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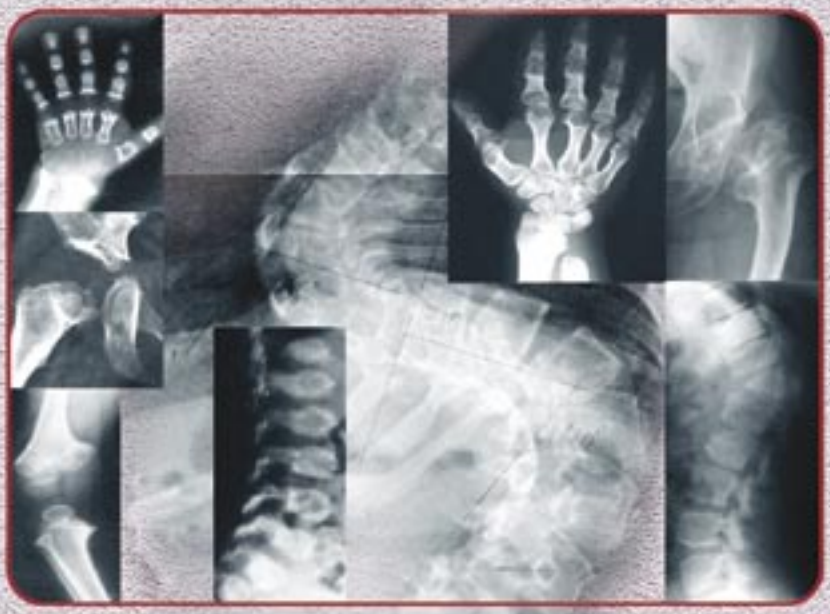
Pokroky ve výzkumu, diagnostice a terapii



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LOCOMOTOR SYSTEM

Advances in Research, Diagnostics and Therapy

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1+2/2003

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Vážení čtenáři, autoři a inzerenti,

V roce 2004 Vás již podruhé oslovujeme, abychom dostáli slibu dohnat zpoždění ve vydávání časopisu *„Pohybové ústrojí – pokroky ve výzkumu, diagnostice a terapii“*. Předpokládáme, že do konce roku 2004 bude vydáno ještě dvojčíslo 3 + 4/2003.

Vzhledem k faktu, že jsme se i přes značné ekonomické obtíže dopracovali k vydávání dvojčísel 10. ročníku, připomínáme čtenářům, že časopis Pohybové ústrojí vznikl za podpory Národní lékařské knihovny – jmenovitě pana PhDr. J. Drbálka – v roce 1994. V letech 1997 – 2002 zajišťovala vydávání časopisu firma Ortotika s.r.o. V roce 2000 převzal počítačovou sazbu pan MUDr. Petr Zubina a stal se odpovědným redaktorem časopisu. Spoluvydavateli jsou od roku 1994 Ambulantní centrum pro vady pohybového aparátu a Společnost pro výzkum a využití pojivových tkání. Od roku 2000 se spoluvydavatelem stala Katedra antropologie a genetiky člověka PřF UK v Praze. V roce 2003 přebírá úlohu vydavatele Společnost pro výzkum a využití pojivových tkání od firmy Ortotika s.r.o. a odpovědným redaktorem časopisu se stal pan Ing. Pavel Lorenc. Dalším spoluvydavatelem časopisu se v roce 2003 stala Česká společnost ortopedicko-protetická ČLS J. E. Purkyně, která finančně podpořila i zpožděné vydání dvojčísel časopisu ročníku 9/2002 a dvojčíslo, kterým právě listujete.

Předmětem a posláním časopisu zůstává publikování prací vycházející z výzkumu pojivových tkání, práce o biochemické, morfologické, genetické a molekulární diagnostice, kostním metabolismu u vrozených poruch i získaných vad. Dále klinické práce, týkající se symptomatické léčby kostních dysplazií, primárních i sekundárních metabolických kostních chorob, osteoporózy, osteo(spondylo)artrózy, končetinových anomálií, kombinovaných (dismorfických) vad pohybového aparátu a genetických syndromů, ale i jiných chorob pojiva. Zvláštní pozornost věnujeme pracem z oblasti biomechaniky, a to neuroadaptivním změnám skeletu, řízené remodelaci, muskuloskeletálním a neuronálním interakcím, dále sdělením antropologickým a paleopatologickým. Ceníme si především interdisciplinárně zaměřených příspěvků. Publikujeme i práce zahraničních autorů. Cenným doplněním jsou zprávy ze sjezdů a konferencí. V rubrice zprávy zveřejňujeme oznámení o životním výročí členů RR.

Jako každoročně uvádíme směrnice pro autory příspěvků. Původní práce a kasuistiky doporučujeme publikovat v angličtině.

Souhrny prací publikovaných v časopisu jsou excerpovány v EMBASE / Excerpta Medica, a proto doporučujeme autorům, aby využili této příležitosti a psali co nejvýstižněji souhrny s klíčovými slovy.

Redakční rada

VÝVOJ VZPŘÍMENÉHO POSTOJE LIDÍ ESEJ ORTOPEDICKÉ ANTROPOLOGIE*

THE EVOLUTION OF MAN'S UPRIGHT POSTURE AN ESSAY IN ORTHOPAEDIC ANTHROPOLOGY*

PHILLIP V. TOBIAS, F.R.S.S.AF., F.L.S. (LOND.), F.R.A.I.

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Dean: Faculty of Medicine, University of the Witwatersrand, Johannesburg

AVANT-PROPOS:

The Francois P. Fouché Lectures honour a well-known South African orthopaedic surgeon. Mr. Francois Fouché was a pathfinder, 'n ware voorloper, in the history of orthopaedics in South Africa. Dear to him was the relief of pain and the restoration of function in cripples and he was an innovator in the founding of cripple care services in South Africa. Indeed, this annual lectureship was endowed by the National Council for the Care of Cripples and was originally known as the Cripple Care Lecture when Sir Harry Platt delivered the first address in 1957.

Fouché was a dedicated professional gentleman. As medical students we attended orthopaedics out patients sessions, under his quiet but effective tutelage. Stories about him were legion: his unremitting hatred of highheeled shoes was proverbial and it was from a window in Chudleigh's Building (now John Orr's), in Eloff Street, Johannesburg, that he was known to hurl especially offending footware to the street below. Sister Welsh, known to hundreds of jittery consultants, quaking housemen and petrified students as 'the old battle-axe', remembers how Francois Fouché once visited a patient in her ward: he did not like the way a pillow had been placed under the patient's knees. 'Put it square, woman!' he cried and flung the pillow at her head. She recalls that he operated superbly, but could be unpredictable and even choleric in theatre.

His work for the disabled led his name to be attached since 1960 to the Cripple Care Lecture, sponsored by the National Council and under the auspices of The College of Medicine of South Africa.

* Republished with permission from the College of Medicine of South Africa.
Transactions of The College of Medicine of South Africa. January – June 1982

I warmly thank the Council of The College of Medicine and the Faculty of Orthopaedics for inviting me to be the 1981 Francois P. Fouché Lecturer. As The College this year celebrates the 25th anniversary of its foundation, it is a singular honour to have been invited to be the Silver Jubilee Lecturer, although this is, in fact, only the 23rd Francois P. Fouché Lecture.* On this jubilee occasion, it gives me great pleasure to congratulate The College on the inestimable contribution it has made to the advancement of medicine in Southern Africa.

THE EVOLUTION OF MAN'S UPRIGHT POSTURE

Human uprightness has long excited the attention of students of human evolution. This posture and its anatomical basis are among the most striking characteristics that distinguish man from the great apes. Yet, in the coming of man, the attainment of erect posture and of a bipedal gait were among the earliest distinctively human features to emerge: for the hominids (the family to which man belongs) have been walking upright for at least 3,5 to 4 million years! Uprightness helped man solve many problems. As Charles Darwin pointed out, it freed the hands from a locomotor function: so the hands became available for sustained use in other directions such as implemental activities. The way in which the body adjusted its structure and its biomechanics to uprightness and bipedalism is little short of ingenious - and I shall devote most of this lecture to a consideration of them. Nevertheless, after perhaps 4 million years or more, we have not yet evolved a fault-free mechanism. Our bodies are still subject to what Sir Arthur Keith (1923) called the ills of uprightness, including flat feet, slipped discs, hernias, prolapses and malposture. These maladies of uprightness account for much that keeps today's orthopaedic surgeons busy.

Thus the mechanism of man's posture and gait, though resourceful and craftily contrived, is imperfect. The first human ancestors to come upright became heir to a host of new problems: so it would be entirely fitting were the profession of orthopaedic surgery to acknowledge the early tottering bipeds as 'Our founders'!

POSTURE AND LOCOMOTION IN LIVING HIGHER PRIMATES

Two main components make for uprightness of the human body: erectness of the trunk and the 2-footed or bipedal stance and gait.

Since the time of Darwin, most students of human evolution have concentrated on bipedalism when speaking of the erect posture. For instance, Washburn and Howell (1960) wrote, 'This process (the development of bipedal locomotion) freed the hands, made possible the use and manufacture of tools, and led to reduction in the size of the teeth and the facial skeleton.' They added, 'Perhaps, as Darwin suggested, tool use is both the cause and the effect of *hominid bipedalism,* and the evolution of *erect posture* occurred simultaneously with the earliest use of tools' (*op. cit.,* p. 37) (italics inserted).

Many others have tried to relate tool-using or tool-making to bipedal locomotion.

* The 23rd Francois P. Fouché Lecture delivered in Durban on 26 May 1981. No lectures were given in 1958 and 1973.

It has long seemed to me that, by stressing the mode of locomotion in relation to tool-making, this approach was focusing on the wrong aspect of uprightness. In April 1965 I drew attention to the importance of *sitting upright* during implemental activities. Studies on the implemental activities of apes had led to the conclusion that habitual bipedalism was not a prerequisite either to effective tool-using or to rudimentary tool-making (Tobias, 1965). For instance, chimpanzees, which are not habitually bipedal, can make tools both in the wild and under experimental conditions.

More important, bipedalism is *not* the only circumstance in which a creature's hands are freed for such activities as tool-using and tool-making: all that an animal needs to do is to *sit* upright, for its hands to be available. In fact, most implemental activities of man and apes are carried' out in a sitting posture. In this position our bodies find greater stability than in the standing attitude. Stability is a most important structural and functional consideration in the development of manual skills. Moreover, sitting upright is widespread throughout the Primates. It characterises arboreal forms such as the capuchin monkey and terrestrial ones such as the baboon. lt is in the sitting position that a great part of the manipulative activities of Jane Goodall's chimpanzees are carried out. Sitting frees, the hands for implemental and other activities as surely as bipedalism does. This means that the postural basis for tool-using and tool-making is widespread in the Primate order, although bipedalism is not.

About the time I was developing these ideas, the importance of erectness of the trunk was being stressed by Straus (1962). He wrote,'It can safely be assumed that primates early developed the mechanisms permitting maintenance of the trunk in the upright position… Indeed, this tendency towards truncal erectness can be regarded as an essentially basic primate character.' It was, he felt, a consequence of a long tree-climbing heritage, that had been argued for so persuasively by Wood Jones (1916).

What makes the sitting position possible is that the body's centre of gravity is well back over the hind-limbs. This posterior placement of the body's centre of gravity was stressed long ago by Morton (1926). In sitting the animal holds its torso more or less upright, though the hind-limbs may be folded in various positions. Poirier (1977) has reminded us that all major groups of non-human primates include species that sit or sleep in an upright position. Thus the distribution in Nature of uprightness of the trunk far exceeds that of uprightness of the whole body. It follows that trunk uprightness probably long preceded the fully orthograde posture and it might well have been present since the emergence of the first primates 50 – 60 million years ago.

Let me turn now to upright posture as generally conceived, i.e. the habitual orthograde posture with 2-footed or bipedal gait.

We should first review the postures and gaits in today's man and his nearest living relatives. Primate posture and locomotion have been studied intensively in the last 30 years. Napier has recognised a number of locomotor patterns among living primates (1963, 1964, 1967, 1971). These include vertical clinging and leaping, especially among lower primates like galagos, tarsiers and lorises (Napier and Walker, 1967). Monkeys and apes are characterised as quadrupeds, semi-brachiators or brachiators.

This classification is based upon both locomotor behaviour and the structural adaptations of the limbs, but these are not hard-and-fast categories. Rather they are convenient reference points in a spectrum of activity patterns **(Table 1)**. They involve variable usages of the upper and lower limbs and diverse morphological adaptations (Oxnard, 1963; Napier, 1964).

The hind-limbs of brachiators are strongly adapted for suspension, though the hind-limb may also support the body from below. In semibrachiators and quadrupedal primates the hind-limb is used habitually to support the body from below and to propel it in locomotion, leaping and jumping. Thus, both semi-brachiators and quadrupeds lack the specializations of the brachiators' hind-limbs; their behaviour rather than their anatomy distinguishes the hind-limbs of semi-brachiators from those of quadrupedal primates.

It is very likely that, from such an unspecialized hind-limb structure, accompanying either quadrupedal or semi-brachiator locomotion, the bipedalism of man arose.

Napier (1967) thus envisages a vertical, clinging ancestral stage, passing next through a quadrupedal stage to the uprightness of modern bipedal man.

The great apes habitually stand or walk in an oblique quadrupedal position **(Fig. 1)**. However, the anatomical constraints are not so rigid that they prevent a non-human primate from standing or walking on occasion on 2 feet. Sporadic bipedal walking has been observed in capuchins, spider and woolly monkeys, baboons, macaques, gibbons, gorillas, chimpanzees and orangs (Hewes, 1961; Napier, 1964). However, the functional relations between hip bones and thigh muscles are such that, when the ape attempts to stand upright, the hip-joint is subjected to stress and the trunk must be bent upon the thigh at the hip. Compared with fully upright man, there is a forward displacement of the centre of gravity. This position is so unstable that, if there were no compensatory mechanism, the animal would be able to maintain the upright position only momentarily and with great muscular effort, before toppling forwards into the quadrupedal position. The compensation is afforded by the bending of the knees. Thus, when an ape stands or walks bipedally, it assumes a bent-hip, bent-knee stance or gait.

There are several varieties of primate bipedalism. First, bipedal *running* occurs sporadically among most non-human Primates: it is marked by a bent-knee gait. Secondly, bipedal walking and standing may occur among apes and again it is in a bent-knee, bent-hip position, almost a crouch. The position approximates to what Dart (1947) has called the *humanoid orthograde posture* and Howorth

	Forelimb		Hindlimb	
	Suspension	Support	Suspension	Support
Brachiators	+++	+(+)	++	+++
Semi-Brachiators	+ (+)	+++	+	+–+
Quadrupeds	(+)	+++	–	+++

The number of plus signs indicates the relative degree to which each limb is used for suspension and for support.

Table l. Limb Function in Monkeys and Apes (after Napier, 1964)

(1946) the *basic dynamic posture.* Thirdly, man is the only primate able to stand erect for long periods of time, with full extension at the knee joint and minimal expenditure of muscular energy. This kind of uprightness is singular to the human species, among all living primates. As Poirier (1977) has put it, 'The distinguishing characteristic of the human locomotor pattern is not erectness, as such, but *prolonged* erectness maintained by straight knees and hind limbs' (*op. cit.,* p. 112). Napier would add to this the special character of human *walking with a striding gait,* which he considers to be 'probably the most significant of the many evolved capacities that separate men from more primitive hominids' (Napier, 1967, p. 56).

