the end of brace treatment was approximately 10 per cent, however, at the follow-up one year later the correction was only 4 per cent, a non-significant improvement when compared with the original curvature.

The curve location was of importance for the result. At the end of the brace treatment the lumbar curves corrected more than 30 per cent, while the thoracic curves did not correct much at all. At the follow-up the curves in the thoracic region had deteriorated very little compared with the original curvature, to 42±10$^{o}$, but in the thoraco-lumbar and lumbar regions the curves improved to 29 ±10$^{o}$.

The original curvature, up to 50$^{o}$, was not significantly correlated with the result at the end of brace treatment. The initial correction of the scoliosis immediately after fitting of the brace was about 20 per cent, and was highly correlated with the result at the end of brace treatment. This initial correction was more valuable as a prognostic sign than the correction of the scoliosis in the supine position, and could serve as a valuable guide to the further treatment program.

The progressive nature of the disease was shown by the fact that the patients in whom the brace treatment was started at an early age - 11-12 years - corrected less than those, who started treatment at an older age. Those, who were near cessation of growth when the treatment was started, however, also corrected less well. The brace had a slight derotating effect on the apical vertebra while used, but the follow-up showed that the rotation was not improved by the brace treatment. The wedge of the vertebral body was not significantly changed by the brace treatment. There was a tendency towards an increase in the vertebral wedge in the younger patients. The correction of the lower compensatory curve was well correlated with the correction of the major curve, but corrected 10-20 per cent less than that curve. The upper compensatory curve often increased in spite of correction of the major curve.

The complications involved with the Milwaukee brace treatment were few and mild. There were two patients with orthodontic complications, which resolved after a change of the brace and after use of occlusion stabilizing devices.

Of the 159 patients seen for two years or more, there were 5 patients, who dropped out of the treatment completely, and 10 patients, who refused to wear the brace according to the full program. Only one of these patients was operated on. 10 of these drop-out patients had reached skeletal maturity at the follow-up. There was a significant deterioration of the scoliosis in these patients compared with those, who completed the full treatment.

In the operated series of 100 patients the mean original curvature was $73 \pm 17^{\circ}$. The mean age at the operation was 15 years. There was an average correction after the second operation of $55 \pm 11$ per cent. The best correction was obtained in the 72 patients with idiopathic scoliosis, 58 per cent.

In the second operation an average correction of about $6^{\circ}$ was obtained, the correction in degrees was larger for the more severe curves, and was well correlated with the total correction after both operations.

The average total loss of correction from the first postoperative supine roentgenogram to the erect roentgenogram of the two-year follow-up was approximately $6^{\circ}$, and the correction maintained was 50 per cent. At the follow-up examinations three and four years after the operations, there was practically no additional loss, - $0.1^{\circ}$ and $0.7^{\circ}$ respectively. (In the idiopathic group $0.7^{\circ}$ and $1.2^{\circ}$ respectively.) The loss of correction was approximately the same in all types of curves, and was neither correlated to the correction result, nor to the original curvature. There was one patient with a loss exceeding $10^{\circ}$ initially at the ambulation, the loss caused by a laminar fracture. Only two patients had a loss exceeding $10^{\circ}$ during the next two years after ambulation. Both were double primary curves operated with two rods, one correcting each curve. In one patient a loss of $5^{\circ}$ a year was due to a lengthening of the curve because of too short a fusion. In five patients with double primary curves operated with two Harrington distraction rods - one correcting each curve - the correction was smaller, and the loss of correction larger than in nine patients operated with the "dollar-sign" method. There was no case of pseudoarthrosis.

The complications with operative treatment were mild. No mortal complications and no deep infections were experienced in these 100 patients. The most serious complication occurred in one patient, who postoperatively developed fecal and urinary incontinence, probably due to a distention of the sacral nerves. She recovered, however, completely during the first postoperative months without further measures.

In a separate analysis of 70 cases with single idiopathic scoliosis, the following was found:
The age of the patients at the time operation (mean 15 years and 2 months) was not of any significant importance for the result. No patients above 26 years of age were operated on. The severity of the curve was of importance, as also has been reported by other authors, the more severe curves corrected to about 50 per cent, while in curves below $80^{\circ}$ the correction averaged 60 per cent.

The correction of the curvature in the supine position was well correlated to the postoperative result. Also well correlated to this result was the "Harrington factor", degrees/numbers of vertebrae which correlated better with the result after the operations than either the preoperative curve or the number of vertebrae in the curve.

The rotation of the vertebrae was better correlated with the final result than the original curvature, the more rotated curves correcting less. The operation had a small but significant derotating effect on the apical vertebra of the spine of about 5 degrees.

The vertebral wedge of the apical vertebra was significantly improved in the follow-up one year after the operations and did not change significantly during the second year after the operations. The disc wedge influenced the correction result, it was in particular with a wedge ratio below 0.4 that correction of the scoliosis was more difficult. The disc wedge was reversed at the operations, but very soon relapsed.

The non-operated compensatory curves were corrected with the major curve, however, usually 10-20 per cent less than that curve. The lower compensatory curve corrected better than the upper curve. In the follow-up 2 years after the operations the compensatory curves had not changed significantly. The lower compensatory curves, even with some rotation up to about $20^{o}$, should not need to be fused.

By the operations, the patients increased considerably in sitting height. This increase in height together with the additional postoperative growth of the non-fused part of the spine was in younger patients, 10-12 years of age, only few centimeters less than the normal expected remaining growth in these age groups. In patients above 13 years of age the total increase in height was larger than the expected remaining growth in normal controls.

It can be concluded that treatment in the Milwaukee brace in growing patients with idiopathic curves of 15-50$^{\circ}$ is effective in preventing further deterioration and is practically free of complications. The greatest problem is to make the patient complete the full brace treatment routine. Important factors for accomplishing this is to produce a good Milwaukee brace, fitting it on an in-patient basis, and to keep a good emotional contact with the patient at regular controls.

Operative treatment in two stages with the Harrington distraction rod in curves exceeding 50$^{\circ}$ can be expected to result in a permanent correction of about 50 per cent of the initial curvature. By a second stage operation, an additional correction of about 6$^{\circ}$ can be reached. It is probable that the second stage operation also partly is responsible for the low incidence of pseudoarthrosis and the low long-term postoperative loss of correction. The Milwaukee brace is effective in the postoperative treatment of these patients, and safely allows early postoperative ambulation.

