Pohybové ústrojí

Pokroky ve výzkumu, diagnostice a terapii

Vydává
Společnost pro pojivové tkáně ČLS J. E. Purkyně z.s.
Odborná společnost ortopedicko-protetická ČLS J. E. Purkyně z.s.
Ambulantní centrum pro vady pohybového aparátu, s.r.o.

ročník 25 / 2018 číslo 2

EMBASE / Excerpta Medica | Bibliographia medica Čechoslovaca

ISSN 2336-4777

Systém výživy kloubů dle výzkumu prof. MUDr. Milana ADAMA, DrSc.

Originální český kolagenní doplněk stravy ***GELADRINK®*** *byl vyvinut na základě výzkumu revmatologa a spoluzakladatele společnosti Orling s.r.o. prof. MUDr. Milana ADAMA, DrSc., člena Akademie věd v New Yorku, oceněného prestižní cenou UNESCO za celoživotní přínos lékařské vědě.*

doplněk stravy

GELADRINK® obsahuje:

- **kolagenní peptidy GELITA®**
  kolagenní bílkovina přispívá k udržování normálních kostí
- **vitamín C**
  přispívá k normální formaci kolagenu pro normální funkci kostí a chrupavek
- **glukosamin a chondroitin sulfáty**
  jsou součástí kloubní chrupavky
- **antioxidanty, MSM a další látky**
  s příznivými nutričními a fyziologickými účinky doplňujícími běžnou stravu a vyživující kosti, chrupavky, klouby a pojivové tkáně

DOPORUČUJÍ

odborné společnosti

SPOLEČNOST PRO POJIVOVÉ TKÁNĚ ČLS J.E. Purkyně

ODBORNÁ SPOLEČNOST ORTOPEDICKO-PROTETICKÁ ČLS J.E. PURKYNĚ PRAHA ČESKÁ REPUBLIKA

ČESKÁ SPOLEČNOST TĚLOVÝCHOVNÉHO LÉKAŘSTVÍ

22 let existence na trhu!

BEZPLATNÁ LINKA - 800 108 999 - WWW.ORLING.CZ

Zdravá a vyvážená strava a zdravý životní styl jsou nezbytné. Doporučená denní dávka 12 kapslí nebo jedna odměrka. Užívejte 3 měsíce.
Není určeno pro děti do 12 let, těhotné a kojící ženy. Doporučenou denní dávku nepřekračujte.


ottobock.
Quality for life
1E95 Challenger
Nové řešení pohybu pro míčové,
raketové a běžecké sporty.
www.mojeproteza.cz

POHYBOVÉ ÚSTROJÍ

ročník 25, 2018, číslo 2 | datum vydání: 28. 2. 2021

REDAKČNÍ RADA

MEZINÁRODNÍ REDAKČNÍ RADA

Pohybové ústrojí. Pokroky ve výzkumu, diagnostice a terapii.

ISSN 2336-4777 (od roku 2013 pouze on-line verze)
Vydává Společnost pro pojivové tkáně ČLS J. E. Purkyně z.s.
& Odborná společnost ortopedicko – protetická ČLS J. E. Purkyně z.s.
& Ambulantní centrum pro vady pohybového aparátu, s. r. o.
Excerpováno v Excerpta Medica a Bibliographia medica Čechoslovaca.
Návrh a grafická úprava obálky Pavel Lorenc.
Časopis je na Seznamu recenzovaných neimpaktovaných periodik vydávaných v České republice. Dvě čísla časopisu vycházejí v elektronické verzi jako ročník s průběžným vydáváním příspěvků po recenzi.
Při příležitosti sympozií je dvakrát ročně vydáváno supplementum.

Pro současné odběratele časopisu PÚ a další zájemce doporučujeme přihlásit se na http://www.pojivo.cz/en/newsletter/, zadat jméno a e-mailovou adresu, na kterou bude časopis posílán.
Na webové doméně SPT ČLS JEP http://www.pojivo.cz/cz/pohybove-ustroji/ naleznete ve formátu PDF všechna jednotlivá čísla a dvojčísla časopisu (včetně Supplement) vydaná od roku 1997.
Rukopisy zasílejte na adresu *profesor MUDr. Ivo Mařík, CSc., Olšanská 7, 130 00 Praha 3, (ambul_centrum@volny.cz)* ve formátu doc. Vydavatel upozorňuje, že za obsah inzerce odpovídá výhradně inzerent. Časopis, jakožto nevýdělečný, neposkytuje honoráře za otištěné příspěvky.

LOCOMOTOR SYSTEM

Advances in Research, Diagnostics and Therapy

Published by The Society for Connective Tissues, Czech Medical Association of J. E. Purkyně, Prague, Society for Prosthetics and Orthotics, Czech Medical Association of J. E. Purkyně, Prague, Czech Republic and Ambulant Centre for Defects of Locomotor Apparatus Prague, Czech Republic

Call for papers

Support this journal by sending in your best and most interesting papers. The issue of the journal is published during whole year after proof acceptation of the reviewers. In occasion of the symposia (twice a year) is published the supplement.

Submitted papers: Locomotor System will review for publication manuscripts engaged in diagnostics and interdisciplinary treatment of genetic and metabolic skeletal disorders, limb anomalies, secondary osteoporosis, osteo/spondyloarthritis and another disorders that negatively influence development and quality of locomotor apparatus during human life. Both papers on progress in research of connective tissue diagnostics, medical and surgical therapy of multiple congenital abnormalities of skeleton mainly in the fields of paediatric orthopaedic surgery and plastic surgery, orthotics and prosthetics treatment, and papers dealing with biomechanics, clinical anthropology and paleopathology are appreciated.

The journal has an interdisciplinary character which gives possibilities for complex approach to the problems of locomotor system. The journal belongs to clinical, preclinical and theoretical medical branches which connect various up-to-date results and discoveries concerned with locomotor system. You can find the volumes of Locomotor System journal at http://www.pojivo.cz/cz/pohybove-ustroji/ since 1997 (free of charge). Since 2013 only electronic edition of the journal is available. That is why we recommend to all subscribers and those interested apply at http://www.pojivo.cz/en/newsletter, enter personal data, titles and e-mail address where the journal will be mailed.

Abstracts of presented papers are excerpted in EMBASE/Excerpta Medica (from the year 1994) and in the Bibliographia medica Čechoslovaca (from the year 2010). We prefer the manuscripts to be prepared according to Uniform Requirements for Manuscripts Submitted to Biomedical Journals (Vancouver Declaration, Brit med J 1988; 296, p. 401–405).

25. ročník časopisu Pohybové ústrojí připomínající odkaz

doc. MUDr. Milana Rotha, DrSc. vědě a medicíně,

je věnován jubilantům členům redakční rady

prof. MUDr. Ctiborovi Povýšilovi, DrSc. (75 let)

doc. RNDr. Pavlovi Bláhovi, CSc. (75 let)

prof. MUDr. Josefovi Hyánkovi, DrSc. (85 let)

a

doc. Dr. Med. Kazimierzovi S. Kozlowskému (90 let)

The 25th volume of Locomotor System journal commemorating a legacy

of Associate Professor Milan Roth, DSc. to science and medicine

is dedicated to the anniversary of

Professor Ctibor Povýšil, DSc. (75 yrs.)

Associate Professor RNDr. Pavel Bláha, PhD. (75 yrs.)

Professor Josef Hyánek, DSc. (85 yrs.)

and

Associate Professor Dr. Med. Kazimierz S. Kozlowski, M.R.A.C.R. (90 yrs.)

POHYBOVÉ ÚSTROJÍ, 25, 2018, č. 2

Pokroky ve výzkumu, diagnostice a terapii

OBSAH

LOCOMOTOR SYSTEM, 25, 2018, No. 2
Advances in Research, Diagnostics and Therapy

CONTENT

SLOVO ČTENÁŘŮM PÚ 2018, ROČNÍK 25, ČÍSLO 2
A WORD TO READERS LS 2018, VOLUME 25, NO. 2

Vážení čtenáři, autoři a inzerenti!

Děkujeme za Vaši pomoc při tvorbě mezioborového odborného recenzovaného časopisu *„Pohybové ústrojí – pokroky ve výzkumu, diagnostice a terapii (dále PÚ)".*

Od roku 2013 je časopis PÚ vydáván pouze v elektronické formě (v roce 2014 bylo přiděleno nové ISSN 2336-4777). Časopis PÚ byl v roce 2008 zařazen Radou pro výzkum, vývoj a inovace vlády ČR na Seznam recenzovaných neimpaktovaných periodik vydávaných v České republice. V souvislosti se změnou v elektronickou formu vydávání v roce 2013 časopis nedopatřením vypadl z tohoto Seznamu. Od roku 2015 je elektronická forma Pohybového ústrojí opět na Seznamu recenzovaných neimpaktovaných periodik.

Všechna čísla a dvojčísla časopisu (včetně Suplement) vydaná od roku 1997 naleznete ve formátu PDF na webové doméně Společnosti pro pojivové tkáně ČLS JEP z.s. http://www.pojivo.cz/cz/pohybove-ustroji/ (bezplatný přístup).

Od roku 2016 vydáváme po recenzi příspěvky v chronologickém pořadí jako číslo 1 a 2, dále dvě samostatná Suplementa s příspěvky ze symposií Kubátovy dny a Prague-Lublin-Sydney-St. Petersburg Symposium.

Nedostatek příspěvků byl a je příčinou zpožděného vydávání. Začátkem roku 2020 byla se zpožděním vydána plánovaná dvě čísla časopisu PÚ, 2019, ročník 26. V období celostátního výjimečného stavu v důsledku koronarovirové choroby jsme vydali Suplementum 1 časopisu PÚ, 27, 2020. Dále číslo 1 časopisu PÚ, 25, 2018 s podtitulem „In memory to Milan Roth" a nosným sdělením ortopeda pana Dr. Pieta van Loona z Holandska s názvem „Milan Roth – Legacy to Medicine", ve kterém autor podal přehled experimentální práce pana doc. MUDr. Milana Rotha, DrSc a jeho publikací vydaných v letech 1965–1999 s cílem oživit a vyzvednout Rothovu prioritní celosvětově nedoceněnou vědeckou práci.

V létě 2020 bylo vydáno číslo 1 časopisu PÚ, 27, 2020 a koncem roku 2020 Suplementum časopisu PÚ, 27, 2020 s abstrakty z konference „Adaptation – interdisciplinary aspects" (12.9.2020, Lékařský dům v Praze).

Začátkem roku 2021 v době 2. vlny koronavirové pandemie vyšlo se zpožděním číslo 2 časopisu PÚ, 27, 2020.

V právě vydaném čísle 2 časopisu PÚ, 2018, ročník 25, které je také určeno k oživení vzpomínky na pana doc. MUDr. Milana Rotha, DrSc., čtenáři mají příležitost ocenit komplexně zpracovanou problematiku idiopatické skoliózy (IS) v podání pana prof. Dr. Mihaila Dudina, DSc. a jeho týmu (Emeritus director of the Children´s Rehabilitation Center of Orthopaedics and Traumatology

„Ogonyok", St. Petersburg, Russia) od historie poznání IS přes biomechanické vysvětlení vzniku idiopatické lordoskoliózy (preklinického a subklinického stadia) k osvědčenému systému konzervativního léčení v „Ogonyoku". Vysvětlení vzniku a progrese IS i principy konzervativního léčení vycházejí z experimentálního průkazu asymetrického růstu páteře a míchy a míšních kořenů – viz přehled prací Milana Rotha publikovaný v čísle 1 časopisu PÚ, 25, 2018.

Jako v letech minulých je posláním časopisu PÚ uveřejňovat vědecké práce zabývající se diagnostikou a symptomatickým mezioborovým léčením genetických kostních chorob, vrozených defektů končetin, sekundární osteoporózy, osteo/spondyloartrózy, ale i jiných chorob, které ve svých důsledcích negativně ovlivňují vývoj a kvalitu pohybové ústrojí v průběhu lidského života. Dále práce vycházející z výzkumu pojivových tkání na všech úrovních poznání, práce orientované na biochemickou, morfologickou, genetickou a molekulární diagnostiku chorob pohybového ústrojí. Zvláštní pozornost je přikládána pracím z oblasti ortopedické a antropologické biomechaniky, neuroadaptačním změnám skeletu v období růstu, řízené remodelaci pojivových tkání, studiím muskuloskeletálních a neuronálních interakcí v závislosti na léčebných metodách (léky kalciotropní aj. , rehabilitace, ortoticko-protetické a operační léčení) a v neposlední řadě sdělením antropologickým a paleopatologickým. Oceňujeme především interdisciplinárně zaměřené práce. V anglickém jazyce jsou publikována sdělení zahraničních i našich autorů. Žádaným doplněním obsahu časopisu jsou zprávy ze sjezdů a konferencí. V rubrice zprávy zveřejňujeme oznámení o životním výročí členů RR časopisu, SPT ČLS JEP z.s., Ortopedicko-protetické společnosti (OPS) ČLS JEP z.s. a významných osobností, sdělení o prioritních pozorováních, ze studijních a poznávacích cest aj.

V každém ročníku naleznete směrnice pro autory příspěvků, kterým věnujte prosím pozornost při tvorbě Vašich vědeckých sdělení. Souhrny prací publikovaných v časopisu jsou excerpovány v EMBASE / Excerpta Medica (od r. 1994) a v Bibliographia medica Čechoslovaca (od r. 2010).

K prosazení časopisu Pohybové ústrojí mezinárodně je velmi významné citovat práce v našem časopisu uveřejněné v příspěvcích posílaných do zahraničních časopisů s impakt faktorem. Pro zvýšení úrovně časopisu PÚ je nezbytné získávat původní kvalitní práce a kazuistiky, které doporučujeme publikovat v angličtině, s cílem zvýšit zájem o náš časopis v odborném světě. Souhrny původních prací doporučujeme psát co nejvýstižněji, strukturovaně, česky a anglicky (objectives, methods, results and discussion), s klíčovými slovy.

Těšíme se na Vaši spolupráci a tvůrčí připomínky.

Redakční rada

SPOLEČNOST PRO POJIVOVÉ TKÁNĚ ČLS J.E.Purkyně PRAHA • ČESKÁ REPUBLIKA

ODBORNÁ SPOLEČNOST ORTOPEDICKO-PROTETICKÁ ČLS J.E. PURKYNĚ PRAHA ČESKÁ REPUBLIKA

ČESKÁ LÉKAŘSKÁ SPOLEČNOST J.E. PURKYNĚ

AMBULANTNÍ CENTRUM pro vady pohybového aparátu s.r.o.

OBRÁZEK NA TITULNÍ STRANĚ ČASOPISU A POPIS NEUROADAPTIVNÍ DEFORMITY KOSTÍ PODLE MILANA ROTHA

Vlevo. neuroadaptivně-dysplastické změny kostry zadních končetin pulce (Rosničky kubánské) chovaného v 0,5 % roztoku alkoholu (nervový teratogen), který vedle známého centrálně-nervového působení zřejmě též narušuje neurální extenzivní růst. Energeticky náročný růst nervového skeletu a příslušných nervových kmenů je působením teratogenů insuficientní. To se projevuje makromorfologicky přímějším průběhem kratšího sedacího nervu vzhledem k ohnutému, tj. delšímu femoru. Rostoucí kosti se musely přizpůsobit příliš krátkým přímo probíhajícím nervům. Nervy jsou výrazně kratší než zakřivené kosti, i když jsou uvolněné v neutrálním postavení (**1, 3**).

Uprostřed. Dislokace v kolenních a hlezenních kloubech u zadních končetin osteolathyrického žabího pulce. Vzorky zadních končetin pulce, zvětšení 6–8x, projasnění speciální technikou (**4, 5**). Osteolathyrismus vzniká u rostoucích živočichů krmením hrachorem vonným (Lathyrus odoratus, sladký hrách) nebo chemickými osteolathyrogeny, které narušují maturaci kolagenu. Osteolathyrogeny narušují neurální růst a působí stejným nepřímým osteoneurálním mechanismem na rostoucí skelet jako klasické kostní teratogeny. To znamená, že způsobují primární porušení růstu míchy, komplexu nervových kořenů a periferních nervových kmenů.

Vpravo. Schéma idiopatické skoliózy jako důsledek "neuroadaptivní" odpovědi obratlového (páteřního) obalu na přehnané zpomalení spinálního nervového růstu (míchy a jejích kořenů). Pozoruhodná

tendence idiopatické skoliózy ovlivnit dolní hrudní páteř může být snadno spojena s faktem, že míšní segmenty Th5–10 jsou nesporně nejdelší, takže jejich růst je více energeticky náročný a více zranitelný než jiné segmenty míchy. Mícha v dolní hrudní krajině je tudíž náchylná k přehnanému zpomalení růstu, což se manifestuje neuroadaptivní deformitou rostoucího obratlového obalu (**2**).

TITLE PICTURE AND DESCRIPTION NEUROADAPTIVE BONE DEFORMITIES BY MILAN ROTH

Left. Neuroadaptive-dysplastic experimental-teratogenic deformities of the hindlimbs in a frog tadpole that was kept in water with alcohol (0.5 per cent alcohol solution). Alcohol is well-known neural teratogen which has not only effect on the central nervous system but also disturb the neural-extensive growth. Growing bones had to adapt to too short, straight coursing sciatic nerves. Even when slackened in neutral posture the nerves are distinctly shorter than the bowed bones (**1, 3**).

Middle. Dislocation at the knee and at the cruro-tarsal joints in an osteolathyric frog tadpole. A tadpole specimen of the hind limbs, magnification about 6–8x, staining amphibian peripheral nerves with Sudan black (**4, 5**). Osteolathyrism is produced in growing animals by ingestion of peas of Lathyrus odoratus or by chemical osteolathyrogens which cause disturbance of collagen maturation. Osteolathyrogens interact with the neural growth and work by the same indirect osteoneural mechanism upon the growing skeleton as the classic skeletal teratogens. It means that cause a primary growth impairment of the spinal cord – nerve roots complex and of the peripheral nervous trunks.

Right. The scheme of idiopathic scoliosis as a consequence of "neuroadaptive" response of vertebral envelope to exaggerated slowness of spinal neural growth. The striking tendency of idiopathic scoliosis to involve the lower thoracic spine may be readily related to the fact that Th5–10 spinal cord segments are by far the longest so that their growth is more energy-consuming and more vulnerable than that of the other segments. The lower thoracic spinal cord is thus prone to undue growth slowness which becomes manifest in neuroadaptive deformity of the growing vertebral envelope (**2**).

REFERENCES

1. ROTH M. Role of neural growth in the pathomechanism of skeletal dysplasias: an experimental study. Locomotor System, 2, 1995, No. 3, pp. 85–111
2. ROTH M. Macroneurotrophic features of growth hormone effects upon the spine and hip. Locomotor System, 3, 1996, No. 2, pp. 72–108.
3. ROTH M. Neuroadaptive Pathomechanism of Bone Dysplasias (in Czech). Locomotor System, 5, 1998, No. 3–4, pp. 127–132.
4. FREIHOFER W.C, COMPAGNO LJV. Additional notes on the use of Sihler technique of staining nerves of small whole specimens of fishes and other vertebrates. Copeia, 1977/3 p. 587–588.
5. NISHIKAWA KC. Staining amphibian peripheral nerves with Sudan black B: Progressive vs. regressive methods. Copeia, 1987/2, p. 489–491.

Professor Ivo Marik, MD, PhD
Centre for Defects of Locomotor Apparatus l.l.c.
Olšanská 7, 130 00 Prague, Czech Republic
E-mail: ambul_centrum@volny.cz

SKOLIÓZA: HISTORIE POZNÁNÍ

SCOLIOSIS: HISTORY OF KNOWLEDGE

Dudin M., Pinchuk D., Pankratova G.

Children's Rehabilitation Center of Orthopaedics and Traumatology "Ogonyok", Saint Petersburg, Russia.

SUMMARY

Abstracting from the medical concept of scoliosis, it is quite permissible to note that this is just an irreversible change in the shape of the spine. In the overwhelming majority of cases, the deformation of this unique segment of the skeleton occurs against the background of complete health in the most important pubertal period of a person's life. It is interesting that the process of transition of the spinal column to a new status is asymptomatic, which practically excludes an immediate visit to a doctor. This is what K.M. Bagnall said in his lecture in 2012 in Poznan (Republic of Poland) "the peculiarities of the onset and development of idiopathic scoliosis are such that 'work' with the patient begins only after the appearance of signs of deformity". He further emphasized that the main unsolved problem in the problem of scoliosis is the so-called "dark period". So, according to this scientist, it should be called the period of time during which the transition of a healthy spinal column to a deformed state occurs.

Steps to finding the causes of initiation and the laws of the subsequent development of scoliosis were taken throughout the "acquaintance" with this lesion. However, it was possible to approach both the key etiological factors and the disclosure of the patterns of its pathogenesis only in the second half of the XXth century, when new diagnostic technologies appeared.

The presented article is devoted to the history of the knowledge of scoliosis, which, with minimal symptoms, cannot be considered a disease, although in the case of progressive development it leads to severe disability that grows in subsequent life.

Keywords: Scoliosis, history, etiology, pathogenesis

INTRODUCTION

Scoliosis. It is difficult to find a term behind which stands so much anxiety, hopes and disappointments, successes and failures. Let us assert that along with the acquisition of the status of Homo erectus by man, which caused a radical change in the position of the spinal column, he had a number of problems in this most important, unpaired segment of the skeleton. As a disease of the spinal column, which the physicians of that time "do not know how to treat" it was mentioned in the papyri of ancient Egypt (Edwin Smith /1822–1906/ collection) (**30**). It is believed that the first description of patients with scoliosis was made two and a half thousand years ago by Ἱπποκράτης βήτα (*lat*. Hippocrates II) /460–370 BC/ from the island of Kos /Κως/. However, the deformity received its personal name "scoliosis" only five hundred years later from Claudio (or Clarissimus) Galenus /129–217/, a Roman doctor of Greek origin from the city of Pergamum /Πέργᾰμον/.

Meanwhile, 3500 years later, scoliosis is still full of mysteries and paradoxes, and we, doctors, are not far from the conclusion of ancient Egyptian healers.

Scoliosis (by Cl. Galenus) is a persistent curvature of the spinal column in the frontal plane (*anc.gr./ lat*. σκολιός, skoliós – curve). And only in the XIXth it was established that scoliosis is a 3D deformity (**34**).

In the process of its development, the deformation of the spine in a sufficient number of cases causes a gradual changes in the anatomy of the thoracic and abdominal cavities, as well as corresponding violations in the topography and functions of organs located in them. Indications of these events can be found even in Abū ʻAlī Husein ibn' Abdallāh ibn Sīnā, 980–1037/ (*pers*. ابو على حسين بن عبدالله بن سينا). As a result, a person becomes disabled both in the aesthetic perception of his body, and in the numerous dysfunctions in their organisms.

Later (most often in the fourth or fifth decade of life) such a patient begins to complain of disorders in the heart, lungs, gastrointestinal tract, etc. After a medical examination they often say to them: "You have scoliosis!" Then an almost classic dialogue takes place. Patient: "But they have never told me about it!" Doctor: "You haven't asked!"

From our point of view, scoliosis with good reason can be included in a number of those diseases of Homo Sapiens, which humanity "encountered" at the "dawn" of its development. To this day, thousands of famous and unknown doctors have taken part in solving its riddle.

It is quite natural that the main goal in this collective work was to find ways to "fight" the deformity of the spinal column. However, to effectively achieve this goal, knowledge about its etiology and pathogenesis was required, but the failure of their search gives a full explanation of the fact – the vast majority of published works turned out to be devoted to the treatment of a disease that mutilates the human body.

However, only very recently, only 500 years ago, the **first** “breakthrough” in its (scoliosis) knowledge took place. This breakthrough is associated with Ambroise Paré /1510–1590/, the First Surgeon of the French Kings François II, Charles IX and Henri III de Valois, who described congenital scoliosis.

By this he showed that not all scolioses are the same. Today no one doubts that this is a heterogeneous group of three-plane deformations of the spine. It should be noted here that the search for the causes of scoliosis from the standpoint of medical knowledge of each era was carried out both before and after A. Paré. The authors believe that most likely the failure of such searches, as well as the clinical monoformity of scoliosis, underlies the widespread use of mechanical conservative, and, since the beginning of the XXth century, surgical correction of the curved spine. We consider it important to note that this trend dominates in modern scoliosology.

WHAT IS SCOLIOSIS?

The answer to this question has been sought since the prehistoric times.

In the third volume of the Russian edition of the Hippocratic Corpus, in the treatise “On the joints” (*lat.* “De articulis”), spine and its diseases are devoted to as many as 8 paragraphs (#41–#48). Paying more attention to the “humpback”, the Great Greek writes: “In some cases, the spine bends to the side – to the left or to the right. All these curvatures or most of them are due to clusters that form on the inner side of σπονδυλική στήλη (*lat.* ridge, *engl.* spine)” [*here and in other quotes, the terminology in the interpretation of the translator*, authors] (**43**).

At the same time, several general remarks should be noted that indicate Hippocrates’ amazing observation of deformations σπονδυλική στήλη: “Kyphotic, or hind, humps are more favorable than lordotic[1], or front”, “When the curvature has occurred in children, the body does not grow, except for the legs, arms and head” and “The formation of a hump in an adult directly relieves the disease, but over time it manifests itself in the same symptoms as in younger ones, but in a less malignant form”. In addition, we note that the Great regarded the curvature of σπονδυλική στήλη as a process and its cause, from the standpoint of knowledge of that time, he saw, first of all, in injuries, in which the muscles on both sides of the spine acquired different severity and thereby caused the deformation[2].

One can only marvel at the insight and fundamental nature of the conclusions of the Great Greek. One of them is his approach to the treatment of scoliosis, based on the mechanical correction of deformity σπονδυλική στήλη. And although at the same time he criticizes unnamed doctors who used methods such as “stretching” on the stairs in the “up” or “down” position with their heads in patients with “humps”, nevertheless, historians associate the “scamnum” method with Hippocrates (translated from *latin* – bench). According to the sketches that have survived up to our time and

1 Since Hippocrates is quoted by V.I. Rudnev, it remains unclear – who is the author of the terminological roots “kyphosis” and “lordosis”? Cl. Galenus, traditionally mentioned in the literature as an author, or Ἱπποκράτης βήτα 400 years before him?

2 This is the forerunner of the muscular genesis of scoliosis!

have been reproduced many times in modern literature, it is clearly seen how the spinal column is stretched on the "bench" and at the same time, with the help of a flat board-lever, pressure is exerted on the rib hump. From sources that have come down to us, it is known that Galen of Pergamon added active and passive exercises, breathing exercises, loud singing and health baths to the therapy of scoliosis" (**53**).

The scope of the article does not allow for a more detailed analysis of the works of famous and unknown doctors of the beginning of our era, but they are reflected in sufficient detail in the works (**40, 41, 49, 58**).

It can only be stated that this principle of mechanical correction of the deformity of the spinal column dominates to this day (**70**).

However, in order to successfully treat the deformity of the spinal column, it is necessary to know the cause and pathogenesis of that lesion. Although in solving this problem, achievements are much less impressive.

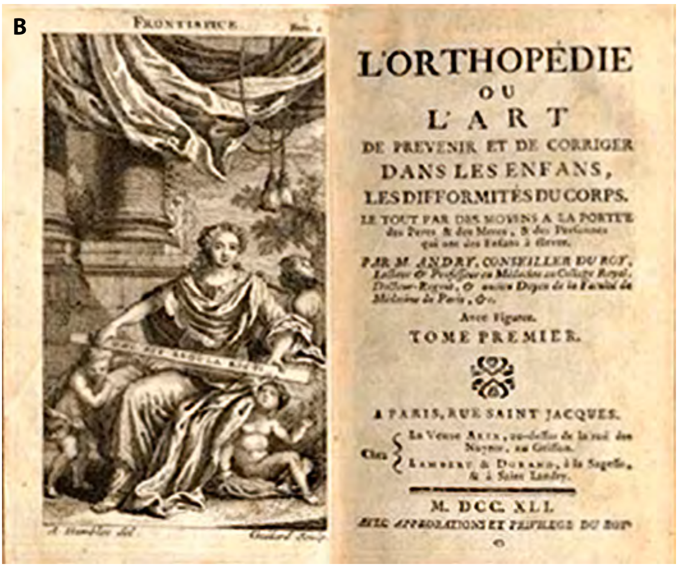
As noted above, the **first "breakthrough"** was made by A. Pare, who added the description of scoliosis "congenital" to the description of the scoliotic spine (Flemish Andreas Vesal /1515–1564/).

The **next stage** in the knowledge of the causes of the curvature of the spinal column is associated with the name Danie Whistler /1619–1684/, who in 1645 rediscovered the phenomenon of "softening of the bones" that was found in the works of Soranus /98–138/ from the city of Ephesus (Ἔφεσος) and Galenus from the city of Pergamum (Πέργᾰμον). He wrote his thesis "De Morbo puerile Anglorum, quern patrio idiomate indigense vocant "The Rickets"". Just five years later, the British physician Francis Glisson /1597–1677/ dedicated to him a whole treatise "De rachitide" (1650). He considered the main risk factors for the development of this lesion in the child's skeleton to be passed down heredity and inappropriate nutrition of the mother, and his contemporary and compatriot, chemist, physician and physiologist John Mayow /1641–1679/ in 1660 directly pointed to the softening of bones, which, in his opinion, and there is a condition for their curvature. Moreover, F. Glisson proposed a soft leather sling under the chin and the back of the head for traction (mechanical!) straightening of the "crooked ridge", which under the name "Glisson's loop" is widely used in traumatology and orthopedics to this day.

NOTE. It is interesting to note that the term "rickets /from anc.gr. ῥάχις + ίτ = ῥαχίτις*) literally means inflammation of the spine. This translation allows us to think that with this disease, doctors first of all paid attention to the deformed spinal column. True, there is speculation that the word "rickets" comes from the Old English "wrickken" ("to twist"), used in 1649 by Arnold de Boot, a Frisian doctor who practiced in Ireland (***72***).*

Thus, it was only in the XVIIth century that a second (after Hippocrates) hypothesis appeared about the cause of the origin of scoliosis – rickets is to blame! This hypothesis turned out to be so simple and understandable that even in the second half of the XXth century it was still quite widespread in

Figure 1:
A – Nicolas Andry de Bois-Regard /1658–1742/.
B – Frontispiece L'ORTHOPÉDE (1741).
C – Orthopedics symbol in which you can "see" the deformed spine.



the medical literature, and in the XXIth century this view remains dominant among hygienists and pediatricians.

In addition, it (this hypothesis) largely explains the activation in the construction of various devices (similar to the "hippocratic" ones) for mechanical resistance to the deformation of the spine. However, the fascination with mechanical devices for the first time led to an undesirable complication – overstretching of the spinal cord. By the way, it still haunts vertebrologists today when they perform surgical, most often one-stage, deformity correction (**84**).

But, on the other hand, this phenomenon has once again shown: scoliosis is far from a simple curvature of the spinal column.

Considering scoliosis as the main theme of this work, it is necessary to recall Nicolas Andry, who in the XVIIIth century, being a helminthologist, became the godfather of the specialty, which he called orthopedics[3]. He wrote his monograph "L'orthopédie ou l'art de prévenir et corriger dans les enfants les difformités du corps" at the age of 82 (!) years. It was first published in Paris (1741) in two volumes (Ith volume – 345 p., IInd volume – 365 p.). But, obviously, the demands of society on this problem were so great that it was soon reprinted in Brussels (1742), and then in English in London (1743) and in German in Berlin (1744). On one of the first pages of the French edition, a tree was drawn, which has become a symbol of our entire specialty (**figure 1**).

A short translation of the title in English reads like this: "Orthopedics or the art of preventing and correcting deformities of the body in children".

However, if you look at the original of this book [it was "digitized" by Google, author], you can come across a number of surprising moments. In the first chapters of the first volume, the author expresses his point of view on the origin of body deformities in children from the height of knowledge of his time. We believe that criticizing these views today is the height of indecency. But there is one fact that is difficult to ignore in the context of our topic we are considering: the author, talking about deformities of the trunk and examining their external manifestations, does not use the term "scoliosis"! The explanation of this peculiarity is seen only in one fact – **N. Andry himself was not an orthopedist.**

Nonetheless, at the same time, he notes that the diagnostics of these deformities is an extremely difficult task for a doctor. Nevertheless, in the recommendations for their prevention and correction, the author gives quite modern advice: how to sit, what devices (analogs of orthoses) to use and what exercises to apply. In general, the nature of scoliosis as the main cause of changes in the shape of the trunk for almost 2500 years was considered from the positions expressed by Hippocrates and the "fight" with the pathological curvature of the spine was reduced to a "pure" mechanical effect on

3 Orthopedics (fr. L'orthopédie, english Orthopedia) – from anc.greek. ορθος, straight, correct + παιδίον, παιδεία, child, education, training.

the spinal column with the help of its stretching and lateral pressure on the costal hump, based on the principles laid down in the construction of the "bench" of Hippocrates (**3**).

The next significant event on the path to knowledge of scoliosis was the publication in 1792 in the city Amsterdam (the Netherlands) of the book "Aanmerkingen over de Wanstaltingheden der Ruggengraat", or "Remarks on spinal deformity", the author of which was David van Gesscher /1735–1810/. We agree with colleagues that this monograph (as presented by Piet J.M. van Loon in Recent Advances in Scoliosis, edited by Theodoros Grivas, IntechOpen: 2012, article #13), written on the basis of author's clinical observations and postmortem research, not only laid the foundations to understand scoliosis [D. van Gesscher, in contrast to N. Andry, already widely uses the term "scoliosis", authors], but also outlined the key directions in the study of the mechanogenesis of three-plane deformation[4]. First, he postulated the importance of posture and balancing forces for correct posture formation in Homo erectus. Secondly, although he did not use the concept of "sagittal balance", he gave the physiological curves of the spinal column a key role in ensuring the "human bipedal way of standing and walking". Third, he proposed a mathematical equation to describe the optimal value of these bends, the task of which is "to maintain the vertical position of the body in an energy-saving mode". In addition, "In postmortem examinations, he discovered ventral disc deformity and vertebral wedging, which has been confirmed today in 'severe' kyphosis and scoliosis". D. van Gesscher associated these phenomena with prolonged sitting, especially in "girls with a "weak constitution" who spend days knitting or doing embroidery in a bent position on chairs or stools, and they more easily develop scoliosis in the area of compressed and easily deformed thoracolumbar vertebrae[5]" (all quotes from P.J.M. van Loon (**79**).

In the XIXth century, we distinguish **five** developments in the study of the nature of scoliosis. Let us remember that this was the century of the opening of various schools and other educational institutions for the people. It was there that the **first** event took place in the form of mass examinations of children by European orthopedists Witzel, Zander, Volkmann and others, as well as by ophthalmologists and pediatricians Berlin, Rembold, Tllinger and Vogt, etc. (**33**). In Russia, the first such examination was carried out by a pediatrician-orthopedist-physiologist N.F.Gagman (**35**).

Based on the results of such examinations, the first statistical data on the frequency of scoliosis among the child population were obtained. At the same time, it turned out that most frequently met deformities of the spinal column are observed in school-age children. This immediately gave rise to the conclusion – "the wrong sitting of children in their educational places is to blame". Thus the so-called the hypothesis of the "school" origin of scoliosis was born. And now, for more than a century and a half, it has dominated the minds of the public. It was this idea which gave rise to the whole world industry for the production of special educational desks. Their prototype was the bureaus created during the Renaissance, which were later transformed into desks and secretaires. A monument to a school desk was erected in Warsaw in 2010 (**figure 2**).

4 Paradoxically, but to them [the ideas of D. van Gesscher] the world scoliosology returned only two centuries later /author/.

5 A hundred years later, a similar conclusion will be made based on the results of mass examinations of children and the hypothesis of the "school" origin of scoliosis will be "born".

Figure 2: Monument to a school desk in Warsaw (Sculptor Wojciech Grinevich).

The **second** event related to scoliosis is the collective discovery of the Delpech – Hueter – Von Volkmann – Wolff Law (effect), which indicated that the bone is highly plastic and not an inert tissue (**19, 39, 82, 85**).

In the context of talking about scoliosis, of particular interest is the part of the law that was discovered independently by German scientists Carl Hueter /1838–1882/ and Richard von Volkmann /1830–1889/. It reflects the reaction of the growing bone tissue to mechanical stress: "compressive forces suppress bone growth, and tensile forces stimulate it". From the standpoint of this law, an explanation has emerged for the lateral curvature of the spine due to the violation of its verticality. By the way, this assumption "nicely" complemented the "school" hypothesis: "a child develops scoliosis because they do not sit correctly (crookedly) at school". But how can one explain the stable incidence of scoliosis in children after, in our progressive time, all children were "put" at special desks? However, let's not be categorical and we shall return to the role of Hueter–Volkmann Law in the pathogenesis of scoliosis.

Figure 3: A – William Adams /1820–1900/. **B** – Gideon Algernon Mantell /1790–1852/

Among the events of the XIXth century around scoliosis, a couple more interesting points should be mentioned. The first of them we consider the descriptions of various systemic diseases in children, in the picture of which scoliosis is present as a syndrome in most cases (Recklinghausen syndrome, or neurofibromatosis, described by the German pathologist Friedrich Daniel von Recklinghausen /1867–1942/ in 1882, Marfan syndrome, which was described in 1896 by the French pediatrician Antoine Marfan /1858–1942/, syndrome Elers-Danlos, described in 1899 by the Danish physician Edvard Laurits Elers /1863–1937/ and supplemented in 1908 by the French dermatologist Henri-Alexandre Danlos /1844–1912/ (to date, 14 variants of this syndrome have been described, authors).

The **third** important development is the fundamental conclusion drawn by William Adams following the observation of his friend Gideon Algernon Mantell, who complained of lower back pain (**figure 3**).

Dr. W. Adams examined G.A. Mantell, but apart from the asymmetry between the right and left paravertebral regions in the form of unilateral swelling (judging by the preparation – on the left, the authors) did not find it. After the death of G.A. Mantell his spine[6] was carefully studied by W. Adams.