To Napier we owe the aphorism, 'Human walking is a unique activity during which the body, step by step, teeters on edge citge of catastrophe.' Here is

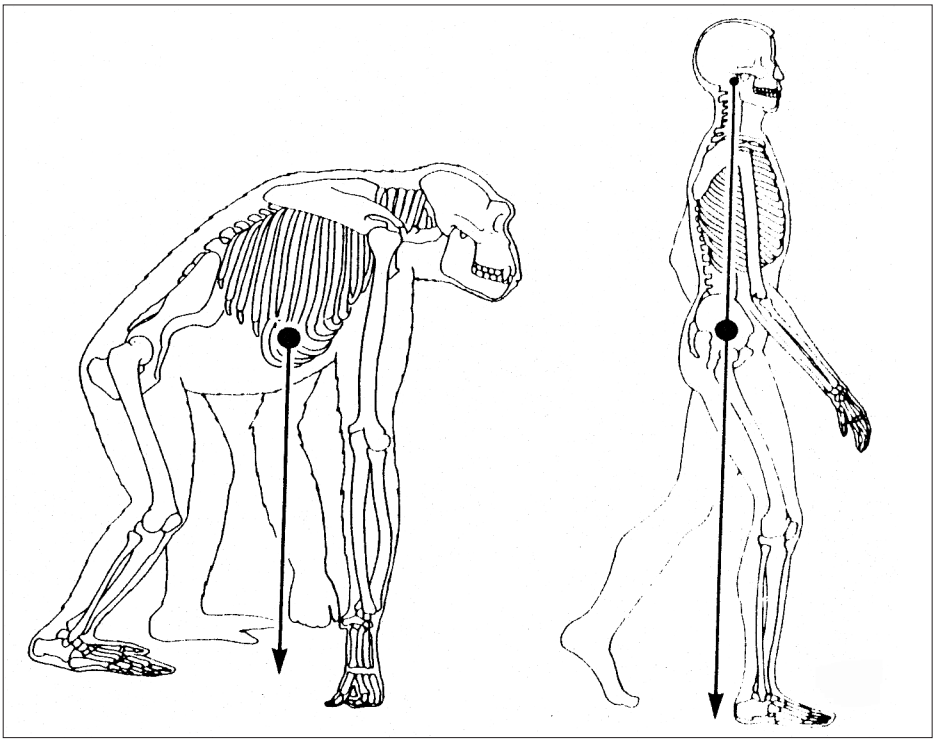

Fig. 1. The weight-line of an anthropoid ape in the oblique quadrupedal position (left) talls between the forelimbs and hind-limbs. In upright standing man, the axis of the body mass (or the centre of gravity) passes from the occipital condyles, close to the vertebral column, through the hip-joints on either side and so to the tripodal feet. During walking the weight-line is constantly moving forwards: in the swing phase of the right lower limb, the line of the body's mass lies between the 2 feet. As the right foot changes from the swing phase to the stance phase – as in the man shown above – the line of weight moves from the space between the feet to the now weight-bearing right foot.

Napier's description of the sequence of events involved in human walking:

'Man's bipedal mode of walking seems potentially catastrophic because only the rhythmic forward movement of first one leg and then the other keeps him from falling flat on his face. Consider the sequence of events whenever a man sets out in pursuit of his centre of gravity. A stride begins when the muscles of the calf relax and the walker's body sways forward… The sway places the centre of the body weight in front of the supporting pedestal normally formed by the two feet. As a result one or the other of the walker's legs must swing forward so that when his foot makes contact with the ground, the area of the supporting pedestal has been widened and the centre of the body weight once again rests safely within it. The pelvis plays an important role in this action: its degree of rotation determines the distance the swinging leg can move forward, and its muscles help to keep the body balanced while the leg is swinging.

'At this point the "stance" leg – the leg still to the rear of the body's centre of gravity – provides the propulsive force that drives the body forward. The walker applies this force by using muscular energy, pushing against the ground first with the ball of his foot and then with his big toe. The action constitutes the "push-off" which terminates the stance phase of the walking cycle. Once the stance foot leaves the ground, the walker's leg enters the "swing" phase of the cycle. As the leg swings forward it is able to clear the ground because it is bent at the hip, knee and ankle. Before making contact with the ground and ending the swing phase the leg straightens at the knee but remains bent at the ankle. As a result it is the heel that strikes the ground first. The "heel strike" concludes the swing phase; as the body continues to move forward the leg once again enters the stance phase, during which the point of contact between foot and ground moves progressively nearer the toes. At the extreme end of the stance phase, as before, all the walker's propulsive thrust is delivered by the robust terminal bone of his big toe.' (Napier, 1967, p. 56).

To appreciate the problem of the evolution of the upright posture of Man we need answers to this question: what changes are necessary to convert a flexed upright position and gait (as in apes) into Man's fully extended stance and his erect, striding gait?

THE ANATOMY OF POSTURE AND LOCOMOTION IN LIVING HIGHER PRIMATES

Habitual uprightness is accompanied by special anatomical adjustments in two main parts of the body: first, the axial skeleton (spinal column surmounted by skull) and, secondly, pelvis and lower limbs.

Spine and Skull

The spinal column of man, looked at teleologically, seems as if designed to project the axis of the body mass, or centre of gravity, in a straight line from the occipital condyles, through or close to the vertebral bodies, to the pelvis **(Fig. 1)**.

Spinal Curvatures

The cervical and lumbar parts of the human spinal column project forward strongly convexly, much more so than in the great apes **(Fig. 2)**. These 2 secondary

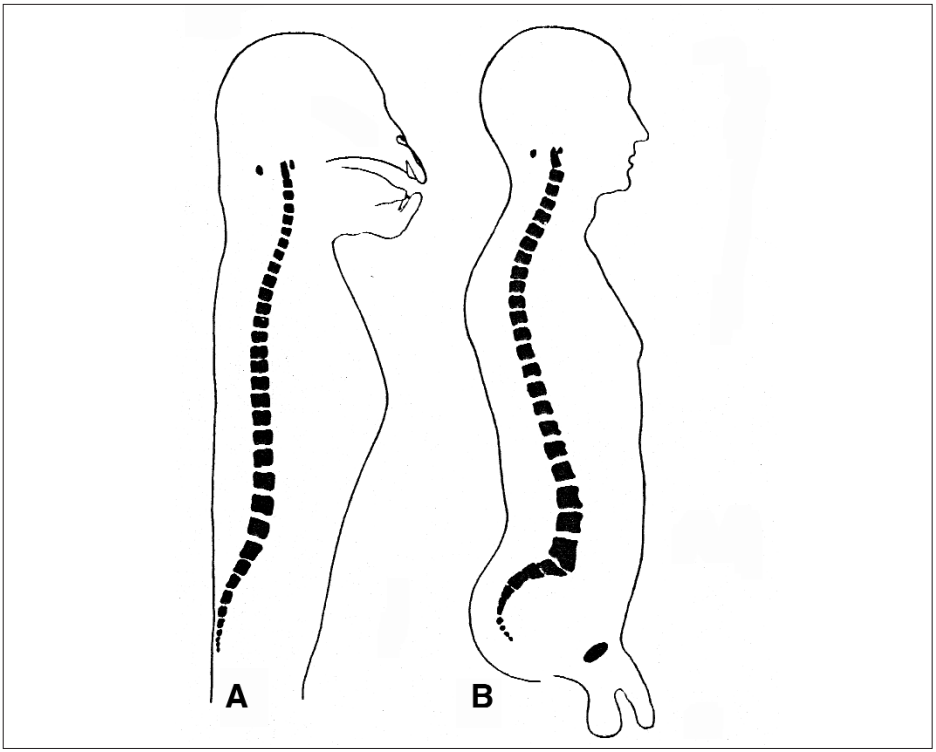

Fig. 2. Median sections through the head and trunk of a chimpanzee (after Cunningham, 1886) and of a man (after Stratz, 1905), to show the differences in the curvatures and in the sequence of vertebral body sizes of ape and man (from Weidenreich, 1941).

curves of the spine are maintained partly by ligaments and muscles and at least in the lumbar region partly by bony adaptation of the vertebrae themselves, so that they are individually wedge-shaped with the thicker diameter to the front. Thus if one articulates 5 human lumbar vertebrae with one another, even without intervertebral discs, they generally form a curve which is convex anteriorly. Two other human features are the marked, anteriorly concave curvature of the sacrum and the powerful promontory at the lumbosacral border, which develops in man even before birth (Schultz, 1936, 1950).

Size Sequence of Vertebrae

In upright man there is a progressive increase in the size of vertebral bodies from the cervical to the pelvic end. This follows because in an upright creature the force of compression increases from head to pelvis. Thus the cross-sectional areas of the vertebral bodies increase from above downwards (Schultz, 1950). Such a size seriation is much less evident in the obliquely walking apes **(Fig. 2)**.

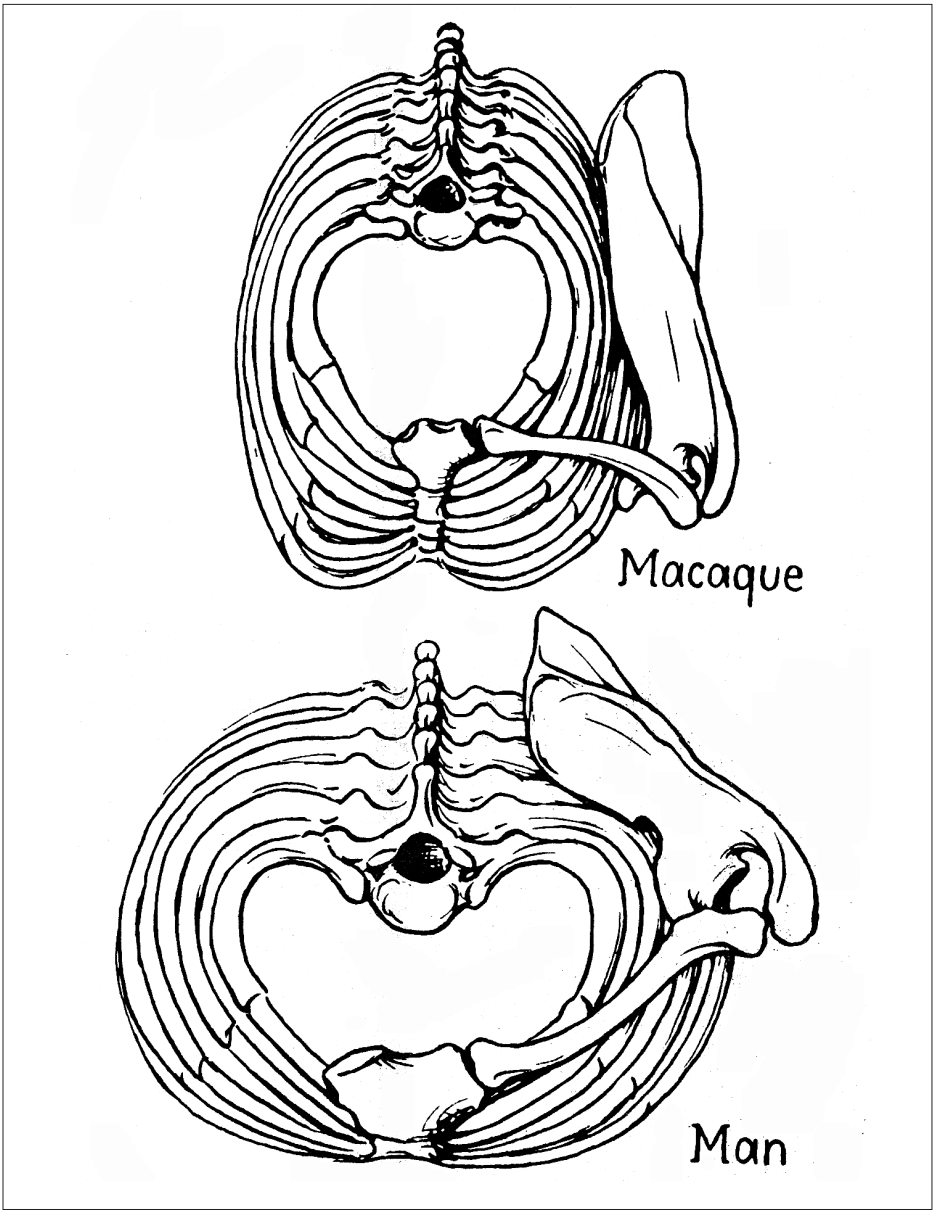

Fig. 3. Thoracic shape, seen from above, in a monkey (macaque) and a man. In the human subject the spinal column projects into the thoracic cavity and the sternum is relatively much nearer the spine than in the barrel-shaped chest of monkeys and apes (after Schultz, 1950).

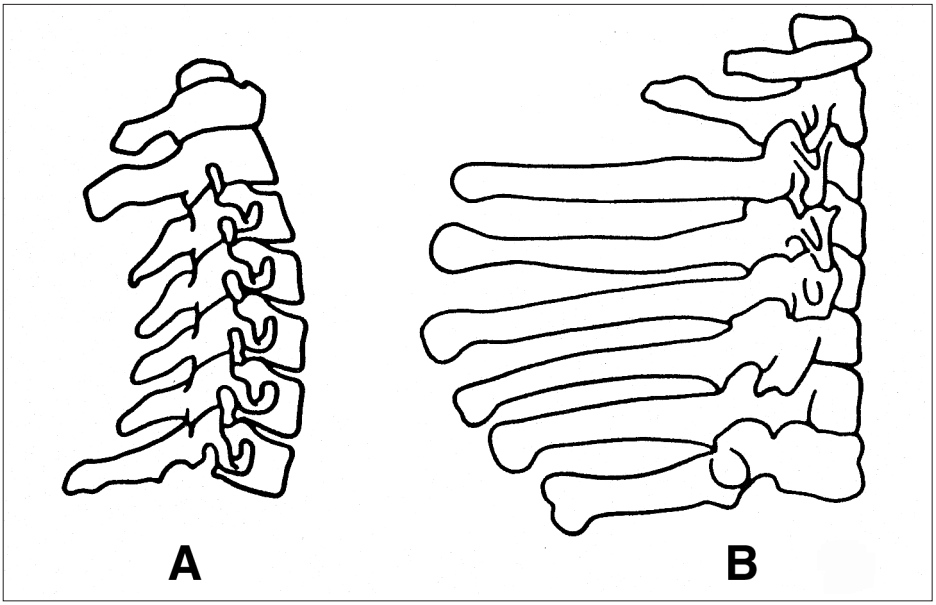

Fig. 4. The spinous processes of the cervical vertebrae in man **(A)** and gorilla **(B)**. Note the large size of the processes in the ape, in keeping with the marked development of the nuchal muscles. In man, the well-balanced head is supported by small nuchal muscles attached to the reduced spinous processes shown above (re-drawn after Campbell, 1974).

Spinal Encroachment with Flattening of the Thorax

The thorax in monkeys and apes is barrel-shaped, the sternum lying well forward of the spinal column and the vertebrae encroaching but little into the chest cavity. Thus, the centre of gravity is carried appreciably in front of the vertebral column. In man, the thoracic cage is flatter, the sternum lying nearer the spine and the vertebrae projecting well forward into the thoracic cavity **(Fig. 3)**. These differences place the centre of gravity in upright man much further back and nearer the vertebral column (Schultz, 1950; Campbell, 1974).

Size of Spinous Processes

The spinous processes of the neck vertebrae give attachment to nuchal muscles and ligaments that help support the head. In apes the spinous processes are large; in man they are reduced, since the human head is more nearly balanced on top of the atlas vertebra than it is in, say, the gorilla **(Fig. 4)**.