## REFERENCES


ABRAHAMS, M.: The mechanical behaviour of tendon in vitro. A preliminary report. Med. biol. Engng 5:433, 1967.

ALEXANDER, M.A., BUNCH, W.H. & EBBESSON, S.O.E.: Can experimental dorsal rhizotomy produce scoliosis? J. Bone Jt Surg. 54-A: 1509, 1972.

AMATO, V.P. & BOMBELLI, R.: Early skeletal and vascular changes in rats, fed on sweet pea (Lathyrus odoratus) seeds. J. Bone Jt Surg. 41-B: 600, 1959.

ANDERS, J.M.: Chromosome studies in scoliosis. In: Proceedings of a symposium on scoliosis, held in ... London 1965. Ed. by P.A. Zorab. London 1965, p. 32.

APLEY, A.G.: Transthoracic approach in the treatment of scoliosis. Proc. roy. Soc. Med. 54:281, 1961.

ARKIN, A.M., PACK, G.T., RANSOHOFF, N.S. & SIMON, N.: Radiation-induced scoliosis, a case report. J. Bone Jt Surg. 32-A: 401, 1950.

ARKIN, A.M.: Correction of structural changes in scoliosis by corrective plaster jackets and prolonged recumbency. J. Bone Jt Surg. 46-A: 33, 1964.

ASBOE-HANSEN, G.: The hormonal control of connective tissue. Int. rev. connect. Tissue 1:29, 1963.

BADGER, V.M.: Correlation studies on muscle in scoliosis: histochemistry, electromyography, electronmicroscopy, and quantitative enzymes. J. Bone Jt Surg. 51-A: 204, 1969.

BALABA, T.Y.: Some biochemical aspects of scoliosis and their pathogenetic significance. Reconstr. Surg. Traum. 13: 191, 1972.

BARASH, H.L. & DEWALD, R.: Milwaukee-brace treatment for idiopathic scoliosis. J. Bone Jt Surg. 53-A: 196, 1971.

BEALS, R.K.: Homocystinuria. A report of two cases and review of the literature. J. Bone Jt Surg. 51-A: 1564, 1969.

BEIGHTON, P.H. & HORAN, F.: Orthopaedic aspects of the Ehlers-Danlos syndrome. J. Bone Jt Surg. 51-B: 444, 1969.

BEIGHTON, P.H.: Articular manifestations of the Ehlers-Danlos syndrome. Semin. Arthr. and Rheum. 1: 246, 1971.

BENSON, P.: Hydroxyproline excretion in scoliosis. In: Proceedings of a symposium on scoliosis, held in ... London 1965. Ed. by P.A. Zorab. London 1965, p. 47.

BERGMAN, I. & LOXLEY, R.: Two improved and simplified methods for the spectrophotometric determination of hydroxyproline. Analyt. Chem. 35: 1961, 1963.

BISGARD, J.D. & MUSSELMAN, M.M.: Scoliosis, its experimental production and growth correction; growth and fusion of vertebral bodies. Surg. Gynec. Obstet. 70: 1029, 1940.

BJORKSTEN, J.: The crosslinkage theory of aging. J. Amer. Geriat. Soc. 16: 408, 1968.

BJURE, J., GRIMBY, G. & NACHEMSON, A.: Correction of body height in predicting spirometric values in scoliotic patients. Scand. J. clin. Lab. Invest. 21: 19, 1968.

BLECKMANN, H., KRESSE, H., WOLLENSAK, J. & BUDDECKE, E.: Glykosaminoglykan- und Kollagenanalysen bei Osteogenesis imperfecta. Z. Kinderheilk. 110: 74, 1971.

BLOUNT, W.P., SCHMIDT, A.C., KEEVER, E.D. & LEONARD, E.T.: The Milwaukee brace in the operative treatment of scoliosis. J. Bone Jt Surg. 40-A: 511, 1958 a.

BLOUNT, W.P., SCHMIDT, A.C. & BIDWELL, R.G.: Making the Milwaukee brace. J. Bone Jt Surg. 40-A: 526, 1958 b.

BLOUNT, W.P.: The Milwaukee brace in non-operative scoliosis treatment. Acta orthop. scand. 33: 398, 1963.

BLOUNT, W.P.: The Milwaukee brace in the treatment of the young child with scoliosis. Arch. orthop. Unfall-Chir. 56: 363, 1964.

BLOUNT, W.P. & BOLINSKE, J.: Physical therapy in the nonoperative treatment of scoliosis. Phys. Ther. (Rev.), 47: 919, 1967.

BLOUNT, W.P., & MOE, J.H.: The Milwaukee brace. Baltimore 1973.

BOUILLET, R. & VINCENT, A.: La scoliose idiopathique. Acta orthop. belgica, 33: 93, 1967.

BOURY, G.: Cited in Bouillet R. & Vincent A. Acta orthop. belg. 33: 112, 1967.

BREITENFELDER, J.: Die konservative Behandlung der Skoliose mit dem Milwaukee-korsett. Z. Orthop. 109: 690, 1971.

BRENTON, D.P. et al.: Homocystinuria and Marfan's syndrome. A comparison. J. Bone Jt Surg. 54-B: 277, 1972.

BROOKS, L. et al.: The epidemiology of scoliosis. A prospective study. J. Bone Jt Surg. 55-A: 436, 1973.

BUTTERWORTH, T.R. Jr. & JAMES, C.: Electromyographic studies in idiopathic scoliosis. Sth. med. J. (Ala) 62: 1008, 1969.

BÖHMER, D.: Enzymuntersuchungen bei jugendlichen Patienten mit Skoliosen. Arch. orthop. Unfall-Chir., 58: 313, 1965.

BÖHMER, D. & NOLTE, B.: Biochemischer Beitrag zur Ätiologie der Skoliose. Z. Orthop. 110: 137, 1972.

CALVO, I.J.: Observations on the growth of the female adolescent spine and its relation to scoliosis. Clin. Orthop. 10: 40, 1957.