6 We only once came across information according to which this was done at the request of G.A. Mantell, which fully corresponded to the traditions of natural scientists of the XIXth century.

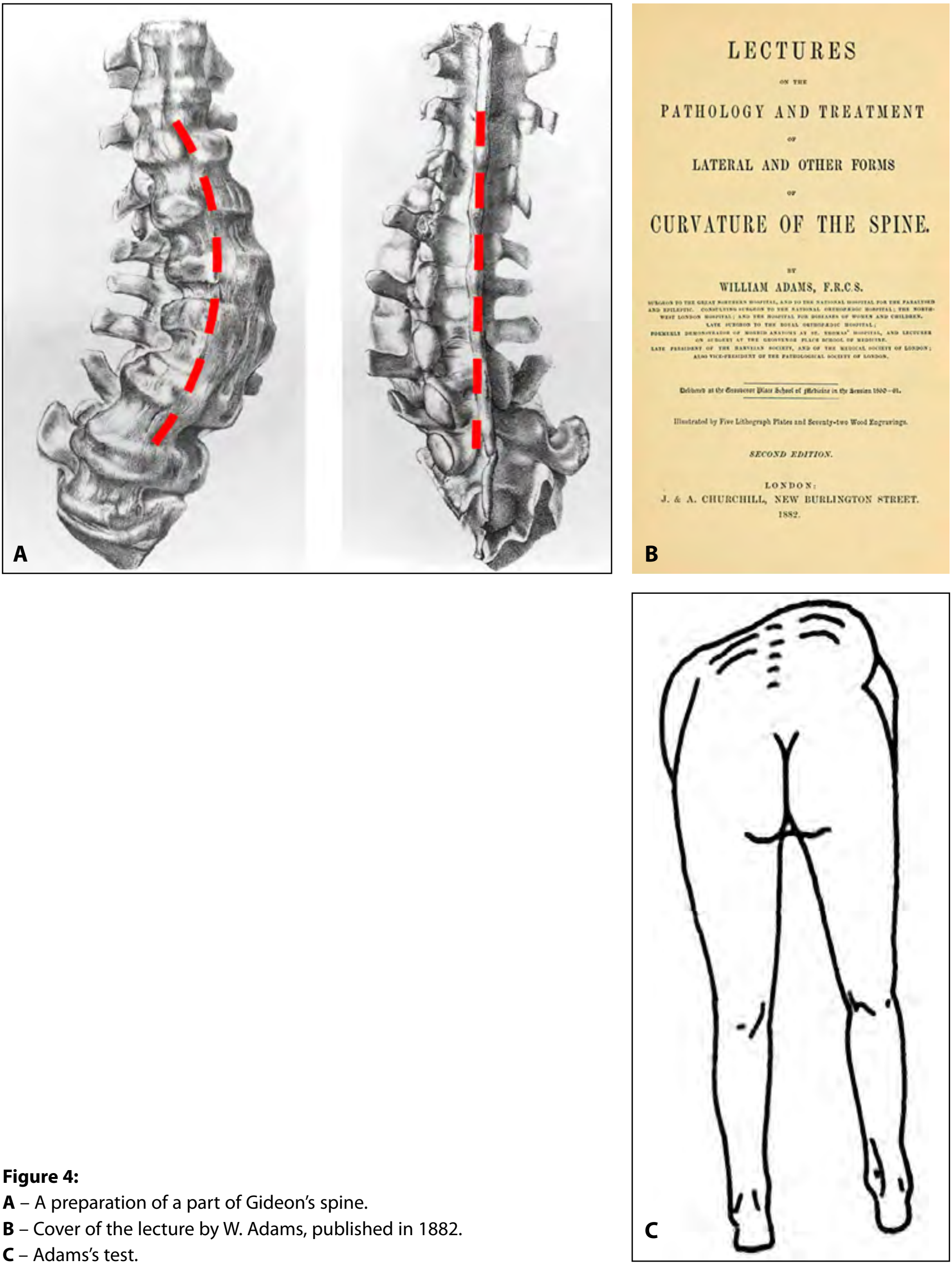
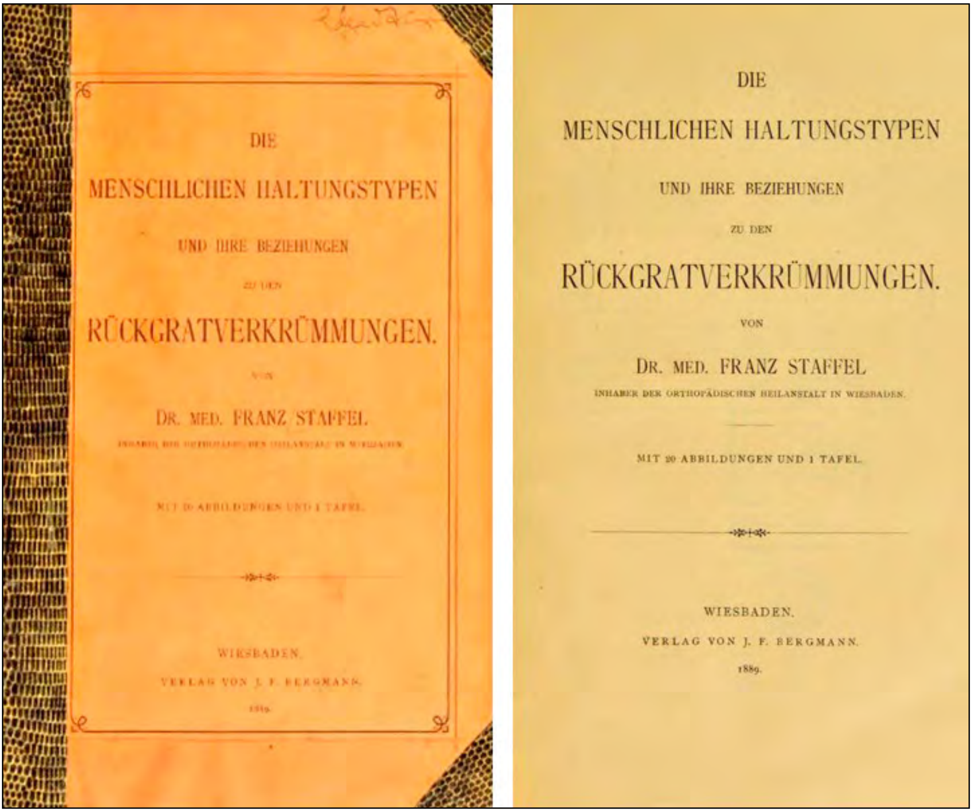


Figure 4:
A – A preparation of a part of Gideon's spine.
B – Cover of the lecture by W. Adams, published in 1882.
C – Adams's test.


DIE
MENSCHLICHEN HALTUNGSTYPEN
UND IHRE BEZIEHUNGEN
ZU DEN
RÜCKGRATVERKRÜMMUNGEN.
VON
DR. MED. FRANZ STAFFEL
WIESBADEN.
VERLAG VON J. F. BERGMANN.

DIE
MENSCHLICHEN HALTUNGSTYPEN
UND IHRE BEZIEHUNGEN
ZU DEN
RÜCKGRATVERKRÜMMUNGEN.
VON
DR. MED. FRANZ STAFFEL
INHABER DER ORTHOPÄDISCHEN HEILANSTALT IN WIESBADEN.
MIT 20 ABBILDUNGEN UND 1 TAFEL.
WIESBADEN.
VERLAG VON J. F. BERGMANN.
1889.

Figure 5: A – Cover and Frontispiece of the monograph F. Staffel.

It turned out that the line of spinous processes ran along the midline, while the midline along the anterior surface of the vertebral bodies had an arched appearance. This gave every reason to conclude that scoliosis is a deformity not only in the frontal plane (**figure 4**) and to identify the rotational component of the scoliosis W. Adams proposed the **Forward Bending Test**, which today bears his name.

At the same time, it is fair to say that W. Adams himself quotes Dr. Dodds from the city of Bath (England), who published back in 1824 the book "Pathological observations of the rotated and contoured spine, usually called lateral curvature", which draws attention to the horizontal rotation of the vertebrae in scoliosis. A little later, Jules Rene Guerin (1801–1886) wrote about this phenomenon, which became one of the motives for myotomies of the paravertebral muscles (**34, 54**).

The **fourth** important event in the late XIXth century in the history of the study of spinal column deformities was the monograph by Franz Staffel (**figure 5**).

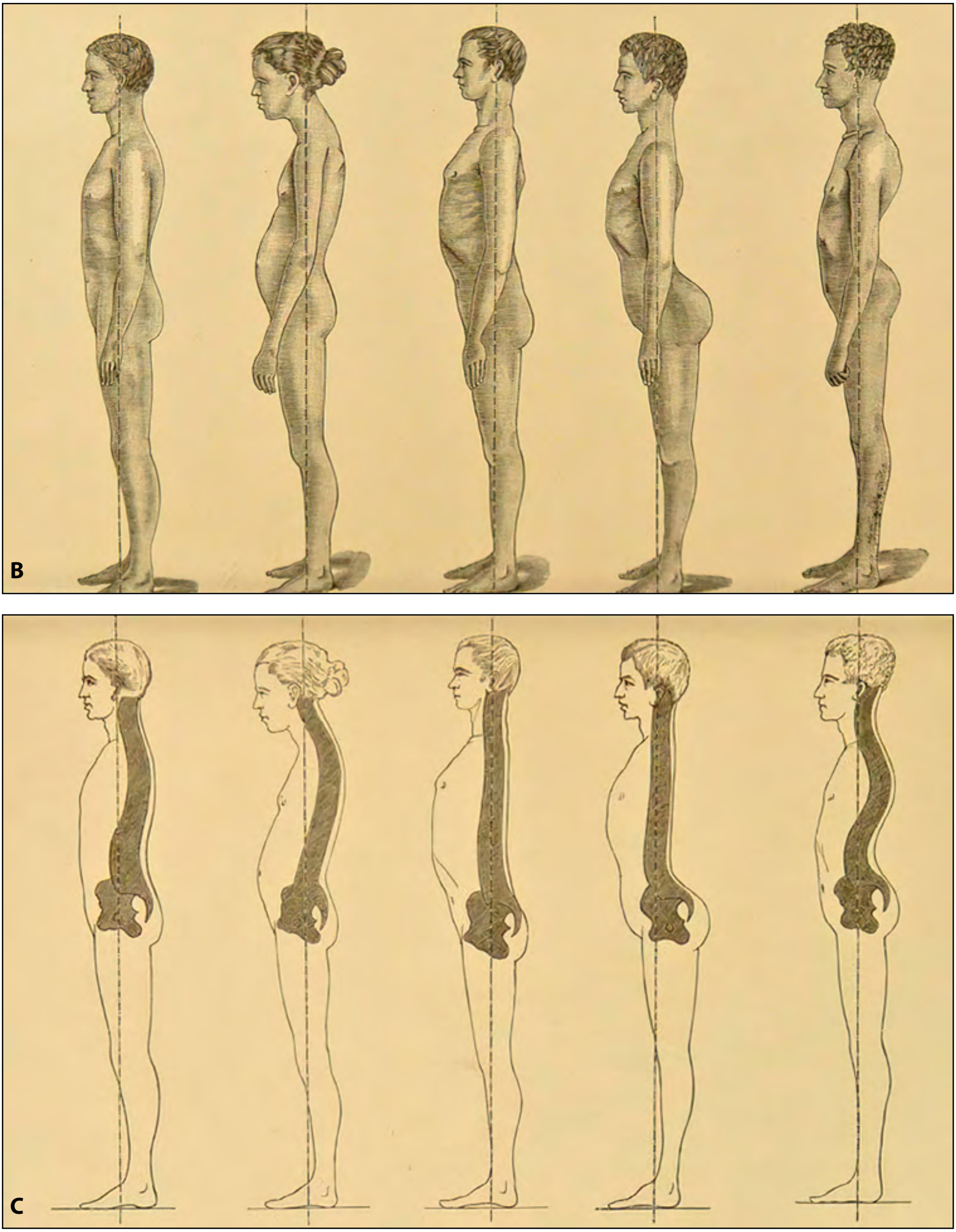


Figure 5: B and **C** – Illustrations on pages 102–103 in the monograph by Franz Staffel.

The value of this monograph is that it has drawn attention to the shape of the spinal column in the sagittal plane. It turned out that various parts of the spine have a fairly wide spectrum in their forms – from pronounced kyphosis where physiological lordosis should be normal to lordosis in the zone of physiological kyphosis (**73**).

As a result, the concepts of "lordoscoliosis" and "kyphoscoliosis" appeared quite naturally, in the clinical picture and evolution of which significant differences were observed. Let us recall what Hippocrates wrote: "The kyphotic, or hind, humps are more favorable than the lordotic ones, or the front ones" (!) (**52**)

And finally, the **fifth** world event was the discovery of Wilhelm Conrad Röntgen /1845–1923/ on the evening of November 8, 1895, the so-called. X-rays. Their practical application in orthopedics has caused a flurry of new information, including about scoliosis. It turned out that with all the external similarity of external manifestations, the anatomy of the deformed spine turned out to be very diverse. Among all scolioses, groups of congenital and dysplastic[7] deformities quickly emerged. A group of lesions of the bone tissue of the vertebral bodies was identified, leading to the development of the so-called "juvenile kyphosis". Scheuermann's disease (Scheuermann kyphosis), described in 1920 by the Danish radiologist Holger Werfel Scheuermann /1877–1960/, took the leading place among them, and a little later (1924) information about it was supplemented by orthopedic surgeon Karl Mai /1890–1958/ from the German city of Cologne (Köln) (**53, 68**).

The flat image of the three-plane volumetric deformation on the X-ray diffraction pattern, in spite of certain distortions, made it possible to obtain numerical estimates of the spine deformation. So, in 1948, John Robert Cobb /1903–1967/, based on the idea of Dr. Robert Lippmann, proposed a method for measuring the frontal and horizontal components, as well as the degree of wedge-shaped vertebrae. Other methods have appeared, but due to its simplicity and sufficient accuracy, it is Cobb's technology that has become the most popular (**16**).

Later, in 1969, C.J. Nash Jr. and J.H. Moe proposed their technique for calculating the rotation of the vertebrae, focused on the position of the projections of the roots of their arches (**55**).

On the basis of a similar idea, rather simple methods of measuring the sagittal component from a lateral radiograph appeared. The most difficult to measure horizontal component is currently successfully calculated on a PC using the Pavel Černý and Ivo Mařik's program (www.anglespine.com) (**13**).

Today, various computer optical-topographic diagnostic techniques are widely used. Their positive characteristics are complete safety and the ability to provide information about changes in the shape of the spinal column in three planes simultaneously. Despite the relative "roughness" of this method, it is very convenient for ongoing observation and control of patients (**54**).

[7] Dysplasia is a violation or perversion of the growth of the vertebrae in the postnatal period of a child's life.

However, let's go back a little. The first half of the XXth century was occupied by two world wars. And any war can be called a traumatic epidemic, so it is quite natural that many orthopedic doctors were involved in the treatment of the wounded and in the course of their service they gained vast experience in spinal column surgery. This experience gave a serious "impetus" for the development of techniques for surgical correction of scoliotic spine on the basis of. Moreover, shortly before the wars, in 1911, American surgeons Russell Aubrey Hibbs /1869–1932/ and Frederick Houdlette Albee /1876–1945/ independently worked and proposed a method for the formation of fusion, leading to the elimination of spine mobility. Despite the fact that this method was first used in patients with tuberculous vertebral lesions, it quickly attracted the attention of scoliosologists, who began to receive encouraging results (**2, 42**).

However, there was also another impact of traumatic epidemics. They gave a great impetus to the development of prosthetics, which also manifested itself in the development of new designs of corsets, including for patients with scoliosis. In fact, these corsets have become a serious step towards the conservative treatment of scoliosis as their use also gave hope for a bloodless victory over the defeat of the spinal column.

Meanwhile, evaluating both of these areas in the fight against scoliosis, we involuntarily come to the conclusion that surgical and corset (conservative) treatment is based on purely mechanical correction of deformity, but only at a higher technological level than Hippocrates and all his followers had.

Here it is pertinent to refer to the results of the work carried out under the patronage of the American Orthopedic Association, led by Alfred Rives Shands Jr. /1899–1981/. The history of observation and treatment of 425 patients with idiopathic scoliosis was analyzed. Of these, 185 patients received conservative treatment, which was based on various corsets with simultaneous exercises. Another 180 patients were operated on for the formation of fusion. Another 80 people did not receive treatment. Of the two non-surgical groups (265 people), 34 (13%) patients were still operated on. In operated patients, in 60% of cases, repeated intervention was required for additional bone grafting. The final result is a 27% correction. The complication rate is 38.2% (pseudarthrosis – 28%, other complications – 10.2%). In untreated patients, a stable clinical picture or its improvement occurred in 88% of cases, and deterioration – only in 12%. In patients treated conservatively (exercise and corset), stabilization and improvement were observed in 64%, and deterioration in 36%. The Risser corset and spinal fusion led to stabilization in 89% of patients, and deterioration occurred in 11%. However, the final conclusion of the A.R. Shands Jr. It sounds like this: "There is no alternative to surgical treatment of scoliosis" (**70**).

The given data are of undoubted value, but at the same time they raise the question – why are they so "motley"? Most likely because all patients with scoliosis are the same, but there are no identical scoliosis. They, scolioses (we are talking about their largest group – idiopathic, authors), arise against the background of complete health, but at different ages, they go to the doctor with both initial and severe symptoms, some have a progressive course, while others remain stable. The impression is that they "say": "we have our own laws." And it is difficult to name those listed by Dr. A.R. Shands Jr. therapeutic measures to the category of pathogenic. Moreover, the number of complications from

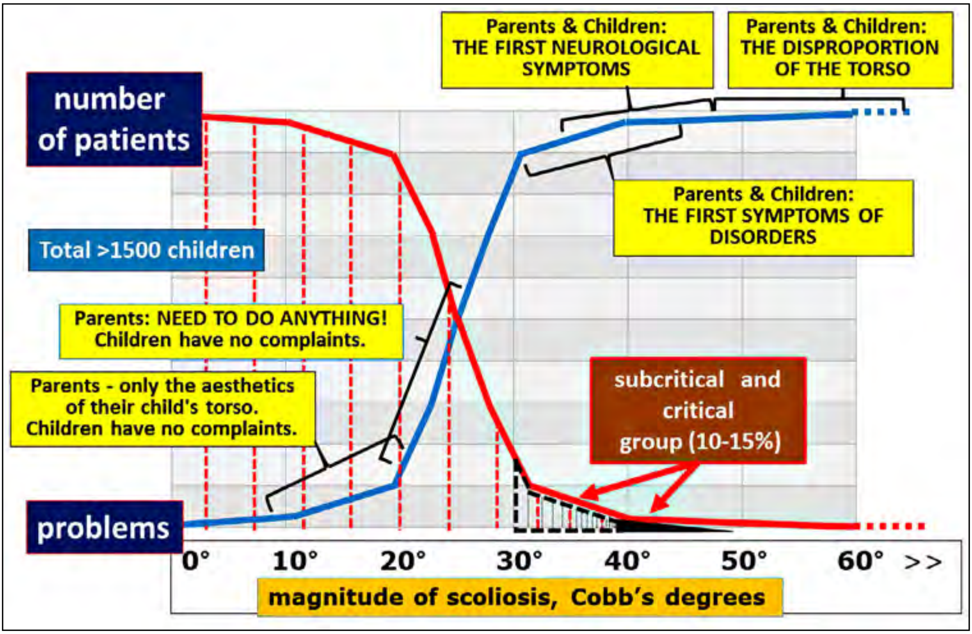


Figure 6: The severity of scoliosis and the problems it causes. From aesthetic ("walks and sits crookedly", "some kind of hump on the back") to serious violations of the cardio-respiratory complex and severe psychological deviations due to imbalances in the trunk (especially in women, "ape-like figure with long limbs and short trunk").

surgical interventions cited by the authors of the Conclusion does not add optimism. We will not discuss this Conclusion, because until the end of the first half of the XXth century, the mystery of the origin of scoliosis in 80–90% of cases was not solved and such group, as "idiopathic" deformities, entered all modern etiological classifications.

It is more likely that information provided by a group led by A.R. Shands Jr., caused an even greater desire to understand the laws by which pathological 3D deformation of the spine arises and develops.

If the **first paradox** of scoliosis is its monoformity with polyetiology (this will be discussed below, authors), then **the second one**, rightfully, can be considered that not all scolioses have a progressive course. And this means that the problems that it creates for a person will differ in their severity.

To clarify the nature of the links between the actual severity of the deformity and the emerging pathological symptoms (recorded both by the patients themselves and their parents, as well as by doctors and teachers), in our clinic in 2015–2017, a medical and psychological examination of 1,500 patients with scoliosis was carried out.

In children included in this group age and gender were not taken into account. The deformation value was taken into account only at the time of examination. As a result, the subgroup with scoliosis of initial severity (up to 20°–25° Cobb's degrees) included both small children, who may have progression of spinal deformity in the future, and older children (14–16 years old) who are unlikely to develop such scoliosis. On the contrary, the subgroup with severe scoliosis (40° and more Cobb's degrees) included both adolescents aged 14–16 years old and children under 10 years old. This allowed us to see a "cut" of the frequency of scoliosis in general in the abstract group of patients (**figure 6**).

As the red graph in figure 6 illustrates, the largest number of patients had an initial symptomatology of three-plane deformity. In a significantly smaller number of children (of all age subgroups), the frontal curves reached 25°–35° Cobb's degrees, and the critical values of scoliosis were recorded in a relatively small subgroup. Note that similar conclusions can be found in the works of M.A. Asher and D.C. Burton (**5**), W.J. Kane (**44**), J.E. Lonstein (**51**) T.N. Sadovaya. (**66**) and many others. Including in the analysis of the data given in the work cited above by A.R. Shands Jr. (**70**).

In addition, if the results of the medical and pedagogical examination of the problems caused by scoliosis (blue graph) are imposed on the red graph, it is clearly seen that at first the parents are concerned only with the aesthetics of the child's torso. In particular, "the asymmetric position of the shoulder girdle and shoulder blades", "an incomprehensible hump on the back", "the child sits unevenly, although we constantly remind them to sit upright!", etc. **Children have no complaints at this time** (!).

In most of these cases, parents are alarmed and go to the orthopedist. However, in the absence of an understanding of the nature of the pathological deformity of the spine, conservative treatment does not guarantee a positive effect. Scoliosis continues to "live" according to its own laws and it progresses in some children. And only at this time, as they grow older (!), adolescents begin to realize that their torso is deformed.

Meanwhile, in most cases, scoliosis either progresses slowly or does not progress at all. Moreover, these cases directly indicate that there are mechanisms in the body of a child that oppose the most unpleasant characteristic of scoliosis – the ability of progressive development. And if these mechanisms are found and used, then the need for the opinion "Perhaps no disease subject to treatment in a <surgical> orthopedic and trauma clinic gives so much concern to an orthopedic surgeon, does not bring as much disappointment to both the patient and the doctor as scoliotic disease" (**77**).

In the 50^{s} of the XXth century, two new directions in the study of idiopathic scoliosis were formed.

The first of these is related to genetics[8]. Its intensive development in the 20–30–40^{s} of the XXth century in the subsequent period was marked by a fairly long series of Nobel Prizes (1933 y. –

[8] The birth of genetics as a science in the middle of the XIXth century was determined by the works of Gregor Johann Mendel /1822–1884/, an Augustinian monk from Brno (german Brünn).

Thomas H. Morgan /1886–1945/; 1935 y. – Hans Spemann /1869–1941/; 1946 y. – Hermann Josef Möller /1890–1967/; 1958 y. – George Beadle /1903–1989/, Edward Tatem /1909–1975/ and Joshua Lederberg /1925–2008/; 1959 y. – Severo Ochoa /1905–1993/ and Arthur Kornberg /1918–2007/; 1962 y. – Francis Crick /1916–2004/, James Watson /b. 1928/ and Maurice Wilkins /1916–2004/; 1965 y. – Francois Jacob /1920–2013/, André Lvov /1902–1994/ and Jacques Monod /1910–1976 /; 1968 y. – Robert Holly /1922–1993/, Har G. Koran /1922–2011/ and Marshall Nirenberg /1927–2010/; 1969 y. – Max Delbrück /1906–1981/, Alfred Hershey /1908–1997/ and Salvador Luria /1912–1991/).

Against this background, in 1934 H.G. Garland (**36**) examines and describes a case of scoliosis in five generations of the same family. The high incidence of scoliosis in children, whose relatives already had scoliosis, was reported by R. Wynne-Davies (**86**) and E.J. Riseborough et al. (**60**).

In Russia, the study of the role of the genetic factor in the origin of idiopathic scoliosis is associated with the name of Madame A.M. Zaydman, under whose leadership several dozen genes associated with idiopathic spine deformity were identified (**88–91**). However, in numerous discussions with Professor A.M. Zaydman authors could not get answers to two questions. The first is why dysfunctional genes begin to be realized only in puberty, i.e. 10 years after birth? Second – what does the chain of processes consist of (what is the sequence) from a dysfunctional gene to formed scoliosis, which differ in the nature of progression? Nevertheless, the topic of the "genetic" theory of the origin of this disease continues to develop.

The second direction was the study of the functional state of the paravertebral muscles[9]. Even Hippocrates considered muscle imbalance as the cause of deformities of the spine. The new equipment, created by the middle of the XX[th] century, made it possible to quickly detect the asymmetry of the bioelectric activity of the muscles serving the spinal column. Its high values (relative to those on the contralateral side) were stably recorded on the convex side of the apex scoliotic arc. Paradoxically, this phenomenon has long remained unsolved. Of the recent works on the study of the role of paravertebral muscles in the pathogenesis of scoliosis, the publication of J. Cheung et al. (2005) (**14**), which reflects the results of longitudinal observation of the paravertebral muscles in children with progressive and non-progressive scoliosis. It turned out that the data obtained at the level of the caudal end of the frontal arch were of the greatest value: the larger the asymmetry coefficient, the more aggressive the deformation behaved. Let us add that our own results of a similar study of the functional state of the transversospinal muscles throughout the entire spinal column allowed us to reveal the mechanism of the formation of two antiturns in the bone-disco-ligamentous-muscular "sheath" of the spinal cord, which, in fact, together form a scoliotic arc (**24, 25, 26, 59, 93**).

In the middle of the XX[th] century, Madame Marie-Jeanne Thillard and her colleagues experimentally found scoliosis in chickens from which the pineal gland was removed – a small hormone-producing formation in the epithalamic zone of the brain (**76**). And although the first mentions of it as a "dwell-

[9] The method of electromyography in humans was first applied by the German scientist Hans Edmund von Piper /1877–1915/ in 1907 y. (Piper H. Elektrophysiologie menschlicher Muskeln, Springer Verlag, Berlin Heidelberg, J. Springer, 1912: 61).

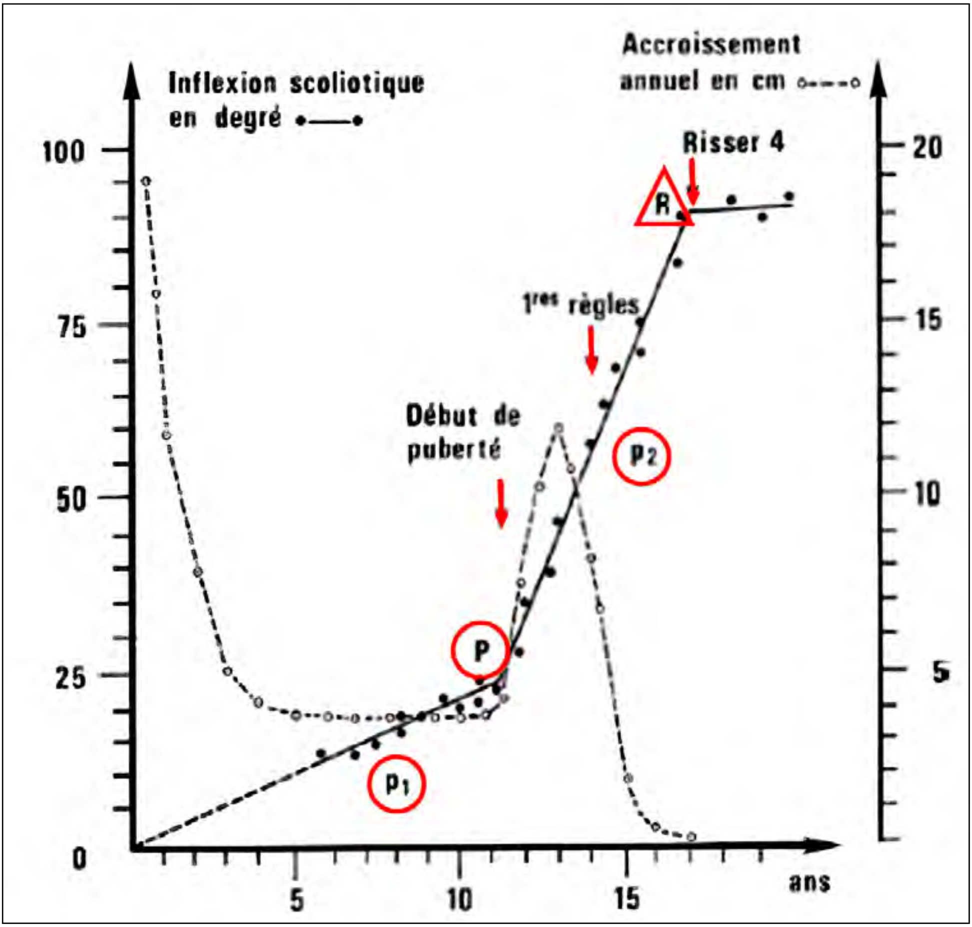


Figure 7: Duval-Beaupère curves. The solid curve represents the worsening of scoliosis curvatures, with the slope $\mathbf{P_1}$ representing childhood until the onset of puberty **P** and the slope $\mathbf{P_2}$, faster between puberty and the end of growth **R**. The dotted curve represents the trunk growth rate. **/**In «Baudoux M. Analyse des fractures de tiges après arthrodèse vertébrale dans les scolioses de l'enfant et de l'adolescent. These pour le diplome d'etat de docteur en medicine: Universite Lille 2 droit et santé Pôle recherche de la Faculté de médecine de Lille, Lille: 19 Juin 2017: 80.»/.

ing place of the soul" are found in the ancient Indian tracts, its main hormone melatonin (*anc.gr./lat./eng.* μελας, melas, black + τοςος, tosos, job) was discovered by Aaron Bunsen Lerner /1920–2007/ only in 1958 (**50**). The importance of these discoveries is that the pineal gland, being a neuroendocrine structure, is the main pacemaker in a living organism and one of its tasks is to coordinate the work of the glandular endocrine system. In other words, in the etiology of idiopathic scoliosis (experiments by Madame M.-J. Thillard and colleagues), a hormonal factor begins to be seen.

It should be noted that in the 50s–60s last century many advanced studies were carried out in France by young scientists, who later became the first magnitude stars in vertebrology. It was they who discovered the most indisputable fact in the theory and practice of idiopathic scoliosis – the connection between its occurrence and development with the child's growth process (**figure 7**) (**17, 28, 29**).

Moreover, the Russian scientist G.G. Epstein in 1981 (**32**) showed that children with scoliosis turn out to be taller than their healthy peers if the absolute length of the spinal column is included in the indicator of their height. Three decades later, completely identical data were published by Zhen Liu et al. (**92**). A simple conclusion followed from these studies – in children with scoliosis, a more intensive longitudinal growth of the axial skeleton is observed.

Meanwhile, in 1968–1969, several works of the outstanding Czech scientist Milan Roth (1923–2006) from Brno were published, which, unfortunately, **did not arouse much interest among "scoliosologists"** (**61–64**).

He was the first to draw attention to the fact that the spine is the most complex segment of the axial skeleton, in which, in the closest anatomical connection, there are two components of different embryonic origin – the spinal cord and its bone-disco-ligamentous-muscular "sheath". Each of them has its own task for Homo erectus. So, the spinal cord, as a department of the directive central nervous system, due to the pathways provides communication (with the exception of the head) with absolutely all organs and tissues of the body, and due to motoneurons – the implementation of the locomotor function (**71**). In the second component of the spine, the "sheath", the most mobile segment of the axial skeleton, the main task is to provide homeostasis for the spinal cord. This task includes:

- mechanical protection;
- support (maintaining longitudinal dimension) with a spring effect;
- mobility with a safety effect,
- in growing children – protection against non-linear growth spurts, first message made by Count Georges-Louis Leclerc De Buffon back in the XVIIth century (as physiological and individual annual, seasonal, monthly and per diem) (18.68).

It follows, and this is what Milan Roth emphasized, that the critical condition in the mutual existence of the spinal cord and its "sheath" is their synchronous (conjugate) axial growth. However, the content of this process is different for each of them. In the department of the central nervous system, it is "extensive (lengthening), and in the second, segment of the axial skeleton, it is cell division (mitotic, or proliferative)" (cited by M. Roth). In other words, between the longitudinal growth of the spinal cord and its "sheath", which differ in embryonic origin and in the mechanisms of regulation of this growth, as well as in the performance of their tasks, a conflict is quite admissible, which can be called "medullo-vertebral". Moreover, M. Roth created a mechanical "SPRING-STRING" model illustrating scoliosis-like deformity.

But such a revolutionary conclusion required proof. It may seem immodest, but it was the work of M. Roth that "provoked" us to create a virtual biomechanical model of scoliosis-like deformity based on the fundamental laws of theoretical mechanics (**21**). This was the second attempt after David van Gesscher from the Netherlands to draw a mathematical apparatus for understanding the nature of scoliosis. The most unexpected and therefore impressive conclusion in our work was the fact of the model's high sensitivity to the change in the difference (ΔL = **L – S**) between the longitudinal dimensions of the "spinal cord" (**S**) and its " bone-disco-ligamentous-muscular sheath" (**L**). For example, note that to obtain a "scoliosis-like" deformity of 15–20° (similar to Cobb degrees) in the model, it was enough to have ΔL equal to <1% of the total model length. A real child's spinal column, the length of which is about **35–40 cm**, this value is **3–4 mm** (!).

Perhaps that is why the methods of intravital atraumatic imaging of the spinal cord, which were invented only in the early 70^{s} of the XXth century, did not immediately provide evidence for the ideas of M. Roth. We are talking about computer (1972) and magnetic resonance (1973) tomography, the creators of which were awarded the Nobel Prizes. For CT in 1979 Godfrey Newbold Hounsfield /1919–2004/ and Allan McLeod Cormack /1924–1998/, and for MRI in 2003 Paul Christian Lauterbur /1929–2007/ and sir Peter Mansfield /1933–2017/ (**26**).

However, let us return to the most indisputable fact in the theory and practice of scoliosis – to the connection between its occurrence and development with the process of growth of the axial skeleton. There is no need to remind that this process is regulated primarily by the endocrine system. And to understand its role, information is needed on the levels of osteotropic hormones in the blood serum (a growth hormone was isolated in 1956, a glucocorticoid cortisone and cortisol – in 1937, a calcitonin – in 1962 and a parathyrin – in 1972). To solve this problem, the radioimmunoassay, was developed in the 50^{s} of the XXth century by Solomon Aaron Berson /1918–1972/ and Rosalyn Sussman Yalow /1921–2011/, by the Nobel laureates in 1977. Soon, the enzyme-linked immunosorbent assay (ELISA) was involved in the direct determination of hormone concentrations, which was first used by Swedish scientists Peter Perlmann /1919–2005/ and Eva Engvall /b. 1940/ (**31**).

If in "pure" endocrinology these technologies caused a real revolution, then in "scoliosology" only individual hormones were studied. The first among them were sex ones (this was motivated by the higher frequency of scoliosis in girls) and, of course, growth hormone. An example of such work is the dissertation of E.N. Bakhtina (1990) (**6**).

The most remarkable result of this work is that it was shown that the concentration of sex hormones and GH significantly differed from their levels in healthy peers. But at the same time, due to the large variance of the actual data, correlations with the clinical and radiological picture turned out to be weak.

Here, as a small digression, we note that the endocrine system, as one of the most ancient regulatory systems of a living organism, is a complex, consisting of glandular and diffuse parts (Amine-Precursor-Uptake-Decarboxylation, or APUD system) (**57**) and it has already established relationships between informons, which, in turn, are accountable to the thalamo-hypothalamic structures of the brain. Therefore, from our point of view, to understand the role of hormones in the etiology and pathogenesis of idiopathic scoliosis, it is always necessary to consider biological effects for antagonist pairs (for example, for GH and its functional antagonist cortisol, Csl). In addition, when analyzing the results obtained, it is necessary to take into account the circadian character in the functioning of both parts of the endocrine system (**69**).

Based on such information about the functioning of the endocrine system, in the period from 1987–1992, we obtained (RIA method) data on the content of two pairs of main osteotropic hormones in more than 400 children aged 10 to 14 years with idiopathic scoliosis of different severity and at the beginning of work – with an unknown nature of progression. The first pair was GH and its functional antagonist Csl, "responsible" for the organic matrix of bone tissue. The second pair is calcitonin (Ct) and its functional antagonist parathyrin (Pt), "responsible" for the mineral component of bone tissue. During the specified period, the levels of these hormones were repeatedly examined in the observed patients with scoliosis (on average, 2 times a year).

Upon acquisition of the first information, we were faced with two phenomena. The first is the high variance of the actual data, which, in fact, was identical to the results of E.N. Bakhtina (**6**). The second phenomenon – in almost all patients, the concentrations of the studied hormones did not go beyond the normal range specified in the annotations to the reagents. If the first phenomenon did not cause much alarm, then the second upset us. Until the mentioned annotations were analyzed. It turned out that the manufacturer of the reagents obtained normal values from 20–40 year old volunteers who had no signs of endocrinological pathology.

But is the age norm in children of puberty the same as in adults? Do children with scoliosis have signs of endocrinological pathology?