The Balance of the Head

The head articulates with the atlas vertebra and so compresses the cervical vertebrae, while it is held up by the nuchal muscles and ligaments under tension. In 4-footed animals the head hangs forward

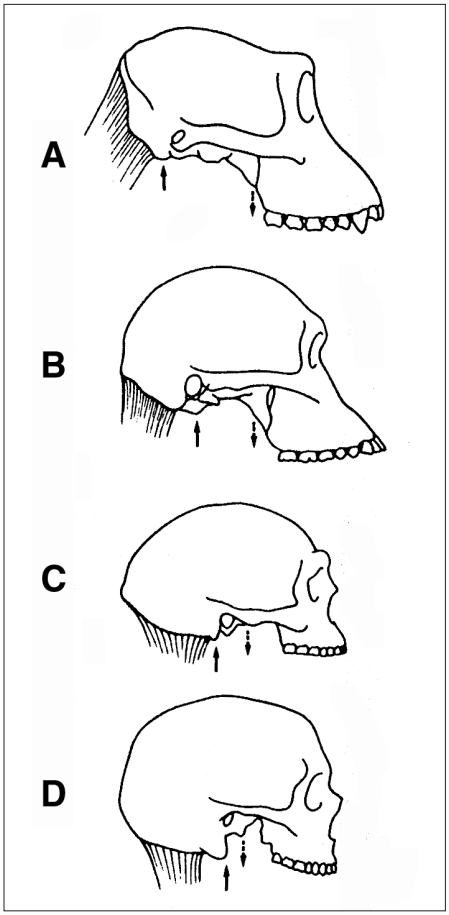

Fig. 5. The crania of 4 higher primates, brought to the same neurocranial length, to show important features of the poise and support of the cranium. The diagrams illustrate the varying degrees of development of the nuchal muscles and direction of the pull of their fibres. The downward-directed, interrupted arrow indicates the approximate position of the centre of gravity of each skull: the upward-directed, continuous arrow represents the position of the supporting occipital condyles. Note that, while the condyles change to a progressively more anterior position, from ape **(A)** through *Australopithecus africanus* **(B)** and *Homo erectus* **(C)** to modern man **(D)**, the position of the cranial centre of gravity moves posteriorly, as the braincase enlarges and the teeth and jaws diminish – until in modern man, the weight-line and the condyles are almost coincident. These adjustments are accompanied by progressive diminution of the nuchal muscles and their purchase on the cranium and cervical spinous processes, and by changes in the relative direction of the pull of their fibres.

from a posteriorly-situated atlas: considerable tension is exerted on the nuchal muscles and ligaments. Six times as much power is needed in the nuchal muscles of monkeys and apes as in those of man. With an erect posture more of the weight of the head is carried directly by the neck vertebrae, whilst the role of the nuchal muscles is correspondingly far less. This diminished role of the nuchal muscles is reflected in man by the smaller cervical spinous processes. There is also in man a dramatically reduced area on the base of the cranium for the attachment of nuchal muscles.

Two other features affect the changed positioning of the head: in apes the jaws and teeth are large and the brain small; in man the jaws and teeth are reduced and the brain and its enveloping brain-case are much enlarged. Hence, the centre of gravity of the human head is much further back than is that of the ape head **(Fig. 5)**. Indeed, in man, the centre of head mass lies only a short distance anterior to the pivot of the head on the spinal column, i.e. the weight of the head falls just in front of the occipital condyles. The small nuchal muscles attaching behind the point of pivot are sufficient to maintain the poise of the human head in the upright posture **(Fig. 5)**.

Dart (1925), in his paper on *Australopithecus africanus,* proposed that the rela-

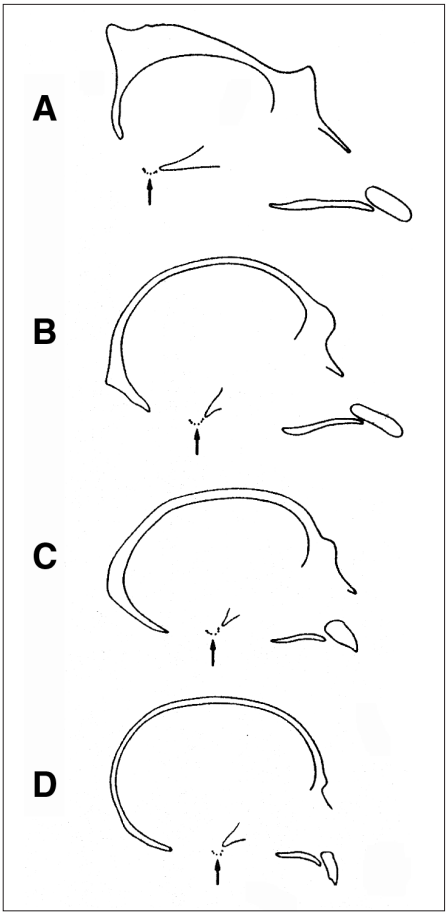

Fig. 6. Median sagittal sections through the crania of gorilla **(A)**, *Australopithecus africanus* **(B)**, *Homo erectus* **(C)** and *Homo sapiens* **(D)**: the position of the foramen magnum is clearly seen. On to this view the position of the occipital condyles has been projected (dotted line indicated by arrow). Note the changes in the position and orientation of the foramen magnum and the forward 'migration' of the condyles, from ape to man.

tive position of the centre of weight of the head, in relation to the occipital condyles, be expressed by a *head-balancing index.* This index in the Taung fossil child was different from that in young apes and had moved in a human direction. Twenty-five years later Le Gros Clark (1950) developed Dart's idea, and proposed a *condylar position index.* This enabled him to confirm that in *Australopithecus* the centre of gravity had moved posteriorly (while the occipital condyle had 'moved' relatively forward) in comparison with apes. Later, I found an even more humanoid value of this index in the very robust East African ape-man. *Australopithecus boisei* (Tobias, 1967), and a still more human pattern in the 2-million-year-old *Homo habilis* (Tobias, 1981a) **(Fig. 6)**.

Pelvis and Lower Limb

The fore-limbs of man, unlike those of quadrupedal creatures, play no part in weight-bearing. All of the body's weight is transmitted downwards through the pelvis to the lower limbs. It is not surprising to find remarkable differences in these parts between man and apes. What is astonishing is how the pelvis adjusted itself to its new role in the hominids without sacrificing its other critical function, to serve as 'the triumphal arch through which every youthful candidate for immortality must pass' (Sir William Osler), viz. the birth canal.

The Pelvis

Compared with that of apes, the human pelvis has become tilted; the ilium is shortened, broadened and bent backwards (Washburn, 1968). The posterior bending of the ilium, the backward displacement of the sacrum and the appearance of the

sacral promontory and lumbar curve, permit the trunk to be held vertically – whilst an adequate birth canal is maintained. The ilia face medially rather than ventrally, while the sacral part of the ilium is greatly enlarged **(Fig. 7)**. The significance of these changes is as follows:

The shortening of the ilium brought the auricular surface vertically nearer to the acetabulum. This enabled the mass of the trunk to be transmitted more directly to the lower limb, thus enhancing stability (Campbell, 1974). But a direct vertical approximation of the 2 joint areas would have greatly attenuated the birth canal. As though to maintain adequate pelvic diameters, the auricular surface not only moved downwards, i.e. nearer the ground, but was displaced posteriorly. This was effected by both the backward bending of the ilium and the expansion of the sacral part of the iliac blade. In this remarkable fashion Nature equipped the human os coxae (innominate bone) the better to transmit

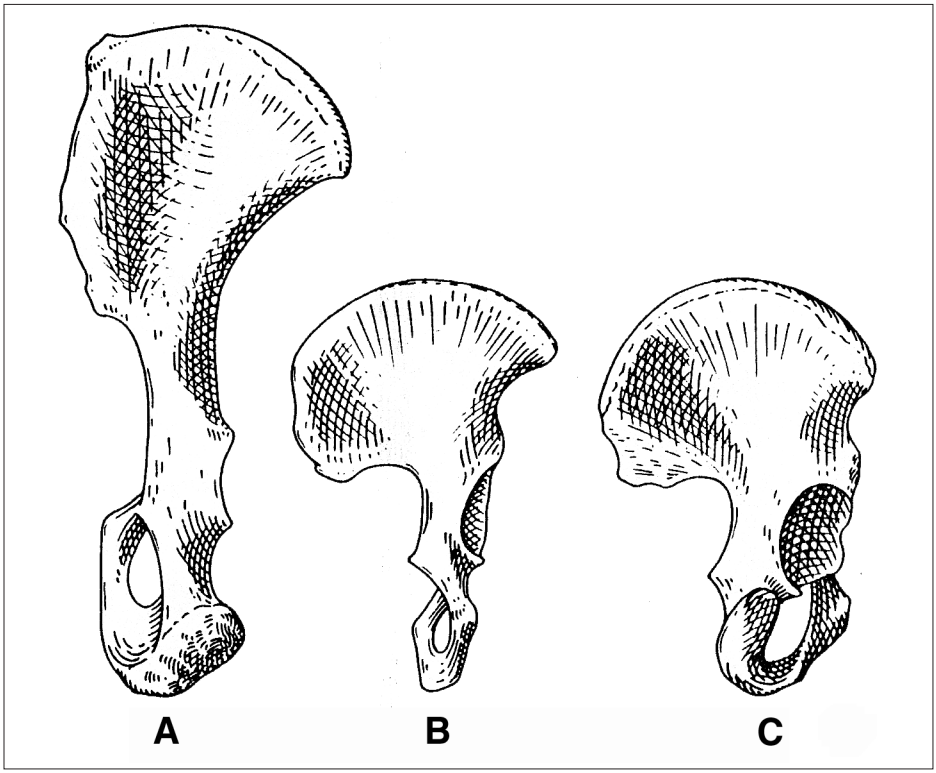

Fig. 7. Ossa coxace (innominate bones) of chimpanzee **(A)**, *Australopithecus africanus* **(B)** and modern man **(C)**. In each instance, the bone is orientated with the plane of the ilium at right angles to the line of sight, and with the anterior superior iliac spine pointing to the right (these are right innominates). The transversely expanded ilia of man and *Australopithecus* contrast with the narrow, vertically expanded ilium of the ape.

the mass of the trunk more directly to the lower limb, while the pelvis continued to provide an adequate birth canal.

Recent studies by one of my doctoral students, I. M. Suzman (1976, 1981), have shown that not only is the external morphology of the ilium adapted to weight-bearing, but the internal architecture, too, presents bony responses to bipedalism. These take the form of specialised trajectories of cancellous bone, especially in regions subject to much compression, like the supra-acetabular region in man (but not in apes). Similarly the head and neck of the femur, to which the body mass is transmitted through the hip-joint, are marked by specialised internal structure.

Another bony adjustment to bipedalism is the development in man of a prominent iliac pillar, which has no formal designation in the Nomina Anatomica and which extends from the tubercle of the iliac crest to the posterior part of the acetabulum. Among living primates, this thickening of the ilium and the tubercle is unique to man (Mednick, 1955) and it occurs, too, in early hominids like *Australopithecus.* The iliac pillar helps to bear the compression exerted by the gluteus medius muscle when it tilts the pelvis during walking. This feature of the human pelvis is thus related more to bipedal locomotion than to static erect posture.

We may thus recognise 2 kinds of bony adaptations of the pelvis and lower limb: one kind related to weight-bearing in the stationary erect posture and one involved in 2-legged locomotion.

Gluteal Muscles

With the adjustment to erect posture and bipedal locomotion, the muscles playing across the hip-joint have become reorganised. Gluteus maximus (as its name indicates) has become in man the largest of the gluteal muscles, whereas in apes, it is smaller than gluteus medius. Gluteus maximus has expanded in man as its attachment area, the posterior part of the iliac blade, has broadened and moved backwards. Man's gluteus maximus has become a most powerful extensor at the hip-joint, of the greatest importance in the maintenance of the upright posture. It was formerly taught that in the great apes gluteus maximus was not an extensor but an abductor. However, detailed analyses have shown that in the apes, too, gluteus maximus is an extensor (Robinson, 1972), though its extensor function is relatively less developed, while the abductors, especially gluteus medius, are more important.

Hamstring Muscles

The hamstring muscles operate across the hip-joint. They are attached above to the ischial tuberosity and inferiorly to the leg-bones below the knee. The leverage of these muscles differs between apes and man. In apes, with a long ischium and relatively short femur, the moment arm (the stable element) is long and the lever arm (the mobile element) is short: hence the muscles act with power; in man the proportions of the moment and lever arms are reversed and the same muscles react with speed rather than power. Powerful contraction, it seems, is of greater value to a clambering pongid, and speedy action to a walking, striding and running biped (Poirier, 1977).

The Femur

As the distribution of weight-bearing has shifted from 4 limbs to 2 hindlimbs, the

head of femur and the acetabulum which lodges it have become larger in man. The femur of man has a relatively long neck, a long, fairly straight and slender shaft and, distally, a narrower condylar surface with the line of weight passing through the lateral condyle. These features seem to provide for more efficient weight transmission than in the apes. In addition, man alone among the higher primates is capable of habitually fully extending his limb at the knee and of locking his knee-joint in the fully extended position.

The Foot

In the foot the most overt difference is the divergence of the great toe in apes and the lack of divergence in man. The foot is a grasping organ in apes and a weight-bearing and locomotor prop in man. Lewis (1980) has proposed that, instead of the hallux of man having been adducted towards the other toes, it appears that during hominid evolution the lateral 4 digital rays have been realigned towards the great toe. The bones of the human first (hallucial) digital ray have become relatively much larger as compared with those of the other 4 rays. One load line runs through the human hallux. Although the foot of man is narrowed, a new secondary load line has evolved along the fifth digital ray. In this manner the weight of upright man, when standing, is distributed through a tripod in each foot, comprising 3 weight-bearing centres, the heel, hallux and small toe.

Summation on Comparative Anatomy

It is clear that the acquisition of the upright posture and bipedal gait of man has been accompanied by a suite of anatomical adjustments that sharply distinguish living man from the extant apes. These extend from the base of the cranium to the distal phalanx of the great toe. Functional and morphometric analyses have furnished evidence that those anatomical adjustments effectively produce a more effective weight-bearing system, in which the centre of gravity passes from the occipital condyles, in a straight line just in front of the spinal column, to the upper sacral vertebrae, and thence, across the ilia and the upper margin of the acetabula, through the femoral heads, down the hind-limbs, to the left and right pedal tripods **(Fig. 1)**. The effect of this concentration of the weight-line is threefold: it enhances stability; it makes for better balance, both in the upright standing position and in man's striding gait; and it enables man to stand upright with a minimal expenditure of muscular energy (and therefore less fatigue).

Hence, almost every bony element from basicranium to foot bears in modern man the clearly recognisable hallmarks of his uprightness and his bipedalism. A bone from almost any part of this axis could, in the hands of a specialist, yield the message; 'I belong to a quadruped' or 'I am part of an upright animal'.

It may safely be assumed that the bones of yesterday's primates also reflected the posture and function of their possessors. Thus, an isolated bone, 2 million years old, might yield sufficient structural details to enable us to make statements about the posture and locomotion of the long-dead primate in question.

Nature of the Fossil Evidence

The raw materials available to students of the fossil record are in the main fossilized

bones of earlier creatures. Bones become fossilized by the gradual replacement of their organic component by inorganic materials. The rate and nature of fossilization, and the faithfulness of preservation, depend on the environmental conditions to which the bones are subjected before they become fossilized. Despite an enormous variety of taphonomic agents, within broad limits bones retain their erstwhile living morphology. This is true whether one examines their external structure, internal form, or even microscopic pattern. A 150-million-year-old dinosaur bone, on histological examination, shows Haversian systems, concentric lamellae, canaliculi and lacunae: only the lacunae and canals, once busy with bone-cell activity, are now quiet and deserted. Yet the structure remains because fossilization is a process of molecular replacement. While the minute building-stones are being superseded, brick by brick, the edifice retains its overall form and even its size. It is thus valid to study the structure of the bones entombed in the earth and (with due allowance for obvious deformation, cracking, fractures, warping, chew-marks, etc.) to interpret the fossilized structure as that which characterised the organism at the time it died.

Isolated fossil bones provide us with information on the form, shape, size and proportions of the bone.

We may learn much about the soft tissues that clothed and were attached to the bones. Muscles, tendons, ligaments, leave their mark. Like Ezekiel in the valley of bones, we may learn, from the position and degree of development of the attachment areas, to clothe the bones in the mind's eye with muscles, sinews and ligaments that were formerly attached to the bones. In this way the fossil record may give one information about musculo-skeletal units that operated in the long-dead body.