CARTER, C. & WILKINSON, J.: Persistent joint laxity and congenital dislocation of the hip. J. Bone Jt Surg. 46-B: 40, 1964.

COBB, J.R.: Outline for the study on scoliosis. Instructional course lect. 5: 261, 1948.

COBB, J.R.: The problem of the primary curve. J. Bone Jt Surg. 42-A: 1413, 1960.

COCHRAN, G.V.B. & WAUGH, T.R.: The external forces in correction of idiopathic scoliosis. J. Bone Jt Surg. 51-A: 201, 1969.

COLLIS, D.K. & PONSETI, I.V.: Long-term follow-up of patients with idiopathic scoliosis not treated surgically. J. Bone Jt Surg. 51-A: 425, 1969.

CONEN, P.E.: Histological study of tissues in scoliotic patients. J. Bone Jt Surg. 53-A: 199, 1971.

COSSETTE, J.W., FARFAN, H.F., ROBERTSON, G.H. & WELLS, R.V.: The instantaneous center of rotation of the third lumbar intervertebral joint. J. Biomech. 4: 149, 1971.

COTREL, Y. & MOREL, G.: La technique de l'E.D.F. dans la correction des scolioses. Rev. Chir. orthop. 50: 59, 1964.

COTREL, Y.: The end results in the treatment of idiopathic scoliosis. Excerpta med. (Amst.), ICS 116: E 77, 1966.

COULSON, W.F., WEISSMAN, N. & CARNES, W.H.: Cardiovascular studies on copper-deficient swine. VII. Mechanical properties of aortic and dermal collagen. Lab. Invest. 14: 303, 1965.

COWELL, H.R., HALL, J.N. & MACEWEN, G.D.: Genetic aspects of idiopathic scoliosis. Clin. Orthop. 86: 121, 1972.

CRONKITE, A.E.: The tensile strength of human tendons. Anat. Rec. 64: 173, 1936.

COX, R.W. & GRANT, R.A.: The structure of the collagen fibril. Clin. Orthop. 67: 172, 1969.

DAVIDSSON, L.: Tensile strength, rupture and regeneration of tendons. Ann. Chir. Gynaec. Fenn. 43, Suppl. 5: 61, 1954.

DE GEORGE, F.V. & FISCHER, R.L.: Idiopathic scoliosis: genetic and environmental aspects. J. med. Genet. 4: 251, 1967.

DENT, C.E.: Scoliotic speculations. In: Proceedings of a second symposium on scoliosis: causation, held in ... London 1967. Ed. by P.A. Zorab. Edinburgh & London 1968, p. 58.

DEWALD, R. & RAY, R.D.: Skeletal traction for the treatment of severe scoliosis. J. Bone Jt Surg. 52-A: 233, 1970.

DOLAN, J.A. & MACEWEN, G.D.: Surgical treatment of scoliosis. Clin. Orthop. 76: 125, 1971.

DUVAL-BEAUPÈRE, G.: Pathogenic relationship between scoliosis and growth. In: Scoliosis and growth; Proceedings of a third symposium, held in ... London 1970. Ed. by P.A. Zorab. Edinburgh & London 1971, p. 58.

DWYER, A.F., NEWTON, N.C. & SHERWOOD, A.A.: An anterior approach to scoliosis. A preliminary report. Clin. Orthop. 62: 192 1969.

EDMONSON, A.S.: Evaluation of Milwaukee brace treatment in idiopathic scoliosis. J. Bone Jt Surg. 51-A: 202, 1969.

EDMONSON, A.S. & MORRIS, J.T.: Follow up study of Milwaukee brace treatment in patients with idiopathic scoliosis. J. Bone Jt Surg. 55-A: 439, 1973.

ELFSTRÖM, G.: Intravital wireless telemetry of forces during and after surgical correction of scoliosis. Chalmers University of Technology. Technical report no. 26 1972.

ELLIOTT, D.H.: Structure and function of mammalian tendon. Biol. Rev. 40: 392, 1965.

ELLIS, D.E.: Cross sectional area measurements for tendon specimens: A comparison of several methods. J. Biomech. 2: 175, 1969.

ENNEKING, W.F. & HARRINGTON, P.: Pathological changes in scoliosis. J. Bone Jt Surg. 51-A: 165, 1969.

FAIT, M. & JANOVEC, M.: Establishing of the rotation angle in the vertebra. Scripta medica, Fac. Med. Brun. 43: 207, 1970.

FARKAS, A.: The pathogenesis of idiopathic scoliosis. J. Bone Jt Surg. 36-A: 617, 1954.

FIELDING, J.W. & WAUGH, T.: Postoperative correction of scoliosis. J. Amer. med. Ass. 182: 541, 1962.

FRANCHI, F., IGNESTI, C., DONNINI, L. & SIMONETTI, E.: Attività antielastica serica nella scolios giovanile essenziale. Reumatismo 15: 30, 1963.

FREDERICH, A., CREYSSEL, R., GILLY, R. & GOLDBLATT, B.: Les anomalies métaboliques observées dans la scoliose essentielle de l'enfant. Lyon méd. 209: 97, 1963.

FRISÉN, M., MÄGI, M., SONNERUP, L. & VIIDIK, A.: Rheological analysis of soft collagenous tissue. P.I. Theoretical considerations. J. Biomech. 2: 13, 1969.

FRY, P., HARKNESS, M.L.R., HARKNESS, R.D. & NIGHTINGALE, M.: Mechanical properties of tissues of lathyritic animals. J. Physiol. (Lond.) 164: 77, 1962.

FUNG, Y.C.B.: Elasticity of soft tissues in simple elongation. Amer. J. Physiol. 213: 1532, 1967.

GALANTE, J.O.: Tensile properties of the human lumbar annulus fibrosus. Acta orthop. scand. Suppl. 100, 1967.

GALANTE, J., SCHULTZ, A., DEWALD, R.L. & RAY, R.D.: Forces acting in the Milwaukee brace on patients undergoing treatment for idiopathic scoliosis. J. Bone Jt Surg. 52-A: 498, 1970.

GALLOP, P.M.: 3, 2, 1: A, B, C, sub-unit hypothesis for the alfa chains of tropocollagen. Nature 209: 73, 1966.