Negative answers to these questions made us optimistic. Since in the reference literature about adolescents we found isolated information about the age-related physiological norm of the concentration of the hormones we selected, it became necessary to find these norms ourselves in the control group. In order to preserve the norms of ethics, blood samples were used, taken according to objective indications from peers who had neither signs of endocrinological diseases, nor signs of scoliosis. Such a control group was formed by children who underwent preparation for planned surgical intervention for an umbilical or inguinal hernia, surgical treatment of clubfoot or improperly fused fracture, etc., which gave us the right to take the information received as the "norm" for adolescents.

After 5 years, all patients of the main group had a natural evolution of their scoliosis. In slightly more than half (<60%) of children, deformities did not progress at all, although among them there were patients with 20°–25° frontal arches (by Cobb), in 30–35% of cases there was an increase in curvature by 10°–15° Cobb's degrees, but the critical values of deformity were reached only in 35 (<10%) patients. It should be noted that in the last subgroup there were children who came under our observation with already expressed severe scoliosis and with progression of only 3–5 Cobb's degrees, their frontal arches quickly reached those critical values. Of this subgroup, 31 people agreed to a surgical correction.

As a result, it was possible to identify correlations between the characteristics of the osteotropic hormonal profile (diagram with the concentration indicators of all four of the above hormones) and the nature of the progression of pathological deformation of the spine (**figure 8**) (**22**).

Since hormones (in this case, the main osteotropic ones) are controlling informons, it is fair to note the connection: what is the level of hormones today, such will be the response of the performer tomorrow. In other words, the given variants of combinations in the content of osteotropic hormones acquire prognostic value.

From the standpoint of understanding the revealed role of GH, the conclusion of E.D. Wang et al. (**83**) based on the results of an attempt to include GH drugs in the scoliosis treatment protocol "We conclude that growth hormone may increase the risk of progression of scoliosis. Furthermore, the progression is frequently rapid and requires special vigilance by the treating physcian".

On the other hand, our findings fully explain the phenomenon of higher stature in children with scoliosis (**32, 92**), as well as the fact of activation of the progression of existing idiopathic deformities during periods of physiological growth spurts (**28, 29**).

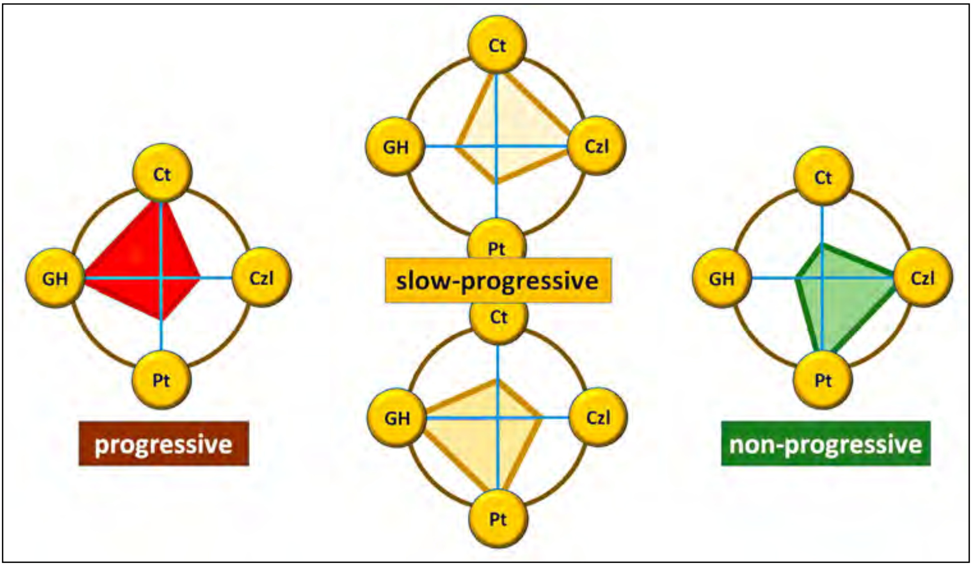


Figure 8: Four typical variants of the osteotropic hormonal profile in patients with different scoliosis progression. For ease of perception, we have kept the normative reference points indicated in the annotations to the reagents: the center in each diagram is the lower level of the endocrinological norm, and the circle is the upper limit of the same endocrinological norm for each hormone. Trapezium lines connect the actual concentration of the studied hormones in a particular patient.

In recent decades, the study of scoliosis has two directions. The first direction is determined by the fact that, against the background of increased attention to early diagnosis, revealing new aspects of the pathogenesis of this lesion, the number of cases with critical deformities remains quite large. Summarizing the experience shared by surgeons-vertebrologists at numerous conferences and congresses, today the best results in ensuring the quality of life of patients are obtained in those of them who underwent surgical correction with deformities of 45°–55° Cobb's degrees. At lower values of the frontal arches, the loss of spine mobility causes much greater inconvenience than a "beautiful" radiograph with 80–90% deformation correction. The authors of this article believe that it is for these patients with critical severity of scoliosis that the conclusion of the Research Committee of the American Orthopedic Association (**70**) remains relevant. Obviously, therefore, in modern operative vertebrology, work continues on the maximum adaptation of surgical techniques and implants used to the anatomy, biomechanics, and physiology of the deformed spine (**54**).

The second direction in modern scoliosology is clearly visible in the growing number of publications, which provide the results of studying the genetic factor, two most important directive systems (nervous and endocrine), as well as metabolic processes (osteogenesis), in which the employees of Charles Rivard Laboratories have been especially successful (Montreal, ULC Senneville, Quebec, Canada) (**80**). Interesting results were obtained when studying the influence of the so-called Factors of Postural Asymmetry (FPA) on the rotator muscles of the spinal column. Surprisingly, they turned out to be the neuropeptides oxytocin and arginine-8-vasopressin. It was previously established (**81**) that with a significant increase in their concentration, these natural regulators acquire properties with new biological effects. The discovery of these effects in patients with scoliosis (**48**) brings us closer to understanding two more phenomena – self-healing – an increase in "scoliosis of newborns" and a higher frequency of right-sided scoliosis in girls. By the way, most authoritative J.F. Dubousset himself agreed with the argumentation of our points of view on these phenomena, to whom they were presented in a private conversation back in 2014.

The undoubted expected result of these studies will be the identification of "weak" links in the pathogenesis of idiopathic scoliosis, some of which may turn into "targets" for conservative therapeutic intervention in order to interrupt the process of deformation of the spinal column.

CONCLUSION

It is quite natural that throughout the history of the study of scoliosis, various theoretical views on its etiology and pathogenesis were formed. On this basis, several sufficiently reasoned hypotheses have been proposed about the nature of this pathological deformity of the spinal column. Which of their authors is right? We allow ourselves to assert that everything. It should be noted that a common drawback of these hypotheses is the "reliance" on only one factor – myogenic, genetic, hormonal, dystrophic, vascular, neurogenic, etc. The number of these factors so far allows us to draw only one conclusion – scoliosis is a polyetiological lesion. But why, with all the agreement with this conclusion, is it (scoliosis) monoform in the form of a pathological three-plane deformity? How can this paradox be explained?

We hope that readers will forgive our certain immodesty, but in conclusion, we want to share our thoughts on the nature of idiopathic scoliosis. It seems that we are lucky. The first "thank you" we have to say to M. Roth, who in the 70ˢ of the XXᵗʰ century "reminded" all of us that the vertebral complex consists of two completely heterogeneous formations. The second "thank you" is addressed to the case of acquaintance with the specialists of theoretical mechanics, who convincingly, on the basis of impassive mathematics, showed that 3D deformations can occur only in two-column structures (models), in which the different sizes of columns appear. But this fact underlies the hypothesis and the real spring-string model of scoliosis by M. Roth. This raised the following question: why does the indicated difference in size appear in a healthy vertebral complex (disproportion – according to M. Roth)? The answer to this question has made us pay attention to the growth process of the bone-disco-ligamentous-muscular "sheath" of the spinal cord. As reflected above, the theoretical foundations of this process justified the choice of the main osteotropic hormones as objects of study. As a result of many years of research, strong correlations were obtained between their levels and the axial growth of the skeleton in general and the sheath of the spinal cord in particular. The clinical manifestation and illustration of these connections are the findings of the works of G.G. Epshtein and Zhen Liu (**32, 92**). After all, hormones that stimulate bone growth (GH and Ct) "work" in all areas of bone growth in the axial skeleton of a growing child.

But what about the spinal cord? With the introduction of CT and MRI diagnostic technologies into clinical practice, it became possible to obtain information about this part of the central nervous system, but they reflected only its anatomy. Perhaps that is why, among the significant results of assessing the state of the spinal cord in children with scoliosis, there was the detection of rather gross lesions such as syringomyelia, diastematomyelia, fixed spinal cord and some others (**65, 78**). But here it should be emphasized that the vast majority of children with scoliosis do not have such gross violations of the anatomy of the spinal cord. Strictly speaking, M. Roth saw the reason for the medullo-vertebral in the simple non-conjugation of the longitudinal growth of the two main components of the vertebral complex, which in fact is a violation of its homeostasis.

Meanwhile, classical neurophysiology testifies that the most important condition in maintaining spinal cord homeostasis is the correspondence between its anatomical dimensions and the same dimensions of its "sheath". If this condition is violated (for example, with an unpredictable seasonal or other growth spurt in the axial skeleton), afferentation of the thalamo-hypothalamic central structures inevitably occurs from the receptor fields of the spinal membranes and the periosteum of the spinal canal (**23, 56**).

In response to such stimuli, these structures "trigger" adaptive mechanisms (**4**). Based on modern knowledge, in this case, the main of these mechanisms can be considered:

- correction of hormonal regulation of osteogenesis (reduction of GH synthesis and activation of the pituitary-adrenal system in order to increase the synthesis of Csl).
- activation of the production of nerve growth factor, NGF (discovered in the 50s of the XXᵗʰ century by Rita Levi-Montalcini /1909–2012/ and Stanley Cohen /1922–2020/, Nobel Prize 1986).

If these answers are enough, then the problem of disproportionate growth (by M. Roth) between the main components of the vertebral complex will be solved.

We made an attempt to register the aforementioned increased afferentation, but due to the individual characteristics of the examined children and the large variance of the data, as well as due to the lack of criteria for the norm, it was unsuccessful.

W.C. Chu et al. (**15**) and M. Deng et al. (**20**) were more successful, who, during a six-month follow-up, found an increase in the difference between the longitudinal dimensions of the spinal cord and its "sheath" in children with scoliosis. According to these authors, this fact is a diagnostic criterion for the progressive development of scoliotic deformity. Note that the information provided added to us confidence that we are on the right track.

Therefore, in order to "bypass" the problem of identifying the response from the spinal cord, which is unsolvable today, we used the results of biomechanical modeling of three-plane deformation on a two-column model. The **pre**- and **sub**clinical stage of transition of a healthy spinal column to a deformed state revealed with its help aroused attention to the muscles serving the vertebral complex. After all, only they are capable of leading to displacement in the spinal motion segments (**25**).

It turned out that after the **pre**clinical stage, which consists in filling the reserves of physiological bends (primarily thoracic kyphosis) with excess osteogenesis and the formation of **flat back syndrome**, unilateral bioelectric activity of the paravertebral muscles appears in the caudal zone of the spine. This was consistently recorded in children with flat back syndrome. The result of this muscle imbalance was the torsion of the spine with the effect of a "spiral staircase" with a maximum rotation of the uppermost "step", which manifested itself in a violation of the parallelism of the frontal axes of the pelvic and shoulder girdles. At the same time, the optical axis of the eyes shifted.

But why does a real scoliotic arch consist of two countercoils?

The "clue" was obtained in the works of the French ophthalmologist J.B. Baron, who in the distant 50^{s} of the XXth century discovered a high asymmetric bioelectrical activity of the right vertebral muscles in patients who had a displacement of the optical axis of more than 4° (!) (**7, 8, 9**)

It turns out that the previous compensatory response to the disproportionate growth of the spinal cord and its "sheath" in the form of torsion of the spine caused a new compensatory response to maintain the physiological normal position of the optical axis, the position of the head and shoulder girdle. The result of this response, which is realized by the paravertebral muscles (they are contralateral to the muscles of the same name, which caused the primary torsion of the spine), is the formation of a countercurrent in the cranial zone of the vertebral complex.

Then comes the time for a process called the "vicious circle" that I.A.F. Stokes brilliantly described (**74, 75**). True, he always emphasized that the implementation of this process requires a primary violation of the verticality of the spinal column. It looks like we managed to find its cause.

Now we can return to existing hypotheses and views on the etiology and pathogenesis of idiopathic scoliosis.

Myogenic hypothesis (Ἱπποκράτης βήτα, A. Parè, N. Andry, J.M. Delpech). In our scheme of patho- and mechanogenesis, it can be seen that it is they who displace the vertebrae in the horizontal plane, forming two countercoils in the spine. The first, at the preclinical stage of the transition of a healthy spinal column to a deformed state, and the second, as a second-order compensatory reaction, restores the physiological position of the cranial part of the entire trunk (pleural girdle and head, which ensures the correct direction of the optical axis).

Genetic hypothesis (A.M. Zaidman 1994; P.F. Giampietro et al., 2003; C. Raggio et al., 2009). There is no doubt that organic and functional deviations in the work of all directive systems and, first of all, in the nervous and endocrine systems can become one of the consequences of violations in the genetic code. As, however, and in the executive organs at the tissue level. An example of this is the results of studying the VitD receptor, the gene of which is located in the short arm of the XIIth chromosome and is characterized by high polymorphism. A "high-quality" receptor in bone tissue, even at low doses of calcitriol, provides a high activity of osteogenesis, and if its quality is poor, the biological effect of VitD will be the same.

Melatonin and neurohormonal hypotheses (M-J. Thillard et al., 1959; M. Machida et al., 1996; A. Moreau et al., 2004; T.B. Grivas, O.D. Savvidou, 2007). Our studies of melatonin allowed us to see not so much its direct metabolic role as the role of a pacemaker. Indeed, at night, inhibiting the biological effect of Csl, it "gives" complete freedom to GH (**38, 69**). Therefore, with a high activity of the neurohormonal pineal gland during the day (we discovered this phenomenon using 3DLocEEG (**10, 11, 24**), it is quite acceptable to continue stimulating osteogenesis not only at night (children grow up in a dream), but also during the day. What is behind this? A disrupted genetic code or a malfunction in the organogenesis of the gland itself? Or maybe there is a functional insufficiency of its regulation by the thalamo-hypothalamic structures?

Central (CNS) genesis of scoliosis (G.M. Jensen, K.B. Wilson, 1979; I. Petersen, T. Sahlstrand, U. Sellden, 1979; R. Herman et al, 1985; P. Rogala, J. Huber, A. Nowakowski, 1998). And one should agree with this view. Firstly, the spinal cord itself is a part of the central nervous system, and secondly, its development and maturation is determined both by the central neural structures of the brain and by glial cells. To this should be added various congenital and acquired abnormalities in the synthesis of the most important regulators of axon growth – numerous NGFs. After all, the main part of the spinal cord is the axons, which make up the ascending and descending (afferent and efferent) conducting tracts.

Vegetative-vascular dysfunctions (J.A. Sevastic, 2002; R.G. Burwell, P.H. Dangerfield, 2006). We fully agree with the opinion that the vegetative-vascular factor plays a role in the pathogenesis of idiopathic scoliosis. And as the initiator of the delay in the longitudinal growth of the spinal cord (and as one of the "weak" links in the most complex process of adaptive (compensatory) response to medullo-vertebral conflict (**1, 94**).

Variants of metabolic disorders (Z.A. Lyandres, L.K. Zakrevsky, 1967; V.A. Pedrini, I.V. Ponseti, C.S. Dohrman, 1973; A.I. Kazmin, V.Ya. Fischenko, 1974; D.B. Parsons et al., 1982; K. Kindsfater et al., 1994). And we agree with this point of view, since such violations can be initiated both by deviations in the directive (regulatory) systems of the body, and high-quality alimentary surpluses. If these circumstances (reasons) cause a disturbance in the normal physiological balance between the pools of osteoblasts and osteoclasts in favor of the former (i.e., lead to a significant predominance of osteosynthesis), then the initiation of the same medullo-vertebral conflict is quite likely. Therefore, the revealed metabolic disorders can be both an objective indicator and the cause of the transition of a healthy spinal column to a deformed state.

Neurodysplastic genesis (E.A. Abalmasova, A.V. Kogan, 1965; V.D. Chaklin, A.A. Korzh, B.I. Simenach, 2004). As can be seen from the list of authors of this view, this view is quite popular in Russia, which is directly related to the detection in the overwhelming number of patients with scoliosis of the so-called. "Status disraficus", or "Bremer's syndrome", it was first described by the German neurologist Friedrich Wilhelm Bremer /1894 –?/ in the 20[s] of the last century (**12**). The classic description of this syndrome directly indicates the anatomical and functional features in the spinal cord. And it is quite likely that one of them is a simple delay in the longitudinal growth of this part of the central nervous system. And already this delay (in relation to the growth rate of his bone-disco-ligamentous-muscular "sheath") "starts" the whole chain of processes that compensate for the arisen medullo-vertebral conflict.

This list does not mention the hypothesis of **rickets genesis** of scoliosis. The fact is that in the small work of the Russian scientist-histologist Madame E.N. Yaroshevskaya (1957) (**87**) convincingly showed a radical difference in the state of bone tissue in children with rickets and with idiopathic scoliosis. On the other hand, we believe that there is no need to argue for the fact that rickets (including all the so-called D-resistant it variants) is manifested in a violation of normal osteogenesis and a lag in the longitudinal growth of the axial skeleton. In the vertebral complex this condition also leads to a medullo-vertebral conflict, but its result will be a completely different variant of three-plane deformity – kyphoscoliosis. This is determined by the fact that the spinal cord will have a relative excess of the longitudinal size (**24**).

The hypothesis of a "short" spinal cord (M. Roth 1965). We believe that almost the entire last half of this article is a commentary for this view of the origin of scoliosis.

The "vicious circle" hypothesis (I.A.F. Stokes, D.D. Aronsson, 2001) and the adjacent biomechanical pathogenesis of AIS by Tomasz Karski (**45, 46, 47**), as well as the "forgotten" but living hypothesis of the school genesis of scoliosis are based on Delpech – Hueter – Von Volkmann – Wolff Law implementation. In accordance with this law, any change in the load on the growth zones of the vertebral bodies (violation of the verticality of the spine according to I. Stokes, or deviations in the position of the pelvis due to imbalance of pelvis-femoral muscle according to T. Karski) invariably lead to plastic disturbances in their anatomy. But at the same time, we note that "scoliosis in a schoolchild does not arise because they are not sitting correctly, they are sitting incorrectly because they have begun to develop scoliosis"

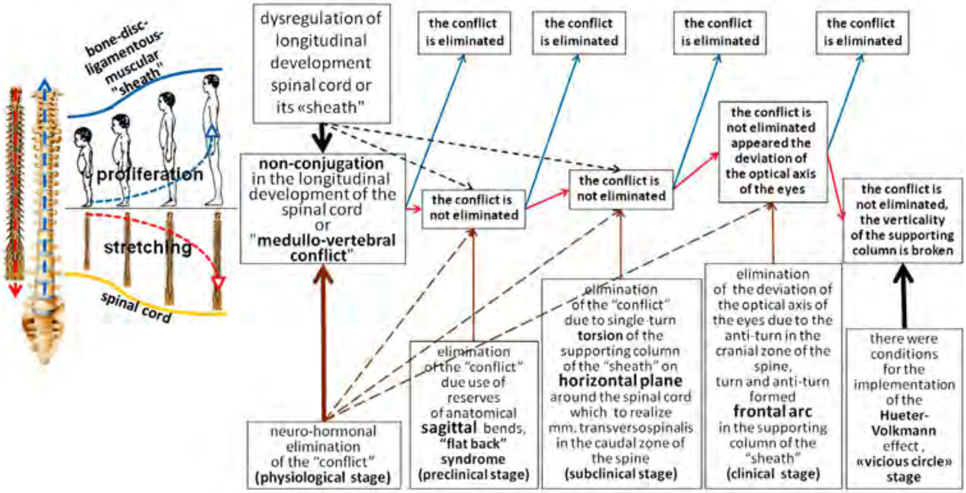


Figure 9: Pathogenesis of lordoscoliosis and the stages of the transition of a healthy spinal column to the "scoliotic" status.

Summarizing all of the above, we offer as a "skeleton" the following scheme of the pathogenesis of lordoscoliosis and the stages of the transition of a healthy spinal column to the "scoliotic" status (**figure 9**).

So, from our point of view, based on a comprehensive analysis of information about three planar deformities of the spine, scoliosis is a clinical manifestation of the adaptive response of a two-component segment of the axial skeleton to the arisen medullo-vertebral conflict. It, this conflict, can be only in a growing person[10]. Its essence is the discrepancy (non-conjugation) between the rates of longitudinal growth in the spinal cord and in its bone-disco-ligamentous-muscular "sheath". As a result, two variants of the relationship may appear. The first is the excess "sheath" length, and the second, on the contrary, is the excess spinal cord length.

There is a fairly long list of reasons for the occurrence of any of these variants of non-conjugation. From congenital and acquired defects in the implementation of the genetic code and the same genesis of dysfunctions in other control systems of the body. Let us dwell on the development of the reaction to the first variant of the medullo-vertebral conflict, since its (of the reaction) result is a typical "lordoscoliosis".

[10] The physiology of a growing child allows us to assert that this conflict is observed in him every day, which is associated with the fact that the axial skeleton predominantly grows at night and during the daytime "correction" if it turns out to be excessive.

The first clinical sign of tension between the two components of the spine is flat back syndrome. This is a **pre**clinical stage as a result of filling the "reserves" of physiological sagittal bends with an excess of "sheath" length.

If the possibilities of reserves are insufficient, then compensation for non-conjugation enters the second stage – **sub**clinical. It manifests itself in the form of a one-sided torsion of the spine with a "spiral staircase" effect, as a result of which the optical axis and the frontal position of the shoulder girdle are disrupted.

It should be emphasized that at any moment the unfortunate conflict can be eliminated and subsequent events will not take place.

Meanwhile, unfortunately, the described result of the subclinical stage necessitates the correction of the position of the optical axis of the eyes and the shoulder girdle. And as described above, there is a need to form a counterturn, which forms one frontal arc with the first turn. The clinical stage is coming, in which one of the key roles will be played by "circulus vitiosus"

It should be emphasized that in this case only lordoscoliosis is considered as a compensatory reaction of the spine to the medullo-vertebral conflict due to the relative excess of the longitudinal size of the "sheath" of the medulla spinal. The second variant of three-plane bone deformity in the form of kyphoscoliosis requires a personal description.

REFERENCES

1. ADAMKIEWICZ, A.: Die Blutgefässe des menschlichen Ruckenmarkes. I. Teil: Die Gefässe der Rückenmarkssubstanz. Sitz. Ber. Math. Naturwiss. Classe Kaiserl. Akad. Wissensch. 1881:84: 469, Google Scholar
2. ALBEE F.H. Transplantation of a portion of the tibia into the spine for Pott's disease: a preliminary report. JAMA. 1911: LVII(11):885–886.
3. ANDRY DE BOIS-REGARD N. Orthopædia: or, the art of correcting and preventing deformities in children: by such means, as may easily be put in practice by parents themselves, and all such as are employed in educating children. To which is added, a defence of the orthopaedia, by way of supplement /by the author/. In two volumes. Translated from the French of M. Andry. London: Printed for A. Millar. 1743: In French edition 1741 Ist vol. 345 p., IInd vol. 365 p.
4. ANOKHIN P.K. Key questions of the theory of functional systems. Moscow: Science. 1980: 197.
5. ASHER M.A., BURTON D.C. Adolescent idiopathic scoliosis: natural history and long term treatment effects. Scoliosis. 2006; 1: 2. Published online 2006 Mar 31. doi: 10.1186/1748-7161-1-2
6. BAKHTINA E.N. Features of puberty in patients with dysplastic scoliosis. Abstract of the thesis diss... cand. med. scienc. Moscow, Central Research Institute of Traumatology and Orthopedics (CITO). Ed. CITO. 1990:19.
7. BARON J.B. Muscles moteurs oculaires, attitude et comportement locomoteur des vertébrés. Thèses de Sciences. Paris. 1955: 158.
8. BARON J.B. Muscles moteurs oculaires, céphalées, déséquilibre et attitude scoliotique. Presse med. 1955: vol. 63, № 20. 407–410.

9. BARON J.B., USHIO N., NOTO R. Oculo-nuco-vestibulo-spinal system regulating tonic postural activity; statokinesimetric study. Agressologie. 1974: № 15: 395–400.
10. BEKSHAEV S.S. Individual-typological features of the statistical structure of the interaction of the rhythms of the human electroencephalogram and its dynamics in the process of adaptation to extreme environmental conditions: Abstract of the thesis diss.... cand. biol. sciences. Inst. Experiment. Medic. Russ. Acad scienc. Leningrad. 1983: 24.
11. BEKSHAEV S.S. Program: "Three-dimensional localization of electrical sources of the brain that generate the spatio-temporal profile of the electroencephalogram" ("3DLocEEG"). State Registration No. 2002611116 dated 02.07.2002.
12. BREMER F.W. Klinische Untersuchungen zur Atiologie der Syringomyelie der "Status Dysraphicus". Deutsche Ztschr. Nervenh. 1926:Bd.95:1–103.
13. ČERNÝ P., MAŘÍK I. The axial pelvis rotation assessment according to the original radiographic method with the use of 3d pelvis model. The 17th Prague-Lublin-Sydney-St.Petersburg Symposium. 24–26 Sept. 2015. – Kroměříž. Czech Republic. www.angelespine.com.
14. CHEUNG J., HALBERTSMA J.P., VELDHUIZEN A.G., SLUITER W.J., MAURITS N.M., COOL J.C., VAN HORN J.R. A preliminary study on electromyographic analysis of the paraspinal musculature in idiopathic scoliosis. J Euro Spine. 2005:14(2): 130–13
15. CHU W.C., MAN G.C., LAM W.W., YEUNG B.H., CHAU W.W., NG B.K., LAM T.P., LEE K.M., CHENG J.C. Morphological and functional electrophysiological evidence of relative spinal cord tethering in adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2008 Mar 15: 33(6):673-80. doi: 10.1097/BRS.0b013e318166aa58.
16. COBB J.R. Outline for the study of scoliosis. In: Edwards J.W., editor. AAOS, Instructional Course Lectures. Vol. 5. Ann Arbor: The American Academy of Orthopaedic Surgeons. 1948: 261-75. [Google Scholar]
17. COTREL Y. Traitement des scolioses essentielles. Rev Chir Orthop. 1957: 43: 331–343
18. DE BUFFON G.-L. L. in the book Kharitonov V.M., Ozhigov A.P., Godin E.Z. Anthropology. Textbook for universities: Vlados. 2004: 272. ISBN 5-691-01068-9
19. DELPECH J.M. De l'Orthomorphie, par Rapport a L'Espece Humaine avec atlas. Paris, Gabon, 1828. In 2 vol. (French). Copy of the 1828 edition of the Barvarian library in German. Editions Medicina Rara. 1980
20. DENG M., HUI S.C., YU F.W., LAM T.P., QIU Y., NG B.K., CHENG J.C., CHU W.C. MRI-based morphological evidence of spinal cord tethering predicts curve progression in adolescent id-iopathic scoliosis.Spine. 2015 Jun 1: 15(6):1391-1401. doi: 10.1016/j.spinee.2015.02.033. Epub 2015 Feb 25.
21. DUDIN M.G., SINITSKIY YU.F. On the mechanogenesis of torsional changes in scoliosis. J. Orthopedics, traumatology. Prosthesis (USSR). 1981: № 2: 33–36.
22. DUDIN M.G. Particular qualities of hormonal regulation of metabolic processes in the bone tissue as etiopathogenic factor of idiopathic scoliosis. Diss... Doctor of medical. Sciences. Saint Petersburg: Institute of Traumatology and Orthopedics by R.R. Vreden. 1993: 195.
23. DUDIN M.G., PINCHUK D.YU. Idiopathic scoliosis: diagnosis, pathogenesis. Saint Petersburg. Man. 2009: 336.
24. DUDIN M.G., PINCHUK D.YU. Idiopathic scoliosis: neurophysiology, neurochemistry. Saint Petersburg. Man. 2013: 304.7
25. DUDIN M.G., BALOSHIN YU.A., POPOV I.YU. et al. Mathematical modelling of three-plane deformity of the human spinal column. Russian Journal of Biomechanics. 2016: vol. 20, № 3: 272–282.
26. DUDIN M.G. Idiopathic scoliosis: prevention, conservative treatment. Saint Petersburg. Man, 2017: 223.

27. DUDIN M., PINCHUK D., MINKIN YU., BALOSHIN YU., POPOV I., ZUBKOV M., POMORTSEV I., BOBERS., UZDENNIKOVA M., LARIONOV N. AIS: Etiology, Pathogenesis. Facts and Reflections. Int J Ortho Res, 2020: vol 3(2):1–13. DOI: doi.org/10.33140/IJOR.03.02.01.
28. DUVAL-BEAUPÈRE G. [Maturation indices in the surveillance of scoliosis]. Rev Chir Orthop Reparatrice Appar Mot. févr 1970:56(1):59–76.
29. DUVAL-BEAUPÈRE G., DUBOUSSET J., QUENEAU P., GROSSIORD A. Pour une théorie unique de l'évolution des scolioses. [A unique theory on the course of scoliosis] Presse méd. 1970: 88: 1141–1146.
30. Edwin Smith's Surgical Papyrus: Hieroglyphic Transliteration, Translation and Commentary James Henry Bristed (editor): 1922, New York Historical Society
31. ENGVALL E., PERLMANN P. Enzyme-linked immunosorbent assay (ELISA). Quantitative assay of immuno-globulin G: Immunochemistry. 1971: 8(9):871-874. doi: 10.1016/0019-2791(71)90454-x.
32. EPSHTEIN G.G. Peculiarities of growth of the spine in idiopathic scoliosis. In: Diseases and injuries of the spine in children: inter-institutional collection of scientific articles. Leningrad. 1981:18–26.
33. EULENBURG A. Real Encyclopedia Of Practical Medicine. Medical – Surgical Dictionary for Practical Physicians. Translation from a completely revised-tango German edition, with additions by Russian authors. Edited by Priv.-Assoc. Imper. Military-Med. Academy of M.B. Blumenau. In 17 volumes. The medicine. St. Petersburg. Publication of the journal "Practical Medicine" (V.S. Ettinger). 1909–1915: vol. XV: 303–503.
34. FAIRBANK J.C.T. William Adams and the spine of Gideon Algernon Mantell: Ann R Coll Surg Eng. 2004: 86: 349-352. doi 10.1308/147870804245
35. GAGMAN N.F. Semiotics of Orthopedics. From lectures for students of higher. courses priv.-assoc. Moscow un-that Dr. med. N.F. Gagman, Director Orthopedist. establishments in Moscow... Moscow: Printing house of M.I. Neubürger. 1888:
36. GARLAND H.G. Hereditary scoliosis. Br Med J. 1934:1(3816):328.
37. GRAUERS A. Inheritance and Genetics in Idiopathic scoliosis. Thesis for doctoral degree (Ph.D.) From the Department of Clinical Science, Intervention and Technology (CLINTEC), Division of Orthopedics and Biotechnology Karolinska Institutet, Stockholm, Sweden: Published by Karolinska Institutet. Printed by AJ E-print AB. 2015: 58. ISBN 978-91-7549-904-8
38. GRIVAS T.B., SAVVIDOU O.D. Melatonin the "light of night" in human biology and adolescent idiopathic scoliosis. Scoliosis. 2007: 2: art.#6. https://doi.org/10.1186/1748-7161-2-6
39. HALL B.K. Bones and Cartilage. Development and Evolution Skeletal Biology. 2nd edition: Academic Press is an imprint of Elsevier: 2015: 891.
40. HARMS J. History of scoliosis therapy. www.harms-spinesurgery.com 2007–2013.
41. HARMS J. RAUSCHMANN M., RICKERT M. Therapy of scoliosis from a historical perspective Unfallchirurg. 2015: Dec;118 Suppl 1:28-36. doi: 10.1007/s00113-015-0097-5.
42. HIBBS R.A. An operation for progressive spinal deformity: a preliminary report of three cases from the service of the orthopaedic hospital. NY Med J. 1911:93:1013-1016.
43. HIPPOCRATES. Selected books. Translated from Greek by prof. V.I. Rudnev. Editorial introductory articles and notes by prof. V.P. Karpov. Moscow: State Publishing House of Biological and Medical Literature. Series "Classics of Biology and Medicine" 1936: 736.
44. KANE W.J. Scoliosis prevalence: a call for a statement of terms. Clin Orthop Relat Res. 1977 Jul–Aug;(126):43–46.
45. KARSKI T. Biomechanical Aetiology of the So-called Adolescent Idiopathic Scoliosis (AIS). Lublin Classification (1995–2007). Causative Influences Connected with "Gait" and "Standing 'at ease' on the Right Leg". J Orthop Bone Res (USA), Scholarena, 2018. vol. 1: 1–10.

46. KARSKI T. Etiology of the so-called "idiopathic scoliosis". Biomechanical explanation of spine deformity. Two groups of development of scoliosis. New rehabilitation treatment; possibility of prophylactics. Stud Health Technol Inform. 2002:91:37–46 PMID: 15457691
47. KARSKI T. Explanation of biomechanical etiology of the so-called idiopathic scoliosis (1995 – 2007). New clinical and radiological classification. Locomotor System (Czech Republic) vol. 17, 2010: №1+2: 26 – 42
48. KHAIMINA T., AVALIANI T., KARPENKO M., DUDIN M., PINCHUK D. Factors of Postural Asymmetry (FPA) At The Pre- and Subclinical Stages of the Development of 3D Deformity of the Vertebral Complex. Research Article. Int J Ortho Res, 2020: Volume 3 | Issue 3 | 111–117. Submitted: 04 Aug 2020; Accepted: 10 Aug 2020; Published: 20 Aug 2020
49. KLADNY B. History of conservative spinal therapy Unfallchirurg. 2015 Dec;118 Suppl 1:12–18. doi: 10.1007/s00113-015-0098-4.PMID: 26542053 German.
50. LERNER A.B., CASE J.D., TAKAHASHI Y. Isolation of melatonin and 5-methoxyindole-3-acetic acid from bovine pineal glands". The Journal of Biological Chemistry. 1960 Jul: 235: 1992–1997. PMID 14415935.
51. LONSTEIN J.E. Adolescent Idiopathic Scoliosis. Lancet. 1994: vol. 344 (iss. 8934):1407–1412.
52. LÜFTINGER M. Aetiology of idiopathic scoliosis: Current biomedical research and osteopathic theories / Master Thesis zur Erlangung des Grades Master of Science in Osteopathie / an der Donau Universität Krems niedergelegt an der Wiener Schule für Osteopathie: Wien, Juni 2008: 76.
53. MIKHAILOVSKY M.V. FOMICHEV N.G. Surgery of spinal deformities. 2nd ed., Rev. and add. – Novosibirsk: Redactio, 2011: 592.
54. MIKHAILOVSKY M.V., DUBOUSSET J.F. Scoliosis: history of the disease. Novosibirsk: Kostyukova. 2020.422.ISBN 978-5-6041549-4-6
55. NASH C.L.JR., MOE J.H. A study of vertebral rotation. J Bone Joint Surg. 1969: 51(2): 223–229.
56. OTELIN A.A. Innervation of the human skeleton. Moscow: Medicine: 1965: 250.
57. PEARSE A. G. The cytochemistry and ultrastructure of polypeptide hormone-producing cells of the APUD series and the embryologic, physiologic and pathologic implications of the concept. J Histochem Cytochem. 1969 May:17(5):303–313. doi: 10.1177/17.5.303. PMID: 19048801 French.
58. REY J.C. History of the treatment of scoliosis. Hist Sci Med. 2008 Jan-Mar:42(1):21–28.
59. RIDDLE Y.F.V., ROAF R. Muscle imbalance in scoliosis: Lancet: 1955 Jun 18: 268(6877): 1245–1247.
60. RISEBOROUGH E.J., WYNNE-DAVIES R. A genetic survey of idiopathic scoliosis in Boston, Massachusetts. J Bone Joint Surg. (Am). 1973:55(5):974–982.
61. ROTH M. Idiopathic scoliosis caused by short spinal cord. Acta Radiologica Diagnosis, 1968: vol.7 №3: 257–271.
62. ROTH M. Idiopathic scoliosis: a special type of osteo-neural growth disproportion. Z. Orthop Ihre Grenzgeb., 1969: 107(1): 37–46.
63. ROTH M. Idiopathic scoliosis and Scheuermann's disease: essentially identical manifestations of neuro-vertebral growth disproportion. Radiol Diagn (Berl) 1981:22(3):380–391. [PubMed] [Google Scholar]
64. ROTH M. Neuro-vertebral and osteo-neural growth relations. A concept of normal and pathological development of the skeleton. J.E. Purkyně University Brno. Medical Faculty: Acta Facultatis Medicae Universitatis Brunensis: 1985: 4.
65. ROYO-SALVADOR M.B. Siringomielia, escoliosis y malformación de Arnold-Chiari idiopáticas. Etiología común. Rev Neurol (Barc.), 1996: 24(132): 937–959.
66. SADOVAYA T.N. Screening, monitoring and organization of specialized orthopedic care for children with spinal deformities: Abstract of the thesis.... doc. med. sciences. SPb. 2010:46.