We may find evidence on the form, position and extent of articular surfaces of bones, from which we may learn about the range of movement of which a joint was capable. Sometimes we find articulated bones and even partial or, more rarely, complete skeletons. Such discoveries permit inferences to be drawn about leverage, balance, weight transmission and other biomechanical properties, as they might have functioned in life.

Happily, among the hundreds of bones of the extinct *Australopithecus* and other early hominids, there are represented all skeletal regions most closely related to posture. Thus we have cranial bases, a good selection of vertebrae, some still articulated; specimens of sacrum, ilium, whole innominate bones, femoral heads, necks and shafts, femoral and tibial components of the knee-joint, and some ankle-, foot-and toe-bones. What light do they throw on the posture and locomotion of the early hominids?

Inferences from the Fossil Record on Posture and Gait

The fossils to which we may apply these functional principles stem in the main from South and East Africa. Hundreds of specimens from these regions fall into 2 genera, *Australopithecus* and *Homo. Australopithecus* extended in time from 4 or 5 million years B.P.* to about 1 million years B.P. and *Homo* seems to have appeared about $2^1/_4$ million years ago and persists to the present. Within each genus several species are recognised.

* B.P. = before present.

Although there are disagreements on points of detail, there is a consensus today that *Australopithecus* as a genus was ancestral to the genus *Homo,* i.e. one or other species of *Australopithecus* was ancestral to the earliest species of *Homo.*

A major inference from these fossils is that the australopithecines were of upright posture. After the first find of *A. africanus* was made at Taung in 1924, Dart was able to infer that this creature had assumed an attitude appreciably more erect than that of modern apes. With remarkable insight and prescience, at a time when only this single child skull from Taung was known, Dart went on to declare:

'The improved poise of the head, and the better posture of the whole body framework which accompanied this alteration in the angle at which its dominant member was supported, is of great significance. It means that a greater reliance was being placed by this group upon the feet as organs of progression, and the hands were being freed from their more primitive function of accessory organs of locomotion. Bipedal animals, their hands were assuming a higher evolutionary role…(Dart, 1925).

Since then, scholars have confirmed on numbers of crania that the occipital condyles were further forward and the centre of mass further back in, *Australopithecus* than in apes, so that the centre of gravity of the cranium fell nearer to the front of the vertebral column **(Figs. 5 and 6)**. The diminished nuchal area confirmed that the head was supported by smaller nuchal muscles. In other words, both the position of the condyes and the size and form of the nuchal area supported the inference that in *Australopithecus* the head was better poised on a more nearly erect spinal column than in apes.

From available lumbar and sacral vertebrae **(Fig. 8)** and innominate bones, Robinson (1972) concluded that a well-developed lumbar curvature had been present and that the pelvis had been orientated as in modern man.

The fossil pelvic bones from 4 Transvaal sites showed the kind of pelvis associated with habitual bipedalism, although not to the degree exhibited by modern man (Clark, 1950, 1955; Napier, 1964, 1967; Robinson, 1972; Campbell, 1974). The pelvic bones contrasted strongly with those of modern apes. Taken in combination, they showed a total morphological pattern that included those very bony adaptations that point to *Australopithecus* as having been habitually bipedal **(Fig. 7)**. A thorough functional analysis of the australopithecine pelvis confirmed that it was fully adapted to efficient bipedalism (Lovejoy, 1973).

In the australopithecine lower limb there are essentially man-like adaptations to upright posture and bipedal gait. There are indications of incomplete adaptation and of some ape-like features, especially in the foot (Lewis, 1980). Thus the evidence of the limb-bones confirms that of the pelvis and of the lumbosacral region, and all of these are independently corroborated by the structure of the cranial base. We may conclude that already, by the emergence of *Australopithecus,* at least about 4 million years ago, the complex series of skeletal and functional adjustments had occurred which gave the earth a primate that was uniquely, habitually and efficiently bipedal. The customary upright posture was clearly an early arrival on the scene of hominization and long preceded the marked and allometric enlargement of the brain and the emergence of tool-making.

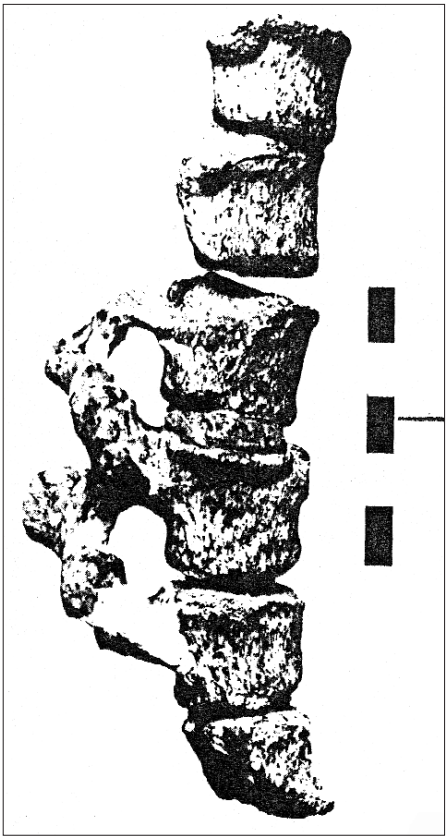

Fig. 8. Six thoracic and lumbar vertebrae of the same spinal column of a skeleton of *Australopithecus africanus* from Sterkfontein, Transvaal. The varying thicknesses of consolidated cave earth (matrix or breccia) fortuitously retained between successive vertebrae give a spurious impression of the spinal curvature.

This early uprightness was anatomically incomplete and imperfect; further structural adjustments were to occur over the next million or two years, until a pattern would emerge in *Homo,* probably in *H. erectus,* which was virtually indistinguishable from that of modern man.

The establishment of bipedalism at an early stage in hominization, and of other features at a later stage, illustrate the principle of mosaic evolution, i.e. a tendency for some features to have been hominising more rapidly, or earlier, or both; and others more slowly, or later, or both. Mosaicism is evident, too, even within various parts of the locomotor complex (cf. the work of Lewis, 1980). Notwithstanding, the occurrence of mosaicism does not detract from the general inference, namely that the earliest species of *Australopithecus* were already bipedal. The 'fossil footprints' discovered by Mary D. Leakey and her team at Laetoli in northern Tanzania provide circumstantial but tell-tale confirmatory evidence (M. D. Leakey and Hay, 1979; Day and Wickens, 1980).

Some Problems and Some Speculations

Uprightness, Gravity and Balance

The musculo-skeletal adjustments to erectness had the effect of bringing the upright body's gravitational axis into a plane more or less equidistant from the 2 primordial surfaces of the body, the dorsal and the ventral. To accomplish this, our ancestors had to achieve a new orientation of the body towards gravity. At the 4-footed stage, the ventral concavity of the body, sheltering the sensitive, relatively hairless, ventral skin, the breasts and the external genitalia, was closest to the ground. In coming upright the early hominids displayed a new frontal exposure to the world. Not only the face looked forward now: so too did glabrous ventral skin, breasts and genitals.

In dragging his ventral aspect away from the earth's gravitational field and

swinging it upwards through 90˚, man attained a stance where his weight line passed in a coronal plane through his 2 firmly planted tripodal feet. His back and front were nearly evenly balanced on either aspect of this plane – unless the subject took to high-heeled shoes, thereby throwing the weight forward, in an apparent endeavour to undo 4 million years of evolution! (No wonder Francois Fouché was moved to wrath!)

Seen in this light, the upright posture is a precariously balanced state. If the body were merely a nerveless framework, with relatively atonic muscles, it could easily be thrown off balance by a push or a gust of wind. Long ago, Schopenhauer said that our walking is admittedly nothing but a constantly prevented falling.

Yet somehow, we do not fall over at a touch. We remain upright under an extraordinary array of conditions – in a high wind; on a moving walkway or an escalator which tries as hard as it can to remove our delicately balancing tripod-feet from under us; on a tightrope, ice-skates or skis; or as a ballet dancer on points. Our success as bipedalists is consummate.

Part of the credit for this success must lie with our sense of balance. It would be reasonable to suppose that a well-developed faculty of balance must have been a prerequisite, or at least an accompaniment, of the development of uprightness. The flocculo-nodular lobe of the phylogenetically most ancient part of the cerebellum, the *archicerebellum,* seems to be essential for the maintenance of uprightness and the bipedal gait. Lesions of the flocculo-nodular lobe produce an *archicerebellar syndrome* in which upright standing and walking are seriously impeded. The bilateral movements used for locomotion and the maintenance of equilibrium are affected. The patient sways and is generally unsteady when standing. When walking he staggers and tends to fall backwards or to either side. Other parts of the cerebellum, such as the palaeocerebellum and the neocerebellum, play a major role in the control of muscle tone, hence indirectly of uprightness, and so too does the vestibulospinal tract.

To this central brain control there travel 2 great sets of sensory inputs, vestibular and proprioceptive. That there is a major vestibular component is undoubted, though in quiet walking and in standing still, it is not the whole story, for uprightness is not simply an exercise in balance. The poise of the sensori-motor system provides the other vital neurological component of uprightness and bipedalism. The proprioceptive messages determine the precise muscle tone and flickering contractions necessary to maintain erect posture and relaxed bipedal walking.

We know little about possible differences between apes ad man in the proprioceptive system, as to both its peripheral development and its central representation. We cannot yet answer the question, whether and to what degree man's success as a biped is to he laid at the door of a more exquisitely developed proprioceptive system.

Muscle Reserves and Bipedalism

In 1950 Washburn assigned to the gluteus maximus muscle, a critical rôle in the development of habitual erect bipedalism. He claimed that its large size in man and its line of function behind the hip-joint as a powerful extensor constituted important differences from the homologous mus-

cle's function in apes, in which it was smaller and said to act mainly as an abductor.

Robinson (1972) assembled much evidence that Washburn's interpretation was not tenable. In chimpanzee and gorilla, for instance, there is a strong posterior part of gluteus maximus which serves as an extensor of the thigh at the hip-joint. Moreover, during relaxed bipedal walking on a level surface, Basmajian (1962) found that gluteus maximus is electrically silent. Other studies have shown that the muscle is used regularly during only a brief part of the walking cycle – as the striding limb reaches its maximal forward position and the foot strikes the ground. Hence during quiet walking, gluteus maximus does not help *propel* the limb; rather it serves to *control* it and halt the forward movement of the swinging limb, and then to stabilise and steady it, as that limb begins to support the weight. However, the great potential of this largest gluteal muscle comes into play when one runs, walks up a steep slope or up stairs, raises the trunk from a bent position, or stands up from a sitting or a squatting position (Wheatley and Jahnke, 1951; Robinson, 1972).

We realise that, in standing still, or in walking in a relaxed manner on the level, man uses only a small part of his available power; he retains enormous muscular reserves. These reserves are ready to be called upon for special actions such as clambering, running and walking up a steep gradient, or for the development of special bodily skills.

It is man's trifling muscular input into the functions of standing erect and of relaxed walking on the level, coupled with the large extent of untapped muscular potential, that have permitted him to develop his bodily techniques in well-postured, poised and skilled movements.

The Skilled Use of the Body

Two examples of the skilled use of the human body are karate and ballet. A study of these 2 sets of skills, so different in effect, yet so similar in the way the body is used, helped me comprehend that to achieve physical skill one must achieve poise. To acquire poise, one must convert malposture to posture. For it is an inescapable fact that despite our 4 million years of standing erect, the human body is still subject to the ills of uprightness (Keith, 1923), including malposture.

Malposture is an insidious and widespread ailment of modern man. Sherrington (1946) drew attention to the fact that in urbanised communities, bad habits in motor acts are especially common. Dart (1947) spoke of the *pandemic condition of malposture* amongst urbanised mankind, and related it to malocclusion and even to aspects of the emotional life.

A difficult balancing feat is the ballet movement of *arabesque:* in its commonest form, the dancer stands on one leg, with the other raised behind her and extended fully. The height of the raised leg is variable and so is the position of the upper limbs. As one watches a dancer performing this feat, one cannot help enquiring: how much of this achievement is peripheral, thanks to bones, ligaments, tendons and muscles, and how much is central, thanks to the control exerted by the brain?

Dancing on the toes or on points is one of the most graceful and poised of all forms of ballet dancing. It was introduced about 1820 and it is awesome to note that Maria Taglioni, who popularised this form of

dancing, wore ballet shoes which were unblocked! Dancing *sur les pointes* is a great challenge to the balancing skill, for the foot is carried up from the tripod stance to a *monopod* stance. Instead of 6 points of contact with the ground, there are only two, while the dancer stands on points, and only *one* when she rises on to the point of one foot at a time. So exacting is this form of dancing that it is dangerous to put a young girl up on points at too young an age. The fascial extensions of the gluteus maximus, quadriceps and gastrocnemius must have been fully established, before any child may dance *sur les pointes* with safety.

Karate is another set of skills associated with intense mental discipline. The poised movements of the periphery are there, just as in ballet, while the mental control is master of the situation. As a Black Belt exponent might show us, in a combination or kata, there are finely co-ordinated actions of eyes, head, upper limbs, trunk and lower limbs, as in ballet. Both of our groups of skilled performers – karate-ka and ballet dancers – use the same muscles, even the same combinations of muscles, to achieve some very similar movements and stances: but while the ballerina's movements stress fluidity and grace, those of the karate-ka stress vigour and power. Both emphasise control.

The speed typist and the pianist require fantastic neuromuscular coordination. To adjust their fingers in space, which means also adjusting the wrist, fore-arm, elbow, arm and shoulder, demands control over 30 different joints and more than 50 muscles for each hand! The visual sense comes into the picture: the pianist reads her music, her eyes dart over the music-sheet and the keyboard; while her fingers dart over the black and white notes, and her feet over the pedals. What amazing co-ordination this demands and what remarkable mechanisms inside the brain, to orchestrate the intricate pattern of movements. The essence of such skilled movements is in the co-ordination of nerve and muscle, of sensory catchment, brain centre and motor periphery.

The Rôle of the Nervous System

In all of these skilled movements, the eyes and the head lead the movements of the body. Yet they are controlled by nerves which are only 5 – 10 cm long, while muscles of the foot and leg are controlled by nerves which are up to 1 metre long. The golden rule of the peripheral nervous system is that the thick, fast nerve fibres connect remote regions, while thin, slow fibres connect neighbouring areas. Hence messages arrive at near and far muscles at about the same time. This is part of the secret of the eye-head-neck-hand-foot co-ordination which is basic to the attainment of human skills, whether they be piano playing, singing and speaking, dancing or karate, standing upright or walking on 2 limbs.

The posture of man and his bipedal walking are 2 of a cluster of human functions all of which require poise and skill in the peripheral executive department of the body, and all of which are subservient to the controlling and co-ordinating mechanisms of the brain.

During the formative aeons from *Australopithecus* to modern man, there has been a trebling of average brain-size. There has been much speculation on the evolutionary advantage of the bigger brain. Teleological as it may sound at this stage of our knowledge, I have been compelled to conclude that the advantage of a larger brain during those evolving millennia was

that not only did it make possible more complex cultural achievements; not only did it provide the central control overseeing our upright posture and bipedal gait, and the ever-increasing range of skilled human physical activities made possible by that posture and gait; but above all it also facilitated the transfer of these survival mechanisms and of other learned behaviour to the offspring: in a word by articulate speech (Tobias, 1980; 1981b).

Speech seems to have come to man at least 2 million years after he had become upright. When it came, it made possible the most advanced form of learning transmission from one generation to the next that had yet appeared among the earth's mammals. So, in a flash of evolutionary insight, the power of speech, added to the pre-existing attainments of uprightness and bipedalism, enhanced man's competency for the skilled use of his body. It gave an immeasurable stimulus to man's ongoing exploration of the limits of his body's capacities. There could be no going back: his first great evolutionary transcendence had been his erect bipedalism; the second came with speech.