GARRET, A.L., PERRY, J. & NICKEL, U.L.: Paralytic scoliosis. Clin. Orthop. 21: 117, 1961.

GAUBERT, J. et al.: Evolution du traitement chirurgical de la scoliose idiopathique. Acta orthop. belg. 33: 713, 1967.

GAUBERT, J. et al.: L'opération de Harrington. Difficultés, indications, résultats. J. Chir. (Paris) 101: 405, 1971.

GLAUBER, A., FERNBACH, J., MASSANYY, L. & MEDGYESI, G.: Protein metabolism in idiopathic scoliosis. J. Bone Jt Surg. 44-A: 1553, 1962.

GOLDSTEIN, L.A.: Surgical management of scoliosis. J. Bone Jt Surg. 48-A: 167, 1966.

- Treatment of idiopathic scoliosis by Harrington instrumentation and fusion with fresh autogenous iliac bone crafts. Results in eighty patients. J. Bone Jt Surg. 51-A: 209, 1969.

- The surgical management of scoliosis. Clin. Orthop. 77: 32, 1971.

- The surgical treatment of idiopathic scoliosis. Clin. Orthop. 93: 95, 1973.

GRATZ, C.M.: Tensile strength and elasticity tests on human fascia lata. J. Bone Jt Surg. 13: 334, 1931.

GREULICH, W.W. & PYLE, S.I.: Radiographic atlas of skeletal development of the hand and wrist. 2 ed. Calif. 1959.

GROSS, J., HIGHBERGER, J.H. & SCHMITT, F.O.: Collagen structures considered as states of aggregation of a genetic unit. The tropocollagen particle. Proc. nat. Acad. Sci. (Wash.) 40: 679, 1954.

GRUCA, A.: The pathogenesis and treatment of idiopathic scoliosis; a preliminary report. J.Bone Jt Surg. 40-A:570, 1958

- Surgical treatment of scoliosis in children. In: Operative treatment of scoliosis. Ed. by G. Chapchal. Stuttgart 1973, p.108.

GÖTZE, H.G., IMMENKAMP, M. & MATTHIASS, H.H.: Die operative Behandlung der Skoliose mit dem Harrington-Instrumentarium. Z. Orthop. 109: 573, 1971.

HALL, J.E.: Idiopathic scoliosis, indications for treatment, and follow-up study of 210 cases treated by Harrington instrumentation. Annual post-graduate course on the management and care of the scoliosis patient. Ed. H.A. Keim, 2: 23, 1970.

HARDY, J.H., TAKAHASHI, R.M. & PETERSON, C.A.: Results of early ambulation following scoliosis spine fusion and Harrington rodding. J. Bone Jt Surg. 55-A: 437, 1973.

HARKNESS, M.L.R. & HARKNESS, R.D.: Changes in the physical properties of the uterine cervix of the rat during pregnancy. J. Physiol. (Lond.) 148 : 524, 1959.

HARRINGTON, P.: Surgical instrumentation for management of scoliosis. J. Bone Jt Surg. 42-A: 1448, 1960.

- Treatment of scoliosis. Correction and internal fixation by spine instrumentation. J. Bone Jt Surg. 44-A: 591, 1962.

- Instrumentation in structural scoliosis. In: Modern trends in orthopaedics. Ed. by W.D. Graham, London 1967, p. 95.

- Technical details in relation to the successful use of instrumentation in scoliosis. Orthop. Clinics Amer. 3: 1, 49, 1972.

HARRINGTON, P.R. & DICKSON, J.H.: An eleven year clinical investigation of Harrington instrumentation: a preliminary report on 578 cases. Clin. Orthop. 93: 82, 1973.

HARRIS, E.D. & SJOERDSMA, A.: Collagen profile in various clinical conditions. Lancet II, 707, 1966.

HAUT, R.C. & LITTLE, R.W.: A constitutive equation for collagen fibres. J. Biomech. 5: 423, 1972.

HEIMENDINGER, J.: Die Ergebnisse von Körpermessungen an 5000 Basler Kindern von 2-18 Jahren. Helv. paediat. Acta, Suppl. 13, 1964.

HENSINGER, R.N. & MACEWEN, G.D.: Evaluation of Cotrel dynamic spine traction in the treatment of scoliosis: a preliminary report. J. Bone Jt Surg. 55-A: 438, 1973.

HERTEL, E. & WÜRFEL, J.: Die Operation der Skoliose und ihre Ergebnisse. Z. Orthop. 106: 85, 1969.

HIBBS, R.A.: A report of fifty-nine cases of scoliosis treated by the fusion operation. J. Bone Jt Surg. 22:3, 1924.

HIGHBERGER, J.H.: The structural stability of the collagen fiber in relation to the mechanism of tanning. J. Amer. Leath. Chem. Ass. 42: 493, 1947.

HIRANO, S.: Electron microscopic studies on back muscles in scoliosis. J. Jap. Orthop. Ass. 46: 47, 1972.

HIRSCH, C. & GALANTE, J.: Laboratory conditions for tensile tests in annulus fibrosus from human intervertebral discs. Acta orthop. scand. 38: 148, 1967.

HIRSCH, G.: Tensile properties during tendon healing. In press 1973.

HODGSON, A.R.: Correction of fixed spinal curves. J. Bone Jt Surg. 47-A: 1221, 1965.

HOPPENFELD, S. & SPIRO, A.: Histochemical findings in the paraspinal muscles of patients with idiopathic scoliosis. J. Bone Jt Surg. 55-A: 435, 1973.

HORAL, J., NACHEMSON, A. & SCHELLER, S.: Clinical and radiological long-term follow-up of vertebral fractures in children. Acta orthop. scand. 43: 491, 1972.

JAMES, J.I.P.: Scoliosis. Edinburgh & London 1967.

- The etiology of scoliosis. J. Bone Jt Surg. 52-B: 410, 1970.

KANE, W.J. & MOE, J.H.: A scoliosis-prevalence survey in Minnesota. Clin. Orthop. 69: 216, 1970.

KARAHARJU, E.O.: Deformation of vertebrae in experimental scoliosis. Acta orthop scand. Suppl. 105, 1967.