67. SCAMMON R.E. The ponderal growth of the extremities of the human fetus. In American Journal of Physical Anthropology, 1930: Vol.15, issue 1: 111–121.
68. SCHEUERMANN H. W. Kyphosis dorsalis juvenilis. Ugeskrift for Læger (in Danish). –1928: 82: 385–93. Republished as: Scheuermann H.W. The classic: Kyphosis dorsalis juvenilis. Clinical Orthopaedics and Related Research, 1977: 128 (128): 5–7. PMID 340099.
69. SCHMIDT R., TEVS G. Human physiology. Translation from English. In 3 volumes. Moscow: Peace. 1996: 877.
70. SHANDS A.R.JR., BARR J.S., COLONNA P.C. et al. Endresult study of the treatment of idiopathic scoliosis: Report of the Research Committee of American Orthopedic Association. J Bone Joint Surg. (Am.), 1941: 22: 963–977.
71. SHERRINGTON CH.S. The Correlation of Reflexes and the Principle of the Common Path // Reports of the British Association for the Advancement of Science, 1904, vol. 74:728–741.
72. SOURNIA J.-CH., POULET J., MARTINY M. (Hrsg) Illustrierte Geschichte der Medizin. Cd-rom. Deutsche Bearbeitung unter der Leitung von Richard Toellner. Digitale Bibliothek Volume 53. Berlin: DirectMedia, 2001.
73. STAFFEL F. Die menschlichen Haltungstypen und ihre Beziehungen zu den Ruckgrat-verkrummungen. J. F. Bergman, Wiesbaden. 1889: 104.
74. STOKES I.A.F., SPENCE H.E., ARONSSON D.D. et al. Mechanical modulation of vertebral body growth. Implications for scoliosis progression. Spine (Phila Pa 1976), 1996: № 21:1162–1167
75. STOKES I.A.F. Hueter-Volkmann Effect. Spine: State of the Art Reviews, 2000: № 14: 349–357.
76. THILLARD M.-J. Vertebral column deformities following epiphysectomy in the chick. CR Hebd Seances Acad Sci. 1959; 248:1238–1240. [PubMed]
77. TSIVYAN YA.L. Scoliotic disease and its treatment. Medicine., Uz. SSR., Tashkent., 1972: 223..
78. ULRICH E.V. Expanding spinal cord syndrome (diastematomyelia). St. Petersburg. Synthesis Beech. 2012:125.
79. VAN LOON PIET J.M. Scoliosis Idiopathic? The Etiologic Factors in Scoliosis Will Affect Preventive and Conservative Therapeutic Strategies. In Recent Advances in Scoliosis. Editor Theodoros Grivas. IntechOpen. 2012, article #13: 211–236. Published: May 9th 2012. DOI: 10.5772/38199
80. VARELA A., JOLETTE J. Bone Toolbox: Biomarkers, Imaging Tools, Biomechanics, and Histomorphometry. In Toxicologic Pathology. 2018: vol. 46, 5: 511–529. First Published July 4, 2018. Review Article. Find in PubMed https://doi.org/10.1177/0192623318779565
81. VARTANIAN G.A., VARLINSKAYA E.I., KLEMENT'EV B.I., TOKAREV A.V., CHERNYAEV S.T., et al. Strukturospetsifichnost factors of chemical regulation of muscle tone at the level of the spinal cord. Bull. Exp. Biol. and Med. 1989: №3: 268–270
82. VON VOLKMANN R. Neue Beitrage Zur Pathologie Und Therapie Der Krankheiten Der Bewegungsorgane: Separat-Abdr. Aus Der Berliner Klinischen Wochenschrift, 1867 u. 1868, (German Edition) (German). Publisher: Nabu Press (January 24, 2012) Paperback 86 pages.
83. WANG E.D., DRUMMOND D.S., DORMANS J.P., MOSHANG T., DAVIDSON R.S., GRUCCIO D. Scoliosis in patients treated with growth hormone: J Pediatr Orthop. 1997: 17(6):708–711.
84. WEINER M-F., SILVER J.R. Paralysis as a result of traction for the treatment of scoliosis: a for-gotten lesson from history. Spinal Cord. 2009: 47:429–434; doi:10.1038/sc.2009.19; published online 7 April 2009.
85. WOLFF J. Das Gesetz der Transformation der Knochen. Herausgegeben mit Unterstützung der Königlichen Akademie der Wissenschaften in Berlin: Berlin, A. Hirschwald, 1892.
86. WYNNE-DAVIES R. Familial (idiopathic) scoliosis. A family survey. J Bone Joint Surg (Br). 1968;50(1):24–30.
87. YAROSHEVSKAYA E.N. Changes in the spine with rickets in young children. J Orthop. Traumatol. and Prosthetics. 1957: № 1: 35–39.

88. ZAIDMAN A.M. Idiopathic scoliosis, morphology, histochemistry, genetics. Novosibirsk. Ed. Novosibirsk State University. 1994: 234.
89. ZAIDMAN A.M., RUSSOVA T.V. KOREL A. V., RYKOVA V. I. Experimental model of spinal deformity: J Bull. of traumatology and orthopedist Priorova. 2003: № 4: 29–73.
90. ZAIDMAN A.M. ALEKSINOVICH T.I. SADOVOY M.A., TREGUBOVA I.L., SHARIPOV R.N. The mechanism of inheritance of idiopathic scoliosis. J Spine surgery. 2005: № 1: 112–121
91. ZAYDMAN A.M., KOREL A.V., RYKOVA V.I., GRIGORIEVA E.V., ESHCHENKO T.YU., SADOVAYA T.N. Etiology and pathogenesis of idiopathic scoliosis: J Spine surgery. 2006: № 4: 84–93
92. ZHEN LIU, ZEZHANG ZHU, JING GUO, SAIHU MAO, WEIJUN WANG, BANGPING QIAN, FENG ZHU, WINNIE CHU, JACK C.Y. CHENG, YONG QIU. Analysis of body growth parameters in girls with adolescent idiopathic sco-liosis: single thoracic idiopathic scoliosis versus single lumbar idiopathic scoliosis: In Technology and Informatics. Research into Spinal Deformities 8 (Editors T. Kotwicki and T.B. Grivas): IOS Press. V. 176. 2012:195–201.
93. ZUK T. The role of spinal and abdominal muscles in pathogenesis of scoliosis. J Bone Joint Surg. 1962: 44B: 102–105.
94. ZÜLCH K.J., KURTH-SCHUMACHER R. The Pathogenesis of "Intermittent Spinovascular in Sufficiency" ("Spinal Claudication of Dejerine") and Other Vascular Syndromes of the Spinal Cord. Research Article. J Vascular and Endovascular Surgery. 1970: V.4, iss. 2: 116–136. First Published June 1, 1970. https://doi.org/10.1177/153857447000400206

Corresponding author's address:
Prof. Mikhail Georgiyevich Dudin, MD, DSc
Children's Rehabilitation Center of Orthopedics and Traumatology 'Ogonyok', St. Petersburg, Russia
E-mail: dudin@list.ru

IDIOPATICKÁ SKOLIÓZA: PREVENCE, KONZERVATIVNÍ LÉČENÍ

IDIOPATHIC SCOLIOSIS: PREVENTION, CONSERVATIVE TREATMENT

Dudin M.G., Pinchuk D.Yu., Pankratova G.S.

Saint Petersburg, Russia, E-mail: mdudin@list.ru

RESUME

The article is devoted to a fundamentally new approach in the "fight" with idiopathic scoliosis (IS). It is based on the use of the most indisputable fact in the theory and practice of this lesion – the direct connection of its occurrence and further evolution with the child's growth process.

Key words: IS, prevention, conservative treatment.

AIM

The main goal of all our work was the development of a system for early diagnostics, prevention and early conservative treatment of children and adolescents with IS in all age groups.

The authors are aware that the goal of the work in the stated formulation is quite ambitious. At the same time, we believe that the accumulated modern knowledge base makes it possible to find the first "targets" and the first technologies for early resistance to the defeat of the vertebral complex, which even at the beginning of the 20th century Konrad Alexander Theodor Biezalski (1868–1930) called scoliosis "the orthopedic cross" (in the preface to A. Farkas "Uber Bedingungen und auslosende Momente bei der Skoliosenentstehung") (**23**).

INTRODUCTION

The exceptional attention of vertebrologists of the world to scoliosis is determined by the fact that it has been studied for several thousand years, but has not yet revealed all its secrets. The limited scope of the article does not allow for a detailed review of already obtained information about the most common lesion of the musculoskeletal system. Nevertheless, the analysis of the accumulated data already provides an objective basis for certain conclusions. The authors of the article believe that the most important of them are:

1. The above-mentioned most indisputable fact in the theory and practice of scoliosis is a direct connection between its occurrence and further evolution with the child's growth process (**10**).

2. The fact of the presence of a little-discussed paradox – with the obvious and generally recognized polyetiology of scoliosis – it is always a monoform three-plane deformity, which indicates the unity of pathomechanogenesis (the exception to this rule is "congenital scoliosis").

3. The available extensive database of statistical data on the characteristics of scoliosis (gender, age, time of onset of symptoms, magnitude of deformation, its direction and length, nature of evolution, etc.) indicates their "sufficient homogeneity, and the observed variance has methodological and, to some degree, population origin" (**4**).

4. As a private opinion, let us note that with all the understanding of the multi-causal nature of IS, in the special literature there is still a desire to find only one "golden key" for discovering the secrets of this lesion of the vertebral complex, which can be illustrated by the presence of numerous and sufficiently reasoned theories and hypotheses about its etiology. But even today, after more than a hundred years, we have the right to agree with the opinion of A. Eulenburg (**22**): "The various theories about the origin of scoliosis make up a rather long and boring chapter <and> most of them are now only of historical interest".

Since this article is intended for specialists in vertebrology, the author considers it possible, to reduce the size of the article, to "omit" references to works in which already generally accepted facts are given (**4**).

Meanwhile, a review of international and national classifications shows that the most problematic are the so-called "Idiopathic three-plane deformities in adolescents" (AIS). In a large heterogeneous group of scoliosis, they account for up to 90% of cases. Their main concerns for patients and doctors are:

1. "Scoliosis is a process" (**25**), which develops over time with different variants of the course, both non-progressive and progressive.

2. The appearance of a manifestation of a deformity against the background of a person's full health at almost any age (infantile, juvenile, adolescent and adult).

3. A wide range of options for the evolution of deformation (from progressive to self-healing), which are difficult to predict in each case.

4. Psychological hopelessness in working with such patients, which is fully explained by the term "Idiopathic" (Anc.gr/Lat/Eng/. ἴδιος, idios, proper + πάθος, pathos, suffering) – the disease itself.

Without dwelling on the history of this term, we note that before its appearance these scolioses were called “habitual” (**22**).

5. The low effectiveness of conservative treatment, which determined the appearance in 1941 of the conclusion of the Group led Alfred Rives Shands Jr. of the Scientific Committee of the American Orthopedic Association “NO ALTERNATIVES TO SURGICAL TREATMENT of SCOLIOSIS” (**39**).

In their totality, the listed problems cause a feeling of fatality (Anc.gr./Lat. μοιραίος fatum, fate, predestination of the gods). However, this feeling is completely disavowed by a little discussed fact – among all patients with AIS, the number of cases with aggressive progressive development of the disease (by K.M. Bagnall – “devastating diseases”) does not exceed 10–15%. And in 70–75% of the remaining observations we are talking about such a deviation in the shape of the spinal column, which does not cause any health problems (by K.M. Bagnall – “cosmetic deformity”) (**5**).

Thus, the IS itself suggests that there are circumstances in the body of a growing child that do not allow deformation to progress. And even if the reader does not consider it pretentious, the search for these circumstances has become a “guiding star” for us.

We will not dwell on the history of our own multilateral study of AIS, as it is summarized in the Research Article “AIS: Etiology, pathogenesis. Facts and Reflections” (**20**). At the same time, we will repeat our main results and conclusions.

The first and most important conclusion follows from the results of the biomechanical analysis of three-plane deformation – it is possible only in a two-column model and arises when a difference appears in the longitudinal dimensions of its columns. As a result, due to the requirement to keep constant at the ends of the model, the deformation of the column with a longer length occurs. The introduction of the boundary conditions into such a model, which are dictated by the anatomy, physiology, and biomechanics of the real vertebral complex, allowed us to obtain a quasi-dynamic scoliosis-like deformity.

However, biomechanical modeling has no emotions and this forced us to consider two options for the relationships in the two-column model. The first is an excess of the length of the column, imitating the bone-disco-ligamentous-muscular “sheath”. The second is the excess of the longitudinal dimension of the second component, which mimics the spinal cord. As a result, two models were obtained. The first, with a relative excess of the length of the “sheath”, turned out to be similar to a typical lordoscoliosis, and the second, with a relative excess of the “spinal cord”, to a typical kyphoscoliosis (**2,15**).

This led to the second fundamental conclusion – all scolioses (**28**) (with the exception of congenital), and above all IS in the form of lordoscoliosis or kyphoscoliosis, are a clinical manifestation of a compensatory reaction of the vertebral complex to the non-conjugation of the longitudinal development of the spinal cord and its bone-disco-ligamentous-muscular “sheath”. It is fair to say that the

Czech scientist Milan Roth (1923–2006) was the first to report this cause of lordoscoliosis back in 1968 (**35**). He showed that due to the peculiarities of the longitudinal development of the two main components of the vertebral complex (the lengthening of the spinal cord runs in the caudal direction, and its "sheath" – in the cranial direction), the occurrence of the so-called "medullo-vertebral conflict". To visualize this conflict, M. Roth even created a "spring-string" model lordoscoliosis-like deformity.

It should be noted that in 2008 an online discussion of Delphi was published, which took place during September 2006 – July 2007 under the patronage of the International Federated Body on Scoliosis Etiology (IBSE) (**9**). However, it seems that even after such a broad and brilliant discussion, all its participants remained unanimous.

Meanwhile, the ideas of the outstanding Czech scientist M. Roth were confirmed in our own research (**17, 18**). As a result, a third important conclusion was obtained: the etiology of IS has two stages. At the first stage, there are numerous congenital and acquired, organic and functional disorders in the mechanisms regulating the longitudinal growth of the spinal cord and its "sheath", in full analogy with the "Principle of the Common Path" of Ch.S. Sherrington (1857–1952, Nobel Prize 1932) (**40**) create the preconditions for non-conjugation in the longitudinal growth of the spinal cord and its "sheath". An example of this is completely different conditions, such as syringomyelia and Arnold-Chiari malformation (**36**) or an unpredictable "spurt" of growth after a summer vacation under the southern sun. It is quite obvious that with a complete difference between these situations, a relative excess of the length of the "sheath" arises. This becomes the "answer" to the main paradox of IS – its obvious monoformality contradicts its generally recognized polyetiology.

A spectacular illustration of the change in the shape (even of a separate area) of the spinal column with rigid fixation of its dorsal section is given by the outstanding vertebrologist Professor J.F. Dubousset (**13**). The "crankshaft" phenomenon was the result of a solid spinal fusion that stopped longitudinal growth in the posterior elements, but the vertebral bodies continued to grow anteriorly. The anterior growth causes the vertebral bodies and discs to bulge laterally toward the convexity and to pivot on the posterior fusion, causing loss of correction, increase in vertebral rotation, and recurrence of the rib hump. This phenomenon can be called "scoliosis in scoliosis", only a metal structure plays the role of a "short" spinal cord.

The reason for the opposite relationship, with an excess of the length of the spinal cord, can be called degenerative-dystrophic processes in the vertebral bodies, which occur in Scheuermann disease (**38**), with stable (according to R. Louis, 1985) compression fractures of the anterior column (type **A** according to F. Denis, 1976), with dystrophic discopathies, etc.

NOTE. The lesions listed in the previous paragraph are manifested by the leading clinical symptom – increased kyphosis. However, it is always accompanied by a poorly expressed frontal arch and a rotational component, which gives the right to classify this type of deformity as a three-plane one. Moreover, they are radically different from typical scoliosis. If typical scoliosis has the classic triad "frontal arc + lordosis of the spinal column + pathological convex side rotation of vertebral bodies", then in this case (let's call it atypical) this triad consists of "frontal arc + kyphosis of the spinal column + pathological concave side rotation of vertebral bodies". In other words, if the values of the frontal arches are equal, the orientation of changes in the sagittal and horizontal planes is completely opposite. In addition to the listed main differences in the anatomy of typical and atypical scoliosis, the main feature is the complete opposite in their evolution: with typical scoliosis, progressive development in the frontal plane is allowed, and atypical scoliosis can be aggravated only in the sagittal plane (**2, 15, 30**).

The emerging two variants of non-conjugation of the longitudinal growth of the spinal cord and its "sheath" in accordance with the strict laws of mathematics cause (as the second stage of etiology) mechanical compensation of the difference in the longitudinal dimensions of the two main components of the vertebral complex in the form of lordoscoliosis and kyphoscoliosis.

Another important result in the development of M. Roth's ideas was the verification of signs (symptoms) of preclinical and subclinical stages in the process of transition of a healthy spine to the status of "scoliotic" one.

Thus, the preclinical manifestation of **possible** lordoscoliosis is the "flat back" syndrome. Its mechanogenesis is quite simple – "excess" length of the supporting column (as a result of excessive osteogenesis or hyperhydration of intervertebral discs (**29**)) is "absorbed" by the reserve, which is thoracic physiological kyphosis with a simultaneous increase in the severity of lumbar physiological lordosis.

After the exhaustion of these physiological reserves and under the condition of an increase in non-conjugation due to the more intensive growth of the vertebral bodies (or hyperhydration of the intervertebral discs), subclinical stages inevitably occur. In full accordance with the laws of mathematics, the compensation of the excess length of the supporting column in the vertical position (the condition of sagittal balance according to J. Dubousset (**12**)) occurs due to its twisting around the spinal cord with the effect of a "spiral staircase". The clinical manifestation of this stage is a violation of the parallelism of the frontal axes of the pelvic and shoulder girdles. It should be noted that this symptom complex is practically not analyzed in the special literature due to its low severity and relative short-term duration.

The preclinical manifestation of **possible** kyphoscoliosis is round back syndrome. Its mechanogenesis is also quite simple – the lack of bone formation causes an increase in physiological thoracic kyphosis and a smoothing of the lumbar lordosis. There is reason to believe that the greater the severity of this symptom complex, the less attention orthopedists-vertebrologists pay to changes in the shape of the spinal column in the horizontal and frontal planes.

However, the development of such pathological kyphosis of the vertebral complex in a growing child has limits (**29**), the most important of which is the volume of the sagittal balance (**12**). These restrictions necessitate a transition to subclinical stages in the development of kyphoscoliosis, and in the case of an increase in the relative insufficiency of the longitudinal size of the supporting column, twisting also occurs, but only the dorsal column, functional (or "posterior supporting" according to F. Holdsworth (**26**)), which includes the spinal cord. The clinical manifestation of this stage is the already mentioned violation of the parallelism of the frontal axes of the pelvic and shoulder girdles. As for the subclinical stages of lordoscoliosis, it can be noted that the manifestation of subclinical stages of kyphoscoliosis, due to its low severity and being relatively short-termed, is also practically not analyzed in the special literature.

Thus, the information about IS obtained as a result of the analysis of the world literature, supplemented by data from our own comprehensive study of this lesion, led to the conclusion that three-plane deformity of the vertebral complex is a process of compensating for non-conjugation in the longitudinal development of the spinal cord and its bone-disco-ligamentous-muscular "sheath". Two objective variants of such non-conjugation cause two absolutely natural responses in the form of clinical symptom complexes of lordoscoliosis and kyphoscoliosis. Since their initiation and further pathogenesis have different bases, the complex of therapeutic and prophylactic measures for each of them should be different.

Due to the fact that of this pair the greatest problems are caused by lordoscoliosis, then due to the limitations in the volume of the journal article, the issues of prevention and treatment of only this type of IS will be considered below. It should be emphasized right away that we will talk about two directions in the prevention of lordoscoliosis. The first is the prevention of lordoscoliosis as a disease, and the second is the prevention of its progressive development.

ETIOLOGY AND PATHOGENESIS

There is no doubt that the search for such "targets" in this case should be based on an understanding of the etiology and pathogenesis of the disease. Here we consider it important to note that the term "etiopathogenesis", which is quite often used in the special literature, is absolutely incompetent, since, as noted above, this lesion of the spinal column is polyetiological, but at the same time monoform, which directly indicates the unity of the pathogenesis of deformation of the spinal column.

It should be emphasized that scoliosis is a process in time. It is only a clinical manifestation of the "struggle" of the whole organism with the medullo-vertebral conflict that has arisen in it. Embryology and physiology, anatomy and biomechanics of the vertebral complex of a growing child suggest that this conflict in a latent form takes place throughout the entire period of their growth in height. In most cases, it ends with a "peaceful resolution" due to timely hormonal correction of the intensity of osteogenesis (due to a decrease in the levels of stimulant hormones and an increase in the supply of their antagonists into the blood) (**27**).

Probably, Nerve Growth Factor (NGF), discovered back in the 1950s by Rita Levi-Montalcini (1909–2012) and Stanley Cohen (1922–2020), who were the Nobel laureates of 1986, is also involved in resolving the conflict. There are proteins synthesized by neurons and glial cells. They, among other effects, stimulate the growth of axons, including those that are part of the pathways of the spinal cord. There is a reason to believe that the cause of the "medullo-vertebral conflict" lies in the imbalance between the biological effects of the regulators of osteogenesis and regulators of axonal growth. And although this assumption has not yet been reflected in the literature, the study of the characteristics of NGF is now one of the directions of our research activities.

Nevertheless, based on the already available information about this lesion of the most important segment of the skeleton, we present as a matrix the scheme of the pathogenesis of lordoscoliosis that we developed (**figure 1**).

This scheme is essentially a description of a multi-stage compensatory reaction (response of the organism) to the non-conjugation of the longitudinal development of the two main components of

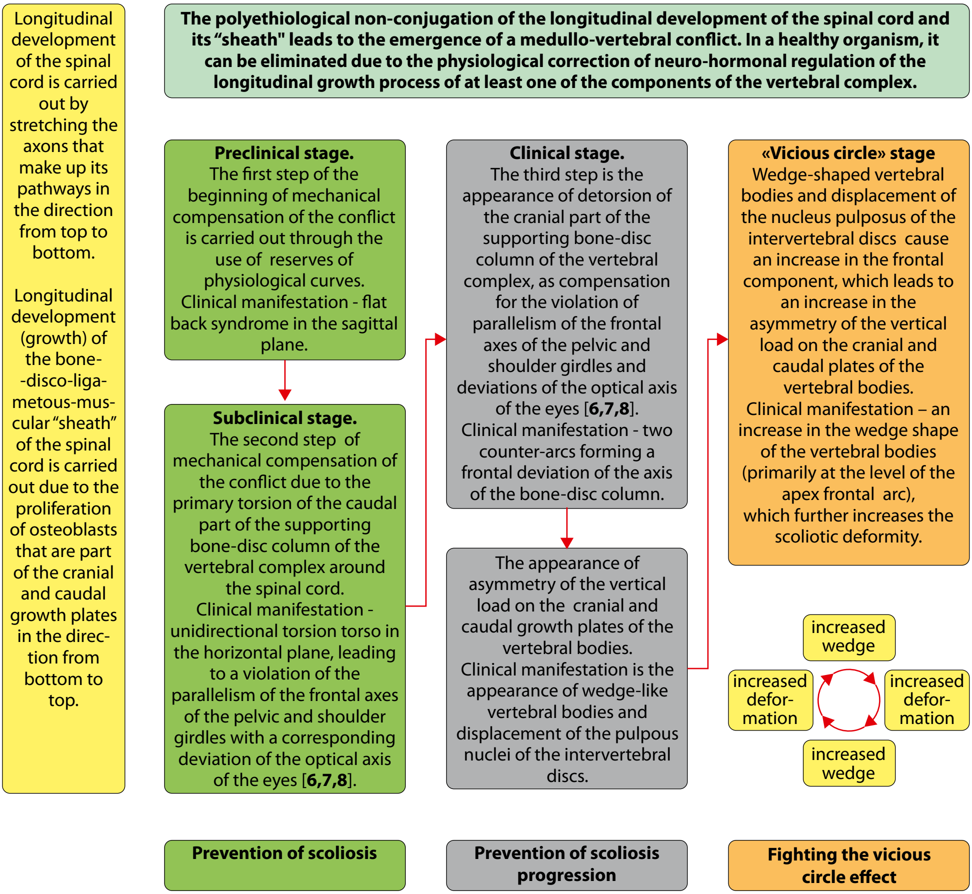


Figure 1. Diagram of the development of IS symptomatology at the stages of transition of a healthy spinal column to the "scoliotic" status (pathogenesis).

the vertebral complex – the spinal cord and its "sheath". In this case, the ill-fated non-conjugation is characterized by a relative excess of the longitudinal dimension of the supporting column, consisting of vertebral bodies and intervertebral discs.

We hope that later we will have the opportunity to publish a similar scheme for a clinical response in the form of kyphoscoliosis to the second variant of medullo-vertebral conflict. Orienting this work to a professional audience, let us not give numerous arguments to substantiate the conclusion that the growth of the vertebral bodies in height, as well as the growth of the entire bone skeleton, is under the control of all directive systems of the body: nervous, endocrine, enzymatic, immune and genetic code. In addition, it is important to note that the normal growth process is non-linear and there are physiological so-called "spurts", from "daily" to "annual" (**11, 24, 27, 37**).

Therefore, when searching for pathogenetic methods of prevention of IS treatment, the first object of attention that plays a huge role in ensuring the normal development of the bone skeleton is the active form of vitamin D, or *calcitriol* [1,25$(OH)_2$D3]. Without dwelling on the extensive information about this multifaceted regulator of the widest spectrum of biological processes, we will single out only one of its roles – the role of an osteogenesis stimulator. Given that the average daily requirement for vitamin D3 at the age of 4–10 years is ≈40μg (≈1600 IU), and for the older ones (10–16 years old) it increases to ≈50μg (≈2000 IU), the actual intake it into the body, primarily from the solar UFO, **is practically not controlled**.

Meanwhile, even such a very brief characterization of vitamin D3 allows us to assert that with an excessive intake of calcitriol (due to excessive exposure to the sun or because of its fashionable consumption in various pharmacological forms, etc.), it is quite real to provoke an unpredictable "spurt" during the child's growth at the beginning of puberty. And if the regulators of the longitudinal growth of a healthy spinal cord are not ready for the appropriate responses to maintain homeostasis of the most important part of the central nervous system, then the very "medullo-vertebral conflict" will arise, which initiates the subsequent chain of compensatory reactions (**20**). Who's guilty? Caring parents? Doctors who prescribe high doses of vitamin D3 for prevention?

CONSERVATIVE TREATMENT

Therefore, there is reason to use as measures aimed at preventing the onset, as well as the subsequent progressive development of the already existing IS, strict restrictions on the child's intake of vitamin D. This is especially important in cases where an absolutely healthy young talent shows signs of risk for scoliosis, in particular – "flat back" syndrome.

Such restrictions are quite simple:

1. Cancellation of calcitriol drugs prescribed to a child for prophylactic purposes. An exception to this requirement are those cases when this drug is used as a therapeutic agent (for example, in

the treatment of rickets or other diseases of the child's skeleton, which are based on impaired osteogenesis).

2. Cancellation (at least for one or two seasons!) of the child's summer vacation in order to "bask in the sun". Otherwise, a child who received a sufficient portion of UFO in July-August, in October–November will have a high probability of having the first signs of IS or an increase in their existing symptoms. The authors believe that this phenomenon is encountered by many practicing orthopedists.

The next most studied regulators of osteogenesis are the effects of the so-called osteotropic hormones (Growth Hormone [GH], insulin-like growth factor I [IGFI] and their functional antagonist Cortizol [Czl], and Calcitonin [Clt] and its antagonist Paratirin [Prt]). For completeness, let us add that adrenocorticotropic hormone [ACTH] and melatonin [Mlt] are regulators of these informons for osteoblasts [Obl], osteocytes [Oct] and osteoclasts [Ocl] (**24, 27**).

In the works (**16, 20**), the characteristics of the levels of the four main osteotropic hormones [GH, Czl, Clt and Prt] have already been published, the concentration combinations of which (the so-called Osteotropic Hormonal Profile or OHP) turned out to be correlated with the nature of scoliosis evolution. Here, let us draw the reader's attention to the fact that fluctuations in the content of these **hormones did not "go" beyond the endocrinological norm,** information about which is given in the annotations to the reagents (**16**).

Here we come to the second "target" in the pathogenetic treatment of IS at all stages of its evolution, starting from the preclinical stage. This is the endocrine regulation of osteogenesis. **Figure 2A** shows variants of the osteotropic hormonal profile with a different nature of the evolution of IS (**16, 20**). It should be noted that the indicators of the actual content of hormones in a particular patient can be given the value of true prognostic criteria. After all, **what level of hormones today will be the intensity of osteogenesis tomorrow**.

As a small digression, let us note that the OHP characteristics of children with progressive IS quite convincingly explain the phenomenon of higher growth of such patients in comparison with healthy peers (**21, 43**), since the former have a higher (compared to the age norm) levels of GH and Clt.

Hence, it is absolutely logical for a particular patient to have such a therapeutic correction of the existing levels of osteotropic hormones, with the help of which an unfavorable variant of the profile will be converted into a favorable one (**figure 2B**).

For this purpose, since the mid-90s of the last century, our clinic has been using medication (non-hormonal) and electrotherapeutic treatment technologies for OHP correction. Based on information about the normal physiology of the endocrine system and the individual characteristics of each of the hormones included in OHP, cortisol was chosen as an object of influence.

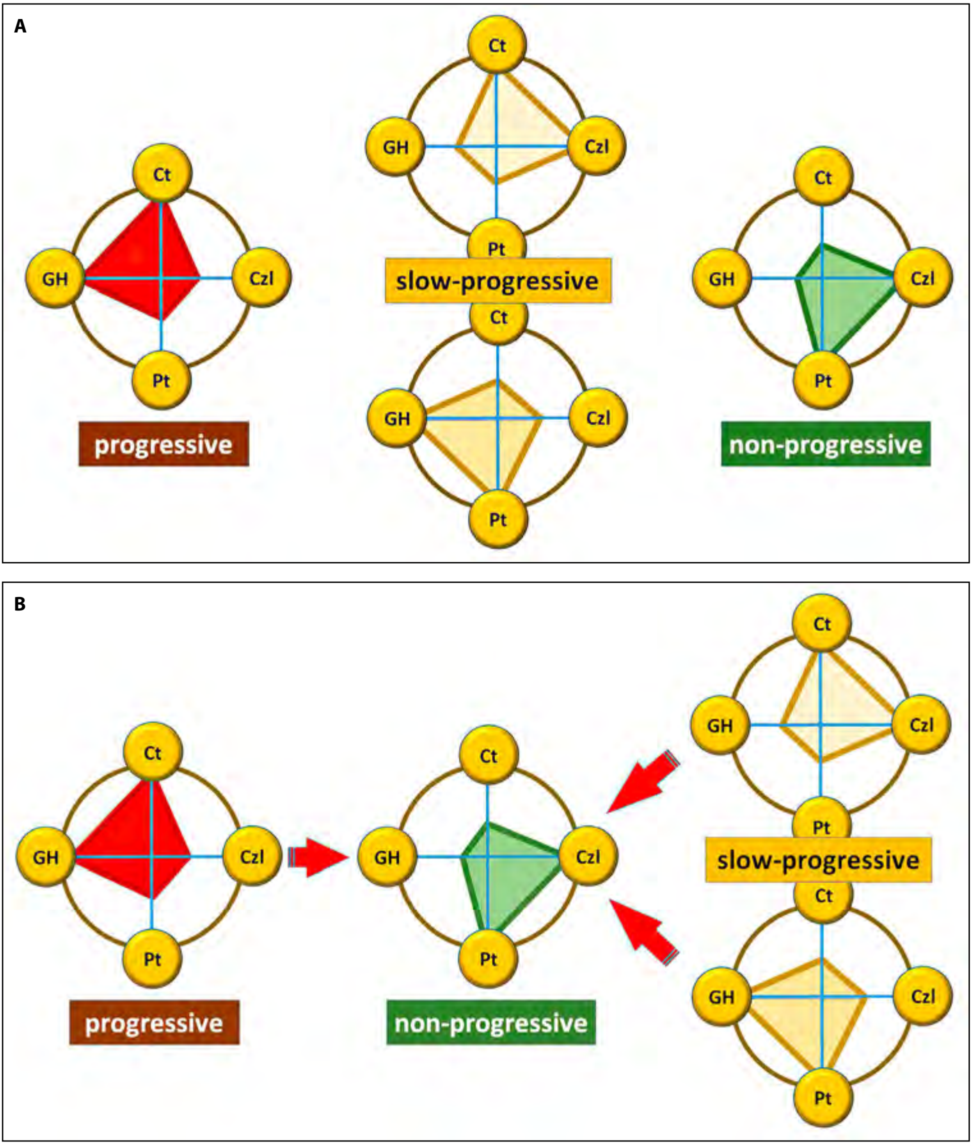


Figure 2. A – four variants of the osteotropic hormonal profile, each of which is typical for different types of IS development; **B** – the task facing hormonal correction of unfavorable types of IS development.

1. Medicinal correction (stimulation) of cortisol levels is carried out by means of preparations containing RADIX GLYCYRRHIZAE or LIQUIRITIAE as a herbal stimulant of the adrenal cortex.

Pharmacology of licorice root preparations: "... the anti-inflammatory and antiulcer effect of licorice preparations is associated with their hormonal (adrenocorticoid) activity" (**31**).

2. Electrotherapeutic correction (stimulation) of cortisol levels carried out using electromagnetic waves of the centimeter and decimeter range (UHF-therapy). Microcurrents penetrate deeply into tissues and organs, affecting the physiological processes taking place in them.

NOTE. Before prescribing UHF adrenal stimulation, ultrasound diagnostics of the renal-adrenal alliance is mandatory (**32**).

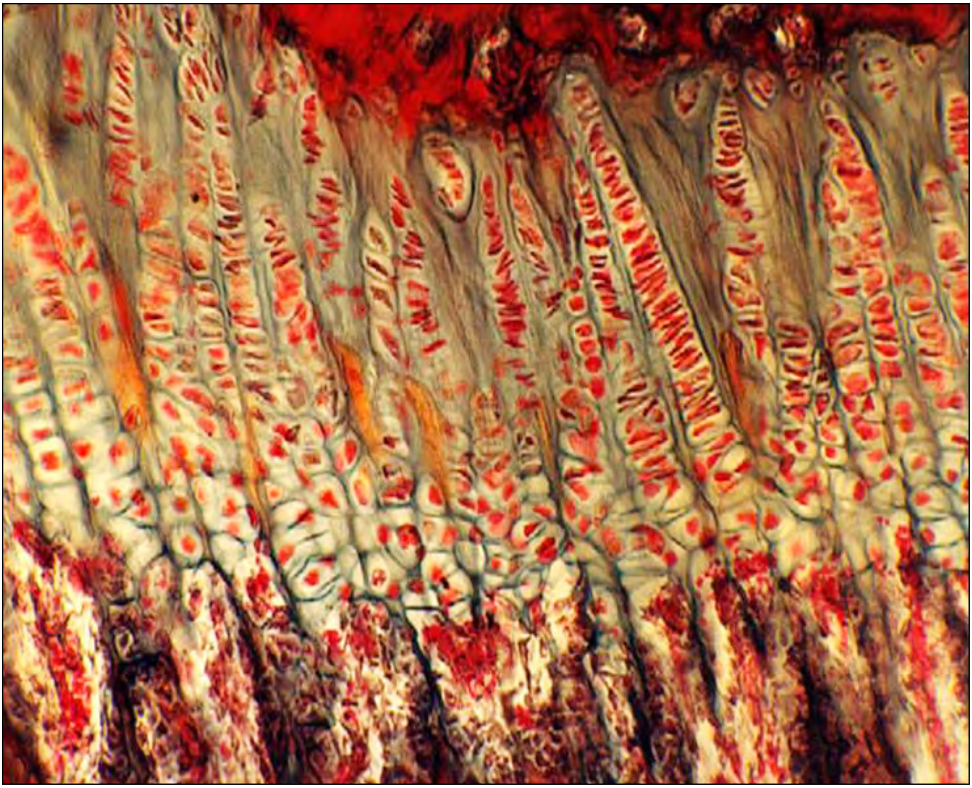


Figure 3. Experiment. The growth plate of the rabbit's thigh. **A** – normal growth plate, control (coloring according to Mallory, x50).

Figure 3. Experiment. The growth plate of the rabbit's thigh. **B** – growth plate after the course of magnetic impulse exposure (coloring according to Mallory, x50).

These treatment technologies are directly shown to children with **pre**-, **sub-** and clinical stages of the IS. The effect of their use in **pre**- and **sub**clinical stages is the prevention of IS as a disease. The effect on clinical stages, regardless of the size of Cobb's degrees, is to prevent the progression of an already established lordoscoliotic deformity of the vertebral complex.

The third "target", but rather for the prevention of the progressive development of IS, are growth plates on the caudal and cranial surfaces of the vertebral bodies. In the clinical-experimental work of MD DPh A. Arseniev (he was a member of our research group) (**3**) it was shown that a pulsed magnetic field (2.5–3.0 Tesla) causes degradation of growth plates in experimental animals (**figure 3**). By the way, the effect obtained by the non-invasive method turned out to be similar to that which is achieved with a fairly common surgical "epiphysiodesis" of the vertebral bodies.

The next "target" in the conservative confrontation with progressive IS was identified based on the results of the disclosure of the mechanism of formation of the rotational component of the 3D deformity of the vertebral complex (**14, 18, 19, 20**). It was found that this mechanism is realized in the caudal-cranial direction of mm. transversospinales, which include mm. rotatores, mm. multifidi and mm. semispinales.

For the last two decades, EMG diagnostics of paravertebral muscles has been included in the protocol of mandatory examination of patients with scoliosis under our supervision. Therefore, the general database contains information on more than 5000 patients. Analysis of the data obtained showed that the muscles serving the vertebral complex in the pathogenesis of IS have two roles. The first, **pathogenic**, manifests itself in the formation of two countercoils, which together form a classic scoliotic deformity in the supporting column of the bone spinal column. The second role is the **ability to resist the development** of an already formed deformation (**14, 19, 20**). Both roles of mm. transversospinales are clearly verified during their EMG diagnostics, which is carried out throughout the entire spine.