Armed with these 2 distinctive attributes, humankind has attained humanity. Man has reached thereby a new phylogenetic plateau - from which he discerns faintly, and gropes towards, the next great step forward in evolution.

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KORZET PRO LÉČENÍ SKOLIÓZY* VÝVOJ NAŠEHO KORZETU OD ROKU 1970. HODNOCENÍ NORMALIZACE ROTACE, STATIKY ŽEBER A KLÍNOVITÉHO TVARU OBRATLŮ

SCOLIOSIS TREATING BRACE* EVOLUTION OF OUR BRACE SINCE 1970. AN EVALUATION OF THE NORMALISATION OF ROTATION, OF RIB STATIC, AND OF THE WEDGE SHAPED VERTEBRAE

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SUMMARY

After having shortly presented Abbott, a genious, the first one who could cure scoliosis, and briefly described the main principles of bracing and the way the other schools are bracing now, the authors present the model that one of them has conceived. It is build with polyethylene. Fifty four zones are taken in account on the scoliosis body. Wide and deep spaces must be managed on concave areas. It has to be adjusted to patient, immediately after delivery, regularly during the duration of a brace and occasionally if necessary. The height, width and breadth as well as many details can be adjusted at least as well as in all other methods. Nine kinds of mechanisms, 4 active and 5 passive, contribute altogether to the correction of scoliosis. A series of short term results is presented, where angle according to Cobb, rotation, rib static and wedge shape are evaluated. All those features had got an average bettering of 41 to 60 %.

Key words: scoliosis, brace, adjustments, wedged vertebrae, nine kinds of mechanisms

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INTRODUCTION

From the far off ages and up to the beginning of the 20th century, man had tried to treat scoliosis, but apparently without any success. The first successful treatment has been made by Abbott and published in 1912 (1). He cured even severe scoliosis even by adults. He reduced the curvatures with an asymmetrical hammock combined with tractions and bending. Then he fixed the gained positions in plaster casts. Wide concave sided hollow spaces were managed in order to allow correction, growth, breathing and a certain range of corrective movements.

Fig. 1. 1909, Abbott straightened scoliotic curvatures by means of traction, bending and lateral pressures. Then patients were placed in plaster casts. Huge expansion spaces were managed on concave sides. Here is the expansion area already filled by migrating concave tissues, which have become convex.

Fig. 2. Three main mechanisms of brace action: Traction, bending, side pressures.

Successors of Abbott tried to better the Abbott technique, but all of them neglected more or less the concave sided expansion, a most important factor. Three mechanisms were used and often combined, traction, side bending and lateral hump pressures. In **Fig. 3** are the main models presented at our times. The Milwaukee brace combines traction and hump pressures. The one of Boston puts the lumbar spine into kyphosis and presses on humps. Others as in Lyon (11) use uprights, bars and plastic pieces based upon hump pressures. They give a certain range of adjustability in height and width, but no one in the antero-posterior plane or in details. The Charleston bending night brace uses a side bending in only one plane of space. As far as we know it, no one could get a valuable result by severe scoliosis with soft bands without hard material.

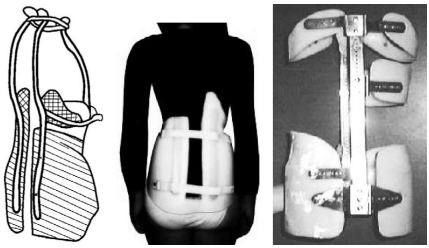


Fig. 3. *Left,* Milwaukee brace, vertical traction + side pressions.
Middle. Boston old style. Strong abdominal pressure causing lumbar Kyphosis; low pressure pads.
R. Brace with uprights and horizontal bars. Side pressures.

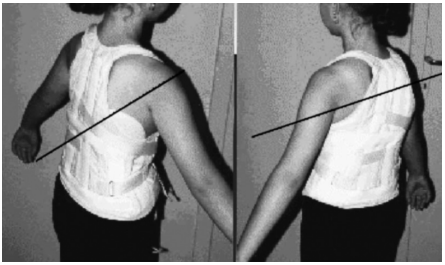


Fig. 4. An attempt to cure scoliosis with only soft bands has never got any success, as long as we are informed. Should a young tree be straightened without a stake? Here an exemple, in Poland. This patient had severely worsened.

Since 1970, one of us (2–8) conceived a brace after Abbott. It is made with plastic material. In the years 1970 to 1992, it consisted in immobilizing shoulders and hip in close relations with one another, then in pressing on humps. Now shoulders and hip are only pressed where they are salient. The correction is mostly active. It combines side bending, lateral hump pressures and very large and deep concave sided expansion rooms. The team must have a complete knowledge of the numerous deformations (We describe 54 of them) caused by scoliosis on the body. The brace has no human shape. It does not envelope the body like a coat does it. It forces it into a corrected position which the treating team aims at giving to the patient after one year. Adjustments must often be made, consisting in widening the expansion rooms and in lengthening the brace upwards and downwards **(Fig. 5).**

Nine kinds of mechanisms interfere and contribute to straighten the curvatures.

1. The cherry stone effect: When laterally pressed, the tissues tend to escape into all possible directions. The team has to let the concave parts of body free and only them.

2. Transfer of tissues along a slide convex-concave. This action is also a cherry stone effect, but is elective.

3. Growth is a severe factor of worsening of scoliosis without brace. It becomes a factor of correction thanks to a marvellous growth-helping action of brace, wished and organized by the team.

A **B** **C** **D**

Fig. 5. A. Adjustments. In **A.** is the front part too low. Breasts have grown and there is not enough room for the right breast. **B.** The front part of brace has been warmed up, then stretched upwards and given a bulged shape. There remain two irregular zones with brownish colour. **C.** After having warmed up again and smoothed the irregular parts between two layers of plastic foam, then polished, no more irregularity can be seen. **D.** Lengthening a brace is quite easy and must be made every time the child growths.

4. Respiration also is a worsening factor without, and a curing factor with brace.

5. Every movement worsens scoliosis as a result of shorter concave sided muscles and of an unfavourable lever arm. With brace, all allowed movements tend to be corrective.

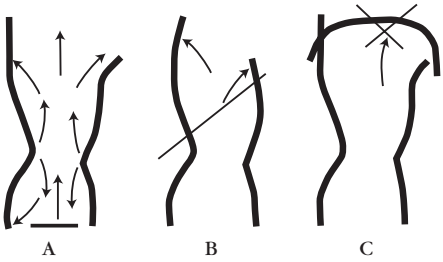


Fig. 6. Cherry stone effect **A,** component forces upwards and downwards. The forces downwards reflect themselves on the ground and reinforce those upwards. **B.** Never make convergent walls. **C.** No shoulder pieces, denial of the most important role of brace, a growth link.

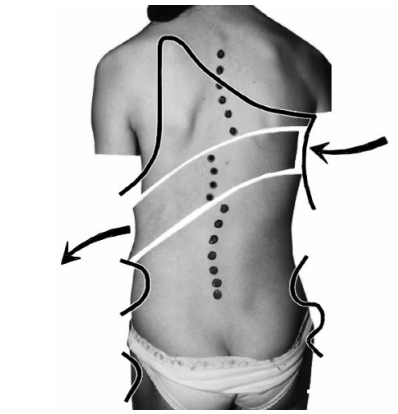


Fig. 7. Elective tissue transfers are an elective case of the cherry stone effect, in which only humps are pressed and a whole slide of deformed tissues migrates towards a concave side. Notice. Thoracic hump is higher than the corresponding concave side.

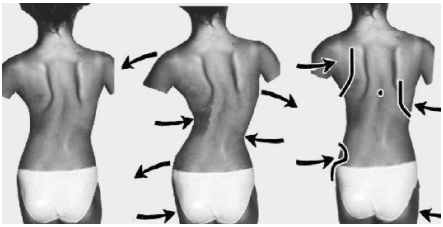


Fig. 8. Scoliosis grows not or only a little in height, but mainly towards humps which are worsened. Brace hinders this bad phenomenon. Then the patient grows towards the concave sides and upward.

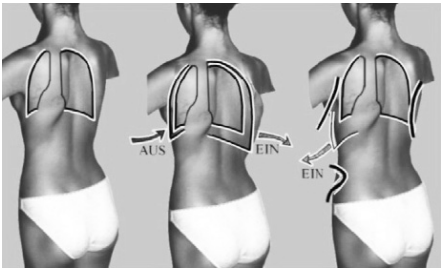


Fig. 9. Breathing without brace towards humps worsens scoliosis. Brace hinders it. The Physiotherapist must teach the patient how to breathe out of his convex, and to breathe in toward his concave sides.

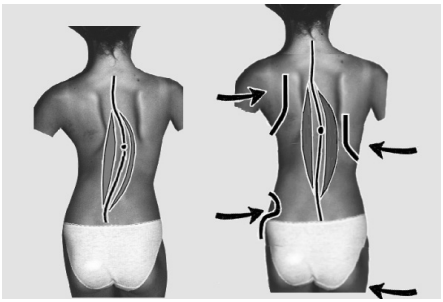


Fig. 10. Every movement has a component in the trunk. Concave sided (worsening) muscles are shorter, then stronger, and the lever arm is worsening. Right. The brace makes the balance of the trunk muscles more symmetrical. So are only the corrective movements allowed.

6. The greater diameter of the **oblique oval thorax** is taken in clamp between the hump pressure parts. Its width decreases. Then the smaller diameter tends to increase. 7. The right anterior brace wall, although concave, serves as a secondary pressure part for the correction of the hollow back.

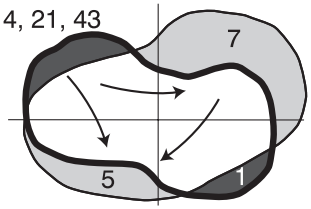


Fig. 11: Section on thoracic apex. Pressed in clamp by back thoracic part 1 and anterior part 4, the greater diameter of thorax diminishes. The trunk derotates and the smaller diameter 5–7 gets larger. Then patient leans on the anterior wall 7, although concave. This „secondary pressure point 7" helps correcting hollow back 5.

8. Bending the upper thorax around the apex helps correcting the thoracic curvature.

9. Anti-gravitational effect corrects the cervico-thoracic and lumbo-sacral curvatures.

Material and methods

We have studied the patients with idiopathic scoliosis in the institute of Amberg, Germany, created in 1995. Patients were provided with brace when Cobb's angle was 21° or more. Then the same measures were maid after one to two years. Non-idiopathic, neurological, as well as small scoliosis combined with Scheuermann's disease and congenital ones have not been taken in account.

We have calculated the **angles according to Cobb,** as all teams use to. We

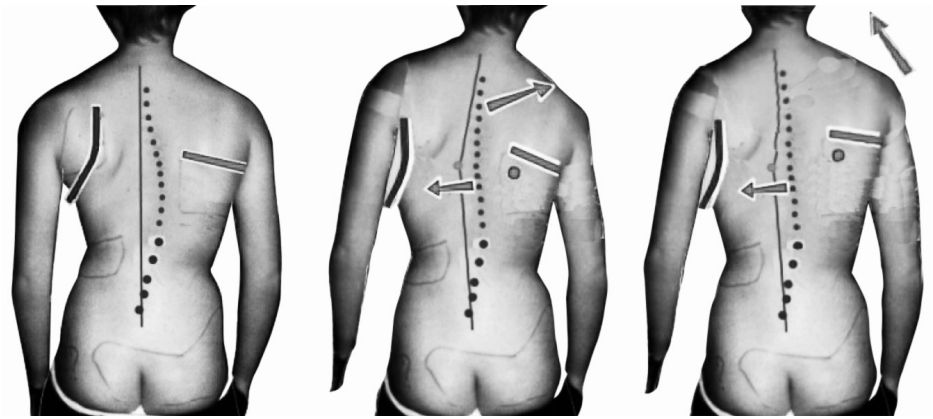


Fig. 12. A Scoliosis **B** – Bending. The cervico-thoracic apex wanders towards right and the thoracic apex migrates towards left. So is the thoracic curve corrected. But the cervico-thoracic curvature still remains, and the right shoulder is lower. **C** – anti-gravitational effect The patient straightens himself, corrects actively the cervico-thoracic curve, rises his right shoulder. Notice the raising of thorax and of the thoracic apex. It is true on this diagram and also true by the braced patient. Pressure part 1 must raise, too.
Anti-gravitational effect is also available in the lower segments of spine. Those lumbar and lumbosacral curvatures are corrected, too, but we have not drawn it there in order not to make this drawing too complex.

wished also to study elements that we never or scarcely had seen published: rotation, rib static and wedging of vertebrae.

Rotation was calculated according to Ponsetti. Based on the position of the more deformed pedicle; we calculed from 0 to 100˚ on the front view of an apex or near-apex vertebral body. The ruler of Perdriolle (12) helps to gain time in this calculation. We never have considered the position of the spinous processes, more deformed than rotated.

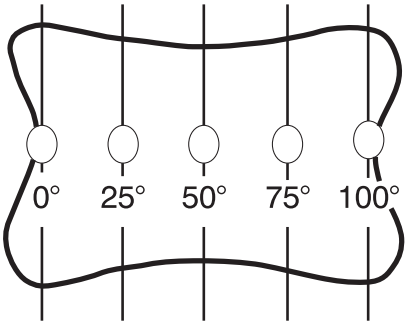


Fig. 13. Rotation, measured by the position of the pedicles, according to Ponsetti. One can help himself with the ruler of Perdriolle. The spinous processes, being more deformed than rotated, have not to be taken in account.

Rib static is measured with the angle according to Mehta **(Fig. 14, 10).** Either the acute angles or the obtuse ones can be measured. What is important is the difference of the angles, and it is the same with the two kinds of measurements.

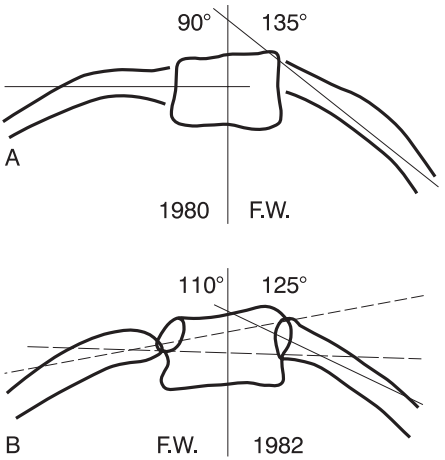


Fig. 14. Rib static. Out of the book of one of us, 1986. A girl of 15 had a Cobb's thoracic angle of 47˚, and a very bad rib static. Brace reduced the angle to 19˚, rotation to 0˚, and the rib static almost completely, although pressure was made on the apex vertebra. Wedge shape remained, as a result of her age and of the technique we would use at this time. Progresses have been made since that time.

Wedged vertebrae. We try to evaluate it that way: Greater height minus smaller height multiplied by 100 and divided by the width. So is the coefficient obtained unaffected by any change of size of the image. **Fig. 15.** (65 – 53) : 98 × 100 = 12,24

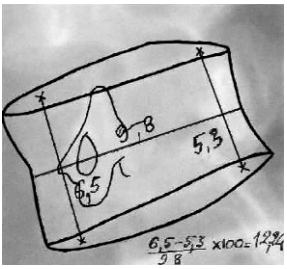


Fig. 15. Measuring the degree of wedging. We divide the difference of heights of the greater and the smaller side by the width. That makes the coefficient indifferent from the size of the image. We multiply by 100 in order to get a whole number. Here the coefficient, 12,24, corresponds to a slight or medium wedging.

Results. A.
Cobb's angle

26 curvatures were analysed. The smallest angle was 21˚, the greatest one 58˚. Average angle before bracing was 38˚, and with brace 15,96˚. The correction reached 58,3 %. No patient worsened during the time of calculation. One of them had been totally corrected from 31˚ to 0˚. This perfect Cobb's correction was accompanied with the normalization of rotation, rib static and wedging. But the patient left brace at 13 years. A setback occurred, which can not be totally mastered.