KARLBERG, P. et al.: In preparation. 1972.

KATZMAN, H., WAUGH, T. & BERDON, W.: Skeletal changes following irradiation of childhood tumors. J. Bone Jt Surg. 51-A: 825, 1969.

KAZMIN, A.N.: [Cuneiform resection of the spine in severe forms of scoliosis.] Acta Chir. orthop. Traum. cech. 29: 368, 1962.

KAZMIN, A.I., FISCHENKO, V.J. & PLOTNIKOVA, J.J.: Surgical treatment of the scoliotic patient by discotomy. Ortop. Travm. Protez. (Moskow), 30, 4: 34, 1969.

KAZMIN, A.I. & MERKURIEVA, R.V.: Biochemical study of the level of aminopolyglycans sialo- and glycoproteids in the spinal tissues of scoliotic patients. Ortop. Travm. Protez. 30, 10: 20, 1969.

KAZMIN, A.I. & MERKURIEVA, R.V.: Role of disturbances of glucosaminoglycane metabolism in the pathogenesis of scoliosis. Ortop. Travm. Protez. 32: 87, 1971.

KIRK, J.A., ANSELL, B.M. & BYWATERS, E.G.L.: The hypermobility syndrome. Musculoskeletal complaints associated with generalized joint hypermobility. Ann. rheum. Dis. 26: 419, 1967.

KLAPP, B.: Das Klapp'sche Kriechverfahren. Stuttgart, 6. überarb. Aufl. 1966.

KNUTSSON, F.: Vertebral genesis of idiopathic scoliosis in children. Acta Radiol. Diagn. N.S. 4: 395, 1966.

KROL, J., POLAKOWSKI, L., LEMPICKI, A. & GARTSKA, J.: Remarks on treatment of scoliosis with Harrington instrumentation. Amer. Dig. 1972, 2: 109.

KYSELKA, R.: Biochemische Vorgänge in Zusammanhang mit der progredienten Skoliose. Beitr. Orthop. Traum. 12: 130, 1965.

KYSELKA, R., SCHRAMM, G. & BECK, O.: Quantitative und qualitative Veränderungen saurer Mukopolsacharide im Urin bei Erkrankungen des Skelettsystems. Beitr. Orthop. Traum. 16: 552, 1969.

LANGENSKIÖLD, A. & MICHELSSON, J.-E.: The pathogenesis of experimental progressive scoliosis. Acta orthop. scand. Suppl. 59, 1962.

LEIDER, L., MOE, J.H. & WINTER, R.B.: Early ambulation after the surgical treatment of idiopathic scoliosis. J. Bone Jt Surg. 54-A: 1792, 1972.

LEVENE, C.I. & GROSS, J.: Alterations in state of molecular aggregation of collagen induced in chick embryos by betaaminoproprionitrile (lathyrus factor). J. exp. Med. 110: 771, 1959.

LINDAHL, O. & RAEDER, E.: Mechanical analysis of forces involved in idiopathic scoliosis. Acta orthop. scand. 32: 27, 1962.

LINDAHL, O.: Resection of vertebral transverse processes in idiopathic scoliosis. Acta orthop. scand. 37: 342, 1966.

LISZKA, O.: Spinal cord mechanisms leading to scoliosis in animal experiments. Acta med. pol. 2: 45, 1961.

LUCAS, D.B. & BRESLER, B.: Stability of the ligamentum spine. University of California Biomechanics laboratory, San Francisco. Technical report 40, 1961.

LUCAS, D.B.: Mechanics of the spine. Bull.Hosp.Joint Dis. 31:115, 1970

MACEWEN, G.D.: Experimental scoliosis. In: Proceedings of a second symposium on scoliosis: causation, held in ... London 1967. Ed. by P.A. Zorab. London 1968, p. 18.

MACEWEN, G.D. & COWELL, H.R.: Familial incidence of idiopathic scoliosis and its implications in patient treatment. J. Bone Jt Surg. 52-A: 405, 1970.

MACEWEN, G.D.: Factors affecting the growth of the vertebral bodies and intervertebral discs. In: Scoliosis and growth. Proceedings of the third symposium, held in ... London 1970. Ed. by P.A. Zorab. Edinburgh & London 1970, p. 40.

MACEWEN, G.D. & KIRSCH, P.: The structural response of the idiopathic curve to the Milwaukee brace. J. Bone Jt Surg. 53-A: 196, 1971.

MC KUSICK, V.A.: Heritable disorders of connective tissue. S:t Louis 1966. 3 ed.

MAROUDAS, A.: Physicochemical properties of cartilage in the light of ion exchange theory. Biophys. J. 8: 575, 1968.

MAROUDAS, A., MUIR, H. & WINGHAM, J.: The correlation of fixed negative charge with glycosaminoglycan content of human articular cartilage. Biochim. biophys. acta 177: 492, 1969.

MAROUDAS, A.: Personal communication. 1973.

MATHEWS, M.B.: The intersection of collagen and acid mucopolysaccharides. A model for connective tissue. Biochem. J. 96: 710, 1965.

MATTHEWS, L.S. & ELLIS, D.: Viscoelastic properties of cat tendon: effects of time after death and preservation by freezing. J. Biomech. 1: 65, 1968.

MICHEL, C.R. & JOUVINROUX, P.: Analyse d'une statistique de 164 scolioses operées suivant la technique de Harrington depuis avril 1963. Acta orthop. belg. 33: 631, 1967.

MICHEL, C. & ONIMUS, M.: L'opération de Harrington dans le traitement chirurgical des scolioses. Rev. Chir. orthop. 56: 703, 1970.

MICHELSSON, J.-E.: The development of spinal deformity in experimental scoliosis. Acta orthop scand. Suppl. 81, 1965.

MINEIRO, J.: Scoliosis. British Orthop. Travelling Club Scientific meetings, Portugal. Proceedings. J. Bone Jt Surg. 51-B: 574, 1969.

MOE, J.H.: The management of idiopathic scoliosis. Clin. Orthop. 9: 169, 1957.

- A critical analysis of methods of fusion for scoliosis; an evaluation of two hundred and sixty-six patients. J. Bone Jt Surg. 40-A: 529, 1958.