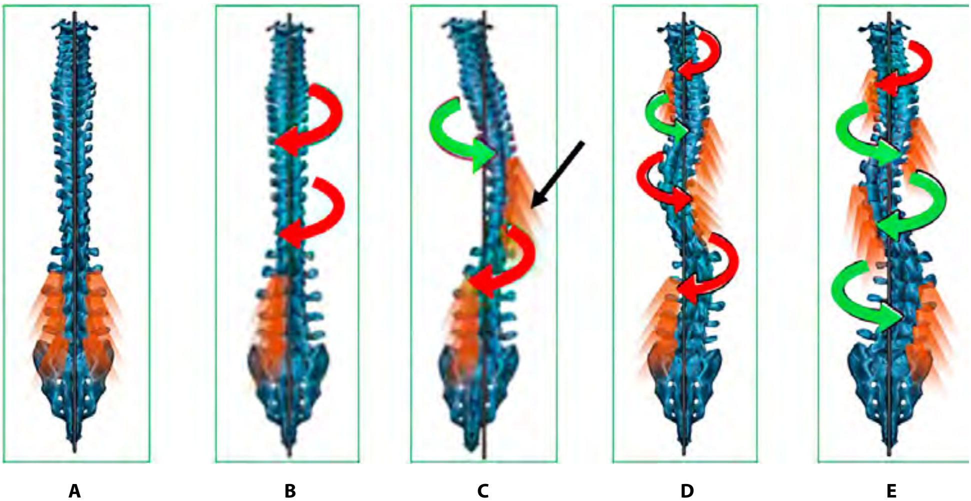


Figure 4. Typical variants of high asymmetric bioelectrical activity (red) mm. transversospinales recorded in patients with IS symptomatology and in their healthy peers (*rear view*); **A** – a healthy spinal column with a symmetrical EMG picture (normal); **B** – high unilateral bioelectric activity of the lumbar mm. transversospinales is observed at the subclinical stage at the base of the vertebral complex and its result is a twisting (torsion) of the entire supporting column of vertebral bodies and intervertebral discs in a "spiral staircase" type (in the direction indicated by the red arrow). The result of this torsion (in the form of a violation of the parallelism of the frontal axes of the pelvic and shoulder girdles with a simultaneous deviation of the optical axis of the eyes) cannot exceed 4° (**6, 7, 8**); **C** – after using this limit, EMGs appear indicating an increase in bioelectric activity located above (straight black arrow) mm. transversospinales on the contralateral side, which indicates the beginning of compensatory detorsion in the overlying vertebrae (in the direction indicated by the green arrow); **D** and **E** – in the process of increasing deformity in conditions of maintaining sagittal balance, the initial picture of the bioelectrical activity of the paravertebral muscles begins to "disintegrate" along the entire spinal column.

The role is considered pathogenic if asymmetrically high electroactivity is recorded from the concave side of the scoliotic arch. In this case, these muscles, with their contraction and "pull" for the spinous processes, increase the pathological rotation of the vertebrae.

If such is recorded on the side of the bulge, then it is quite obvious that the "pull" for the spinous processes during muscle contraction counteracts the growth of the already existing pathological rotation of the vertebrae (**figure 4**).

It is important to note that the given typical variants **D** and **E** are practically identical in the clinical and radiological picture. However, the bioelectric activity of mm. transversospinales in variant **D** plays a pathogenic role, since their contraction leads to an increase in pathological rotation of the vertebrae (red arrows). At the same time, in variant **E**, the muscles of the same name on convex side, on the contrary, in their work prevent (green arrows) an increase in the horizontal component of 3D deformation, i.e. progression of IS.

It is these patterns described above that made it possible to include IS technologies based on magnetic and electrical stimulation of the muscles serving the vertebral complex into the protocol of prevention and complex conservative treatment.

The implementation of such procedures is quite simple, but there are several prerequisites. So, they are prescribed and carried out **only after the EMG diagnosis** of the state of mm. transversospina-

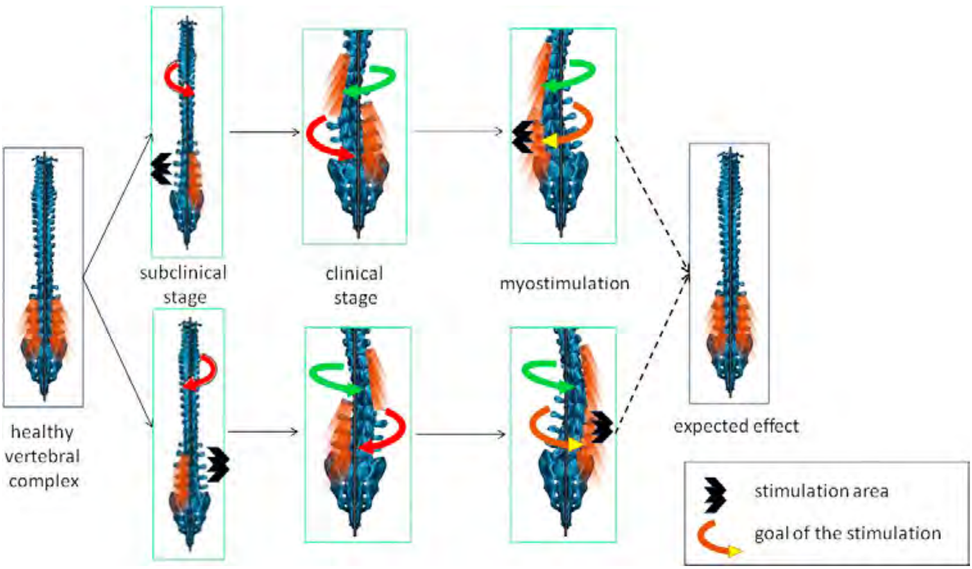


Figure 5. Basics of myostimulation on subclinical and clinical stages in the evolution of IS. In the bottom row, right--sided scoliosis occurs and is equalized, and in the top row, left-sided.

les and correct "reading" of the data obtained. This requirement allows for targeted stimulation of precisely those muscles that are able to resist the growth of pathological rotation of the vertebrae.

Prescribing myostimulation therapy is permissible at any stage of the transition of a healthy spinal column to the "scoliotic" status. In practice, in the overwhelming majority of cases, these are patients with already existing deformities. However, if the practicing doctor manages to "catch" the moment the first signs of imbalance in the bioelectric activity appear, mm. transversospinales in the lumbar region, then it will be entitled to apply the specified treatment technology even at the subclinical stage (**figure 5**).

The equipment widely used in the world for electrical or magnetic stimulation is used. It can be noted that in our practice we give preference to an almost painless magnetic factor.

Figure 5 shows the paravertebral areas on which it is necessary to apply the active element of the stimulating apparatus.

From these positions, we looked in a new way at the good old (therapeutic) Physical Education, which (in particular, in Russia) has always pinned great hopes in the fight against IS. Now, based on the correct "reading" and understanding of the results of EMG diagnostics, physical exercises can be determined for training of quite specific muscles.

We call the lumbar mm. multifidi the first in the list of such muscles, almost all muscles of the posterior abdominal wall, as well as mm. iliopsoas (muscles that attach to the anterolateral surfaces of Th12–L1 and when the hip is fixed, their unilateral contraction rotates the vertebrae bodies to the ipsilateral side). All these muscles have a specific task to exert such an influence on muscle activity that will lead to a decrease in pathological rotation of the vertebrae. Muscles attached to the spinous processes are activated on the CONVEX side scoliotic arc, and mm. iliopsoas – on the CONCAVE side. For the same purpose, we use biofeedback technology (**34**), which two years ago was supplemented by the method of "managing" the state of lumbar lordosis and thoracic kyphosis, which was introduced to us by Professor Piet Van Loon from the Netherlands [priv. communication].

Corset therapy, which is one of the oldest medical technologies, is widely used in our clinic. Here the corset by Ambroise Paré (1510–1590) is a textbook example. However, the authors believe that the real "breakthrough" in corsetology was made by Jacques Pierre Joseph Cheneau. He was able to consider the unique possibilities of a new material – thermoplastic plastic, which made it possible to reveal new possibilities in products for external correction of a deformed spinal column and to practically fully implement the principles of Edville Gerhardt Abbott (1871–1938): **E**longation-**D**erotation-**D**eviation (**EDD**).

It should be added here that during the manufacturing process and at the stages of using the Cheneau-type corset, with the help of inserts, we began to apply the second maneuver proposed by Piet van Loon that increases the lumbar physiological lordosis in patients with lumbar scoliosis, which led to a significant decrease in the frontal component of 3D deformity. A result near to this effect was obtained with the same maneuver for thoracic scoliosis. We completely agree with the author that due to the proposed excessive lordosis of the deformed parts of the spinal column, the "depth" of the medullo-vertebral conflict decreases.

NOTE. It is important to note that the Piete van Loon maneuver is indicated for patients only with typical lordoscoliosis and is contraindicated for benign atypical scoliosis – kyphoscoliosis.

Today, corsets like Cheneau, both in the original and in its various modifications, can be found in almost any country. We will allow ourselves not to enter into a discussion about the advantages of this revolutionary product as a whole, but will only express our point of view on the mechanism of its operation and the expected effect.

The basic premise for our conclusions is three facts in the natural nature of IS. The first is the relationship between its progression and the growth process. The second is the "vicious cycle" phenomenon analyzed in detail by I.A.F. Stokes (**41, 42**). The third is the Hueter-Volkmann Effect described in the 19th century.

From our point of view, the rigid corset created by J.P.J. Cheneau in the 20th century and the most implementing the principles of E.G. Abbott, is an absolutely adequate way to resist asymmetric loading on the cranial and caudal surfaces of growing vertebral bodies. This creates the conditions for reducing the Hueter-Volkmann Effect, thereby interrupting the above-mentioned "vicious cycle". It can be noted that there are already publications in the literature that report a decrease in the wedge shape of the vertebral bodies as a result of prolonged use of a Cheneau-type corset. We observed absolutely identical effect in our practice.

We would like to draw your attention to one moment. Our current vision on the mode of wearing a corset. We prescribe it for scoliosis (not only idiopathic) with a deformity of 15–20 Cobb's degrees for 23 hours a day. There are two reasons for operating in this mode. The first is that the child grows during the night sleep. It is in a dream, when, as a result of active osteogenesis, the deformity has all the conditions for progression, the rigid corset still restrains the unfavorable tendency. The second circumstance is the perception of scoliosis as a **process** that "wants to spoil the mood of both the patient and the doctor". Therefore, it is not at all justified to give this process even minimal freedom or indulgence.

CONCLUSION

In the academic Conclusion of a scientific and practical article dedicated to the treatment of a particular disease, it is necessary to provide illustrations and rejoice at your wonderful results. But we will allow ourselves to deviate from such a tradition for the following reasons.

First. IS is a global problem and many doctors have excellent results in their background in the fight against this lesion of the spinal complex. However, it is fair to say that the main goal of such work (we can judge from the results of discussions with our Russian colleagues) was the deformity of the spinal column itself – they corrected the curved spine, and did not control the processes that caused it. In other words, until recently, we ourselves "treated radiographs".

Remembering the words of the Great Hippocrates that "scoliosis is a process in time", we managed to "look inside" the IS and see some patterns and paradoxes.

As has been mentioned more than once above, the most indisputable fact in the theory and practice of scoliosis is the connection between its occurrence and further development with the child's growth process.

And although the main paradox of this lesion of the spinal column, we consider the monoformality of its clinical manifestation in the form of 3D deformity, the second <paradox> standing next to it should be considered the fact of the presence of a sufficiently large group of patients with distinct signs of scoliosis, but which in them does not have a progressive development despite continued growth. If the first paradox directly indicates the presence of a single last link in the pathogenesis of the disease in the preclinical period, then the second paradox inspires optimism – there are conditions under which the process of pathological deformation stops.

Disclosure of these paradoxes led to the identification of "targets" for curation impact. And the experience we have gained allows us to assert that the prevention of IS as a disease and the prevention of the progression of an already existing deformity is no longer a dream. We allow ourselves to assert that the uncontested surgical treatment of <"severe"> cases has already had an alternative in the form of preventing deformities before the indicated <"severe"> clinical symptoms. We believe that it is better to have scoliosis of 25-30 Cobb's degrees in a mobile spine than a fixed complex with 15-25 Cobb's degrees deformity after a perfectly done surgical correction of <"severe"> scoliosis. As a final generalized illustration of the application of the protocol below for the diagnosis and conservative treatment of IS, we inform you that **none of our patients** who came under our observation with scoliosis up to 35 Cobb's degrees reached the operating table.

As a small digression, we will tell you that, according to our proposal, the analysis of the state of posture in children at the beginning of puberty (the beginning of the second, pubertal growth "spurt") was first carried out by young doctors in a typical Russian city called Perm in 2010–2012. It showed that in this age category their posture is not constant and can change from "normal" towards the development of "flat back" and "round back" syndromes, and in the presence of these syndromes, on the contrary, towards "normal" (**33**). From our point of view, during this period, the medullo-vertebral conflict is not expressed and our own temporary functional adaptive systems successfully cope with it (**1**). But on the other hand, the findings indicated that there are factors in the child's body that oppose the development of pathological deformation of the vertebral complex.

Based on Russian and foreign experience, we have formed a protocol for monitoring a growing child with the aim of preventing IS or treating it, if it does appear.

Today this protocol is offered for professional discussion.

The reason for the planned clinical examination of the child is his reaching the age of 5–7 years (the period of the first growth push ("spurt")) and the second mandatory clinical examination at the beginning of puberty (9–11 years).

If during these inspections the child is diagnosed with a "flat back" (**pre**clinical stage) syndrome, verified on any optical topography installation, the following actions should be taken immediately:

1. Make a superficial (total) electromyography of the paravertebral muscles throughout the entire spinal column;

2. Conduct consultations with colleagues (pediatricians and endocrinologists) in order to identify subclinical signs of adrenal hypofunction (for example, food and non-food allergic reactions, mild but frequent colds, etc.).

3. Based on the information received, it is concluded that the child is included in the "risk group" for lordoscoliosis, and measures are taken to reduce the intake of calcitriol into their body (primarily due to the prohibition of solar insolation) with the simultaneous prescription of licorice root preparations.

4. If an imbalance is detected in the bioelectrical activity of the paravertebral muscles in the lower half of the vertebral complex, conduct 2 courses of electro- or magnetic stimulation of these muscles in accordance with the diagram in **figure 5**.

5. After 6 months, it is necessary to repeat the medical and instrumental diagnostics (optical topography, EMG) and, based on its results, make a decision on the treatment tactics. So, if the signs of belonging to a risk group for scoliosis are eliminated, the child can be "released" to a free regimen.

   In 2012–2013, this part of the short protocol was first tested by our young Permian colleagues (this step allowed us to obtain an independent expert assessment of its effectiveness). As a result, a significant decrease in the number of children with signs of the "risk group" for lordoscoliosis was obtained.

   Meanwhile, it was quite expected that in a small group of children the "flat back" symptom complex was preserved and even slightly increased its severity. In addition, the signs of torsion of the trunk "added" to it. From our point of view, such a picture indicated the preservation of non-conjugation in the longitudinal development of the spinal cord and its "sheath" the course of the IS passed into the subclinical stage. Therefore, it became necessary to continue monitoring and treatment in accordance with the following part of the protocol.

6. In cases where the desired result is not entirely convincing and the doctor has the slightest doubts about the complete elimination of unfavorable symptoms, then it is necessary to repeat everything that was done in the previous six months.

7. In this case, the complex of diagnostic measures, including optical topography and total EMG, should be supplemented with:
   a) X-ray of the spinal column in the anteroposterior and lateral projections in the "standing" position (in order to identify congenital malformations of the elements of the spinal column);
   b) determination by ELISA of the osteotropic hormonal profile (in order to determine the potential of hormonal regulation of osteogenesis, typical variants of which are shown in **figure 2A**);
   c) sonographic diagnosis of the anatomy and topography of the renal-adrenal alliance (to ensure accurate targeted UHF stimulation of cortisol synthesis in the adrenal glands);
   d) MRI diagnostics (if there are appropriate symptoms and indications) of the state of the spinal cord (in order to confirm the clinical symptoms of spinal cord lesions, such as syringomyelia, fixed spinal cord syndrome, Arnold-Chiari malformation, etc.).

8. After carrying out the diagnostic procedures specified in the previous paragraph and obtaining new information about the participation of other systems and organs of the child's body in the pathogenesis of IS, we "include" in the multicomponent conservative treatment (this is the prevention of, unfortunately, practically completed scoliosis) "heavy artillery", including the above mentioned technologies. From UHF adrenal stimulation to the appointment of a corset like Cheneau.

NOTE. The described magnetic-pulse effect (due to its effect in the form of irreversible degradation of growth plate) is used only in older children.

Thus, in the brief protocol of measures for the prevention of possible IS and prevention of the progression of the existing deformity, three groups of measures can be seen (D. Tesakov, priv. communication)

The first, pathogenic, is a conservative technology influencing the growth process of the bone-disco-ligamentous-muscular "sheath" of the spinal cord. Unfortunately, there are no such technologies yet to control the longitudinal development of the latter.

The second, auxiliary, is corsetting, which, with the support of magnetic impulse exposure, we consider so far the only non-invasive way in the fight against the "vicious cycle".

The third, background, is physiotherapy exercises in the form of a wide variety of techniques (including breathing exercises by K. Schroth), as a way to create a background for controlling the muscle corset, the most important participant in the mechanogenesis of three-plane deformity of the vertebral complex.

In one of his brilliant presentation, K.M. Bagnall (Poznan, 2012) definitely noted that work with patients begins when the child has a complete clinical and radiological picture of IS. He explained this fact by the lack of understanding of “how the healthy spine passes into the “scoliotic status””. He named the time of such transition “dark period”. Our understanding of the content of this period allows us to recommend the above protocol not only at the beginning of the indicated first growth “spurt”, but also at the beginning of the second (9–11 years). Our experience has shown that the confrontation with IS should begin from the moment the “flat back” symptom complex is identified. And in those cases when the effect will be obtained even from those still few technologies, the child and their parents will not even know about the danger that threatened them.

Acknowledgement

It should be noted that this publication is a generalization of the results of many years of work by a team of specialists from various fields of science and practice who have studied scoliosis in children under the guidance of the authors, and for this they all deserve the most sincere gratitude.

REFERENCES

1. ANOKHIN P.K. Key questions of the theory of functional systems. Moscow: Science, 1980: 197.
2. ARMSTRONG G.W.D., LIVERMORE N.B., SUZUKI N. et al. Non-standard vertebral rotation in Scoliosis Screening Patients: Its prevalence and relation to the clinical deformity. Spine,1981: № 7: 50–55.
3. ARSENIEV A.V. Idiopathic scoliosis: the use of a pulsed magnetic field in complex conservative treatment (clinical experimental study). Diss ... Cand. of medical sciences. Saint Petersburg: Institute of Traumatology and Orthopedics by R.R. Vreden, 2005: 101.
4. ASHER M., BURTON D. Adolescent idiopathic scoliosis: natural history and long term treatment effects. Scoliosis, 2006; 1: 2. Published online 2006. https://doi: 10.1186/1748-7161-1-2
5. BAGNALL K. How can we achieve success in understanding the aetiology of AIS? In: Studies in Health Technology and Informatics, volume 135: Editor T.B.Grivas. The Conservative Scoliosis Treatment. IOS Press, 2008: 61–74.
6. BARON J.B. Muscles moteurs oculaires, attitude et comportement locomoteur des vertébrés. Thèses de Sciences, Paris, 1955: 158.
7. BARON J.B. Muscles moteurs oculaires, céphalées, déséquilibre et attitude scoliotique. Presse med., 1955: vol. 63 (20): 407–410.
8. BARON J.B., USHIO N., NOTO R. Oculo-nuco-vestibulo-spinal system regulating tonic postural activity: statokinesimetric study. Agressologie, 1974: № 15: 395–400.
9. CHU W.C.W., LAM W.M.W., NG B.K.W. et al. Relative shortening and functional tethering of spinal cord in adolescent scoliosis – Result of asynchronous neuro-osseous growth, summary of an electronic focus group debate of the IBSE. In: Scoliosis and Spinal Disorders. Scoliosis, 2008 Jun 27;3:8. https://doi: 10.1186/1748-7161-3-8.
10. COTREL Y. Treatment of essential scoliosis. Rev. Chir. Orthop. 1957: № 43: 331–343.
11. DE BUFFON G.-L. L. in the book Kharitonov V.M., Ozhigov A.P., Godin E.Z. Anthropology. Textbook for universities: Vlados. 2004: 272 p. ISBN 5-691-01068-9
12. DUBOUSSET J.F. Tree-dimensional analysis of the scoliotic deformity. Editor S.L. Weinstein. The pediatric Spine: Principles and Practice. New York: Raven Press. 1994: 479–496.

13. DUBOUSSET J.F., HERRING A., SHUFFLEBARGER H. The crankshaft phenomenon [Case Reports]. J. Pediatr. Orthop. 1989: 9(5):541-550. doi: 10.1097/01241398-198909010-00008.
14. DUDIN M.G., SINITSKII Y.F. About mechanogenesis torsion changes in scoliosis. J. Orthop., Traumatol. and Prosthetic. 1981: № 2: 33–36.
15. DUDIN M.G. Scoliosis with atypical pathological rotation of the vertebrae – diagnosis, course, treatment tactics. Diss. ... Cand. med. sciences. Leningrad: Institute of Traumatology and Orthopedics by R.R. Vreden. 1981: 210.
16. DUDIN M.G. Particular qualities of hormonal regulation of metabolic processes in the bone tissue as eti-opathogenic factor of idiopathic scoliosis. Diss ... Doctor of medical. Sciences. Saint Petersburg: Institute of Traumatology and Orthopedics by R.R. Vreden. 1993: 195.
17. DUDIN M.G., PINCHUK D.YU. Idiopathic scoliosis: diagnosis, pathogenesis. Saint Petersburg: Man, 2009: 336.
18. DUDIN M.G., PINCHUK D.YU. Idiopathic scoliosis: neurophysiology, neurochemistry. Saint Petersburg: Man, 2013: 304.
19. DUDIN M. Idiopathic scoliosis: prevention, conservative treatment. Saint Petersburg: Man, 2017: 223.
20. DUDIN M., PINCHUK D., MINKIN YU. et al. AIS: Etiology, pathogenesis. Facts and Reflections. Int. J. Orthop. Res., 2020: vol. 3. Published online 2020. doi:10.33140/IJOR.03.02.01.
21. EPSHTEIN G.G. Features of growth of the spine in idiopathic scoliosis. In Diseases and injuries of the spine in children: inter-institutional collection of scientific articles. Leningrad: Medicine. 1981: 18–26.
22. EULENBURG A. Real Encyclopedia of Practical Medicine. Medical-Surgical Dictionary for Practical Physicians [Translation from a completely revised German edition]. Edited by M.B. Blumenau. In 17 volumes. Medicine. Saint Petersburg. Publication of the journal "Practical Medicine" (V.S. Ettinger), 1909: vol. XV: 303–503.
23. FARKAS A. Uber Bedingungen und auslosende Momente bei der Skoliosenentstehung. Stuttgart: Encke, 1925: 224.
24. HAM A.W. (author), CORMACK D.H. (editor). Histology. In 5 vol., Lippincott Williams and Wilkins; 8th Revised edition. Translated from English. Moscow: Mir, 1982-1983 (in Russian).
25. HIPPOCRATES. Compositions. [Transl. in Russian by V.I. Rudnev, in 3 volumes. vol. 3, "Treatise on joints". Paragraphs 41–48]. Moscow: Medgiz, 1941: 364.
26. HOLDSWORTH F. Fractures, dislocations, and fracture-dislocations of the spine. J. Bone Joint Surgery (Am.), 1970: 52 (8): 1534–1551.
27. Human Physiology. Editors: SCHMIDT R. F., THEWS G., in 3 vol. Berlin Heidelberg: Springer-Verlag, 1989: 827.
28. JAMES J.I.P. Idiopathic scoliosis. The prognosis, diagnosis, and operative indications related to curve patterns and the age at onset. J. Bone Joint Surgery (British), 1954: vol. 36-B, Issue 101: 36–49.
29. KAPANDJI A.I. Fisiología articular. V. 3 "Tronco y Raquis". 6th Edicion. Maloine: Editoreal medica Panamericana, 2012: 370.
30. LÜFTINGER M. Aetiology of idiopathic scoliosis: Current biomedical research and osteopathic theories. Master Thesis zur Erlangung des Grades Master of Science in Osteopathie. Wien: An der Donau Universität Krems nie-dergelegt an der Wiener Schule für Osteopathie, 2008: 76.
31. MASHKOVSKY M.D. Medicines: A guide to pharmacotherapy for doctors. In 2 vol. 10th ed. Moscow: Medicine, 1985: 624.
32. PANKRATOVA G.S. Assessment of the state of the urinary system in hildren with idiopathic scoliosis. Thesis ... Cand. of medical. Sciences. Medical University by I.P. Pavlov, 2017: 21.
33. PECHERSKY V., LIHACHEVA L., CHICHERIN A. et al. The evolution of state of vertebral column in three planes. In: Studies in Health Technology and Informatics, Volume 176. Editors T. Kotwicki and T.B. Grivas. Research into Spinal Deformities 8. IOS Press, 2012: 164–167.

34. PINCHUK D.YU., DUDIN M.G. Biofeedback electromyogram in orthopedics et in neurology. Saint Petersburg: Man, 2002: 120.
35. ROTH M. Idiopathic scoliosis caused by short spinal cord. Acta Radiologica Diagnosis, 1968: vol.7 №3: 257-271.
36. ROYO-SALVADOR M.B. Siringomielia, escoliosis y malformación de Arnold-Chiari idiopáticas. Etiología común. Rev Neurol (Barc.), 1996; 24(132): 937–959.
37. SCAMMON R.E. The ponderal growth of the extremities of the human fetus. American Journal of Physical Anthropol., 1930: vol. 15, issue 1: 111–121.
38. SCHEUERMANN H. W. Kyphosis dorsalis juvenilis. Ugeskrift for Læger (in Danish). –1928: 82: 385–93. Republished as: Scheuermann H.W. The classic: Kyphosis dorsalis juvenilis. Clinical Orthopaedics and Related Research, 1977: 128 (128): 5–7. PMID 340099.
39. SHANDS A.R.JR., BARR J.S., COLONNA P.C. et al. End result study of the treatment of idiopathic scoliosis: Report of the Research Committee of American Orthopedic Association. J. Bone Joint Surgery (Am.), 1941: vol. 23(4): 963–977.
40. SHERRINGTON CH.S. The Correlation of Reflexes and the Principle of the Common Path. Reports of the British Association for the Advancement of Science, 1904, vol. 74: 728–741.
41. STOKES I.A.F., SPENCE H.E., ARONSSON D.D. et al. Mechanical modulation of vertebral body growth. Implications for scoliosis progression. Spine (Phila Pa 1976), 1996: № 21:1162–1167.
42. STOKES I.A.F. Hueter-Volkmann Effect. Spine: State of the Art Reviews, 2000: № 14: 349–357.
43. ZHEN LIU, ZEZHANG ZHU, JING GUO, et al. Analysis of body growth parameters in girls with adolescent idiopathic scoliosis: single thoracic idiopathic scoliosis versus single lumbar idiopathic scoliosis. In Studies in Health Technology and Informatics, Volume 176. Editors T. Kotwicki and T.B. Grivas. Research into Spinal Deformities 8. IOS Press, 2012: 195–201.

Corresponding author's address:
Prof. Mikhail Georgiyevich Dudin, MD, DMSc
Children's Rehabilitation Center of Orthopedics and Traumatology 'Ogonyok', St. Petersburg, Russia
E-mail: mdudin@list.ru

POZNÁMKA EDITORA | LETTER TO THE EDITOR

concerning the published review paper titled

IDIOPATHIC SCOLIOSIS: PREVENTION, CONSERVATIVE TREATMENT

by authors: Dudin M.G., Pinchuk D.Yu., Pankratova G.S., Saint Petersburg, Russia)

The reviewers do not agree with authors´ suggestion *"the excessive intake of calcitriol (due to excessive exposure to the sun or because of its fashionable consumption in various pharmacological forms, etc.), is likely to provoke an unpredictable "spurt" during the child's growth at the beginning of puberty and thus contribute to the further development of scoliosis."*

Can you cite some publications on vitamin D (D2 and D3) to confirm these your opinions? We do not know any scientific paper where is written that vitamin D provokes an unpredictable "spurt" during the child's growth at the beginning of puberty. Your empiric assumption was not scientifically proved!

Reply of professor Mihail Dudin: I agree with your remark about vitamin D. However, its action is very diverse. Therefore, focusing on the AVERAGE VALUES OF ITS NEEDS is focusing on the average temperature in the hospital. After all, its biological effect largely depends on the quality of vitamin D RECEPTORS. It has already been established that it (the receptor) is very polymorphic. Therefore, children who are at RISK for lordoscoliosis (FLAT BACK SYNDROME) so called PREVENTIVE vitamin D will only accelerate the growth of the axial skeleton, which will lead to an increase in the MEDULLO-VERTEBRAL CONFLICT. Looking ahead, I will say that children from the RISK GROUP for KYFOSCOLIOSIS, with the "ROUND BACK" syndrome, need this vitamin in large quantities and not only as a prophylaxis, but also for therapeutic purposes. Vitamin D should stimulate the longitudinal growth of the "sheath" spinal cord.

Proposition od Professor Dudin: We can continue this discussion a little later on the pages of the journal "LOCOMOTOR SYSTEM". Do you agree? I think that it (such a DISCUSSION) can begin after the article on LORDOSCOLIOSIS and KYFOSCOLIOSIS.

As the empiric assumption that vitamin D provokes an unpredictable "spurt" during the child's growth at the beginning of puberty was not scientifically proved, the **reviewers recommend** the following formulation: *"We suggest that the excessive intake of calcitriol (due to excessive exposure to the sun or because of its fashionable consumption in various pharmacological forms, etc.), is likely to provoke an unpredictable "spurt" during the child's growth at the beginning of puberty and thus contribute to the further development of scoliosis. However, other authors (Ahuja et al 2018, Ng et al 2018, Zhu et al 2019) have stated that vitamin D-deficiency and impaired calcium-phosphate metabolism further contribute to the development of scoliosis. Therefore, further controlled studies and meta-analyses are necessary to confirm or reject such a hypothesis."*

REFERENCES

1. AHUJA K et al. A Comparative Analysis of the Metabolic and Coagulative Profiles in Patients with Idiopathic Scoliosis, Congenital Scoliosis and Healthy Controls: A Case–Control Study. Asian Spine J 2018;12(6):1028-1036
2. NG SJ et al. The Role of Vitamin D in the Pathogenesis of Adolescent Idiopathic Scoliosis. Asian Spine J 2018; 12: 1127-1143
3. ZHU Q et al. Association between calcium-phosphorus balance and adolescent idiopathic scoliosis: A meta--analysis. Acta Orthopaedica et Traumatologica Turcica 2019; 53: 468e473

CONCLUSION

Discussion on intake of calcitriol in patients with lordoscoliosis and kyphoscoliosis will continue later in frame of next paper titled „ADOLESCENT IDIOPATHIC KYPHOSCOLIOSIS AND LORDOSCOLIOSIS" that was accepted for review at the Journal Locomotor System, 28, 2021, No. 1.

BIOMECHANICKÉ MODELOVÁNÍ PŘECHODU ZDRAVÉ PÁTEŘE DO STAVU „SKOLIOTICKÉHO“

BIOMECHANICAL MODELLING OF THE TRANSITION OF A HEALTHY SPINE TO THE STATUS "SCOLIOTIC"

Dudin M.[1], Pinchuk D.[1], Minkin Yu.[2], Baloshin Yu.[3], Popov I.[3], Bober S.[1], Lisitza N.[3], Pomortsev I.[3], Uzdennikova M.[1]

[1] Children's Rehabilitation Center of Orthopaedics and Traumatology "Ogonyok", Saint Petersburg, Russia. E-mail: mdudin@list.ru

[2] State University of Railways of Emperor Alexander I, Saint Petersburg, Russia

[3] National Research ITMO University, Saint Petersburg, Russia

ABSTRACT

The presented work is focused on the biomechanical analyses of the transitions of a healthy vertebral complex to the scoliotic state. The subject of the analyses were space deformations (3D) of the spine, from the physiologically normal state to the scoliosis. The spine was considered as a two-column quasi-dynamic biomechanical model in which one column is formed by the spinal cord, while the other column is formed by the bone and fibrous “sheath” of the spinal cord (bone-disco-ligamentous-muscular “sheath”). Each of these anatomical elements – columns has a different length during and after growth. Biomechanical analyses are based on research by the Czech scientist M. Roth, who showed that spinal deformities (scoliosis) are a response (or compensatory reaction) to different growth rates, different gradients of growth accelerations to significant differences in the length of “columns” (i.e. from a biomechanical point of view scoliosis of spine is a response to primary non-physiological loads/deformations on both spinal “columns”).

The limit conditions of the genetically predetermined healthy spine made it possible to select from a number of analysed variants for the “columnar” biomechanical model only those disorders (scoliosis) that correspond to clinical case reports (findings) at children. Biomechanical studies of space (3D) deformities of the spine contribute to the estimation of gradients of pathological disorders of the physiologically unhealthy spine (physiological preclinical, subclinical and complete clinical stages in the onset and subsequent development of scoliosis) and subsequently to the elimination or alleviation of disorders in the processes of scoliotic mechanogenesis.

Keywords: scoliosis, two-column spine model, preclinical – subclinical and clinical stages, healthy spine transition to scoliotic status

INTRODUCTION

The spine is the most complex segment of the supporting human skeleton. This conclusion is determined by its embryonic origin, anatomy, functionality and tasks solved by it (**19**). From our point of view, the more traditional and widely used term "vertebral column" veils[1] these facts. After all, the "column" is perceived as a vertical, stationary and homogeneous object. As a result, it misses the fact that it consists of two completely heterogeneous components[2] – the spinal cord and its bone-disco-ligamentous-muscular "sheath". The first is a part of the directive Central Nervous System, and the second is a multi-element formation that solves the following tasks:

1. mechanical protection of the spinal cord;
2. support (resistance to vertical loads with spring effect);
3. mobility[3] (plasticity) with safety for the spinal cord;
4. in growing children to the listed tasks is added:
   a) protection against non-linear (daily, seasonal, annual and gender) growth spurt;
   b) maintaining the homeostasis[4] of the complex during the entire time of its growth and maturation.

The most important condition[5] of homeostasis of the vertebral complex is the conjugation between the longitudinal development of the most important part of the nervous system and the growth of its "sheath". However, in the processes of elongation of these two components there are fundamental physiological differences and the first to pay attention to this fact was the great Czech scientist Milan Roth (1926–2006) (**24, 25**). The longitudinal development of the most important part of the nervous system, he associated with the "stretching" axons, which consist of afferent and efferent pathways. The basis of the same development of the "sheath" Milan Roth called "proliferation" in the apophyseal zones of the vertebral bodies.

It is important to note here that the solution of these problems occurs in the conditions of a single-support stable vertical balance[6] in the sagittal, frontal and horizontal planes, which, among other things, should provide a perpendicular direction of the optical axis to the horizon line with a strictly horizontal position of the mandibular joints (**19**).

---

[1] Veiling-intentionally to do something not quite clear, as if covering with a veil [Dictionary of the Russian language: in 4 vols. /4th ed. Edited by A. P. Evgenieva // RAS, In-t Linguistic. research. – M.: Rus. lang.: Polygraph resources. – 1999].

[2] Component – a part of something [Dictionary of the Russian language: in 4 vols. /4th ed. Edited by A. P. Evgenieva // RAS, In-t Linguistic. research. – M.: Rus. lang.: Polygraph resources. – 1999].

[3] A.I. Kapanji gives the following volumes of movements for the vertebral complex: flexion+extension in the sagittal plane ≈ 250°; lateroflexion for each side ≈ 75-85°; total rotation (rotation) in the horizontal plane ≈ 90°.

[4] Homeostasis /anc. gr. ὅμοιος + στάσις = ὁμοιοστάσις, state constancy/ – the ability of a system to maintain the constancy of its internal state. The idea of this was formulated in 1878 by french scientists Claude Bernard /1813–1878/, and the term as the name for "coordinated physiological processes that maintain stable States in the body systems" was proposed by american physiologist Walter Bradford Cannon /1871–1945/ in his book "The Wisdom of the Body" (**9**).

[5] Condition /lat. conditio/ – quality, which must have the object or process

[6] The body of Homo erectus in space is in a stable (balanced) position, which is similar to the Kapitsa pendulum (**20**), but its description in posturology has not yet been reflected

Despite the fact that the shape of the "sheath" is determined by the genetic code[7], in the process of its growth and maturation (**26**) under the influence of internal and external factors, the relations between the numerous elements of the vertebral complex can change, which naturally affects its integral characteristic, called "posture"(**30**). It should be noted that the very assessment of "posture" has an emotional and aesthetic basis, which, in turn, "blurs" the boundaries of "norm" and "pathology". An example of this conclusion is the completely opposite attitude of orthopedic professionals and professional choreographers to posture with "flat back". The first attribute this type of posture to the sub-pathological syndrome, and the second put a stamp on the "professional unfitness" of young dancers who have this variant of posture is missing! Meanwhile, despite the mentioned "blurring" of the boundaries of "norm" and "pathology", we believe that there is no need to look for arguments for the statement – the most unpleasant variant of deviation from the usual form of the vertebral complex is its 3D deformation, or scoliosis.

And the last one. It is fair to say that in the specialized literature there are, although not numerous, publications on the biomechanical description of scoliosis. However, all of them are based on the static characteristics of deformations (**1, 4, 5, 10**). We did not see any work on biomechanical modelling of scoliosis as a dynamic process to deform of the vertebral complex.

The main aims

1. To determine the main causes and mechanisms of the transition of a healthy vertebral complex to a "scoliotic" state from a biomechanical and clinical point of view.
2. On the basis of biomechanical analyses to confirm the adequacy of biomechanical description of dynamic processes of vertebral complex deformations in connection with the explanation of the main paradox of scoliosis, i.e. its monoformity with general inclusion in the poly-aetiology of the disease.

Part 1

Aim of part 1.