B. Rotation.

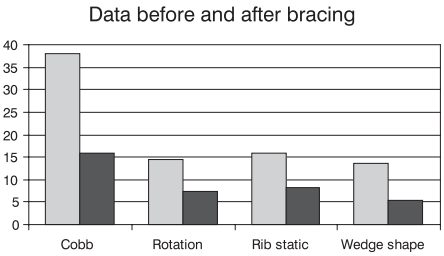
30 vertebrae had their rotation measured. Average rotation angle was 14,5˚. After bracing was it of 7,33˚. Among those patients, 19 had a rotation before bracing., the maximal rotation being of 40˚, the average one 22,9˚, the one after one or two years of brace 11,48˚. Three patients had a rotation unchanged after having been braced. Five were completely corrected. No one worsened. Average correction was 49 %.

C. Rib static.

Twelve patients had their rib static measured. Five of them had symmetric ribs, no one having worsened after bracing. Seven had asymmetric ribs. After one year of bracing; 5 of them were very much bettered, and two of them worsened. Average bettering however was near 47,75 %, from 15,86˚ to 8,29˚.

D. Wedged vertebrae

We have calculated the wedge state of 33 vertebrae. Their average coefficients were 13,51 before and 5,25 one to two years after bracing. Five of them were not wedged before being braced. 28 vertebrae were previously wedged, with coefficients 15,92 before and 6,19 after one or two years of bracing. The common percentage of correction was 61,14 %. No apex or near-apex vertebra has worsened its wedging after one year of brace.



DISCUSSION

A. Cobb's angle. Owing to the fact that our experience is relatively recent, and therefore that only few patient has got a brace compared with older centres, our Cobb's angle result have no great value. We have given them in order to do like all other teams. Results are near the best ones all over the world, although the mean angle of the curvatures, was 38°; far greater than the initial angle of the other schools, 28 to 33°. (11). Another very unfavourable fact is the one, that many of our patients had previously got a brace before, sometimes with success, often without any improvement and without any stop of worsening. When the brace before ours was good, we cannot have any hope to get a much better angle. When it was bad, scoliosis is so much changed that often a good brace can only get poor results.

B. Rotation is often considered as less important in publications. On radiographs presented on papers and on internet, we very scarcely could see radiographs detailed enough to allow a rotation calculation. For some 40 years, rotation was evaluated by deviation of the apex and neighbouring spinous processes. But the spinous processing are mostly much more deformed than rotated. Moreover, spinous processes between Th. 7 and Th. 12 are often very much reduced in size (Karski, 9). Therefore we do no more take this kind of mark in account when evaluating rotation. Now we evaluate the position of pedicles. The pedicles being also distorted in scoliotic patients, the angles calculated are not precise. But that gives an idea of the rotative deformation and a basis for a comparison all along the course of the treatment and after its end.

C. Rib static has scarcely been measured in publications. We have found two pages of internet, unsigned, not precise and without any number. One was http//:www.posna.org and the other http//:sauk.org.uk/scoliosis. They say, in harmony with Mehta (10) that a trouble of rib static can give indications for the evolution potential as well as for the time when brace can be quitted. Measuring rib static is not precise, because ribs are curved, and because the vertebra concerned often is oblique. But it gives an approximate idea of the rib static and an approach for a further comparison. In our series, most patients had got their rib static improved after having been braced. Let us remember. Pressure part 1 pushes horizontally at the level of the apex vertebra. Many specialists allege, that such a pressure should worsen the rib static. The contrary occurs, because a correction leaded that way is far more active than passive thanks to the concave sided expansion rooms. Two patients had their rib static worsened after being braced. They had very severe scoliosis, initially being over the limit of bracing indication. In spite of this bad angle, both have got a very good correction of their Cobb's angle, rotation and wedging. Only the rib static had worsened.

D. Wedged vertebra. We heard some critics concerning the evaluation of wedging. According to those critical persons, wedging should not be a true one, but the consequence of rotation. The concerned vertebra being seen obliquely, its wedging were only apparent. There are our answers.

1. Does a vertebra look like wegded when seen obliquely? No. Please look at oblique radiogaphs **(fig. 16 A).**
2. When a scoliotic vertebra is seen obliquely and seems to be wedged, could this

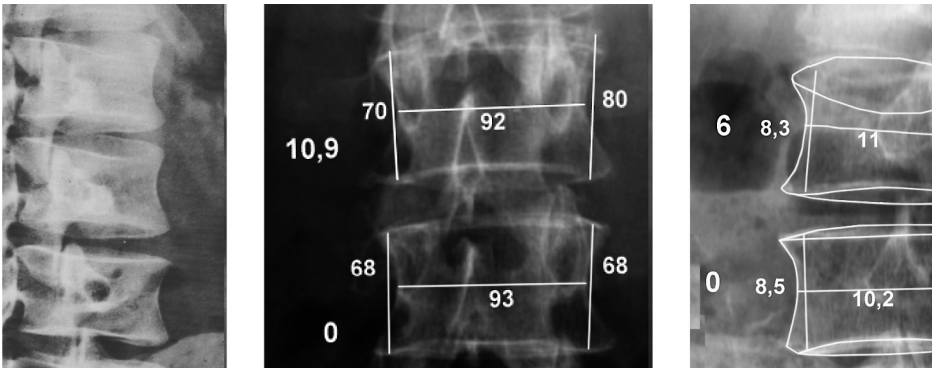
aspect be only due to a notch in a side of the vertebra? With Doctor Kotwicki in Poznan, Poland, we have controlled this fact on a rotated and wedged vertebra. Doctor Kotwicki has derotated the apex with his hands, then radiographed it again. The wedge shape had remained unchanged. That signed a true wedging and not only a single notch. A similar demonstration is made **fig. 16 B and C**.

Notice. The wedging of a single vertebra has a value only in relation with the wedging of the neighbouring vertebrae. Often is the vertebra under the apex one the most wedged. When only one vertebra is wedged, the deformation is maximal and the prognosis bad. All that could be the matter of a further study.

Please notice, that all our wedged and unwedged vertebrae have been either unchanged or bettered under action of brace. Many of them had been completely unwedged, into a symmetrical state. That is one more proof of the beneficial action of the good braces. This part of our work concerning wedged vertebrae is the most important one, because we never saw similar facts published.

CONCLUSION

Although the number of patients in our series is low, we believe that our count has a certain value as far as rotation, rib static and above all wedging of vertebrae have been measured and compared. Such measures had scarcely been published up to our times. With only two exceptions which concern rib static by two very severe scoliosis, no feature, Cobb's angles, rotation, rib static and wedged vertebrae has worsened after bracing. Most have bettered to an important percentage. The most important data concern the normalization of wedging. These normalizations of structure



A B C

Fig. 17. A. The vertebral bodies do not look like being wedged in this incidence. B and C. Scoliosis with small Cobb's angle as a result of a lower limbs difference. L2 is wedged, both in front view and in oblique one. The greater side is left on front view, and right on oblique one, as a result of the change of position. Notice, the upper plate of L2 is very oblique backwards and downwards : its oval shape is large. L3 is not wedged, neither on front, neither on oblique views.

prove once more, that a well done brace with expansion rooms is a determining condition for curing a scoliosis. We intend to continue to do evaluations with more cases, more delayed time and more factors taken in account. We hope that others teams will measure the wedging of apex and near-apex vertebrae in a near future, being a sure sign of bettering of scoliosis by brace.

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BOSWELLIN – NOVÉ ANTIREVMATIKUM?

BOSWELLIN – A NEW ANTIRHEUMATIC DRUG?

M. ADAM

Revmatologický ústav, Praha

V roce 1991 americká společnost SABINSA se započala věnovat výzkumu *Boswellinu*®. Nejprve došlo ke standardizaci extrakční metody ze stromu *Boswellia serrata* tak, aby výsledný produkt obsahující boswellové kyseliny vyhovoval předpisům FDA. Pro látky s protizánětlivým účinkem, které jsou přítomné v tomto extraktu se ujal název boswellin. Standardizované extrakty, které jsou tč. na trhu, obsahují podle titračních analýz 50 – 70 % boswellových kyselin (1).

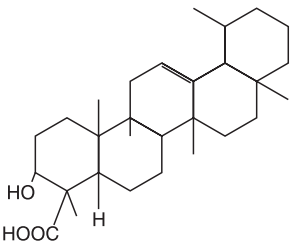
Boswellia serrata je velký rozvětvený strom s opadavým listím, který roste v suchých kopcovitých částech Indie. Pryskyřice kaučukovitého charakteru, v místním jazyce známá jako „Salai guggal“, je v domorodém lékařství užívána pro léčbu dýchacích onemocnění, jaterních poruch a revmatismu. Rovněž v evropské medicíně je boswellin podáván k potlačení revmatických a zánětlivých chorob. Léčebné účinky extraku ze stromu Boswellia serrata jsou připisovány již zmíněným boswellovým kyselinám, které inhibují dva prozánětlivé enzymy – 5-lipoxygenázu, generující zánětlivé leukotrieny a lidskou leukocytární elastázu (HLE), což je serinová proteináza iniciující tkáňové poruchy, které spouštějí zánětlivé pochody. Přestože aktivitu 5-lipoxygenázy nejúčinněji potlačuje acetyl-11-keto-β-boswellová kyselina, komerční preparáty obsahují jen nevelká množství této kyseliny. *Boswellin*® obsahuje, pokud je známo:

- monoterpeny (α-thujen)
- diterpeny (makrocyklické diterpenoidy jako incesol, incesol oxid, diterpen alkohol (serratol))
- triterpeny (např. α- a β- amyriny)
- pentacyklické triterpenové kyseliny (tirukal-8,24-dien-21-oiková kyselina).

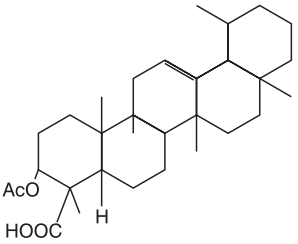
V kyselém extraktu z pryskyřice Boswellia serrata (Boswellin®) jsou přítomny čtyři větší pentacyklické triterpenové kyseliny:

- β-boswellová kyselina (I)

chemicky: 3α-hydroxy-urs-12-en-23-oiková-kyselina
molekulový vzorec: C30H48O3
molekulová váha: 456.7



● acetyl-β-boswellová kyselina (II)
chemicky: 3α-acetoxy-urs-12-en-23-oiková kyselina
molekulový vzorec: C32H50O4
molekulová váha: 498.74



● 11-keto-β-boswellová kyselina (III)
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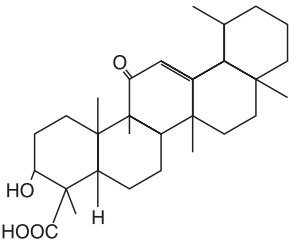
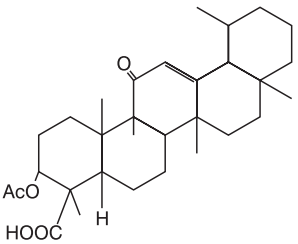
molekulová váha: 470.69

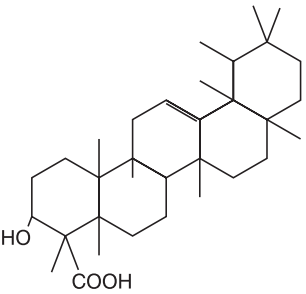


● acetyl-11-keto-β-boswellová kyselina (IV)
chemicky: 3α-acetoxy-urs-12-en-11-keto-23-oiková kyselina
molekulový vzorec: C32H4805
molekulová váha: 512.73

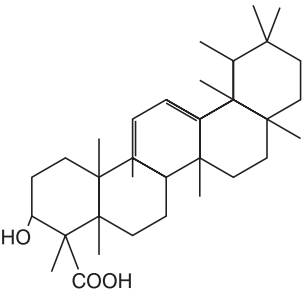


Kromě toho byly ještě isolovány dvě další triterpenové kyseliny:

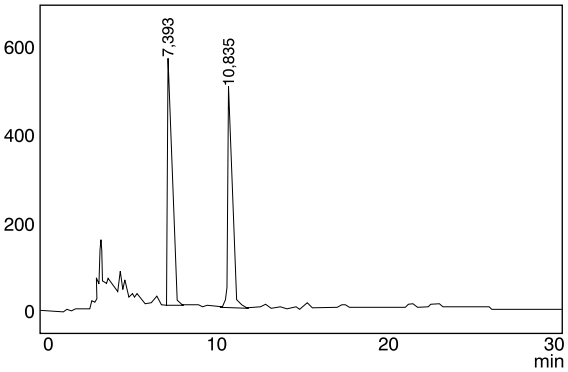
● α-boswellová kyselina (V)



● γ-boswellová kyselina (VI)



Dodatečně bylo izolováno ještě pět triterpenových kyselin, a dále čtyři tetracyklické triterpenové kyseliny. Při přípravě Boswellinu® byly získány dvě frakce: kyselá frakce a neutrální frakce. Souhrn kyselých frakcí má farmakologický význam a všechna klinická pozorování jsou založena na pozorování vlivu těchto frakcí při jejich podávání nemocným. Výzkumy z devadesátých let ukazují, že v kyselém extraktu jsou boswellové kyseliny farmakologicky nejaktivnější a mají nevíce vyjádřený léčebný účinek. Jedná se o 11-keto-β- a acetyl-11-keto-β-boswellové kyseliny.

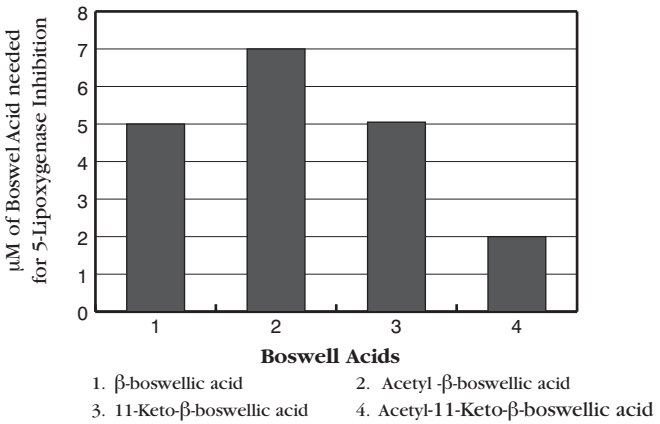


Obr. 1. Porovnání HPLC chromatogramů dvou β-boswellových kyselin (UV detekce 254 nm).
Retenční čas: 7,393 – 11-keto-β-boswellová kyselina
Retenční čas: 10.835 – acetyl-11-keto-β-boswellová kyselina

VÝSLEDKY KLINICKÝCH STUDIÍ

Klinické studie uváděné v literatuře uvádějí zkušenosti s kyselou frakcí boswellinu®. Čistá boswellová kyselina představuje asi 25 % protizánětlivých látek účinkujících u nemocných. Shao a spol. (1998) popsal inhibiční vliv boswellinu na buňky lidské leukémie HL-60. Boswellin podle Safayhi a spol. 1997 (2) inhibuje také lidskou leukocytární elastázu.

Další klinickou studií byla práce autorů Kulkarni et al. 1991 (3), kteří testovali



Obr. 2. Inhibiční účinek různých boswellových kyselin na 5-lipogenasu.

přípravek obsahující výtažek z kořene Withania somnifera, kmene stromu Boswelia serrata a zinek. Randomizovaná dvojitě zaslepená studie u OA pacientů, kontrolovaná placebem. Do výzkumu bylo zařazeno 42 pacientů s osteoartrózou, přípravek byl podáván 3 měsíce a po 14 denní pauze další 3 měsíce. Klinická účinnost byla vyhodnocena na základě míry bolestivosti, ranní ztuhlosti, míry invalidity a síly stisku. Sedimentace a RTG snímkování bylo prováděno každý měsíc. Ve skupině léčené herbominerálním přípravkem byl proti placebu pozorován signifikantní pokles v míře bolestivosti ($p<0,001$) a v míře invalidity ($p<0,05$). Radiologická vyšetření nevykazovala signifikantní rozdíly mezi skupinami. Nebyly pozorovány vedlejší účinky.