- Changing concepts of the scoliosis problem. J. Bone Jt Surg. 43-A: 471, 1961.

MOE, J.H. & GUSTILO, R.B.: Treatment of scoliosis. J. Bone Jt Surg. 46-A: 293, 1964.

MOE, J.H. & KETTLESON, D.H.: Idiopathic scoliosis. Analysis of curve patterns and the preliminary results of Milwaukee brace treatment in one hundred sixty-nine patients. J. Bone Jt Surg. 52-A: 1509, 1970.

MOE, J.H.: Methods of correction and surgical techniques in scoliosis. Orthop. Clinics of N. Amer. 3, 1: 17, 1972.

MORGAN, F.R.: The mechanical properties of collagen fibres. Stress-strain curves. J. Soc. Leath. Trades' Chem. 44: 170, 1960.

MORSCHER, E.: Möglichkeiten und Grenzen der Methode nach Harrington in der operativen Skoliosebehandlung. Arch. orthop. Unfall-Chir. 70: 136, 1971.

MOVSHOVICH, I.A.: [On the problem of the pathogenesis of scoliosis.] Ortop. Travm. Protez. 21: 23, 1960.

NACHEMSON, A.: A long term follow-up study of non-treated scoliosis. Acta orthop. scand. 39: 466, 1968.

NACHEMSON, A.L. & EVANS, J.H.: Some mechanical properties of the third human lumbar interlaminar ligament (ligamentum flavum). J. Biomech. 1: 211, 1968.

NACHEMSON, A. & ELFSTRÖM, G.: A force-indicating distractor for the Harrington rod procedure. J. Bone Jt Surg. 51-A: 1660, 1969.

NACHEMSON, A. & ELFSTRÖM, G.: Intravital wireless telemetry of axial forces in Harrington distraction rods in patients with idiopathic scoliosis. J. Bone Jt Surg. 53-A: 445, 1971.

NACHEMSON, A.: Personal communication 1972.

NACHEMSON, A.: Personal communication 1973.

NACHEMSON, A. & ELFSTRÖM, G.: Telemetry recordings of forces in the Harrington distraction rod. Clin.Orthop. 93:113, 1973

NACHLAS, I.W. & BORDEN, J.N.: The cure of experimental scoliosis by directed growth control. J. Bone Jt Surg. 33-A: 24, 1951.

NASH, C.L. Jr. & MOE, J.H.: A study of vertebral rotation. J. Bone Jt Surg. 51-A: 223, 1969.

NEUBERGER, A.: The structure of collagen. In: Proceedings of a symposium on scoliosis, held in ... London 1965. Ed. by P.A. Zorab. London 1965, p. 42.

NEUGEBAUER, H.: Cobb oder Ferguson. Eine Analyse der beiden gebräuchlichsten Röntgenmessmethoden von Skoliosen. Z. Orthop. 110: 342, 1972.

NEUMAN, R.E. & LOGAN, M.A.: The determination of hydroxyproline. J. biol. Chem. 184: 299, 1950.

NILSONNE, U. & LUNDGREN, K.D.: Long-term prognosis in idiopathic scoliosis. Acta orthop. scand. 39: 456, 1968.

NILSONNE, U.: Vertebral epiphyseodesis of the thoracic curve in the operative treatment of idiopathic scoliosis. Acta orthop. scand. 40: 237, 1969.

O'BRIEN, J.P., YUA, P.C.M.C. & HODGSON, A.R.: Halo pelvic traction; a technique for severe spinal deformities. Clin. Orthop. 93: 127, 1973.

PARTINGTON, F.R. & WOOD, G.C.: The role of non-collagen components in the mechanical behaviour of tendon fibres. Biochim. biophys. Acta (Amst.) 69: 485, 1963.

PEACOCK, E.E., Jr.: Comparison of collagenous tissue surrounding normal and immobilized joints. Surg. Forum 14: 440, 1963.

PEREY, O.: Discography in early cases of idiopathic scoliosis. Acta orthop. scand. 33: 392, 1963.

PICARD, J.J., BONAFOS, M., FERVAL, E. & CANARD, R.: Le traitement chirurgical des scolioses. A propos de 80 interventions. Montpellier chir. 12: 509, 1966.

PIEZ, K.A.: Cross-linking of collagen and elastin. Ann. Rev. Biochem. 37: 547, 1968.

PIGGOTT, H.: Posterior rib resection in scoliosis. A preliminary report. J. Bone Jt Surg. 53-B: 663, 1971.

PONSETI, I.V. & FRIEDMAN, B.: Changes in the scoliotic spine after fusion. J. Bone Jt Surg. 32-A: 751, 1950.

PONSETI, I.V. & SHEPARD, R.S.: Lesions of the skeleton and of other mesodermal tissues in rats fed sweet pea (Lathyrus odoratus) seeds. J. Bone Jt Surg. 36-A: 1031, 1954.

PONSETI, I.V.: Skeletal lesions produced by aminonitriles. Clin. Orthop. 9: 131, 1957.

- Studies on the nature of skeletal lesions produced by aminonitriles. Bull Hosp. Joint. Dis. 20: 1, 1959.

PONSETI, I.V., VON NOORDEN, G.K. & BURIAN, H.M.: Anterior chamber angle of the eye in patients with idiopathic scoliosis. J. Bone Jt Surg. 42-A: 1448, 1960.

PONSETI, I.V.: The pathogenesis of adolescent scoliosis. In: Proceedings of a second symposium on scoliosis: causation, held in ... London 1967. Ed. by P.A. Zorab. Edinburgh & London 1968, p. 60.

PONSETI, I.V., PEDRINI, V. & DOHRMAN, S.: Biochemical analysis of intervertebral discs in idiopathic scoliosis. J. Bone Jt Surg. 54-A: 1793, 1972.

PROCKOP, D.J. & KIVIRIKKO, K.I.: Relationship of hydroxyproline excretion in urine to collagen metabolism. Biochemistry and clinical applications. Ann intern. Med. 66: 1243, 1967.

RAMACHANDRAN, G.N. & KARTHA, G.: Structure of collagen. Nature (Lond.) 174: 269, 1954.