The purpose of the first part of this work was to substantiate and formulate the problem of biomechanical modelling[8] of scoliosis-like deformation.

[7] Since physiological bends of the spine occur even in children who, due to circumstances, have never been verticalized, this gives the right to disagree with the common point of view that they, these bends, are a manifestation only of adaptation to an upright position (**19**).

[8] Mathematical model – a mathematical description of reality, the study of which allows you to obtain information about the objective laws in this reality, i.e. the real object of research is replaced by a mathematical model, the construction of which is called mathematical modeling. It describes, as a rule, an ideal object whose properties can be expressed by mathematical symbols or equations. The main property of the mathematical model is that it allows you to predict the behavior of a real object. Biomechanics is a special section of mathematical modeling. In humans, it studies the statics and movements of both his skeleton as a whole and in individual segments.

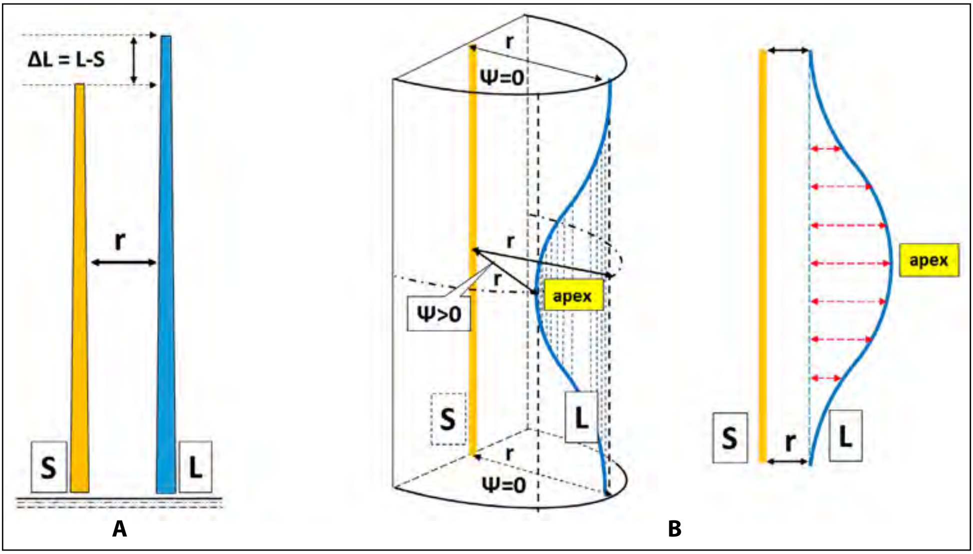


Figure 1: **A** – is a scheme of the two-column model; **B** – is a scheme for compensating for different sizes in a two--column model at the expense of two "anti-turns".

The conditions of the problem.
Based on the above circumstances, in which the vertebrate complex solves the problems facing it and for the maximum approximation of the model to the real conditions, the following boundary conditions[9] are introduced into it:

#1. vertical position of the model;
#2. constant distance between columns throughout their length;
#3. the same level of "cranial" and "caudal" ends of the columns;
#4. preservation of parallelism of sagittal and frontal axes of the ends of columns. Deformation of the vertebral complex.

Problem solution

Despite the fact that the term "scoliosis"[10] is translated as "lateral curvature", only relatively recently, in 1882, W. Adams (**2**) showed: scoliosis is always a 3D deformation. This fact, which is not disputed by anyone, formed the basis of the main paradox of spinal lesion – with its polyetiology, the clinical manifestation is monoform.

[9] Boundary conditions are additional conditions of the problem arising from the real characteristics of the process under study. Their value is determined by the fact that an equation (or a system of equations) has a large number of solutions and only the introduction of boundary conditions allows us to select a particular case of interest from them (**32**).

[10] **Scoliosis** /anc. gr. σκολιός/, proposed Cl. Galen /anc. gr. Cl. Γαληνός, 129-217 AC/, in addition, he is the author of the terms **lordosis** /anc. gr. λορδός/, **kyphosis** /anc. gr. κύφος/ and **strophosis** /anc. gr. στροφός, lat. strofoz or torsionis/.

It was this paradox that raised the question: "Why do many sufficiently convincingly reasoned etiological factors lead to one result – three-plane deformation of the vertebral complex?".

In the late 70s of the XXth century, it was given to associate Professor Yuri G. Minkin, an employee of the Department of theoretical mechanics of the Leningrad Institute of railway engineers (now – "Saint Petersburg State University of Railways of Emperor Alexander I").

Despite the problems of "translating medical terminology into the language of mathematics", he gave an unambiguous answer – a three-plane deformation can be obtained only in a two-column model! (**12**). This answer was convincingly argued by the mathematical equation (1), showing a direct relationship between the occurrence and subsequent development (increase) of the characteristics of 3D deformation with the value of the difference in the longitudinal size of the two columns of the model (**Figure 1**).

$$L = S\int_0^1 \left[\sqrt{1 + k(\xi^2 - \xi)^2}\right] d\xi \qquad (1)$$

Equation (1) shows the relation between the lengths of two parallel segments, one of which changes its longitudinal dimension provided that the distance between them is maintained, where: **S** [short, author notice] – is a column that does not change its longitudinal size; **L** [long, author notice] – is a column that changes its longitudinal size; **r (↔)** – is a rotation radius or distance between **L** and **S** (constant throughout the model); **ψ** – is the largest angle rotation observed when **L** is twisted around **S**; **k = 144γ2/S²**, **γ** = **rψ** and **ξ = 2x/S** (**γ** – is an accessory parameter, **x** – is a variable for the **x**-axis in **Figure 1B**, **dξ** – is integrable variable).

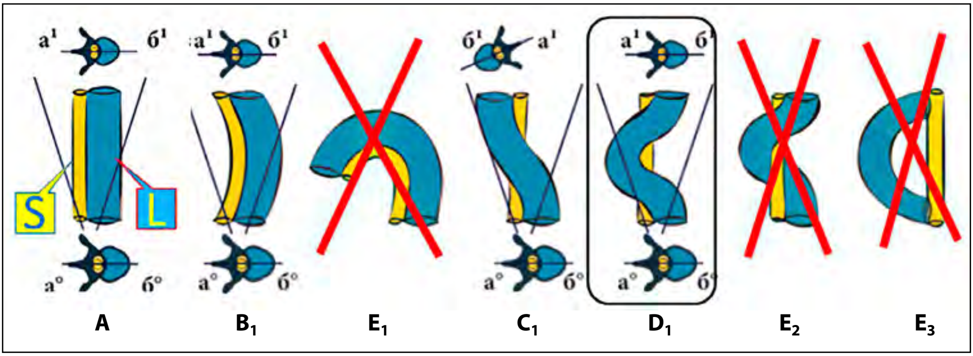


Figure 2: The options for compensating for the difference in length in the two-column model in which the length of the column **L** increases. **A** – is the starting position (**S=L**); $\mathbf{B_1}$ – is the single-plane (sagittal) deformation (**S<L**); $\mathbf{C_1}$ – is the two-plane (horizontal & frontal) single-turn deformation (**S<<L**); $\mathbf{D_1}$ – is the two-plane (horizontal & frontal) double-turn deformation (**S<<<L**); $\mathbf{E_1}$, $\mathbf{E_2}$ and $\mathbf{E_3}$ – are the options that do not comply with the boundary conditions («ring», «360° turn», «bow»).

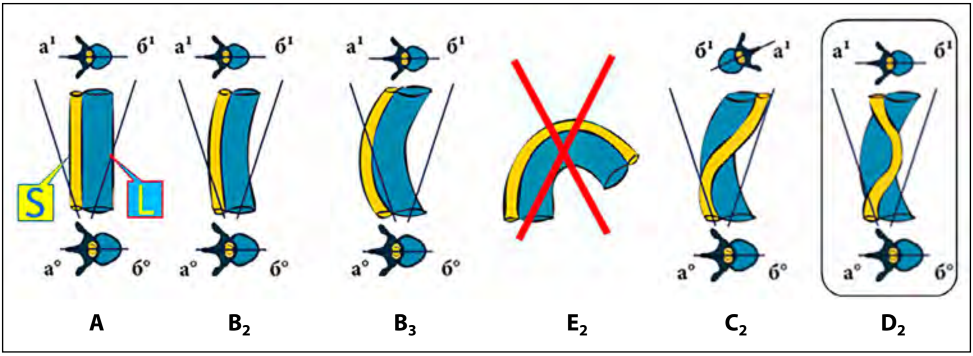


Figure 3: The options for compensating for the difference in length in the two-column model in which the length of the column **S** increases. **A** – is the starting position (**S=L**); $\mathbf{B_2}$ – is the single-plane (sagittal) deformation (**S>L**); $\mathbf{B_3}$ – is the aggravation of the single-plane (sagittal) deformation (**S>>L**); $\mathbf{C_2}$ – is the two-plane (sagittal & horizontal) single-turn deformation (**S>>>L**); $\mathbf{D_2}$ – is the two-plane (sagittal & horizontal) double-turn deformation (**S>>>>L**); $\mathbf{E_2}$ – is the option which do not comply with the boundary conditions.

It should be noted that this result with mathematical objectivity confirmed the position previously formulated by Milan Roth (**24**, **25**) about the relationship of scoliosis with the features of "the osteo-neural growth disproportion". And although he considered only the variant with an overly long "sheath", biomechanical modeling pointed to two kinds of compensatory reaction caused by two situations in the column ratio**:** **L > S** and **L < S**.

The clinical experience of the authors suggests that in each of these cases, on the abstract two-column model, it is possible to observe the coincidence of individual forms of compensation for the different size of the columns with the symptoms encountered in real patients. Thus, with a relative excess of the longitudinal size of the "sheath" of the spinal cord (as a columns **L** in **Figure 2**), the compensatory process leads to a prototype of "lordoscoliosis" ($\mathbf{D_1}$ in **Figure 2**). In the opposite situation (with an excess of the longitudinal size of the spinal cord (as a column **S** in **Figure 3**), a prototype of "kyphoscoliosis" ($\mathbf{D_2}$ in **Figure 3**) develops.

Info. *If the description of the symptoms of lordoscoliosis has a long history, the modern full description of the clinical and radiological picture of kyphoscoliosis, indicating their benign course, was made only in the second half of the XXth century* (**3,13,14, 21**) *and they were called "atypical scoliosis", "non-standard scoliosis", "scoliosis with atypical rotation of the vertebrae".*

It is fair to recall here that the difference between the "front" and "rear" humps[11] drew the attention of Hippocrates (anc. gr. Ἱπποκράτης, 460–370 BC). He stressed that the second have a less aggressive course (**18**). *The fact of benign development of kyphoscoliosis was noticed in the XIXth century (Meijer H.,*

[11] In "The Hippocratic Corpus" the expression "hump of the ridge" /anc.gr. καμπούρα κορυφογραμμ/ was used, but the term "scoliosis" will emerge only in 500 years, yet the derivatives "kyphoscoliosis" and "lordoscoliosis" will begin to be used much later.

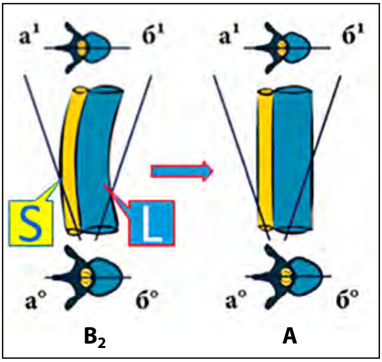


Figure 4. The transition $\mathbf{B_2} \rightarrow \mathbf{A}$ as the first stage of compensation of increase of the longitudinal size of a column **L** at initial excess of length of a column **S** that as a result leads to equality **L=S**.

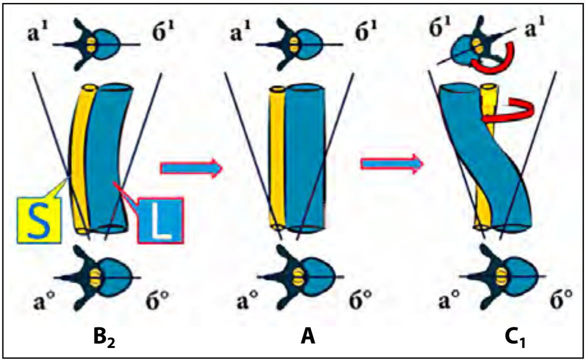


Figure 5: The transition $\mathbf{A} \rightarrow \mathbf{C_1}$ as the second stage of compensation of the continuing increase in the longitudinal size of the column **L** after reaching the equality **L=S**, which eventually leads to the appearance of the primary one-sided turn of the column **L** around the column **S**.

1866, in the thesis M. Lüftinger (**22**)*). The author, to whom the dissertation refers, emphasizes that the aggravation of such deformation of the spinal column as a whole is possible, but not catastrophic, only in the sagittal plane. For completeness, we add that the description of individual symptoms of kyphoscoliosis can be found in the works of the great orthopedists W. Schulthess* (**27**)*, A. Steindler* (**31**) *and J.C. Risser* (**23**).

DISCUSSION

If one takes into account that the actual spine is a physiological bending in the thoracic spine (the most frequent localization of the scoliosis), which corresponds to option $\mathbf{B_2}$ (**S>L**) on **Figure 3**, when increasing the length of the column **L** in the two-column model first comes the equality **S=L**, i.e. introduced into the conditions of the problem "physiological kyphosis" will start to decrease in the sagittal plane until the complete disappearance and the whole structure will gain a vertical position, as shown in **Figure 4**.

The continuation of the increase in the longitudinal size of the column **L** will require, according to the laws of theoretical mechanics, compensation of the difference (**L–S=ΔL**) in the option $\mathbf{B_1}$ (in **Figure 2**). But in this case there is a loss of verticality, which violates the first important boundary condition ["verticality of the model"]. Therefore, it is quite natural to become to option $\mathbf{C_1}$ (in **Figure 2**), i.e. the process of **ΔL** compensation becomes possible only by twisting a long column around a short one (in **Figure 5**).

However, as option $\mathbf{C_1}$ illustrates, the primary one-sided turn of column **L** leads to a change in the position of the sagittal axis $\mathbf{a^1b^1}$ relative to $\mathbf{a^0b^0}$, which violates the last **(#4**) of the above boundary

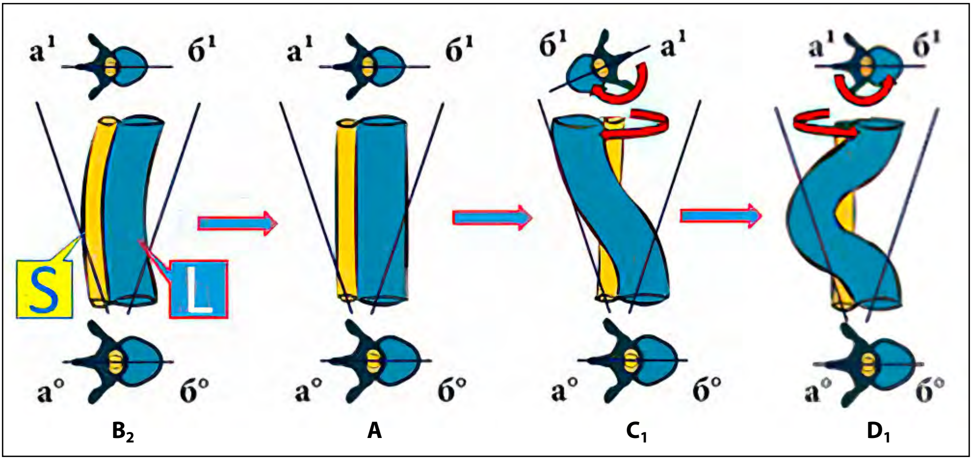


Figure 6: As shown in figure 5, the transition **A → C_1** leads to a violation of the parallelism of the axes **a^0b^0** and **a^1b^1**. The arisen circumstance for preservation of compliance to a boundary condition **(#4)** demanded compensation at the expense of transition **C_1 → D_1**. The result of this process is the appearance of a secondary one-sided turn with the opposite direction (anti-turn).

conditions – "parallelism of the sagittal and frontal axes of the "cranial" and "caudal" ends of the model". This circumstance, while maintaining compliance with other boundary conditions, already requires its compensation. In other words, the realized primary compensation **ΔL** due to a one-sided turn causes the need for compensation of its effect, or compensation of the second order. Theoretical mechanics suggests that for compliance with all boundary conditions it is carried out according to the option **D_1** in **Figure 6**.

If we consider **Figure 6** through the eyes of an orthopedic, then among the above types of compensation for the different size of columns **L** and **S**, we can see prototypes of the main symptoms of lordoscoliosis as a result of **3D-deformation** of the vertebral complex. Thus, option **A** is a "flat back" syndrome, which develops as a result of a change in the original shape (option **B_2**) of the spine in the sagittal plane. Option **C_1** is a syndrome of unilateral torsion of the torso, reflecting the beginning of changes in the shape of the supporting column of the vertebral complex in the horizontal plane. Option **D_1** – is the development of the anti-turn in the cranial part of the supporting column of the model, caused by the preservation of the boundary condition **#4.** This effect, together with the primary turn in its caudal half, leads to the appearance of an arc in the frontal plane, which completes the formation of a complete symptom complex of "lordoscoliosis". It is hard not to notice that **Figure 6** illustrates the basic stages of transition of a healthy vertebral column (option **B_2**) to the status of "scoliotic" (option **D_1**).

In the growing vertebral complex the first stage is absolutely physiological – it compensates for the excess length of the "sheath" due to reserves of physiological bends. It has the right to be called "**pre**clinical", during which it is quite possible to stop the progressive development of "medullo-vertebral conflict", or "the osteo-neural growth disproportion" by Milan Roth. During the development of this stage, the **sagittal component** of 3D deformation (in this case – **lordoscoliosis**) is formed. The second stage is the beginning of the formation of the **horizontal component** of scoliosis (so far only possible, but not mandatory, author notice). This stage may be called **sub**clinical, since torsion compensation **ΔL** cannot be considered physiological. The last, third stage is the formation of two, caudal and cranial, turns of the **frontal arc** in the supporting column, which completes (despite the minimal severity, author notice) the formation of a clinical picture of 3D deformation in the form of lordoscoliosis. Therefore, this stage should be called **clinical**.

Since lordoscoliosis is the most formidable in its possible consequences, further stages of biomechanical modeling will relate to this type of 3D deformation.

So far, we only note that another type of 3D deformation, or **kyphoscoliosis**, according to the results of the general analysis of the biomechanical model, has a fundamentally different key cause of its occurrence. This is an excess of the longitudinal size of the spinal cord relative to the same index for its bone-disco-ligamentous-muscular "sheath". Meanwhile, the anatomy of the vertebrae and the boundary conditions in which kyphoscoliosis develops do not allow it to reach critical values, which has already been noted by clinicians (**3, 13, 14, 18, 21, 22**). Although in the process of forming a complete symptom complex of this type of lesions of the vertebral complex, it is possible to observe **pre**-, **sub**- and purely **clinical** stages characteristic only for it.

Part 2

As follows from the results of the general analysis of the biomechanical model, the first stage of the transition of a healthy vertebral column to the status of "scoliotic" should be considered the formation of the "flat back" syndrome, illustrated by the transition $\mathbf{B_2} \rightarrow \mathbf{A}$ in **Figures 4, 5** and **6**.

Aim of part 2

The purpose of this part of the work is to identify the conditions for the development of "flat back" syndrome in growing children.

Achieving this goal as a task describing the process of development of this syndrome required the introduction into the biomechanical model (**12, 15**) of an array of data on the real size of the structures of the vertebral complex of children aged 9–14 years (**17**), as the period of the greatest detectability of lordoscoliosis.

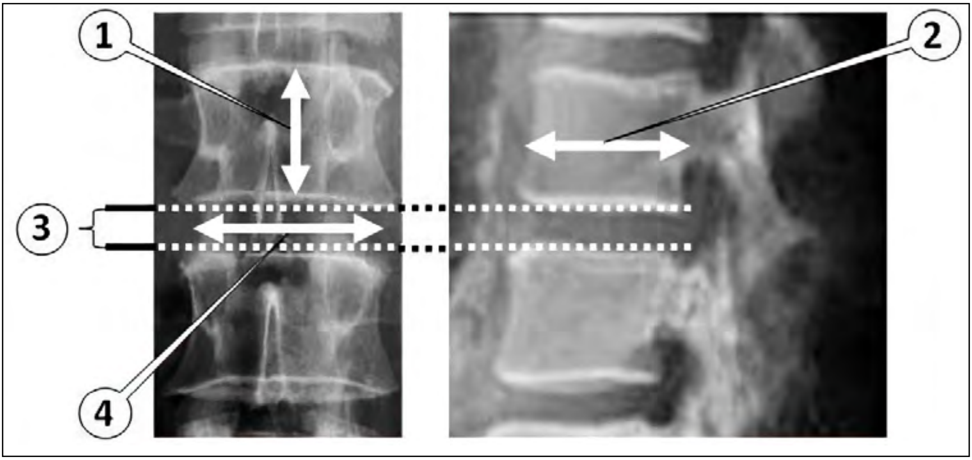


Figure 7: 1 – vertebral body height (**hv**); **2** – anterior-posterior vertebral body size (**aps**); **3** – intervertebral disc height (**hd**); **4** – disc width (**wd**).

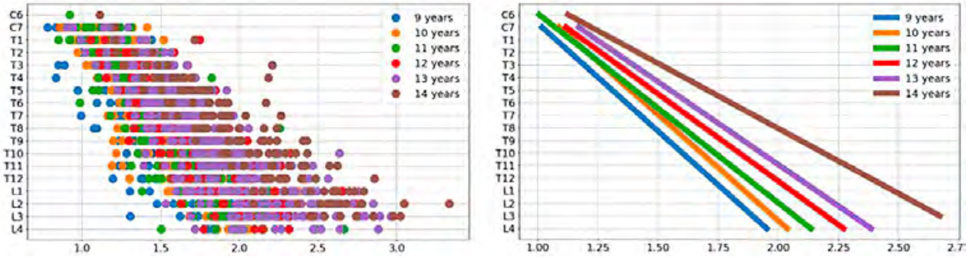
However, as it is surprising, in reference literature such data practically were absent. This caused the need to obtain their own data on the linear dimensions of the main elements of the musculoskeletal "sheath" of the spinal cord – vertebral bodies and intervertebral discs (**Figure 7**).

To solve this problem, we analyzed 497 spondylogramms in two projections obtained in patients aged 9–14 years with stable compression fractures (**A-1** according to the classification of F. Denis (**11**)). The compressed (damaged) vertebrae were not measured and their data were not taken into account (**table 1**).

To determine the average values of longitudinal vertebral dimensions of children separately by age groups, the method of polynomial regression was applied (**28**). As a result, the average values of heights (**hv**) and anteroposterior dimensions (**aps**) of vertebral bodies, as well as linear dimensions

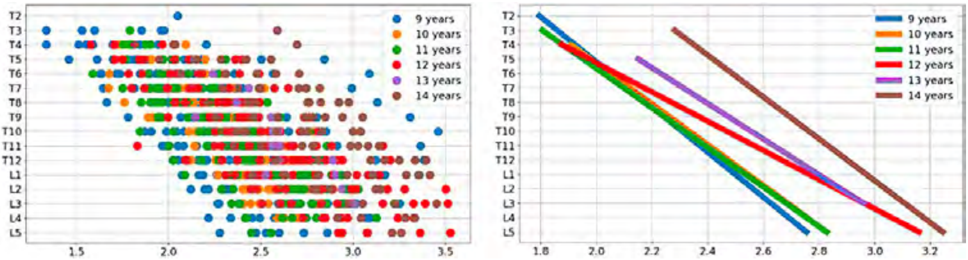
Projection	**Age**						
	9 y.	10 y.	11 y.	12 y.	13 y.	14 y.	Total
Axillary projection	40 X-rays	50 X-rays	52 X-rays	38 X-rays	56 X-rays	22 X-rays	258 X-rays
Lateral projection	50 X-rays	45 X-rays	76 X-rays	44 X-rays	12 X-rays	12 X-rays	239 X-rays
Total	90 X-rays	95 X-rays	128 X-rays	82 X-rays	68 X-rays	34 X-rays	497 X-rays

Table 1: Number of X-rays used to acquire data /material for study/.



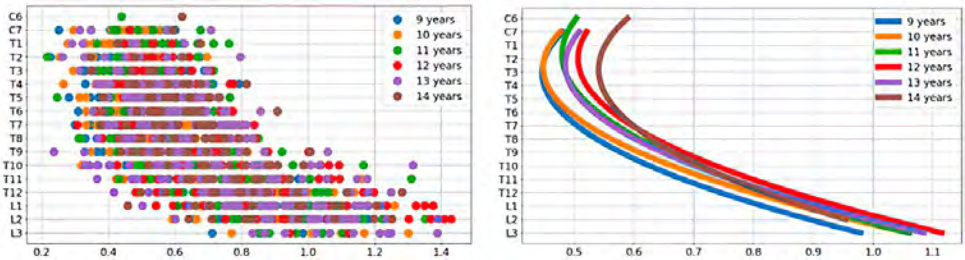
A **B**

Graph 1: A – the height (**cm**) of vertebral bodies (**hv**), **#1** in **Figure 7**; **B** – the average height (**cm**) of vertebral bodies (**hv**) in different age groups (linear relationship).



A **B**

Graph 2: A – the antero-posterior size (**cm**) of vertebral bodies (**aps**), **#2** in **Figure 7**; **B** – the averaged antero-posterior size (**cm**) of vertebrae (**aps**) in different age groups (linear relationship).

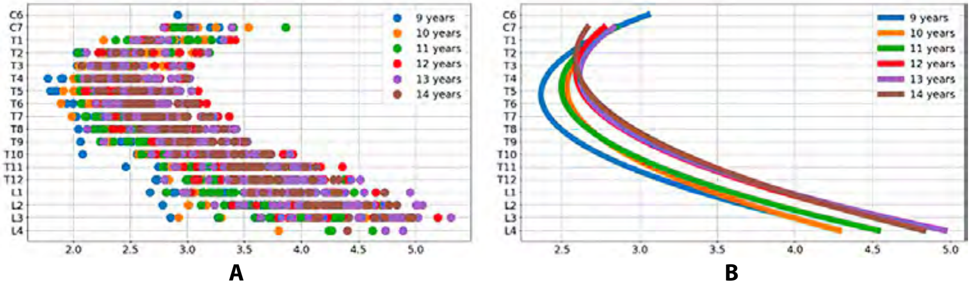


A **B**

Graph 3: A – the height, or thickness, (**cm**) of intervertebral discs (**hd**), **#3** in **Figure 7**; **B** – the averaged height, or thickness, (**cm**) of intervertebral discs (**hd**) in different age groups (nonlinear relationship).

of heights (**hd**) and width (**wd**) of intervertebral discs[12] were obtained in accordance with their position (**#)** in the supporting column (it should be noted that the **Th1** vertebra corresponded to disk **#6**, and the **L5** vertebra to disc **#23**) (**graphs 1, 2, 3, 4**).

[12] Since intervertebral discs are not visualized on radiographs, based on the anatomy of the vertebral-motor segments (**19**), the boundaries of vertebral bodies were taken as guidelines for obtaining data on their linear dimensions.



Graph 4: A – the width (**cm**) of intervertebral disks (**wd**), **#4** in **Figure 7**; **B** – the average width (**cm**) of intervertebral disks (**wd**) in different age groups (nonlinear relationship).

The graphs show the following patterns:

1. The height (**hv**) and antero-posterior size (**aps**) of the vertebral bodies were linear with the increase of these indicators in the caudal direction. The same rule (linearity of increase) was observed with age of patients.
2. The height (**hd**) and width (**wd**) of the intervertebral discs had a non-linear increase in the actual size both during the complex and as the children grew older. Attention is drawn to the minimal increase in the height of the discs as the growth and maturation of the vertebral complex.

These regularities and numerical values of the linear dimensions of the main elements of the supporting column of the spinal cord "sheath" were used as initial (normative) data in the construction of dynamic biomechanical models of both the normal children's vertebral complex and in the modeling of its various deformations.

The calculation of these (due) parameters for vertebrae and discs can be carried out using equations (2)

$$\boxed{hv_N = k_1 \cdot N_v + k_2} \quad 2_1$$

$$\boxed{aps(v_N) = k_3 \cdot N_v + k_4} \quad 2_2$$

$$\boxed{hd_N = k_5 \cdot N_d^{2} \cdot k_6 \cdot N_d + k_7} \quad 2_3 \qquad (2)$$

$$\boxed{wd_N = k_8 \cdot N_d^{2} \cdot k_9 \cdot N_d + k_{10}} \quad 2_4$$

The first two formulas in (2) solve the problem of calculating the height (**hv_N**) and anterior-posterior size (**$apsv_N$**) of the vertebral body, where: **k_1**, **k_2**, **k_3** and **k_4** are the calculated coefficients obtained from the parameters of the studied group, **N_v** is the vertebral index. The second pair of formulas in (2) solve the problem of calculating the height (**hd_N**) and width (**wd_N**) of the intervertebral disc, where: **k_5 – k_{10}** – the calculated coefficients obtained by the parameters of the studied group, **N_d** is the index of the disc.

The calculated coefficients ($\mathbf{k_1}$ – $\mathbf{k_{10}}$) obtained by the parameters of the studied group are presented in **tables 2** and **3**.

age	the body vertebrae height (**hv**)		the body vertebrae antero-posterior size (**aps**)	
	$\mathbf{k_1}$	$\mathbf{k_2}$	$\mathbf{k_3}$	$\mathbf{k_4}$
9 y.	0.058	0.67	0.064	1.28
10 y.	0.059	0.73	0.070	1.20
11 y.	0.066	0.67	0.073	1.15
12 y.	0.072	0.68	0.099	0.88
13 y.	0.076	0.71	0.081	1.26
14 y.	0.097	0.64	0.069	1.66

Table 2: The coefficients $\mathbf{k_1}$–$\mathbf{k_4}$ used to calculate the height (**hv**) and antero-posterior size (**aps**) vertebral bodies of children in different age groups

age	the intervertebral disc height (**hd**)			intervertebral disc width (**wd**)		
	$\mathbf{k_5}$	$\mathbf{k_6}$	$\mathbf{k_7}$	$\mathbf{k_8}$	$\mathbf{k_9}$	$\mathbf{k_{10}}$
9 y.	0.0037	0.067	0.75	0.017	0.392	4.58
10 y.	0.0040	0.069	0.75	0.014	0.294	4.10
11 y.	0.0033	0.051	0.67	0.016	0.342	4.32
12 y.	0.0036	0.058	0.74	0.015	0.278	3.91
13 y.	0.0038	0.065	0.76	0.016	0.305	4.10
14 y.	0.0033	0.058	0.80	0.012	0.208	3.47

Table 3: The coefficients $\mathbf{k_5}$–$\mathbf{k_{10}}$ is used to calculate the height (**hd**) and width (**wd**) of intervertebral discs of children in different age groups

The use of the equations (2) and the coefficients $\mathbf{k_1}$ – $\mathbf{k_{10}}$ allowed us to find the normative longitudinal dimensions of the supporting column of the bone-disco-ligamentous-muscular "sheath" for children of the age in which the appearance of scoliosis is most likely.

This information is shown in **table 4**.

age	thoracic area			thoracic + lumbar area		
	Σ hv **Th_1–Th_{12}** (cm)	Σ **hd** **Th_{1-2}–Th_{12}-L_1** (cm)	Σ **(hv+hd)** **Th_1–Th_{12}** (cm)	Σ hv **C_7–L_5** (cm)	Σ **hd** **C_7-Th_1–L_5-S_1** (cm)	Σ **(hv+hd)** **C_7–L_5** (cm)
9 y.	**16,74**	**6,35**	**23,09**	**27,20**	**11,74**	**38,94**
10 y.	**17,61**	**6,72**	**24,33**	**28,54**	**12,57**	**41,11**
11 y.	**17,94**	**7,05**	**24,99**	**29,29**	**12,84**	**42,13**
12 y.	**18,96**	**7,44**	**26,40**	**31,03**	**13,55**	**44,58**
13 y.	**19,92**	**7,43**	**27,35**	**32,62**	**13,33**	**45,95**
14 y.	**22,23**	**7,56**	**29,79**	**36,84**	**13,35**	**50,19**

Table 4: The longitudinal dimensions of two (**Th1–Th12** and **C7–L5**) segments of the supporting column of the vertebral complex of children in different age groups

Given the actual data allow us to obtain information on the longitudinal size of the columns supporting the "sheath" of the spinal cord and the contribution to it of the sum of the heights of vertebral bodies and intervertebral disks we are interested in age groups.

As a result, all the conditions for calculating the actual reserve of physiological thoracic kyphosis, the filling of which leads to the formation of the "flat back" syndrome, are obtained.

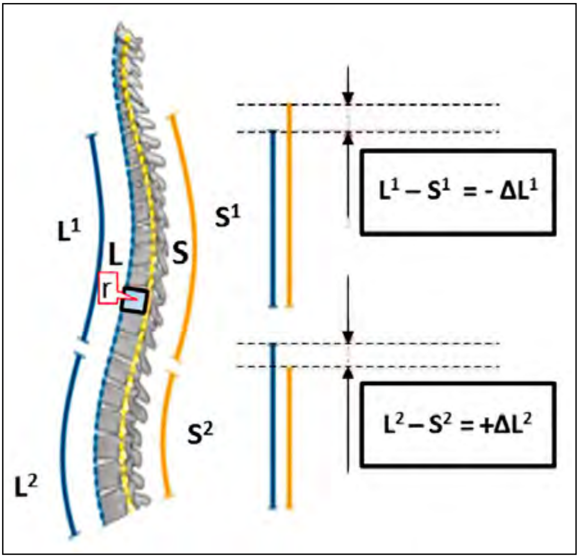


Figure 8: Size ratio of ventral **L** and dorsal **S** columns in the model with "thoracic kyphosis" and "lumbar lordosis".

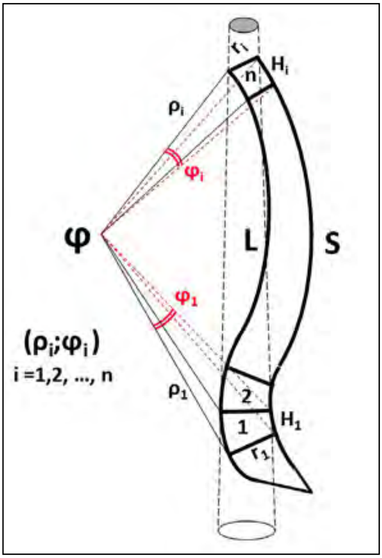


Figure 9: A scheme of the two-column model with "sagittal bends";

As it was noted in the **Introduction** when describing the tasks that the musculoskeletal "sheath" solves in growing children, its sagittal physiological curves resist nonlinear and unpredictable (daily, seasonal, annual and gender) growth spurt. From our point of view, this mechanism is the first physiological barrier preventing the immediate development of medullo-vertebral conflict.

It can be argued that during the evolution of Homo erectus for this purpose in the real vertebral column formed sagittal physiological curves during which initially there are differences (**$-\Delta L^1$** in the zone of thoracic kyphosis and **$+\Delta L^2$** in the zone of lumbar lordosis) between the corresponding areas of the ventral **L** and dorsal **S** columns, since **$S^1>L^1$**, and **$S^2<L^2$** (**Figure 8**).

Because of this circumstance, the process of compensation **ΔL** cannot be described only by equation (1), which requires for itself the initial equality between **L** and **S**. This "alignment" of **L** and **S** for "thoracic kyphosis", for the area of the most frequent localization of scoliosis in humans, is already described by equation (3), solving the problem – what **ΔL** must be added to **L**, so that **L = S**, and the angle **φ** = 180°. It, this equation, allows us to find the value **ΔL**, making the model strictly straight.

$$\Delta L = \sum_{i=1}^{n} \varphi_i \left[\sqrt{r_i^{\,2} + \rho_i^2 + \frac{2 r_i \rho_i}{\sqrt{1+\mathrm{ctg}^2 \varphi_i}}} - \rho_i \right] \tag{3}$$

Equation (3) allows one to determine the value **ΔL**, necessary to obtain the straightness of the two-column model, where: **ΔL=L–S**, as the desired difference in the lengths of the two part departments; **r** – is the distance between the columns; **n** – is the size of the grid (large enough); **ρ(φ)** – is a function interpolating the ventral curve **L** on a given grid (at the point **$\varphi(\rho_i) = \varphi_i$**, where **$\varphi_i$** and **$\rho_i$** are the partition of the curve representing one of the parts of **S** into the grid).

The proposed version of the model for solving this particular problem is based on the following provisions:

1. Only one posteriorly curved (kyphosis-like) segment of the projection of the vertebral complex on the sagittal plane is considered, represented as a figure enclosed between the arcs of two concentric circles (**Figure 10A**).
2. The difference in the radii of these circles coincides with the anterior-posterior size of the bearing column of the complex in the sagittal plane.
3. In the model there is already mentioned a number of boundary conditions arising from the anatomy of the real vertebral column:
   a) its vertical orientation, ensuring the coincidence of the projections of the upper (cranial) and lower (caudal) ends on the horizontal plane;
   b) continuity of communication between columns **S** and **L** at all levels;
   c) only column **L** has the "ability" to lengthen.
4. The model straightening process (**Figures 10B, 10C** and **10D**) is subject to investigation.