Reddy et al. 1989 (4) provedli výzkum efektu kyseliny boswellové na metabolismus glykosaminoglykanů u samců laboratorních potkanů albino. Biosyntéza sulfátových glykosaminoglykanů byla vyhodnocena dle příjmu [35S]sulfátu. Obsah glykosaminoglykanů byl měřen v kůži, játrech, ledvinách a slezině. Byla pozorována signifikantně redukovaná syntéza glykosaminoglykanů. V porovnání s kontrolní skupinou byla pozorována markantně snížená degradace glykosaminoglykanů.

U nás bude v nejbližší době boswellin dodáván na trh v přípravku GELLADRINK FAST® firmy Orling s.r.o., v němž zvyšuje účinnost hlavní složky tohoto nutraceutika tj. kolagenního hydrolyzátu. Možnost placebo efektu odpadá při veterinární aplikaci – velmi se osvědčilo jeho podávání závodním koním, u nichž urychluje hojení zánětů šlach eventuelně namožení kloubů.

Závěrem je možno říci, že boswellin představuje nadějnou látku pro humánní i veterinární medicinu.

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FAMILIÁRNÍ OSTEOCHONDRITIS DISSECANS

FAMILIAL OSTEOCHONDRITIS DISSECANS

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SUMMARY

Two cases of osteochondritis dissecans in a boy and his father are described. The boy was asymptomatic in spite of multifocal osteochondritis-like changes involving the knees, spine and wrist bones. The father showed early degenerative hip disease with left sided total hip replacement. Radiograms documented the wide spectrum of radiological abnormality which occurs in this condition.

Key words: Familial osteochondritis dissecans – osteoarthritis

INTRODUCTION

Osteochondritis dissecans (OD) is a well-known knee disorder. One or both knee joints may be affected. The elbow and ankle lesions are less common and have often a „silent" clinical course (1, 2). Lesions limited to one or two symmetrical joints may be classified as localised cartilage/bone dysplasias (7, 8). Patients with OD localised to the elbows usually show some discreet bony changes at the bones which form the elbow joint (7). OD may be observed in some bone dysplasias especially spondylo-epiphysealis and polyepiphysealis dysplasias (8). Random association of OD with Scheuermann disease, Osgood-Schlatter disease, carpal tunnel syndrome, tibia vara and idiopathic scoliosis has also been reported (5,9,13).Familial OD has been described in over 40 families. Inheritance is autosomal dominant (4-6, 9, 11–13). Males are affected about 3 times as commonly as females. In familial OD short stature and endocrine disorders are distinctive, not uncommon features (9, 13). We report familial OD in a 39 year-old man and his 12 year-old son.

CASE REPORT

PATIENT 1

This 39 year-old man has a long history of bilateral hip pain. At the age of 9 years he experienced knees swelling and at 15 his hips were stiff and at 16 he had a cliff fall with fracture of the left femur. He was moderately sport active at school. He had no other medical history. He developed OD changes in both hips and secondary osteoarthritis. Because of increasing walking difficulties and left hip pain at 24 years of age he underwent a left total hip replacement **(Fig. 1).**

PATIENT 2

This 12 year-old boy presented with short stature and a prominent lumbar lordosis. He was 130 cm tall (below 3rd centile). He was asymptomatic. After assessment of bone age skeletal survey was performed **(Fig. 2A-D).** Clonidine stimulation growth hormone, pancreatic and salivary amylase isoenzymes, and routine biochemistry were all normal.

DISCUSSION

We do not have the early hip radiographs of the father but the present hip

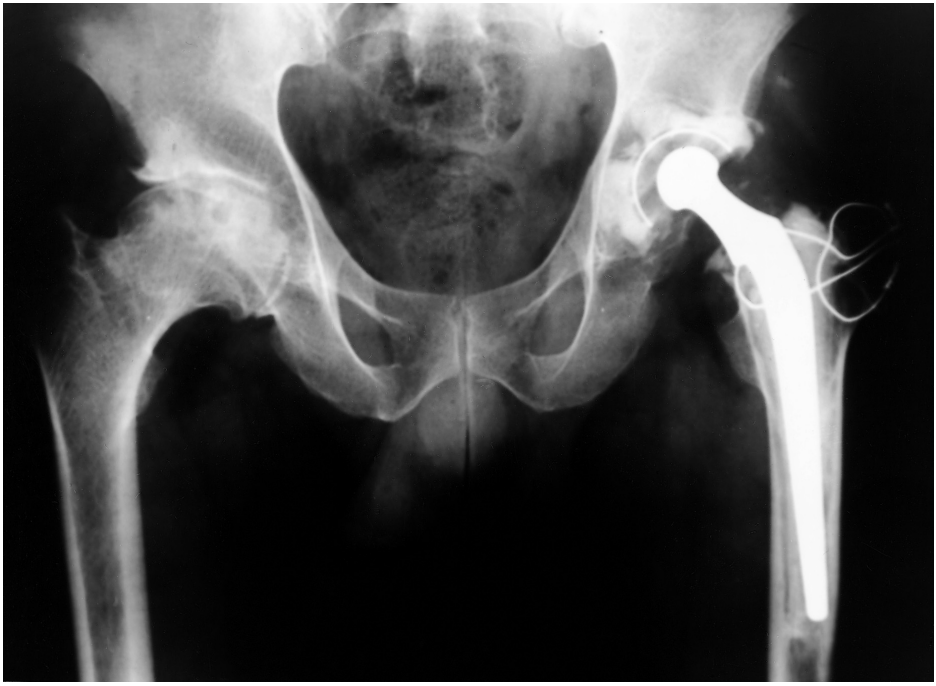
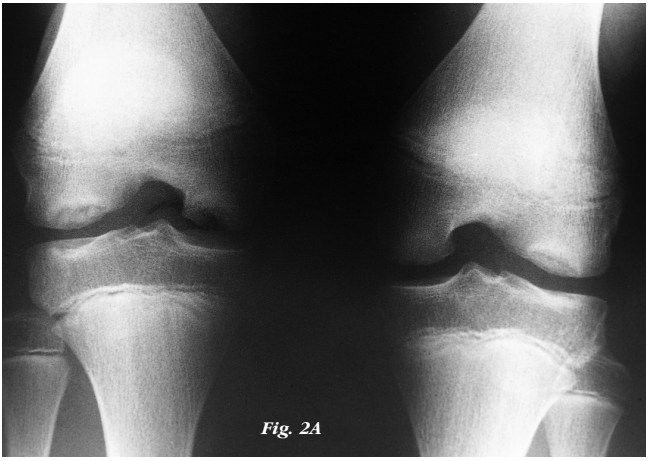
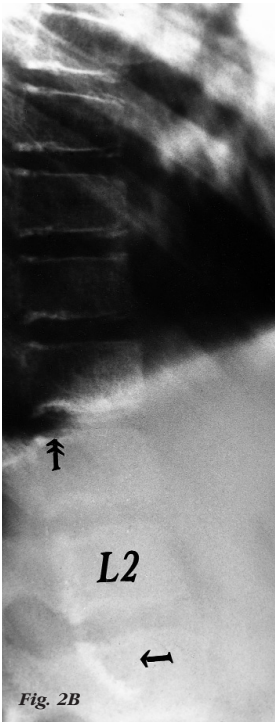


Fig. 1. Familial Osteochondritis dissecans. Man 39 year-old. Pelvis. Severe osteoarthritis with narrowing of the right joint space and subchondral cyst formation.





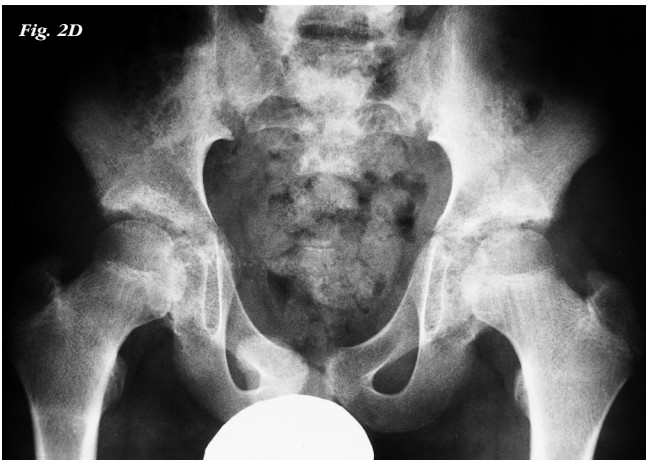


Fig. 2. A-E. Familial Osteochondritis. Boy 12 year-old. A. Knees. OD affecting both the femoral condyles and the lateral part of the tibial epiphyses. B.Spine. There is irregularuty of the vertebral plates most marked posteriorly. Minor flattening of the thoracic spine. Narrowing of the intervertebral spaces in the lumbar spine with small, OD like defects at the level Th12/L1(double arrow), and a large radiolucent defect in the posterior, upper part of the vertebral body of L3 (single arrow). C. Hands. Some intercarpal joint spaces are narrowed. The hamates and triquetra are slightly sclerotic with radiolucent defects in the proximal hamates and distal triquetra. Bone age corresponds to the chronological age of 12 years. D.Pelvis. Indistinct, irregularly sclerotic acetabular outline. Axial sclerosis of sacrum. Grossly normal capital femoral epiphyses. E. Left shoulder. Abnormal trabecular pattern of the acromion, glenoid and the proximal humeral metaphyses. Radiolucent defect of distal part of glenoid is seen. Note the broad medial clavicular end with narrowing of the medullary cavity above the coracoid process.

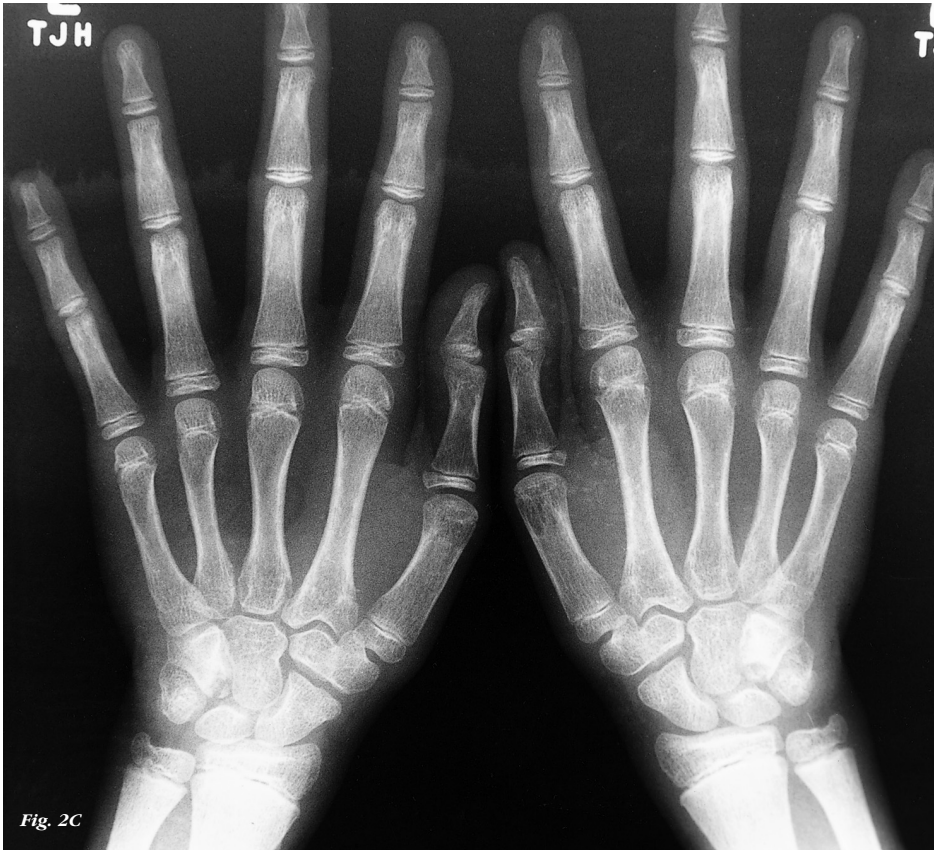


Fig. 2C

X-ray documents OD-like changes and severe osteoarthritis in the right hip. He had been severely affected as a total hip replacement in a 24 year-old man with little sport activity is unusual indeed. In the boy short stature was the cause for medical check up. Surprisingly in spite of bilateral knee OD changes **(Fig. 2A)** he was without clinical symptoms. Noteworthy are his spine changes characterised by minor irregularity of the vertebral plates with OD-like radiolucent defects in the lumbar vertebrae **(Fig. 2B)** and osteolytic changes in the hamates and triquetra **(Fig. 2C).** Involvement of the carpal bones has to the best of our knowledge not have been reported in any forms of OD.

The etiology of OD is uncertain. Although OD is usually regarded as a post-traumatic disorder (2, 3,10) a primary cartilage metabolic deficiency is a possible main reason (6) with trauma as an important contributing factor. Anomalous ossification resulting from enchondral dysostosis with localised interruption of blood supply and underlying bone abnormalities were also

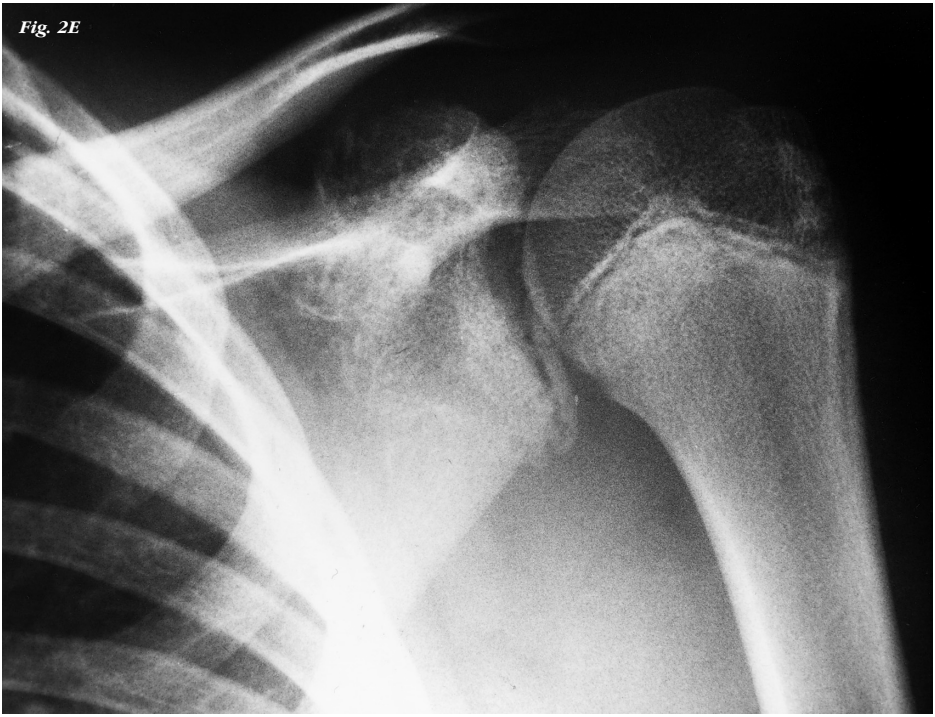


Fig. 2E

implicated as etiologic factors of OD (4). In children with OD of the knees radiograms of the areas of silent OD – elbows, ankle joints or skeletal survey – should be considered depending from medical history and clinical examination. Familial studies are necessary if locomotor apparatus related disorders are present in the family.