RAMACHANDRAN, G.N.: Structure of collagen at the molecular level. In: Treatise on collagen. I. Ed. by G.N. Ramachandran. London & New York 1967, p. 103.

RELTON, J.E.S. & HALL, J.E.: An operation frame for spinal fusion. A new apparatus designed to reduce haemorrhage during operation. J. Bone Jt Surg. 49-B: 327, 1967.

RICH, A. & CRICK, F.H.C.: The molecular structure of collagen. J. molec. biol. 3: 483, 1961.

RIGBY, B.J., HIRAI, N., SPIKES, J.D. & EYRING, H.: The mechanical properties of rat tail tendon. J. Gen. Physiol. 43: 265, 1959/60.

RISKA, E.B.: End results in the treatment of scoliosis. A survey of 57 cases. Acta orthop. scand. Suppl. 102, 1967.

RISSANEN, P.M.: The surgical anatomy and pathology of the supraspinous and interspinous ligaments of the lumbar spine with special reference to ligament ruptures. Acta orthop. scand. Suppl. 46, 1960.

RISSER, J.C.: Important practical facts in treatment of scoliosis. Instructional course lect. 5: 248, 1948.

- The application of body casts for the correction of scoliosis. Instructional course lect. 12: 255, 1955.

- Modern trends in scoliosis. Bull Hosp. Jt Dis. 19: 166, 1958.

- Scoliosis: Past and present. J. Bone Jt Surg. 46-A: 167, 1964.

ROAF, R.: Wedge resection for scoliosis. J. Bone Jt Surg. 37-B: 97, 1955.

- Rotation movements of the spine with special reference to scoliosis. J. Bone Jt Surg. 40-B: 312, 1958.

- The late results of unilateral growth arrest of the spine for scoliosis. Acta orthop. scand. 33: 393, 1963.

- The basic anatomy of scoliosis. J. Bone Jt Surg. 48-B: 786, 1966.

ROLLHÄUSER, H.: Konstitutions- und Alterunterschiede in Festigkeit kollagener Fibrillen. Gegenbaurs morph. Jt 90: 157,

SAKO, K. & MARCHETTA, F.C.: Delayed autogenous bone and callus transplants and prepared host beds. (An experimental study.) Arch. Surg. 92: 771, 1966.

SCARPELLI, D.G. & GOODMAN, R.M.: Observations on the fine structure of the fibroblast from a case of Ehlers Danlos syndrome with the Marfan syndrome. J. invest. Derm. 50: 214, 1968.

SCHEIER, H.J.G.: Zur Verwendung des Instrumentars von P. Harrington zur Korrektur und internen Fixation von Skoliosen. Z. Orthop. 106: 253, 1969.

SCHULTZ, A.B., LAROCCA, H., GALANTE, J.O. & ANDRIACCHI, T.P.: A study of geometrical relationships in scoliotic spines. J. Biomech. 5: 409, 1972.

SCHULTZ, A.B. & HIRSCH, C.: Mechanical analyses of Harrington rod correction of idiopathic scoliosis. Read at the American Orthopaedic Association, January 1973.

SCHÖNENBERGER, F., TAILLARD, W. & BERGER, H.: Beitrag zur Aminosäuren-Ausscheidung bei Epiphyseolysis, Perthesscher Krankheit und Skoliose. Z. Orthop. 95: 73, 1961/62.

SEVASTIKOGLOU, J.A. & BERGQUIST, E.: Evaluation of the reliability of radiological methods for registration of scoliosis. Acta orthop. scand. 40: 608, 1969.

SHANDS, A.R., Jr., BARR, J.S., COLONNA, P.C. & NEALL, L.: End result study of the treatment of idiopathic scoliosis. J. Bone Jt Surg. 23: 963, 1941.

SHANDS, A.R., Jr. & EISBERG, H.B.: The incidence of scoliosis in the state of Delaware: A study of 50.000 minifilms of the chest made during a survey for tuberculosis. J. Bone Jt Surg. 37-A: 1243, 1955.

SIFFERT, R.S. & BARASH, E.S.: Delayed bone transplantation. An experimental study of early host-transplant relationships. J. Bone Jt Surg. 43-A: 407, 1961.

SINCLAIR, R.J.G., KITCHES, A.H. & TURNER, R.W.D.: The Marfan syndrome. Quart. J. Med. N.S. 29: 19, 1960.

SINEX, F.M.: Cross-linkage and aging. Advanc. geront. Res. 1:165, 1964.

SJOERDSMA, A., DAVIDSON, J.D., UDENFRIEND, S. & MITOMA, C.: Increased excretion of hydroxyproline in Marfan's syndrome. Lancet II: 994, 1958.

SMITH, A.D., VON LACKUM, W.H. & WYLIE, R.: An operation for stapling vertebral bodies in congenital scoliosis. J. Bone Jt Surg. 36-A: 342, 1954.

SMITH, A.D.: Scoliosis. J. Bone Jt Surg. 40-A: 505, 1958.

SMITH, D.J. & SHUSTER, R.C.: Biochemistry of lathyrism. I. Collagen biosynthesis in normal and lathyritic chick embryos. Arch. Biochem. 98: 498, 1962.

SMITH, J.W.: The elastic properties of the anterior cruciate ligaments of the rabbit. J. Anat. (Lond.) 88: 369, 1954.

STAGNARA, P. et al.: Contribution à la discussion du rapport de R. Bouillet et A. Vincent. Acta orthop. belg. 33: 610, 1967.

STAGNARA, P.: Utilization of Harrington's device in the treatment of adult kyphoscoliosis above 100°. In: Operative treatment of scoliosis. Ed. by G. Chapchal. Stuttgart 1973, p. 61.

STEARNS, G., CHEN, J.Y.T., MCKINLEY, J.B. & PONSETI, I.V.: Metabolic studies of children with idiopathic scoliosis. J. Bone Jt Surg. 37-A: 1026, 1955.

STEGEMANN, H.: Mikrobestimmung von Hydroxyprolin mit Chloramin-T und p-Dimethylaminobenzaldehyd. Z. physiol. Chem. 311: 41, 1958.

STUCKE, K.: Sehnenbelastung und -ruptur im Tierversuch. Chirurg 22: 16, 1951.