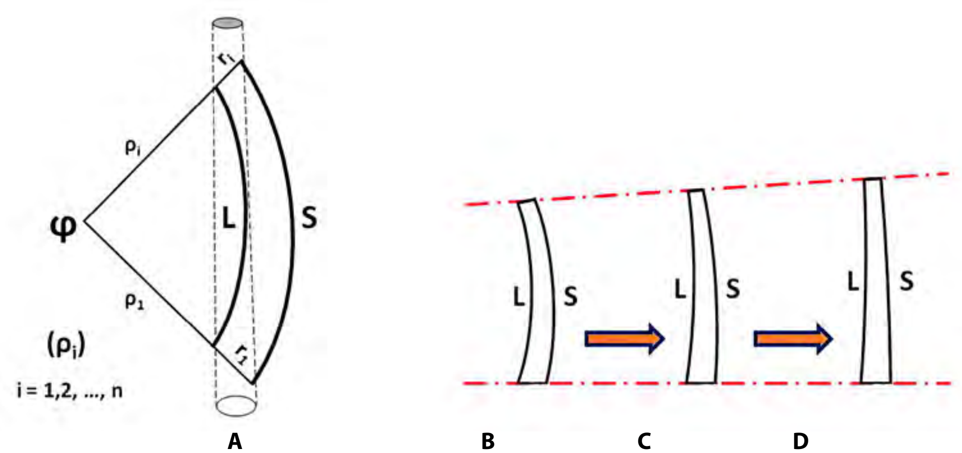


Figure 10. A – is the projection of the "spine" on the sagittal plane, reflecting the thoracic vertebral complex of the region the most frequent localization lordoscoliosis deformation; **B**, **C**, and **D** – are the graphic representation of the process of development of the syndrome of "flat back" in a healthy thoracic spine complex (**B** – source, **C** – intermediate, **D** – outcome).

It is fair to note that idiopathic three-plane deformities occur in the lumbar part (in the cervical and lumbo-sacral such can be only against the background of malformations). However, the preliminary analysis has shown – a number of anatomical and biomechanical features of Homo erectus begin to play a role in their mechanogenesis, which require the introduction of our basic biomechanical model of additional parameters. However, this is another problem and we will not give its solution here.

The key parameters of the model given in picture 10 are the angle (**φ**) of the arc of the "spine" section and its thickness (**r**). The length of the arc **S** is considered constant. The transition to "flat back" consists in straightening these two arcs, which in mathematics is equivalent to leaving the common center of the circles to infinity (with the angle **φ** tends to **180°**). In the initial (corresponding to the form of normal thoracic kyphosis) arcuate state (**Figure 10B**), the length of the inner arc **L** is less than the length of the outer arc **S**. When the lengths of both arcs are equal (**Figure 10D**), the initial deformation disappears.

In this version of the model, the arc length difference (**ΔL**) is given by equation (4):

$$\Delta L = \varphi \cdot r \tag{4}$$

This equation (4) gives one a correlation between column length difference (**ΔL = L–S**) and model parameters – angle (**φ**) reflecting sagittal bend of model and (**r**) reflecting distance between columns.

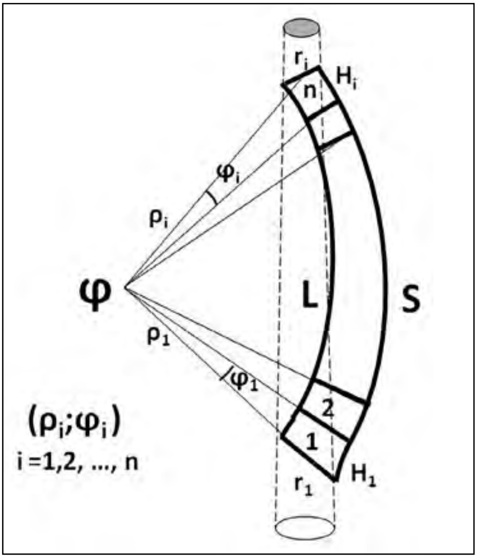


Figure 11: Graphical division of the area between the arcs **L** and **S** into areas corresponding to individual vertebrae.

Note that in this model, the average values of the heights of the vertebral bodies and their anterior-posterior dimensions are taken into account. To solve the problem, the **L** and **S** arcs are divided into sections corresponding to individual "vertebrae" (**Figure 11**).

Now, each vertebra corresponds to the angle ($\boldsymbol{\varphi_i}$):

a) the proportion ($\boldsymbol{\varphi_i}$) in the angle ($\boldsymbol{\varphi}$) is proportional to its height $\mathbf{H_i}$ (in our case $\mathbf{H_i = hv_i + hd_i}$)[13]

b) the value of (**r**) for each vertebra (anteroposterior size of the vertebral body in the sagittal plane).

Thus, the difference in the lengths of two arcs **ΔL = L−S** and the angle ($\boldsymbol{\varphi}$) can be used as numerical parameters when describing both the process causing the uniplanar deformation of the model and the process of its elimination, if it is initially present.

The applied value of solving this problem is to provide actual (numerical) criteria for objective forecasting of the development of the "flat back" syndrome. The length difference **ΔL** in this case can be calculated using equation (5), obtained as a result of transformation of equation (3) (**15, 17**).

$$\Delta L = \sum_{i=1}^{n} \varphi_i r_i = \varphi \cdot \frac{\sum_{i=1}^{n} H_i r_i}{\sum_{i=1}^{n} H_i} \tag{5}$$

Here: **Δ**L is the desired difference in the lengths of **L** and **S**; **φ** is the value of the bending angle of the model; **H** is the value of the model segment corresponding to the sum of the heights of the vertebral body and intervertebral disc; **r** is the size of the anteroposterior vertebral body size.

[13] This technique is justified by the fact that a separate introduction to the mathematical calculations of the height of the vertebral bodies (**hv**) and (**hd**) will only complicate the narrative.

RESULTS

The solution of equation (5) allows us to calculate the difference between the lengths of the arcs, or the desired **ΔL**, from the angle (**φ**) and the parameters of a particular section of the model. Since breast kyphosis, taken as an object of our study, has its own strict anatomical boundaries, we determined the range of possible **ΔL** values in different age groups for sites including 12, 10, 8, 6 and 4 vertebrae.

This step is justified by the fact that 12 vertebrae are their total number in this department of the spinal column. However, according to clinical observations, a change in the severity of breast kyphosis can be caused by an increase in heights and their other number (10, 8 and 6 vertebrae, but not less than 4).

In this regard, for the selected ("thoracic") section of the model, a change in the angle **φ** (dependence argument) from "norm" to "flat back" can be associated with the number of vertebrae in the following relation (6).

$$\varphi = 180° - 2{,}5 \cdot n \tag{6}$$

Equation (6) shows the dependence of the angle (**φ**) on the number of vertebrae, the increase in height of which leads to "flat back" syndrome, where: **φ** – is the total angle of the curved segment of the model; **180°** – is the angle of the model segment that reflects the end of the formation of the "flat back" syndrome; **2,5** – is the correction factor for the thoracic region of the real spinal column; **n** – is the number of vertebrae due to which the model is straightened.

For the vertebrae **n=12** (full thoracic region), **ΔL** values for the angle **φ**=150° can be considered the "absolute norm" of these values for the thoracic region.

For the vertebrae n = 10, 8, 6, and 4, according to formula (5), we obtain $\boldsymbol{\varphi_{10}}$, $\boldsymbol{\varphi_{8}}$, $\boldsymbol{\varphi_{6}}$, and $\boldsymbol{\varphi_{4}}$, respectively, equal to **155°**, **160°**, **165°**, and **170°**. The "norms" obtained by $\boldsymbol{\varphi_{n}}$ in tables 5, 6, 7, 8 and 9 are shown in bold. The values in italics reflect the "threshold" of the angle **φ** as the beginning of the clinical appearance of the "flat back" syndrome.

The results obtained, taking into account the real linear dimensions of the vertebrae in each age group, are presented in the form of actual values in **tables 5–9**.

age	kyphosis angle									
	150	153	156	159	162	165	168	171	174	177
9 y.	**0.89**	0.8	0.71	0.62	0.53	0.44	*0.35*	0.26	0.17	0.08
10 y.	**0.89**	0.8	0.71	0.62	0.53	0.44	*0.35*	0.26	0.17	0.08
11 y.	**0.88**	0.79	0.7	0.61	0.53	0.44	*0.35*	0.26	0.17	0.08
12 y.	**0.9**	0.81	0.72	0.63	0.54	0.45	*0.36*	0.27	0.18	0.09
13 y.	**0.97**	0.87	0.78	0.68	0.58	0.48	*0.39*	0.29	0.19	0.09
14 y.	**1.08**	0.97	0.87	0.76	0.65	0.54	*0.43*	0.32	0.21	0.1

Table 5: The **ΔL** values (**cm**) obtained for kyphosis of varying severity (in degrees) of the entire thoracic region, including **12** vertebrae (**cm**) in children aged 9 to 14 years.

age	kyphosis angle									
	150	153	**156**	159	162	165	168	171	174	177
9 y.	0.96	0.87	**0.77**	0.67	0.58	0.48	*0.38*	0.28	0.19	0.09
10 y.	0.97	0.87	**0.77**	0.67	0.58	0.48	*0.38*	0.28	0.19	0.09
11 y.	0.97	0.87	**0.77**	0.67	0.57	0.48	*0.38*	0.28	0.19	0.09
12 y.	0.98	0.88	**0.78**	0.68	0.58	0.49	*0.39*	0.29	0.2	0.1
13 y.	1.06	0.95	**0.85**	0.74	0.63	0.53	*0.42*	0.31	0.21	0.1
14 y.	1.18	1.07	**0.95**	0.82	0.71	0.59	*0.47*	0.35	0.23	0.11

Table 6: The **ΔL** values (**cm**) obtained for kyphosis of varying severity (in degrees) in the area including 10 vertebrae (**cm**) in children aged 9 to 14 years.

age	kyphosis angle									
	150	153	156	**159**	162	165	168	171	174	177
9 y.	1.18	1.06	0.95	**0.82**	0.7	0.59	*0.47*	0.35	0.23	0.11
10 y.	1.17	1.06	0.94	**0.82**	0.7	0.58	*0.47*	0.35	0.23	0.11
11 y.	1.16	1.06	0.93	**0.83**	0.7	0.58	*0.47*	0.35	0.23	0.11
12 y.	1.19	1.08	1.0	**0.83**	0.71	0.59	*0.47*	0.36	0.24	0.12
13 y.	1.29	1.17	1.03	**0.9**	0.77	0.64	*0.51*	0.38	0.25	0.13
14 y.	1.44	1.29	1.2	**1.0**	0.86	0.72	*0.57*	0.43	0.28	0.14

Table 7: The **ΔL** values (**cm**) obtained for kyphosis of different severity (in degrees) in the area including 8 vertebrae (**cm**) in children aged 9 to 14 years.

age	kyphosis angle									
	150	153	156	159	162	**165**	168	171	174	177
9 y.	1.51	1.36	1.2	1.06	0.8	**0.75**	*0.6*	0.45	0.3	0.15
10 y.	1.41	1.35	1.2	1.05	0.9	**0.75**	*0.6*	0.45	0.3	0.15
11 y.	1.49	1.35	1.2	1.05	0.9	**0.75**	*0.6*	0.45	0.3	0.15
12 y.	1.51	1.37	1.21	1.06	0.91	**0.76**	*0.61*	0.46	0.3	0.16
13 y.	1.64	1.48	1.31	1.15	0.99	**0.82**	*0.61*	0.49	0.32	0.17
14 y.	1.84	1.66	1.47	1.29	1.1	**0.92**	*0.73*	0.55	0.36	0.18

Table 8: The **ΔL** values (**cm**) obtained for kyphosis of different severity (in degrees) in the area including 6 vertebrae (**cm**) in children aged 9 to 14 years.

age	kyphosis angle									
	150	153	156	159	162	165	168	**171**	174	177
9 y.	2.11	1.89	1.68	1.48	1.23	1.06	*0.84*	**0.63**	0.42	0.2
10 y.	2.11	1.9	1.69	1.48	1.23	1.06	*0.84*	**0.65**	0.42	0.21
11 y.	2.11	1.9	1.69	1.48	1.23	1.06	*0.84*	**0.65**	0.42	0.21
12 y.	2.11	1.9	1.69	1.49	1.27	1.06	*0.85*	**0.65**	0.42	0.21
13 y.	2.3	2.08	1.84	1.61	1.39	1.15	*0.92*	**0.69**	0.46	0.23
14 y.	2.57	2.32	2.06	1.8	1.54	1.24	*1.04*	**0.76**	0.51	0.26

Table 9: The **ΔL** values (**cm**) obtained for kyphosis of different severity (in degrees) in the area including 4 vertebrae (**cm**) in children aged 9 to 14 years.

Now, having information about the linear dimensions of the main elements of the supporting column of the vertebral complex, we come to the main clinical goal of the biomechanical modeling of 3D deformation in the form of lordoscoliosis at its first **pre**clinical stage.

Achieving this goal is the answer to the question: How long does the doctor have to carry out therapeutic measures aimed at preventing further progressive development of lordoscoliosis?

The answer to this question can be obtained by comparing data on the actual severity of chest physiological kyphosis (in Coob's degrees) with a value of **+ΔL**, the appearance of which will cause the development of the "flat back" syndrome (**tables 5, 6, 7, 8** and **9**) and the predicted elongation of the bearing column in accordance with the age of the child (**table 4**).

Example 1. The child is 10 years old. Particular attention is paid to the severity of breast kyphosis. Its value is **170°** by Cobb. To develop the "flat back" syndrome, it is sufficient to lengthen the supporting column by **ΔL** equal to **0.26 cm** (**table 5**), or **0.28 cm** (**table 6**), or **0.35 cm** (**table 7**), or **0.45 cm** (**table 8**), or **0.65 cm** (**table 9**).

Meanwhile, it is normal for a child of this age group to predict that by the age of 11, the thoracic support column will grow by only **42.13 – 41.11 = 1.02 cm** (**table 4**). Here we note that we still cannot predict the number of vertebrae that can take part in a possible lordoscoliotic 3D deformation, therefore, suppose their maximum number is **12** vertebrae (**table 5**).

A sufficient, almost four-fold excess of the predicted growth gain (**1.02 cm**, **table 4**) over the value of the sagittal bend reserve (**0.26 cm**, **table 5**) allows us to conclude that the doctor has only three "quiet" months to monitor the condition the spine of the patient. And if by the end of this period a decrease in the severity of physiological chest kyphosis appears, the child should be included in the "scoliosis risk group" with the appointment of therapeutic measures (and in this case they become **preventive**!, author notes) so that the growth rate of the supporting column does not go out beyond the specified values.

Example 2. **The child is 12 years old.** Actively engaged in a choreographic school. The value of physiological kyphosis is **177°** by Cobb, i.e. with the practically formed "flat back" syndrome and the remaining “reserve” for confronting a possible medullo-vertebral conflict is not more than **2 millimeters** (**tables 5, 6, 7, 8** and **9**). At the same time, the expected increase in the thoracic region of the supporting column by **13** years old will be **45.95–44.58 = 1.37 cm** (**table 4**), which greatly exceeds the patient’s compensatory capabilities.

In this situation, the doctor has almost no time for contemplative observation of the patient, since it is necessary to immediately begin the implementation of a treatment program aimed at preventing any, even the smallest, unpredictable growth shocks in the supporting skeleton as a whole and in the vertebral complex in particular (**16**).

If such a program is not implemented, then the likelihood of maintaining the medulo-vertebral conflict increases, and for objective reasons, the next, **sub**clinical stage of compensating for the excess length of the “sheath” of the spinal cord will begin. In the model, this process, with the above limitations, manifests itself in the form of torsion (twisting) of a long column **L** around a short **S**, which illustrates the transition **A** → $\mathbf{C_1}$ in **Figures 5** and **6**.

Part 3

Aim of part 3.

The purpose of this part of the work is to identify the conditions for the development of the torsion component of 3D deformation in the two-column model of the vertebral complex after it has acquired strict verticality (“flat back” syndrome).

Conditions of the problem

The model found strict straightness of the columns while maintaining all the boundary conditions mentioned above:

a) its vertical orientation, ensuring the coincidence of the projections of the upper (cranial) and lower (caudal) ends on a horizontal plane;
b) the continuity of communication between columns **S** and **L** at all levels;

The process of twisting a long elongating column **L** around a column **S**, which retains its longitudinal size, is subject to investigation, since it reflects the first step in the formation of the horizontal component of 3D deformation, or scoliosis.

The solution to this problem is carried out using equation (7).

$$\Psi = \sqrt{\frac{L_0}{r^2} \cdot \Delta L}, \text{ or } \Psi = \sqrt{\frac{\sum_{i=1}^{n}(hv_i + hd_i)}{r^2} \cdot \Delta L}, \text{ where } \sum_{i-1}^{n}(hv_i + hd_i) = L_0 \qquad (7)$$

Equation (7) describes the relationship between **ΔL** and **ψ**, where: $\mathbf{L_0 = \Sigma\ (hv_i + hd_i)}$; **r** – is the distance between the columns, **ΔL** – is the value of the “growth” of the column **L**.

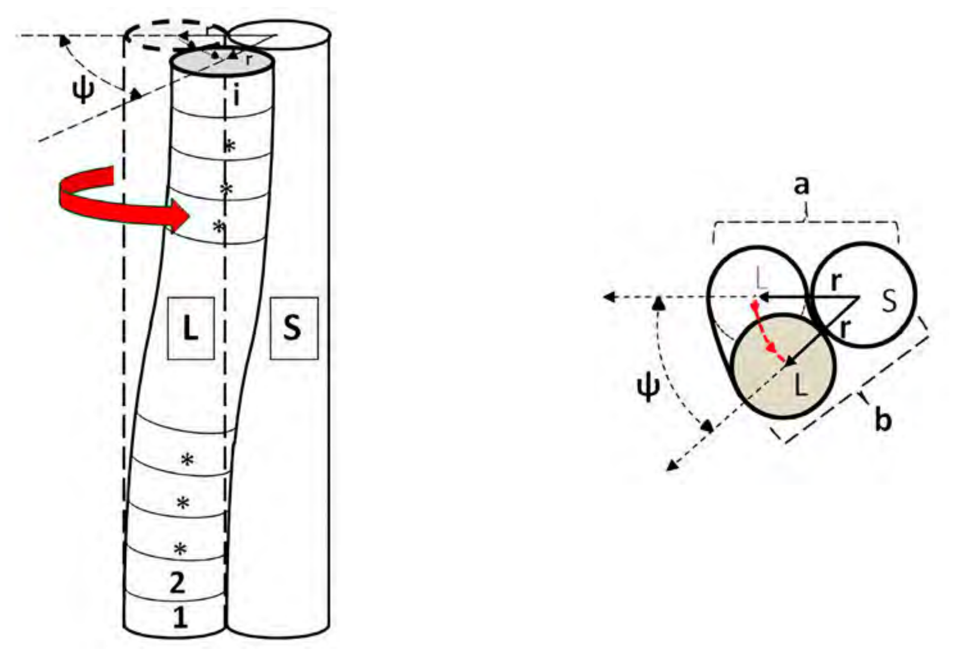


Figure 12: Volumetric (**A**) and flat (**B**) torsion schemes in a two-column model. In scheme **B**: **a** – is the initial position of the columns after the transition $\mathbf{B_1} \rightarrow \mathbf{A}$, reflecting the "flat back" syndrome; **b** – is the result of the transition $\mathbf{A} \rightarrow \mathbf{C_1}$.

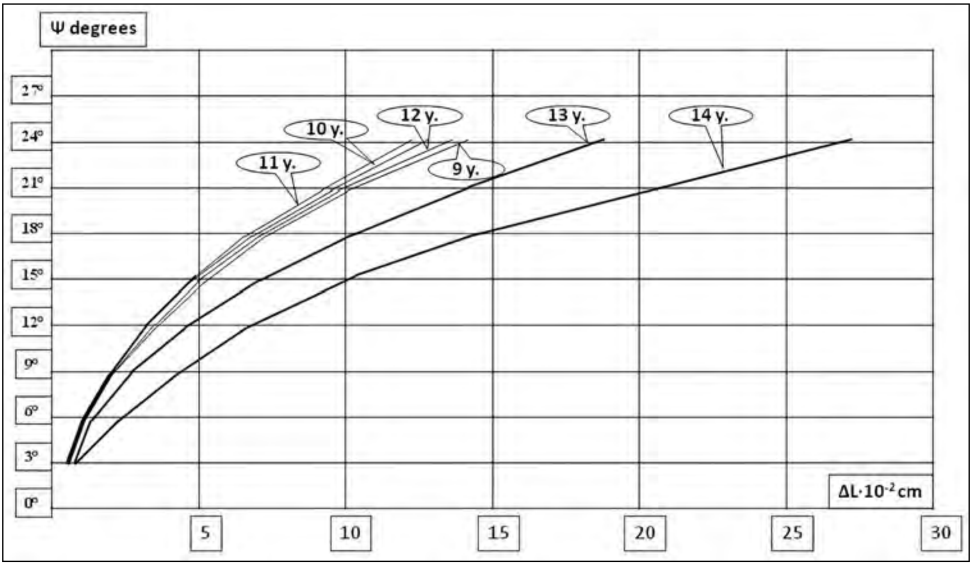
Equation (7) describes the patterns of the appearance and evolution of torsion in an empirical (virtual, abstract) model of the vertebral complex, and this allows one to establish a numerical relationship between the ongoing increase in **ΔL** and (**ψ**), as the angle of rotation (torsion) of the highest point of a long column. A scheme of this process is shown in **Figure 12**.

The obtained calculated numerical values of the angle (**ψ**) (in degrees) for a model whose external dimensions are close to the actual dimensions of the children's vertebral complex are presented in **tables 10** and **11**, and for greater clarity, they are shown in **graphs 5** and **6**.

age	total of vertebrae	*L_0 cm	rotation angle (ψ)				
			0,1 rad	0,21 rad	0,31 rad	0,36 rad	0,41 rad
			6°	12°	18°	21°	24°
9 y.	18	38,94	$\Delta L = 4{,}5 \cdot 10^{-3}$ = 0,0045 cm	$\Delta L = 2{,}0 \cdot 10^{-2}$ = 0,02 cm	$\Delta L = 4{,}5 \cdot 10^{-2}$ = 0,045 cm	$\Delta L = 6{,}0 \cdot 10^{-2}$ = 0,06 cm	ΔL=7,8·10-2 = 0,078 cm
10 y.	18	41,11	$\Delta L = 4{,}3 \cdot 10^{-3}$ = 0,0043 cm	$\Delta L = 1{,}9 \cdot 10^{-2}$ = 0,019 cm	$\Delta L = 4{,}2 \cdot 10^{-2}$ = 0,042 cm	$\Delta L = 5{,}7 \cdot 10^{-2}$ = 0,057 cm	$\Delta L = 7{,}5 \cdot 10^{-2}$ = 0,075 cm
11 y.	18	42,13	$\Delta L = 5{,}0 \cdot 10^{-3}$ = 0,005 cm	$\Delta L = 2{,}2 \cdot 10^{-2}$ = 0,022 cm	$\Delta L = 4{,}9 \cdot 10^{-2}$ = 0,049 cm	$\Delta L = 6{,}6 \cdot 10^{-2}$ = 0,066 cm	$\Delta L = 8{,}6 \cdot 10^{-2}$ = 0,086 cm
12 y.	18	44,58	$\Delta L = 6{,}0 \cdot 10^{-3}$ = 0,006 cm	$\Delta L = 2{,}6 \cdot 10^{-2}$ = 0,026 cm	$\Delta L = 5{,}9 \cdot 10^{-2}$ = 0,059 cm	$\Delta L = 8{,}0 \cdot 10^{-2}$ = 0,08 cm	ΔL=0,104 cm
13 y.	18	45,95	$\Delta L = 6{,}4 \cdot 10^{-3}$ = 0,0064 cm	$\Delta L = 2{,}8 \cdot 10^{-2}$ = 0,028 cm	$\Delta L = 6{,}3 \cdot 10^{-2}$ = 0,063 cm	$\Delta L = 8{,}6 \cdot 10^{-2}$ = 0,086 cm	ΔL=0,111 cm
14 y.	18	50,19	$\Delta L = 9{,}1 \cdot 10^{-3}$ = 0,0091 cm	$\Delta L = 4{,}0 \cdot 10^{-2}$ = 0,04 cm	$\Delta L = 8{,}9 \cdot 10^{-2}$ = 0,089 cm	ΔL=0,121 cm	ΔL=0,158 cm
average		43,82	0,006	0,026	0,058	0,069	0,102

* data on the longitudinal size of the supporting column for **$C_7 – L_5$** are taken from **table 4**.

Table 10: The angle of rotation (torsion) of the cranial end of the column **L** in the two-column model with an increase in **ΔL** over **$L_0 = C_7 – L_5$**

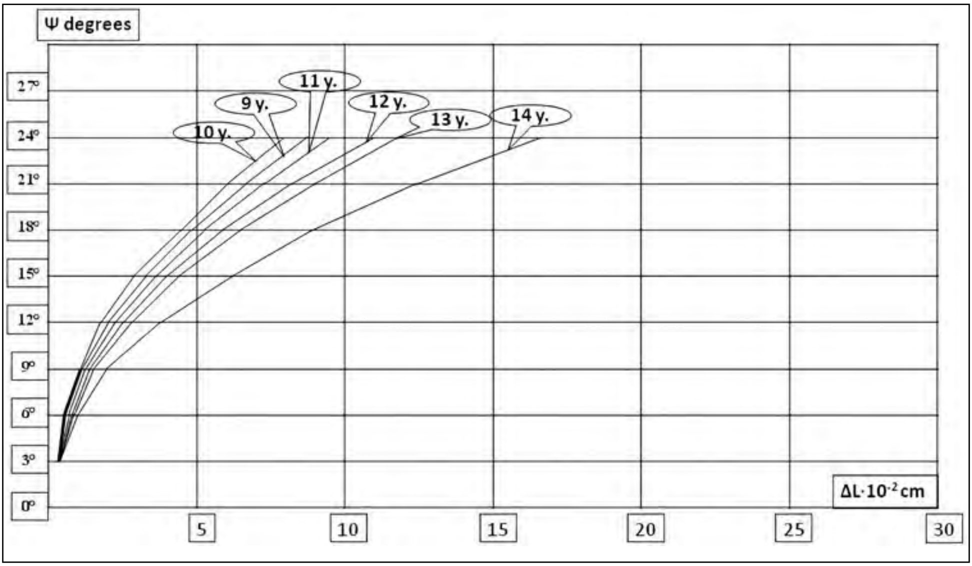


Graph 5: Graphs of the function **ψ = ƒ(ΔL)** with the participation of **18** vertebrae in the deformation (**$C_7 – L_5$**).

age	total of vertebrae	*L_0 cm	rotation angle (ψ)				
			0,1 rad	0,21 rad	0,31 rad	0,36 rad	0,41 rad
			6°	12°	18°	21°	24°
9 y.	12	23,09	$\Delta L=7{,}6\cdot10^{-3}$ = 0,0076 cm	$\Delta L=3{,}3\cdot10^{-2}$ = 0,033 cm	$\Delta L=7{,}4\cdot10^{-2}$ = 0,074 cm	ΔL=0,1 cm	ΔL=0,131 cm
10 y.	12	24,33	$\Delta L=7{,}2\cdot10^{-3}$ = 0,0072 cm	$\Delta L=3{,}1\cdot10^{-2}$ = 0,031 cm	$\Delta L=7{,}0\cdot10^{-2}$ = 0,07 cm	$\Delta L=9{,}6\cdot10^{-2}$ = 0,096 cm	ΔL=0,124 cm
11 y.	12	24,99	$\Delta L=7{,}0\cdot10^{-3}$ = 0,007 cm	$\Delta L=3{,}0\cdot10^{-2}$ = 0,03 cm	$\Delta L=6{,}9\cdot10^{-2}$ = 0,069 cm	$\Delta L=9{,}3\cdot10^{-2}$ = 0,093 cm	ΔL=0,121 cm
12 y.	12	26,40	$\Delta L=7{,}2\cdot10^{-3}$ = 0,0072 cm	$\Delta L=3{,}2\cdot10^{-2}$ = 0,032 cm	$\Delta L=7{,}0\cdot10^{-2}$ = 0,07 cm	$\Delta L=9{,}6\cdot10^{-2}$ = 0,096 cm	ΔL=0,125 cm
13 y.	12	27,35	$\Delta L=1{,}0\cdot10^{-2}$ = 0,01 cm	$\Delta L=4{,}5\cdot10^{-2}$ = 0,045 cm	$\Delta L=10\cdot10^{-2}$ = 0,1 cm	ΔL=0,138 cm	ΔL=0,179 cm
14 y.	12	29,79	$\Delta L=1{,}6\cdot10^{-2}$ = 0,016 cm	$\Delta L=6{,}8\cdot10^{-2}$ = 0,068 cm	ΔL=0,15 cm	ΔL=0,21 cm	ΔL=0,27 cm
average		25,99	0,009	0,040	0,089	0,122	0,158

* data on the longitudinal size of the supporting column for **Th_1 – Th_{12}** are taken from **table 4.**

Table 11: The angle of rotation (torsion) of the cranial end of the column **L** in the two-column model with an increase in Δ**L** over **$L_0 = Th_1 - Th_{12}$**



Graph 6: The graphs of the function **ψ = ƒ(ΔL)** with the participation of **12** vertebrae in the deformation. (**$Th_1 - Th_{12}$**).

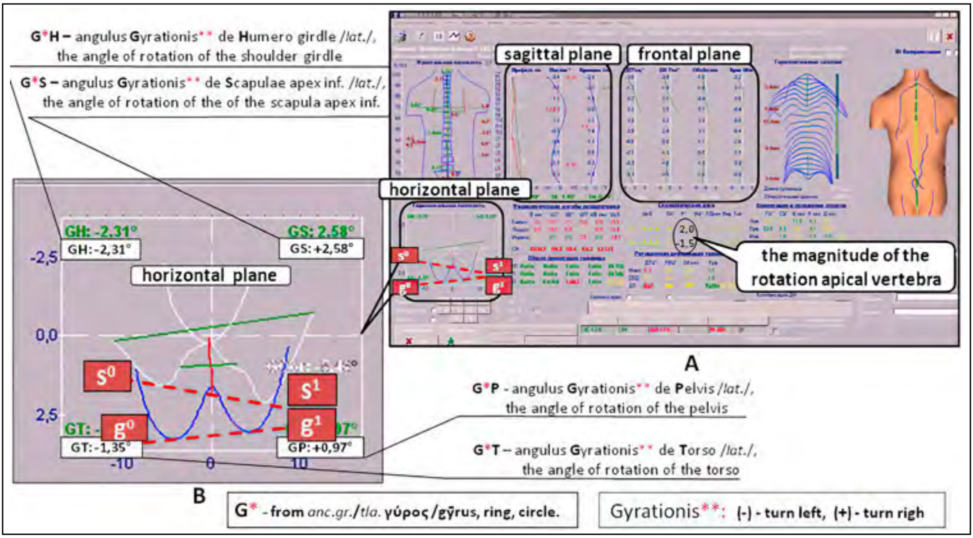


Figure 13: A – is the final protocol of computer-optical topographic diagnostics. On the horizontal plane, the line $\mathbf{s^0s^1}$ is the position of the frontal axis at the level of the shoulder girdle (scapules) and the line $\mathbf{g^0g^1}$ is the position of the frontal axis at the level of the gluteus (pelvic girdle); **B** – is the fragment of the final protocol with digital values of changes in the horizontal plane at various levels of the body. In this fragment can clearly see the collapse of the parallelism of the $\mathbf{s^0s^1}$ and $\mathbf{g^0g^1}$ axes against the background of the absence of a frontal arc in the spinal column, which is illustrated by the picture in the upper left part of the protocol **(A).**

The data shown in **tables 10** and **11** and illustrated in **graphs 5** and **6** indicate two obvious facts:

- a direct correlation between the **ΔL** value and the rotation angle (**ψ**) of the cranial end of the elongating column;
- high "sensitivity" of the column torsion mechanism **L** to minimal changes in its length under the existing boundary conditions. So, for example, to obtain a significant angle **ψ** ≈ 12° (**0.21 rad**) it is enough to have **ΔL** ≈ 0.03 cm, which is less than **1% (!)** of ≈35[14] cm.

But these are calculated values and in the abstract model they easily make it possible to obtain types of compensation **ΔL** (see **Figure 2**) that are unnatural for a real spinal column, which clearly "go beyond" the boundaries of the permissible.

[14] These values reflect the conventional average values of **ΔL** and the same average values of the longitudinal size of the supporting column for all age groups with participation in the deformation from 12 to 18 vertebrae (**tables 10 and 11**).

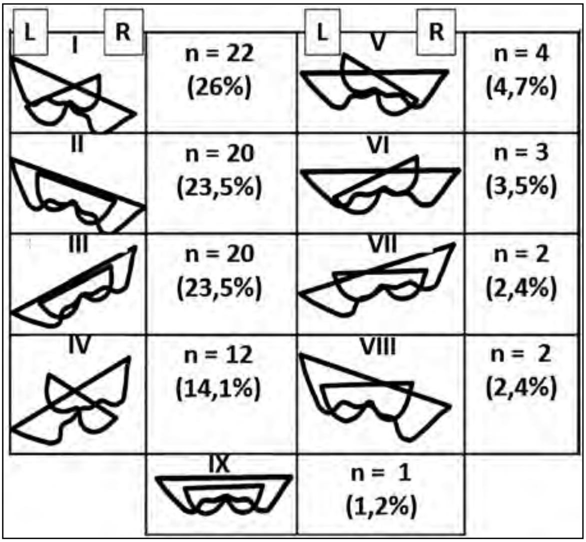


Figure 14. Variants of the rotation shoulder and pelvic girdles in the horizontal plane (results of the examination 86 children).

In addition, **two phenomena** were discovered:

1. The first one is that for the same number of interested vertebrae (both **18** and **12**), for turning the cranial end of the column **L** by the same angle (**ψ**), children 9 years old require a larger value **ΔL** than children **10** years old (**tables 10 and 11**).
2. The second one is that if only the thoracic vertebrae are interested (**n=12**) in children of **11** years, to obtain the same angle (**ψ**), a smaller **ΔL** value is required than in children of **10** and **12** years old.

We attribute the explanation of these phenomena to age-related changes in the anteroposterior size of the vertebral bodies (**aps**), which, with a certain degree of conditionality, are assumed to be (**r** – is the distance between the columns **L** and **S**). However, it should be noted that despite the linear pattern of increase in the (**aps**) parameter over one averaged spinal column for an individual age, distinct differences are observed between the age groups themselves (**Figure 9**)

We believe that since the parameter (**r**), calculated using equation ($\mathbf{2_2}$) with the coefficients from **table 2**, is a "participant" in the calculations in equation (**7**), its age-related variability undoubtedly has a significant impact on the final results.

It should be emphasized that we are not categorical in the above explanation and these phenomena require further study.

Therefore, in order to determine the values of the angle (**ψ**) observed in practical scoliosology, we studied the results of computer-optical diagnostics (**Figure 13**) of a group of children with initial subclinical and clinical symptoms of lordoscoliosis.

The software features of this technology are that it provides the ability to obtain actual values of the vertebral torsion at any level of the vertebral complex[15]. As a result of a survey of a group of children (86 people, girls : boys – 48 : 38, aged from 7 to 17 years, average – 12.8 years), the following variants of the ratios of the frontal axes at the level of the shoulder (line $\mathbf{s^0s^1}$) and pelvic (line $\mathbf{g^0g^1}$) girdle directions (**Figure 14**).

As follows from the above figure, the most common options were $\mathbf{I^{st}}$ (25%), $\mathbf{II^{nd}}$ (23.5%) and $\mathbf{III^{rd}}$ (23.5%). Option **IV** was less common (14.1%). Moreover, the horizontal displacement between the pelvic and shoulder girdles (**ψ**) ranged from 0.2° to 17° (average value – 4.5°). Thus, the torsion volume (**ψ**) observed in real patients is significantly less than mathematical calculations predict. This circumstance, not without reason, can be taken as an additional boundary condition, which must be taken into account to ensure the adequacy of the proposed model

DISCUSSION

To explain the phenomenon of the variety of variants of the ratios of the frontal axes s0s1 and $\mathbf{g^0g^1}$ of the vertebral complex, obtained in a real clinic, it is necessary to proceed from the fact that a multi-element vertebral column, for all the complexity of its structure, is just a part of the supporting structure called the "human skeleton"[16]. It, as noted above, is an accomplice to the complex task of maintaining a stable sagittal, frontal and horizontal balance of the human body, the position of which in space is similar to the inverted "Kapitsa pendulum" (*footnote #6*).

Moreover, in the skeleton of Homo erectus one can see several of these "pendulums" with an oscillating suspension point. The first is the feet and lower legs, relying on the earth. The second is the hips resting on the oscillating lower legs. The third is the pelvic girdle with a multi-element vertebral complex, based on the hips. Finally, the head rests on the most mobile section of the spine – the cervical. All these segments together form the already generalized "Kapitsa pendulum" (**20**).

We believe that only from these positions can one explain the variety (9 options) of the ratios of the frontal axes of the shoulder and pelvic girdles in the horizontal plane. But since the connections between the numerous "pendulums" in posturology have not yet been considered in detail (we

[15] Undoubtedly, this diagnostic method has certain limitations in its sensitivity, but there is no other solution for the children contingent.

[16] The "human skeleton" is a closed biomechanical system. It consists of bones, muscles and ligaments. The first – provide rigid support and protection of soft tissue organs. The second is the movement between the bones. Third, on the contrary, these movements limit. It is important to note that with the apparent heterogeneity of the tasks facing each of the components of the skeleton, their execution is synchronized to the highest degree.

believe that a specific solution to this problem is for future research, author notice), then for now let us dwell on what has been achieved and take into account the average angle (**ψ**).

The values of the angle (**ψ**) obtained during the diagnosis, which can be regarded as an integral indicator of the functioning of the very generalized Kapitsa pendulum, become the boundary condition dictated by the real anatomy. In other words, if mathematical calculations show a sufficiently large range for the angle (**ψ**), then in reality it falls into the range from 0.2° to 17° (with an average value of 4.5°)[17].

This circumstance clarifies the condition for the problem solved in this article. Now it sounds like this: what **ΔL** should be in order to cause primary torsion of the vertebral complex within **17°**. From **tables 10** and **11** it follows that to achieve an angle (**ψ**) of this value, it is enough to have **ΔL** in the range from **0.045** to **0.089 cm** with **18 vertebrae** participating in scoliotic deformation and from **0.07** to **0.15 cm** for the thoracic region of **12 vertebrae**. A simple translation of this data in %% of the longitudinal size of the supporting column shows an amazing picture – this is an average of only **0.34%** for an arch of **18 vertebrae** and **0.132%** for the thoracic region. By the way, these data obtained using modern computer technology completely coincided with the results of calculations on the simplest calculators back in 1981 (**12**).

With all the surprise that these minimum **ΔL** values cause, which cause torsion compensation for the inconsistency of the longitudinal growth of the spinal cord and its "sheath", they clearly see the goal for therapeutic measures at this stage of the transition of a healthy vertebral complex to the "scoliotic" status. This is a conservative "containment" of the very longitudinal extension of the bone-disco-ligament-muscular "sheath" (envelope) of the spinal cord. After all, it is only necessary to eliminate the effect of "extra" millimeters in the size of the supporting column, which can appear in the most commonplace situation in the life of a child.

As objects of influence of such procedures, growth zones of vertebral bodies and hydrophilic intervertebral discs can be called.

But back to the effect that was described. J. B. Baron in the middle of the XXth century is an asymmetric increase of the tonic activity in the paravertebral muscles (**6, 7, 8**). As noted above, the clinical manifestation of this effect is the elimination of a stable deviation of the optical axis of the eyes that occurs during surgery on oculomotor muscles. Meanwhile, the same deviation of the optical axis of the child's eyes occurs during primary torsion of his vertebral complex, in which the head is turned in the ipsilateral (from lat. ipse, self + latus, lateral) side. It is this circumstance, from our point of view, that will quite reasonably lead to the realization of the effect of J. B. Baron with clinical detorsion of the cranial part of the vertebral complex by the angle (**ψ**). In other words, primary torsion, as the sec-

[17] It is important to note here that the angle (**ψ**) obtained in real "scoliosology" completely coincides with the conclusion of the French ophthalmologist J.B. Baron (**6, 7, 8**), who showed that a shift of the optical axis of the eye by 4° in patients after surgery on oculomotor muscles causes unilateral bioelectric activity in the paravertebral muscles. The clinical effect of this phenomenon is the torsion of the cranial part of the spinal column (and the head!, author notice), due to which the aforementioned bias is eliminated.

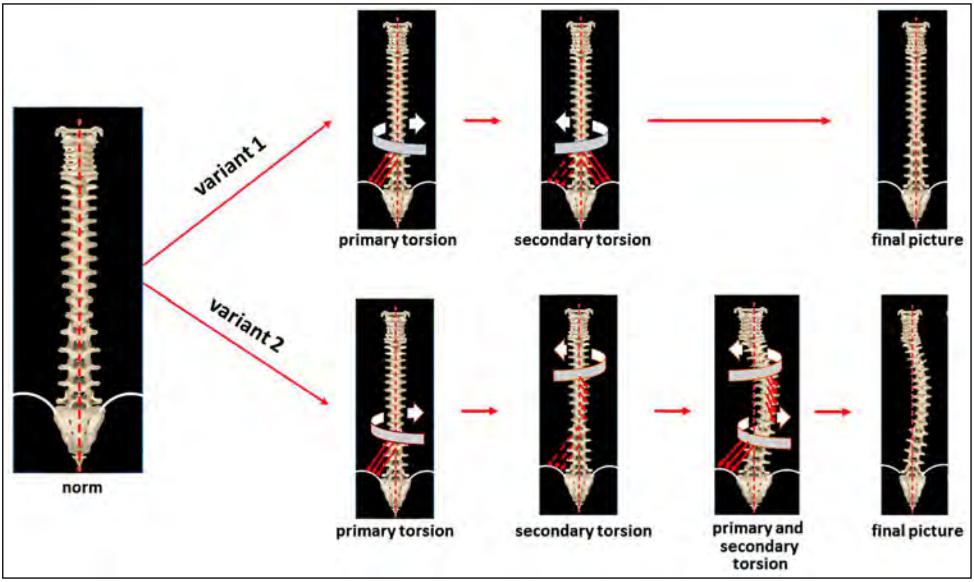


Figure 15: The scheme of the mechanism of primary torsion of the vertebral complex and two options for compensating for its result. **Variant 1** – compensation due to symmetrical contralateral mm. transversospinales. **Variant 2** – compensation due to cranially located contralateral mm. transversospinales.

ond[18] clinical response to the "medullo-vertebral conflict", makes it necessary to compensate for its clinical result in the form of detorsion. Now, attention – if detorsion is carried out by the symmetrical transversospinal muscles of the contralateral side, then the primary torsion will be completely eliminated (**Figure 15**, variant 1). In another case, if only contralateral transversospinal muscles of the cranial zone are included in the detorsion process, then detorsion of the vertebrae accountable to them will lead to the formation of the anti-turn. It, together with the primary turn, will appear in the final clinical picture in the form of frontal curvature of the supporting column (**Figure 15**, variant 2).

It should be noted here that on an isolated two-columned biomechanical model to restore the strict sagittal location of its cranial region (axes $\mathbf{a^0b^0}$ and $\mathbf{a^1b^1}$ on **Figure 4, 5, 6**), which corresponds to the normal direction of the conditional "optical axis", the magnitude of detorsion (secondary, author notice) should be equal to the angle (ψ) of the primary torsion. However, in a real musculoskeletal system, which appears to be several Kapitsa pendulums located one above the other (**20**). In this design, it is permissible that the detorsion process can be carried out not only in the cranial region of the vertebral complex, but also in the caudal, due to its moving point of support (see *footnote #6* and second paragraph in the Discussion of the part 3, author notice). From this position, the phenomenon of several options in the relationship of the frontal axes of the shoulder and pelvic girdles (**Figure 14**) identified in real patients, can be explained.

18 Recall that the first clinical response we consider the development of the "flat back" syndrome (author notice)

CONCLUSION

In literature one can find works that take steps to use the mathematical apparatus for an objective description of scoliosis and the first of them is "Aanmerkingen over de Wanstaltingheden der Ruggengraat" by the Dutch scientist D. van Gesscher (1735–1810) [as presented by Piet JM van Loon in "Recent Advances in Scoliosis", 2012. www.intechopen.com]. Meanwhile, the ideology of this study is significantly different from the publications of our colleagues, since it examines a quasi-dynamic[19] two-column model of scoliosis like deformation. Since we have not met analogues of this approach, we allow ourselves to believe that this is possibly the first study of the kind in the world that reveals the fundamental biomechanical laws in the occurrence and evolution of one of the most mysterious lesions of the musculoskeletal system in growing children and adolescents. The authors hope that it will provoke a discussion among competent specialists around the following issues.

1. The adequacy of the biomechanical model. Our first steps in "creating" of the two-column spine model described above were based on only one non-alternative conclusion of theoretical mechanics: "3D deformations (three-plane deformation) of the spine can be formed only in a two-column structure in which one of the columns is able to increase its longitudinal size". Such deformation is found among many compensation options arising under the condition of rigid fixation of the ends of the columns.

   The clinical basis of this approach is the most indisputable fact in the theory and practice of scoliosis – the relationship of its occurrence and subsequent evolution with the process of child growth.

2. From the anatomical, embryological and biomechanical point of view, the spine is composed of two basic and functionally very different elements, i.e. of the spinal cord and its bone-disco-ligament-muscular sheath. Each of these two anatomical elements has a different growth rate and different length, which biomechanically greatly affect the properties and behaviour of the whole healthy/pathological vertebral complex. This conclusion demonstrates our full agreement with Milan Roth (**24**, **25**) that "Idiopathic scoliosis is caused by short spinal cord" (1968) and "Idiopathic scoliosis: a special type of osteo-neural growth disproportion" (1969). However, it can be noted that this great Czech scientist considered only one option in their relationship – a short spinal cord in combination with large its "sheath", while our model allows us to consider the second natural option, in which there is a deficit in "sheath" longitudinal size relative to the spinal cord[20].

3. The presented biomechanical model takes into account three combinations of the lengths of the spinal cord (**S)** and the bone-muscle "sheath" (**L)** and simultaneously combines two types of spatial (3D) deformations:

---

19 Quasi /lat. quasi/ – adverb corresponding in meaning to the words "imaginary", "false", "fake".

20 Despite the universality of the two-column model, the conditions of a particular problem and its solution for the **L<S** variant (with a deficit of the length of the "sheath") differs significantly from the conditions for the **L>S** variant. For this reason, independent research will be devoted to it.

(1.) **L < S**; (2.) **L = S**, (3.) **L > S**.

There is the **kyphoscoliosis** described in combination (1.) and in (3.) the **lordoscoliosis** is described. It is therefore clear that the considered biomechanical model simultaneously includes two very common cases in orthopaedic practice.

4. Scoliosis is a pathological process of the compensatory response of the vertebral complex to the non-conjugate during the longitudinal growth of its two main components, i.e. the spinal cord (related to the directive nervous system) and its bone muscle “sheath”. The presented biomechanical study contributed to two views on deformation processes from the point of aetiology view and from the point of pathogenesis view.

Note: The team of authors is coming up with an idea to create a program for computer prediction of the evolution of scoliosis, incorporating our factual and theoretical results reflected above into it.

Acknowledgement

The authors thank Professor Ivo Mařik and Professor Miroslav Petrtýl (Prague, Czech Republic) for their invaluable help in editing this article.

LITERATURE

1. ABEDRABBO G., FISETTE P., ABSIL P.-A. et al. A multibody-based approach to the computation of spine intervertebral motions in scoliotic patients. In Studies in Health Technology and Informatics, Volume 176. Editors T. Kotwicki and T.B. Grivas. Research into Spinal Deformities 8. IOS Press. 2012: 95–98.
2. ADAMS W. Lectures on the pathology and treatment of lateral and other forms of curvature of the spine. London: Churchill. 1882.
3. ARMSTRONG G.W.D., LIVERMORE N.B., SUZUKI N. et al. Non-standard vertebral rotation in Scoliosis Screening Patients: Its prevalence and relation to the clinical deformity. Spine. 1981: № 7: 50–55.
4. AUBIN C.-É., DANSEREAU J., PETIT Y. et al. Three-Dimensional Reconstruction of Vertebral Endplates for the Study of Scoliotic Spine Wedging. In: Studies in Health Technology and Informatics, Volume 37. Editors J.A. Sevastik and K.M. Diab. Research into Spinal Deformities 1. IOS Press. 1997: 165–168.
5. AUBIN C.-É, LOBEAU D., LABELLE H. et al. Planes of Maximum Deformity in the Scoliotic Spine. In: Studies in Health Technology and Informatics. Volume 59: Editor I.A.F. Stokes. Research into Spinal Deformities 2. IOS Press. 1999: 45–48.
6. BARON J.B. Muscles moteurs oculaires, attitude et comportement locomoteur des vertébrés. Thèses de Sciences. Paris. 1955: 158.
7. BARON J.B. Muscles moteurs oculaires, céphalées, déséquilibre et attitude scoliotique. Presse med. 1955: vol.63, № 20. 407–410.
8. BARON J.B., USHIO N., NOTO R. Oculo-nuco-vestibulo-spinal system regulating tonic postural activity; statokinesimetric study. Agressologie. 1974: № 15: 395–400.
9. CANNON W.B. Wisdom of the body. New York: W.W. Norton & Co, 1932: 312.

10. CHIGRIK N.N. Geometric modeling of multiparametric processes of scoliotic deformities of the spine in order to create a system of diagnostics and prognosis. Diss. … Cand. technical sciences. Omsk: Omsk University, 2002: 294.
11. DENIS F. The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine (Phila Pa 1976). 1983: vol.8, № 8: 817–831.
12. DUDIN M.G., SINITSKIY Yu.F. On the mechanogenesis of torsional changes in scoliosis. J. Orthopedics, traumatology. Prosthesis (USSR). –1981: № 2: 33–36.
13. DUDIN M.G., SINITSKIY YU.F., SADOFIEVA V.I. Differential diagnosis of idiopathic scoliosis with atypical pathological rotation of the vertebrae. Diseases and injuries of the spine in children: Interinstitutional collection. scientific works. Editor V.L. Andrianov. Leningrad. Medicina. –1981: 77–80.
14. DUDIN M.G. Scoliosis with atypical pathological rotation of the vertebrae – diagnosis, course, treatment tactics. Diss. ... Cand. med. sciences. Leningrad: Institute of Traumatology and Orthopedics by R.R. Vreden, 1981: 210.
15. DUDIN M.G., BALOSHIN YU.A., POPOV I.YU. et al. Mathematical modeling of three-plane deformity of the human spinal column. Russian Journal of Biomechanics. 2016: vol. 20, № 3: 272–282.
16. DUDIN M.G. Idiopathic scoliosis: prevention, conservative treatment. Saint Petersburg: Man, 2017: 223.
17. DUDIN M., BALOSHIN Y., POPOV I. et al. On the linear dimensions of vertebrae and intervertebral discs of children in the beginning of puberty. J. Craniovert Jun Spine, 2018: № 9: 246–249.
18. HIPPOCRATES. Selected books. Translated from Greek by prof. V.I. Rudnev. Editorial board, introductory articles and notes by prof. V.P. Karpov. Moscow: State Publishing House of Biological and Medical Literature. Series "Classics of Biology and Medicine" 1936: 736.
19. KAPANDJI A.I. Spine. Physiology of the joints. 6th edition. Eksmo. 2014: 539.
20. KAPITSA P.L. Dynamic stability of a pendulum at an oscillating suspension point. J. Exp. and theor. physics, 1951: vol. 21, № 5: 588–597.
21. KOROTAEV E.V. Diagnostics and treatment of early stages of idiopathic scoliosis in children. Thesis … Cand. med. science. Saint Petersburg: Institute of Traumatology and Orthopedics by R.R. Vreden, 1999: 16.
22. LÜFTINGER M. Aetiology of idiopathic scoliosis: Current biomedical research and osteopathic theories. Master Thesis zur Erlangung des Grades Master of Science in Osteopathie. Wien: An der Donau Universität Krems niedergelegt an der Wiener Schule für Osteopathie, 2008: 76.
23. RISSER J.C. Scoliosis: past and present. J Bone Joint Surg., 1964: 46-A: 167–199.
24. ROTH M. Idiopathic scoliosis caused by short spinal cord. Acta Radiologica Diagnosis, 1968: vol.7 №3: 257–271.
25. ROTH M. Idiopathic scoliosis: a special type of osteo-neural growth disproportion. Z. Orthop Ihre Grenzgeb., 1969: 107(1): 37–46.
26. SCAMMON R.E., The ponderal growth of the extremities of the human fetus. Am. J. of Phys. Anthropol., 1930: vol.15 (1): 111–121.
27. SCHULTHESS W. Ueber die Lehre des Zusammenhanges der physiologischen Torsion der Wirbelsaule mit lateraler Biegung und ihre Beziehungen zur Skoliose unter Berucksichtigung der Lovett'schen Experimente. Z.Orthop., 1902: № 10: 455–494.
28. SEBER J.A.F. Linear regression analysis. Ttransl. from English: Moscow: Mir, 1980: 456:
29. SHANDS A.R.JR., BARR J.S., COLONNA P.C. et al. Endresult study of the treatment of idiopathic scoliosis: Report of the Research Committee of American Orthopedic Association. J Bone Joint Surg (Am.), 1941; 22: 963–977.
30. STAFFEL F. Die menschlichen Haltungstypen und ihre Beziehungen zu den Ruckgratverkrum-mungen. Wiesbaden: J. F. Bergman, 1889: 92.
31. Steindler A. The compensation treatment of scoliosis. J Bone Joint Surg (Am.), 1929: 11(4): 820–830.

32. TIKHONOV A.N., SAMARSKY A.A. Equations of Mathematical Physics. Moscow: Main edition of Phys. Series "Mathematical Literature", 1953: 680.

Corresponding author's address:
Prof. Mikhail Georgiyevich Dudin, MD, DSc.
Children's Rehabilitation Center of Orthopedics and Traumatology 'Ogonyok', St. Petersburg, Russia
E-mail: mdudin@list.ru

INFORMACE O SPOLEČNOSTI PRO POJIVOVÉ TKÁNĚ ČLS J. E. PURKYNĚ (SPT)

Vážená paní kolegyně, vážený pane kolego,

dovolujeme si Vás informovat o možnosti stát se členem **Společnosti pro pojivové tkáně** (SPT), která v roce 2004 navázala na plodnou desetiletou činnost Společnosti pro výzkum a využití pojivových tkání vedenou panem prof. MUDr. M. Adamem, DrSc. Posláním SPT je podpora rozvoje výzkumu pojivových tkání, šíření nových poznatků týkajících se všestranných analýz tkání z obecného pohledu, moderních klinických přístupů k diagnostice a léčbě. Dalším posláním SPT je usnadnění styků mezi jednotlivými odborníky navázáním spolupráce s různými vědeckými, odbornými, výrobními a farmaceutickými společnostmi.

Vědecké poznání a aplikace nejnovějších poznatků v klinické praxi nabyly v posledních letech nebývalého zrychlení, a to nejenom v zahraničí, ale i u nás. Tato skutečnost bezprostředně souvisí s kvalitativním rozvojem poznání i v nebiologických vědách a v moderních inženýrských přístupech. Stále více se prokazuje, že vše se vším souvisí – není náhodou, že nové poznatky a objevy vznikají na rozhraní oborů a různých vědních disciplín. Lidská společnost v posledních desetiletích dosáhla nové civilizační kvality – ve vědě a v jejich aplikacích zcela jistě, avšak v morálce a etice ne tak příliš. Biomedicína je v současné době rozsáhlou interdisciplinární vědou, která bez kooperace s jinými vědními obory by byla odsouzena ke stagnaci. Proto cílem SPT je nejenom integrovat odborníky v biomedicíně, ale i v technických sférách.

Prioritní snahou SPT je presentovat odborné veřejnosti a specialistům v klinické praxi nejnovější poznatky v oblasti pojivových tkání. SPT je i společenskou organizací klinických pracovníků, vědců, pedagogů, která si klade za cíl společensky sblížit nejenom pracovníky v aktivní službě, ale i kolegyně a kolegy v důchodovém věku a v neposlední řadě i studenty a mladé doktorandy z vysokých škol, universit a akademických ústavů.

SPT organizuje během každého roku alespoň dvě odborná a společenská setkání, kde vedle odborných přínosů je kladen důraz také na společenské – přátelské diskuse všech vás, kteří nechtějí stagnovat a kteří nechtějí přemýšlet o nových poznatcích izolovaně a osamoceně.

Pro uhrazení nejzákladnějších nákladů na korespondenci se členy společnosti, jejich informovanost a pořádání odborných kolokvií, symposií a společenských odborných setkání byl stanoven **roční členský příspěvek pro aktivní kolegyně a kolegy 200 Kč** a **pro studenty a důchodce 100 Kč.**
SPT vydává časopis Pohybové ústrojí – pokroky ve výzkumu, diagnostice a terapii, do kterého se i vy můžete aktivně zapojit odbornými články a vašimi zkušenostmi. **Pro současné odběratele časopisu PU a další zájemce doporučujeme přihlásit se na http://www.pojivo.cz/en/newsletter/, zadat jméno a e-mailovou adresu, na kterou bude časopis posílán. Na webové doméně SPT ČLS JEP http://www.pojivo.cz/cz/pohybove-ustroji/ naleznete ve formátu PDF všechna jednotlivá čísla a dvojčísla časopisu (včetně Suplement) vydaná od roku 1997 (bezplatný přístup).**
Milí kolegové, nestůjte opodál a připojte se k české inteligenci – v oblasti pojivových tkání, ke které i Vy zcela jistě patříte. V naší krásné české zemi je třeba, aby prameny poznání byly stále živé a permanentně udržované. Poslání každého z nás není náhodné. Jsme velice zavázáni našim předkům, kteří rozvíjeli kvalitu odbornosti v naší zemi. Nepřipusťme útlum vědy u nás. Nenechme se zmanipulovat programovanou lhostejností, vyrůstající z neodbornosti, závisti a z patologického prosazování ekonomicko-mocenských zájmů.

Těšíme se na Vás a na Vaše zkušenosti – přijďte mezi nás!

Za výbor společnosti:

Prof. MUDr. Ivo Mařík, CSc. – předseda
Prof. MUDr. Josef Hyánek, DrSc. – čestný předseda
Prof. Ing. Miroslav Petrtýl, DrSc. – místopředseda
RNDr. Martin Braun, PhD – vědecký sekretář
Ing. Jana Zelenková – pokladník

Přihlášku do Společnosti pro pojivové tkáně ČLS JEP, z.s. najdete na adrese:
http://www.pojivo.cz/cz/wp-content/uploads/2020/02/PrihlaskaCLS_JEP_SPT_form.pdf

Přihlášku do Ortopedicko-protetické společnosti ČLS JEP, z.s. najdete na adrese:
http://www.pojivo.cz/cz/wp-content/uploads/2020/02/PrihlaskaCLS_JEP_OPS_form.pdf

INFORMATION ABOUT SOCIETY FOR CONNECTIVE TISSUES CMA J. E. PURKYNĚ (SCT)

Dear Sir/Madam, dear Colleagues,

We have great pleasure to inform you about the possibility of joining the **Society for Connective Tissues** (SCT) that was established in 2004 in order to continue the ten-year fruitful activities of the Society for Research and Use of Connective Tissue headed by Professor M. Adam, MD, DSc. The activities of the SCT are aimed at supporting the research development in the field of connective tissues, the dissemination of knowledge related to the all-purpose analyses of the tissues in general, and the application of the up-to-date approaches to the diagnostics and clinical practice. Further, the SCT is determined to facilitate contacts between the respective specialists by means of collaboration with various research, professional, production and pharmaceutical companies.

In the last few years, the scientific knowledge and the application of the latest findings in the clinical practice have accelerated on an unprecedented scale, not only abroad, but also in this country. This fact is closely connected with the qualitative development of the knowledge in the non-biological sciences and in the up-to-date engineering approaches. The fact that all things are mutually connected is becoming more and more evident. It is fairly obvious that the new knowledge and discoveries arise on the dividing line between the different fields and disciplines of science. In the last few decades, the human society has reached the new qualities of civilization. This applies, in particular, for the disciplines of science and their applications; however, this statement can hardly be used with reference to the moral and ethical aspects of the human lives. At present, the biomedical science is a wide-ranging interdisciplinary science which, in case of lack of cooperation with other scientific disciplines, would be condemned to stagnation. That is the reason why the SCT is aimed at integrating the specialists both within the biomedical science and within the engineering fields.

The priority objective of the SCT is to present the professional public and specialists involved in the clinical practice with the latest knowledge in the field of connective tissues. The SCT is also a civic society whose aim is to bring people close together by joining members of the clinical staff, researchers and teachers including the retired ex-colleagues and, last but not least, the undergraduates and PhD students from universities and academic establishments.

The SCT is planning to organize at least two professional and social meetings each year. Beside the professional contribution of these meetings, emphasis will be laid on social activities – informal discussions of all those who do not want to stagnate and who do not want to acquire the new knowledge in solitary confinement.

The annual membership fee is 200 Czech crowns for full workers, and 100 Czech crowns for students and pensioners. This membership fee shall be used to cover the basic costs on correspondence with the members of the Society in order to inform them about organizing colloquiums, symposiums and social meetings.

The SCT is also engaged in publishing of the interdisciplinary journal entitled Locomotor System – Advances in Research, Diagnostics and Therapy. You are invited to contribute to the journal writing professional articles, exchanging experience or, simply sharing your opinions. **You can find the volumes of Locomotor System journal at http://www.pojivo.cz/cz/pohybove-ustroji/ since 1997 (free of charge). Since 2013 only electronic edition of the journal is available. That is why we recommend to all subscribers and those interested apply at http://www.pojivo.cz/en/newsletter, enter personal data, titles and e-mail address where the journal will be mailed.**

Dear Colleagues, do not stand aside (suffering from terrible lack of time) and join the professional people in the field of connective tissues to whom you undoubtedly belong. In this beautiful country, the sources of knowledge should be kept alive and maintained permanently. Our role in this process is not accidental. We are much obliged to our ancestors who had developed the qualities of proficiency in this country. Do not allow the decline of science. Do not let the programmed indifference arising from lack of professionalism, enviousness, and pathological promotion of economic and power interests manipulate us.

We are looking forward to meeting you. We will be pleased if you join us and share your experience with us.

On behalf of the committee of the Society for connective tissues:
Professor Ivo Marik, MD, PhD – chairman
Professor Josef Hyánek, MD, DSc – honorary chairman
Professor Miroslav Petrtýl, MSc, DSc – vice-chairman
Braun Martin, Dr, PhD – research secretary
Zelenková Jana, Eng – treasurer

Membership application form of the Society for Connective Tissues, Czech Medical Association J.E. Purkynje, Prague you can find on the following link:
http://www.pojivo.cz/cz/wp-content/uploads/2020/02/PrihlaskaCLS_JEP_SPT_form.pdf

Membership application form of the Orthopaedic-Prosthetic Society, Czech Medical Association J.E. Purkynje, Prague you can find on the following link:
http://www.pojivo.cz/cz/wp-content/uploads/2020/02/PrihlaskaCLS_JEP_OPS_form.pdf

TÉMATIKA PŘÍSPĚVKŮ

K uveřejnění v časopise Pohybové ústrojí se přijímají rukopisy prací z oblasti pohybového ústrojí člověka, které se týkají především funkce, fyziologického i patologického stavu kosterního a svalového systému na všech úrovních poznání, diagnostických metod, ortopedických a traumatologických problémů, příslušné rehabilitace a léčebné i preventivní péče. Předmětem zájmu jsou týmové práce z oboru dětské ortopedie a osteologie, dále problémy z oboru biomechaniky, patobiomechaniky a bioreologie, biochemie a genetiky. Časopis má zájem publikovat články kvalitní, vysoké odborné úrovně, které přinášejí něco nového, jsou zajímavé z hlediska aplikací a nebyly doposud nikde uveřejněny s výjimkou publikace ve zkrácené formě.

Redakce přijímá původní práce a kazuistiky, souborné články, které informují o současném stavu v příslušných oblastech souvisejících s pohybovým ústrojím a abstrakty příspěvků z národních a mezinárodních konferencí, věnovaných hlavně pohybovému ústrojí. Původní práce a kazuistiky doporučuje publikovat v anglickém jazyce. Rukopisy jsou posuzovány 2–3 oponenty redakční rady. Redakční rada si vyhrazuje právo provádět recenze a drobné úpravy, případně zkrácení rukopisu. Je velmi žádoucí, aby autor reagoval na případné připomínky.

Nevyžádané rukopisy ani přílohy se nevracejí. Redakce si před uveřejněním prací vyhrazuje rovněž právo na určení pořadí umístění v časopise i jazykovou korekturu.

Příspěvky, uveřejňované v časopise, jsou excerpovány v periodických přehledech EMBASE/Excerpta Medica, vydávaných nakladatelstvím Elsevier a Bibliographia medica Čechoslovaca. Při výběru příspěvků k uveřejnění dáváme přednost rukopisům, zpracovaným podle jednotných požadavků pro rukopisy, zasílané do biomechanických časopisů – Uniform Requirements Submitted to Biomedical Journals (Vancouver Declaration, Br. Med. J., 1988, 296, pp. 401–405).

ÚPRAVA RUKOPISŮ

Rukopis se píše v textovém editoru Word ve formátech doc, docx nebo rtf. Na titulní straně uveďte název článku, pod ním jméno autora, případně autorů, úřední název jejich pracoviště a konečně adresu prvního autora. U českých rukopisů uvádějte název článku a pracoviště také v angličtině.

Na další straně uveďte stručný souhrn (do 250 slov), který má informovat o cílech, metodách, výsledcích a závěrech práce, doplněný překladem do angličtiny. Za ním připojte nejvýše šest klíčových slov v češtině resp. angličtině.

Vlastní text je u původních prací obvykle rozdělen na úvod, materiál a metodiku, výsledky, diskusi, závěr a případné poděkování. Souborné referáty, diskuse, zprávy z konferencí apod. jsou bez souhrnu a jejich členění je dáno charakterem sdělení. Před začátky jednotlivých odstavců nevkládejte žádné mezery ani tabelátory, odstavce by měly mít alespoň čtyři řádky.

TABULKY A OBRÁZKY

Tabulky a obrázky doplněné legendou vkládejte do dokumentu na zvláštním listě s příslušným označením nahoře, příp. jako samostatný soubor. Vyobrazení se číslují v pořadí, v jakém jdou za sebou v textu. V dokumentu označte jejich předpokládané umístění v textu. U českých rukopisů uvádějte texty k obrázkům i v angličtině. Obrázky by měly mít rozlišení 150 dpi u perokreseb (schémata a grafy 600 dpi) a uložené jako typ TIFF File (*.tif) nebo JPEG Bitmap File (*.jpg) tabulky a grafy uložené ve formátech Microsoft Excel (*.xls) nebo jako vektorové obrázky ve formátech (*.eps, *.pdf).

Pojmenování souborů

Název souboru by neměl obsahovat znaky s diakritikou a znaky: „.“ „,“, „:“, „;“, „!“, „?“. Pro lepší následnou orientaci v záplavě souborů je vhodné v názvu souboru uvádět **verzi, jméno autora** (bez diakritiky) a **název článku** (bez diakritiky).

LITERATURA

Seznam odkazů na literaturu se připojí v abecedním pořadí na konci textu. Odvolání na literaturu uvádějte ve vlastním textu příslušnými čísly v kulatých závorkách.

V seznamu citované literatury uvádějte údaje o knihách v pořadí: příjmení a iniciály prvních tří autorů s případným dodatkem „et al.“, název knihy, pořadí vydání, místo vydání, nakladatel, rok vydání, počet stran: Frost HM. The Laws of Bone Structure. 4 ed. Springfield: C.C.Thomas, 1964, 167 s.

Citace z časopisů uvádějte tímto způsobem: příjmení a iniciály prvních tří autorů (u více autorů vložte za jménem třetího autora et al., za jmény autorů nemusí být tečky), název článku, název časopisu nebo jeho uznávaná zkratka, ročník, rok vydání, číslo, strany: Sobotka Z, Mařík I. Remodelation and Regeneration of Bone Tissue at some Bone Dysplasias. Pohybové ústrojí, 2, 1995, č. 1:15–24.

Příspěvky ve sbornících (v knize) se uvádí v pořadí: příjmení a iniciály prvních tří autorů, název článku, editor, název sborníku, díl, místo, nakladatelství a rok vydání, strany ve sborníku (knize): Mařík I, Kuklík M, Brůžek J. Evaluation of growth and development in bone dysplasias. In: Hajniš K. ed. Growth and Ontogenetic Development in Man. Prague: Charles University, 1986, s. 391–403.

KOREKTURY

Redakce považuje dodaný rukopis za konečné znění práce. Větší změny při korekturách nejsou přípustné. Prosíme, aby autoři pečlivě zkontrolovali text, tabulky a legendy k obrázkům. Pro zkrácení publikační lhůty je možno připojit prohlášení, že autor netrvá na autorské korektuře. V rámci časových možností je snahou redakce všechny příspěvky zaslat autorům zpět k odsouhlasení konečné úpravy prací. Korektury prosím provádějte s využitím korekturních nástrojů v programu Acrobat Reader. Prosíme o co nejrychlejší zpětnou vazbu redakci časopisu.

ADRESA PRO ZASÍLÁNÍ PŘÍSPĚVKŮ

Rukopisy zasílejte na adresu:

Prof. MUDr. Ivo Mařík, CSc.
Ambulantní centrum pro vady pohybového aparátu s.r.o., Olšanská 7, 130 00 Praha 3
Tel.: (+420) 222 582 214, e-mail: ambul_centrum@volny.cz

SUBJECT MATTER OF CONTRIBUTIONS

The journal Locomotor System will publish the papers from the field of locomotor apparatus of man which are above all concerned with the function, physiological and pathological state of the skeletal and muscular system on all levels of knowledge, diagnostic methods, orthopaedic and traumatologic problems, rehabilitation as well as the medical treatment and preventive care of skeletal diseases. The objects of interest are interdisciplinary papers on paediatric orthopaedics and osteology, further object of interest are problems of biomechanics, pathobiomechanics and biorheology, biochemistry and genetics. The journal will accept the original papers of high professional level which were not published elsewhere with exception of those which appeared in an abbreviated form.

The editorial board will also accept the review articles, case reports and abstracts of contributions presented at national and international meetings devoted largely to locomotor system. The papers published in the journal are excerpted in EMBASE / Excerpta Medica and Bibliographia medica Čechoslovaca.

MANUSCRIPT REQUIREMENTS

Manuscripts should be submitted in text editor Microsoft Word in format *.doc, docx or *.rtf. While no maximum length of contributions is prescribed, the authors are encouraged to write concisely. The first page of paper should be headed by the title followed by the name(s) of author(s) and his/her (their) affiliations. Furthermore, the address of the corresponding author should be indicated to receive correspondence and proofs for correction. Papers are reviewed by two (and/or three) reviewers.

The second page should contain a short Abstract(up to 150 words) followed by the key words (no more than 6). The proper text of original paper is laid out into introduction, material and methods, results, discussion and if need be acknowledgement. The reviews, discussions and news from conferences are without summaries and their lay-out depends on the character of communication. The paragraphs should not begin with any spaces from the left margin nor tabs and should contain at least four rows.

ILLUSTRATIONS AND TABLES

Authors should supply illustrations and tables on separate sheets in the document. They should be numbered in the same order as is their desired location in the text. The figures should include the relevant details. Pictures should have resolution min. 150 dpi, drawings and graphs in bitmap resolution 600 dpi. They should be saved as tif or jpg format, tables and graphs in Microsoft Excel or as vector graphics in formats *.eps or *.pdf. Figure legends should be provided for all illustrations on a separate page and grouped in numerical order of appearance. On the back of figures, their number and name of the author should be indicated.

REFERENCES

References must be presented in a numerical style. They should be quoted in the text in parentheses, i.e. (1), (2), (3, 4), etc. and grouped at the end of the paper in alphabetical order.

The references of books should contain the names and initials of the first three authors, with eventual supplement „et al.", title of book, number of edition, place of publishing, name of publisher, year of appearance and number of pages, for instance: Frost HM. The Laws of Bone Structure. 4. ed. Springfield: C.C.Thomas, 1964, 167 p.

The references of papers published in journals should be arranged as follows: the names and initials of the first three authors (eventually after the name of the third author introduce et al.), title of the paper, journal name or its abbreviation, year, volume, number and page numbers, for instance: Sobotka Z, Mařík I. Remodelation and Regeneration of Bone Tissue at Some Bone Dysplasias. Locomotor System 1995: 2, No.1:15–24.

The references of papers published in special volumes (in a book) should be arranged in the following order: names and initials (there do not have to be dots after initials) of the first three authors, title of paper, editor(s), title of special volume (a book), place of publication, publisher, year of publication, first and last page numbers, for instance: Mařík I, Kuklík M, Brůžek J. Evaluation of growth and development in bone dysplasias. In: Hajniš K. ed. Growth and Ontogenetic Development in Man. Prague: Charles University, 1986:391–403.

Manuscripts and contributions should be sent to the Editor-in-chief:

Professor Ivo Mařík, M.D., Ph.D.
Ambulant Centre for Defects of Locomotor Apparatus
Olšanská 7
130 00 Prague 3
Czech Republic
Phone: (+420) 222 582 214
e-mail: ambul_centrum@volny.cz

OBSAH ROČNÍKU 2017

KASUISTIKY

ZPRÁVY

CONTENTS OF VOLUME 2017

CASE REPORTS

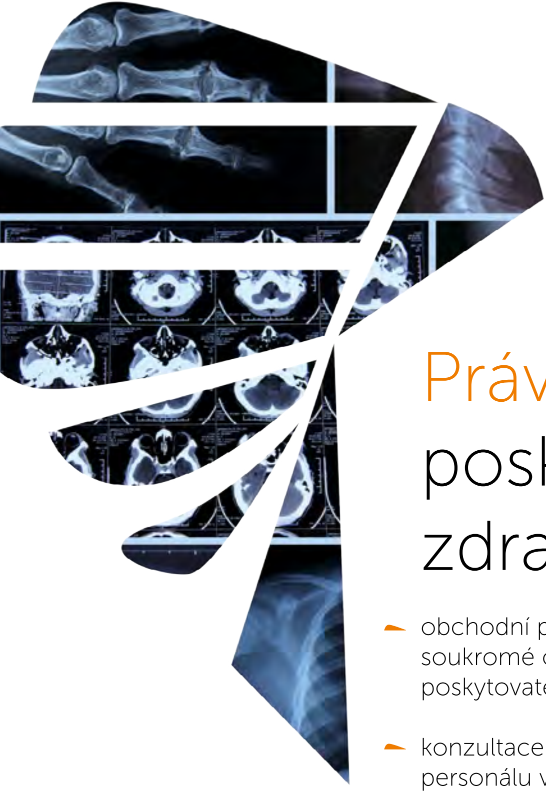
NEWS

OBSAH ROČNÍKU 2018

PŮVODNÍ PRÁCE

ZPRÁVY

CONTENTS OF VOLUME 2018



Právní služby poskytovatelům zdravotních služeb:

- obchodní právo - založení společnosti, transformace soukromé ordinace na společnost, registrace poskytovatele zdravotních služeb,
- konzultace v oblasti medicínského práva – školení personálu ve věcech vedení a nakládání se zdravotnickou dokumentací, informovaný souhlas pacienta,
- smluvní agenda – nájemní smlouvy, kupní a úvěrové smlouvy, smlouvy o službách,
- smlouvy se zdravotními pojišťovnami – úprava smluvních dokumentů, korekce plateb,
- otázky náhrady škody na zdraví a z titulu zásahu do osobnostních práv – konzultace vznesených nároků, jednání s pacienty, zastupování v soudním řízení,
- a všechny další otázky, s nimiž se poskytovatelé zdravotních služeb v praxi setkávají

V případě zájmu o nezávaznou konzultaci a poskytnutí bližších informací nás neváhejte kontaktovat.

SVOBODA & KUČERA | ADVOKÁTI

AK Kučera s.r.o. | Stavitelská 1099/6, 160 00 Praha 6 | T +420 222 965 861 | M +420 731 510 675 | E kucera@sk-advokati.cz | W www.sk-advokati.cz