TAMBORNINO, J.M., ARMBRUST, E.N. & MOE, J.H.: Harrington instrumentation in correction of scoliosis. J. Bone Jt Surg. 46-A: 313, 1964.

TANZER, M.L.: Experimental lathyrism. Int. Rev. Connect. Tissue Res. 3: 91, 1965.

THOMPSON, R.C. & BALLOU, J.E.: Studies of metabolic turnover with tritium as a tracer. V. The predominantly non-dynamic state of body constituents in the rat. J. biol. Chem. 223: 795, 1956.

TKACZUK, H.: Tensile properties of human lumbar longitudinal ligaments. Acta orthop. scand. Suppl. 115, 1968.

TRAVAGLINI, F.: The surgical treatment of scoliosis by Harrington's method. Some avoidable complications. Scalpello 1: 217, 1973.

URBANIAK, J.R. & STELLING, F.H.: Progression of the scoliotic curve after completion of the excursion of the iliac epiphysis. A preliminary report. J. Bone Jt Surg. 51-A: 205, 1969.

VELISKAKIS, K.P.: Increased generalized ligamentous laxity in idiopathic scoliosis. J. Bone Jt Surg. 55-A: 435, 1973.

VERZAR, F.: Veränderungen der thermoelastischen Eigenschaften von Sehnenfasern beim Altern. Experientia (Basel) 11: 230, 1955.

- The stages and consequences of ageing of collagen. Gerontologia (Basel) 15: 233, 1969.

VIIDIK, A., SANDQVIST, L. & MÄGI, M.: Influence of postmortal storage on tensile strength characteristics and histology of rabbit ligaments. Acta orthop. scand. Suppl. 79 1965.

VIIDIK, A., LEWIN, T.: Changes in tensile strength characteristics and histology of rabbit ligaments induced by different modes of postmortal storage. Acta orthop. scand. 37: 141, 1966.

VIIDIK, A.: Biomechanics and functional adaptation of tendons and joint ligaments. In: Studies on the anatomy and function of bone and joints. Ed. by F.G. Evans, Berlin 1966, p. 17.

- The effect of training on the tensile strength of isolated rabbit tendons. Scand. J. plast. reconstr. Surg. 1: 141, 1967.

- Function and structure of collagenous tissue. Göteborg 1968.

- Functional properties of collagenous tissues. Int. Rev. Connect. Tissue Res. 61, 1973.

VIIDIK, A. & ÖRNDAHL, G.: Physical properties of skin collagen in Marfan's syndrome. To be published in 1973.

VOGEL, H.G.: Zur Wirkung von Hormonen auf physikalische und chemische Eigenschaften des Binde- und Stützgewebes I, II, IV. Arzneimittel-Forschung 19: 1495, 1732, 1981, 1969.

VON LACKUM, W.H. & MILLER, J.P.: Critical observations of the results in the operative treatment of scoliosis. J. Bone Jt Surg. 31-A: 102, 1949.

WALKER, L.B., HARRIS, E.H. & BENEDICT, J.V.: Stress-strain relationship in human cadaveric plantaris tendon: a preliminary study. Med. Electron. biol. Engng 2: 31, 1964.

WATERS, R.L. & MORRIS, J.M.: An in vitro study of normal and scoliotic interspinous ligaments. J. Biomech. 6: 343, 1973.

WAUGH, T.R.: Intravital measurements during instrumental correction of idiopathic scoliosis. Acta orthop. scand. Suppl. 93, 1966.

WERTHEIM, M.G.: Mémoire sur l'élasticité et la cohésion des principaux tissus du corps humain. Ann. Chem. Phys.-21, 385, 1847, cited in: Viidik, A.: Function and structure of collagenous tissue, 1968.

WESTGATE, H.D.: Pulmonary function in thoracic scoliosis before and after corrective surgery. Minn. Med. 53: 839, 1970.

WHITE, A.A., III: Analysis of the mechanics of the thoracic spine in man. An experimental study of autopsy specimens. Acta orthop. scand. Suppl. 127, 1969.

WILLNER, S.: Factors contributing to structural scoliosis. Lund 1972.

WILSON, R.L., LEVINE, D.B. & DOHERTY, J.H.: Surgical treatment of idiopathic scoliosis. Clin. Orthop. 81: 34, 1971.

WOESSNER, J.F., Jr.: Biological mechanisms of collagen resorption. In: Treatise on collagen. 2. Ed. by B.S. Gould. London & New York 1968, p. 253.

WRIGHT, D.G. & RENNELS, D.C.: A study of the elastic properties of plantar fascia. J. Bone Jt Surg. 46-A: 482, 1964.

WYNNE-DAVIES, R.: Familial (idiopathic) scoliosis. A family survey. J. Bone Jt Surg. 50-B: 24, 1968.

- Acetabular dysplasia and familial joint laxity; two etiological factors in congenital dislocation of the hip. A review of 589 patients and their families. J. Bone Jt Surg. 52-B: 704, 1970.

- Familial joint laxity. Prod. roy. Soc. Med. 64: 689, 1971.

YAMADA, H.: Strength of biological materials. Ed. by F.G. Evans. Baltimore 1970.

YAMADA, K. et al.: Equilibrium function in scoliosis and active corrective plaster jacket for the treatment. Tokushima J. exp. Med. 16: 1, 1969.

ZAOUSSIS, A.L. & JAMES, J.I.P.: The iliac apophysis and the evolution of curves in scoliosis. J. Bone Jt Surg. 40-B: 442, 1958.

ZORAB, P.A., CLARK, S., COTREL, Y. & HARRISON, A.: Bone collagen turnover in idiopathic scoliosis estimated from total hydroxyproline excretion. Arch. Dis. Childh. 46: 828, 1971.

ZUK, T.: Ätiologie und Pathogenese der idiopatischen Skoliose aus der Sicht elektromyographischer Untersuchungen. Beitr. Orthop. Traum. 12: 138, 1965.

ZWEYMÜLLER, K. & PLENK, H.: Reissfestigkeitsuntersuchungen an Sehnen-Knochenverbindungen bei lathyritischen Ratten. Med. exp. (Basel) 18: 65, 1968.

ÅKERBLOM, B.: Standing and sitting posture. Stockholm 1948.