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DYNAMIKA HOUSTNUTÍ KORTIKALIS

DYNAMICS OF GROW OF BONE CORTICALIS DENSITY

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SOUHRN

Předložená práce je zaměřena na exaktní analýzy procesů houstnutí kortikalis. Rychlosti biochemických reakcí jsou ovlivňovány nejenom chemickými a genetickými účinky, ale i *účinky mechanickými* (biomechanickými). Houstnutí nastává když celkové objemové změny η_j (sledované reaktantní složky) jsou záporné ($\eta_j < 0$), tj. když se objem hmotnostní jednotky zmenší. Proces houstnutí ve tkáni závisí nejenom na dominanci objemových změn, ale i na změnách napětí v elementu kostní tkáně. Změnou napětí Δp lze proces houstnutí ve tkáni urychlit nebo zpomalit. Jestliže změny mechanického napětí ve tkáni jsou *záporné,* dochází k retardaci houstnutí ve tkáni. Jestliže změny mechanického napětí ve tkáni jsou *kladné* dochází k akceleraci houstnutí ve tkáni.

Klíčová slova: biomechanika, houstnutí kosti, regulátory houstnutí, rychlost houstnutí, změny napětí, objemové změny, retardace houstnutí kosti, akcelerace houstnutí kosti.

SUMMARY

Limit cycles of bone remodelling are regulated biomechanically and biochemically (genetically). The speeds of biochemical reactions (i.e. the speeds of intense metabolic processes) depend on the volume changes of molecular mixtures and on the stress changes in a bone element. Processes of density depend on both the dominant volume changes of molecular mixtures and stress changes. The resultant speed of the j^{th} biochemical reaction, which forms part of biochemical (metabolic) processes in the bone tissue (in the remodelling limit cycle) is dependent on the **product of speeds** of the biochemical reaction *initiated biochemically (resp. genetically)* and on the speeds of chemical reaction *initiated biomechanically.* The j^{th} biochemical reaction is influenced by the internal – primary chemical

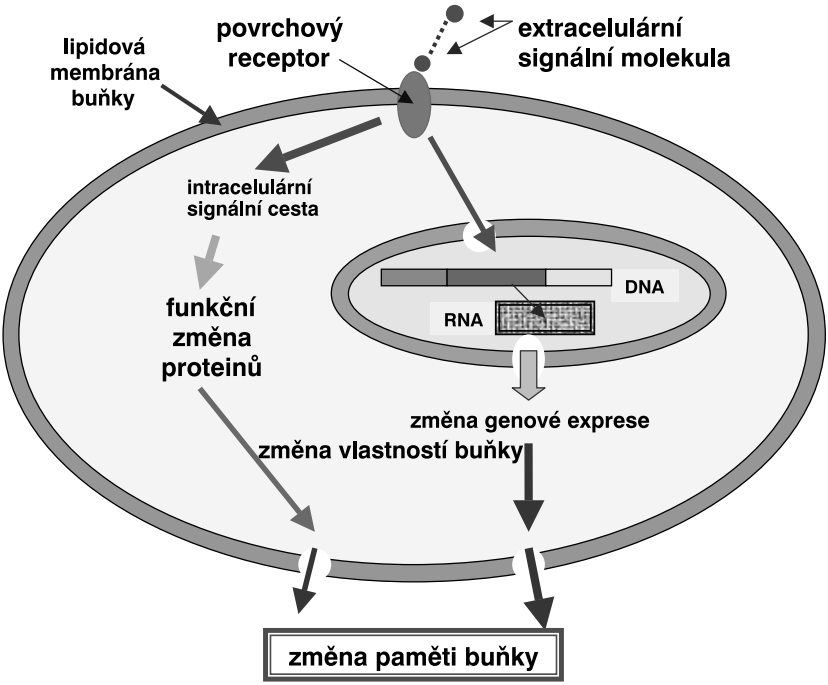
(genetical) effects and the external – biomechanical effects, i.e. stress changes Δp. The density of bone can be increasing when the stress changes in bone have the positive signum.

Key words: biomechanics, grow of bone density, bone regulators, speed of bone density, stress changes, volume changes, retardation/acceleration of bone density.

1. ÚVOD

Remodelační procesy v kortikalis probíhají v limitních cyklech střídáním intenzivních biochemických reakcí se slabě stacionárními stavy [1], [2]. Existence slabě stacionárních stavů je podmínkou následných intenzivních metabolických procesů (biochemických reakcí) a naopak.

Během biochemických reakcí dochází v uvažovaném objemovém mikro/nanoelementu kosti ke změnám koncentrací molekulárních směsí. Tyto změny koncentrací jsou důsledkem komplexních metabolických procesů, které jsou *ovlivněny změnami exprese genů (tj. v případech, kdy se uplatňuje genomový mechanizmus) nebo jsou důsledkem změn proteinů (tj. změn jejich funkcí uvnitř buňky), a to bez vlivu genů,* **obr. 1.**



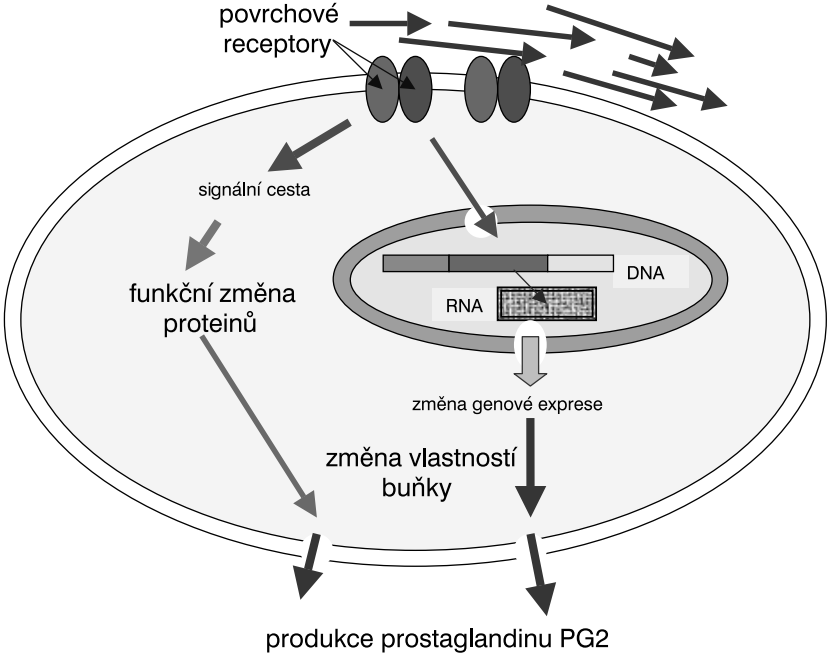
Obr. 1. Dvě cesty buněčné exprese, vlevo – funkční změnou proteinů (bez vlivu genomového mechanizmu), vpravo – změnou genové exprese (vlivem „zapojeného" genomového mechanizmu).

Kromě těchto dvou fundamentálních procesů (bez vlivu genů a s vlivem genů), existuje velmi důležitý **primární vliv mechanický/biomechanický.** Klein-Nulend, E. Burger dokázali [3], že při mechanicky iniciovaném toku extracelulární tekutiny v lakunách osteocytů, dochází ke stimulaci osteoreceptorů α, β (integrínů) a následně k produkci prostaglandinu PG2, který přispívá k resorpci kortikalis, **obr. 2.**

Souhrnně řečeno, biochemické reakce mohou být ovlivněny: *a) chemickými účinky (bez genetických vlivů), b) chemickými účinky u nichž se uplatňuje genomový mechanizmus, c) biomechanicky.*

Role biomechanických účinků při metabolických procesech v kortikalis byla dosud zkoumána a ověřována velmi sporadicky. Nejsou exaktně popsány rychlosti metabolických procesů houstnutí nebo řídnutí kosti v závislosti na objemových změnách molekulárních směsí (verifikovaného elementu tkáně) a není známá role vlivu změn napětí ve tkáni při jejím houstnutí nebo řídnutí.

Tato práce je zaměřena na *analýzu dynamiky metabolických procesů houstnutí kortikalis.* Cílem je **formulovat fundamentální axiomy, týkající se vlivu mechanického/biomechanického namáhání na akceleraci/retardaci houstnutí.**



Obr. 2. Schéma produkce prostaglandinu PG2, který přispívá k resorpci kortikalis. Deformace mineralizované matrice (vyvolané mechanickým/biomechanickým zatížením skeletu) iniciují tok extracelulární kapaliny v lakunách osteocytů. Smykový tok této kapaliny (v tangenciálním směru k povrchu lipidové membrány) namáhá povrchové buněčné osteoreceptory, které přenášejí biomechanochemický signál do intracelulárního prostoru osteocytu [3].

Práce je zaměřena i na racionální využití presentovaných závěrů v klinické praxi.

Pozastavme se nejdříve a ve stručnosti u některých hlavních regulátorů vlastností a struktur kosti. *Genové regulátory* jsou důležité pro tvorbu tkání, na příklad pro její mineralizaci [4]. Geny jsou citlivé na specifické hormony [5]. Na příklad parathyroid je hormonálním peptidem, který reguluje buněčný metabolismus v kosti, snižuje kolagenní syntézu (osteoid) a naopak zvyšuje osteokalcinovou genovou expresi [6]. PTH v kosti stimuluje mnohonásobné buněčné signály [6] a reguluje kalciovou homeostázu v séru [8]. *Hormony iniciují genovou aktivitu* a prostřednictvím genů ovlivňují (sekundárně regulují) biochemické procesy v kosti.

Jiným příkladem je genová regulace kostního sialoproteinu (BSP), která je důležitá pro diferenciaci osteoblastů a pro mineralizaci osteoidu [9]. BSP je (spolu s osteopontinem OSP) hlavním proteinem kostní matrice [4]. BSP je regulován hormonálním peptidem (PTH, viz výše) pomocí genového mechanizmu [10]. Je produkován osteoblasty, během mineralizace osteoidu [4] a je prominentní komponentou, při formaci kosti [11], [12], [13], [14]. Sialoprotein má funkci nejenom regulační, ale i strukturální [10].

Hormonální regulace sehrává důležitou roli i při produkci osteopontinu (OSP). Osteopontin je fosforečný mimobuněčný protein (nalezající se v matrici kosti) [15]. Vzniká v kostní matrici při její mineralizaci z transformovaných buněk [13], [14], [4]. Spolu s kostním sialoproteinem (BSP) je jejím dominantním proteinem.

Mineralizace osteoidu je regulována též D-vitaminem [1.25-$(OH)_2D_3$], [16]. Vitamin D moduluje a řídí uvolňování kalciových iontů buňkami do mineralizované tkáně [16], [17].

Velmi významnou roli při rekonstrukci kosti (její regenerace) sehrávají růstové faktory. *Růstové faktory, spolu s kalciovou fosfatázou,* akcelerují regeneraci tkáně [18].

Výše uvedené biochemické (metabolické) procesy v kosti byly většinou sledovány bez zohlednění biomechanických vlivů, které sehrávají zcela neopominutelnou roli při houstnutí a řídnutí kosti. A právě na tyto vlivy je zaměřena tato práce.

Při analysách změn hustoty je třeba velmi důsledně rozlišovat houstnutí ve tkáni a houstnutí tkáně. Při změnách hustot ve tkáni jsou sledovány vždy jen jisté komponenty (j-té biochemické reakce), zatímco při analysách hustoty tkáně je tkáň posuzována z globálního (celkového) pohledu.

Procesy houstnutí (aposice) tkáně jsou popsány třemi stechiometrickými rovnicemi (1), (2) a (3), [1], [2]:

$$D_2 + D_5 \Rightarrow D_7 + D_8 \quad (1)$$

$$D_9 + D_7 \Rightarrow D_{10} + D_{11} \quad (2)$$

$$D_{12} + D_{10} \Rightarrow D_{13} + D_{14} \quad (3)$$

kde

- $\mathbf{D_2}$ jsou mononukleární buňky (krevního původu) a preosteoblasty,
- $\mathbf{D_5}$ jsou enzymy a zbytné produkty resorbce „staré“ mineralizované kostní tkáně,
- $\mathbf{D_7}$ jsou vznilé osteoblasty a jimi produkované enzymy a další syntetické složky, regulující a spoluvytvářející organickou (nemineralizovanou) matrici – osteoid,
- $\mathbf{D_8}$ jsou zbytné produkty vzniklé degradací organické a anorganické složky kostní tkáně důsledkem aktivity mononukleárních buněk,
- $\mathbf{D_9}$ je substrát (extracelulární tekutina) obsahující na příklad metabolity, látky energeticky podporující funkce remodelace, ionty, cukry, aminokyseliny, kalcitonin (produkovaný štítnou žlázou a vnesený do krve) a soubory dalších látek,

$\mathbf{D_{10}}$ je osteoid, tj vysokomolekulární (kolagenní) komponenta mezibuněčné hmoty neobsahující minerální složky.

$\mathbf{D_{11}}$ je odpadový substrát, který je produktem biochemických oxydačních procesů a ostatních reakcí při vzniku přebytečných (odpadních) nízkomolárních produktů, jako je např. kyselá fosfatáza aj.

$\mathbf{D_{12}}$ je substrát, který obsahuje komponenty iniciující následnou mineralizaci osteoidu.

$\mathbf{D_{13}}$ je substrát, tj. komplex nadbytečných a odpadních produktů z předchozích biochemických reakcí, umožňujících a regulujících remodelační procesy.

$\mathbf{D_{14}}$ je vysokomolekulární mineralizovaný kolagen, tvořící novou extracelulární matrici.

2. RYCHLOSTI BIOCHEMICKÝCH PROCESŮ

Rychlosti jednotlivých biochemických reakcí dle rovnic (1) až (3) jsou výrazně závislé na objemových změnách dominantních komponent (reaktantech, molekulárních směsích) a na změnách napětí. Pro první biochemickou reakci (1) je rozhodující (kromě změn napětí) objemová změna osteoblastů η_3, pro druhou biochemickou reakci (2) je rozhodující (kromě změn napětí) objemová změna osteoidu η_4, pro třetí biochemickou reakci (3) je rozhodující (kromě změn napětí) objemová změna mineralizovaného osteoidu η_5. Tyto závislosti *jsou nástrojem pro exaktní analýzy procesů houstnutí v kostní tkáni. Důsledkem těchto dílčích procesů (tj. souborů všech biochemických reakcí (1) až (3)) je celkové houstnutí kostní tkáně, v globálním pohledu.*

Globální houstnutí tkáně (v integrálním, celkovém pohledu na tkáň) je posloupnost (řetězec) biomechanochemických procesů (tj. biochemických reakcí a stacionárních stavů), jejichž výsledkem je *zvětšení hustoty kostní tkáně* (ve sledovaném jejím elementu).

Proces houstnutí kostní tkáně (v jejím objemovém elementu) je charakteristický tím, že hmotnostní jednotka této tkáně (na příklad 1g nebo 1 mg) zaujme menší objem než měla na počátku houstnutí. *To znamená, že došlo k nárůstu kostní tkáně, tj. zvětšila se její hustota, tzn., že výsledná objemová změna kostní tkáně je menší jak nula.*

Rychlosti biochemických reakcí jsou ovlivňovány nejenom chemickými a genetickými účinky, ale i **účinky mechanickými** (biomechanickými). Obecně řečeno, rychlost $\mathbf{k_j}$ (j-té biochemické reakce, j = 1, 2, 3) je funkcí jak objemových změn η_j *sledovaných reaktantních složek* molekulární směsi tkáně, tak i změn napětí $\Delta p = p - p_e$, kde p_e je napětí v elementu kostní tkáně za stacionárního stavu.

Pro rychlost j-té biochemické reakce (j =1, 2, 3) remodelované kostní tkáně platí exponenciální rovnice (4), viz [1], [2], [19], [20], [21]:

$$k_j = A_j \, e^{-\eta_j \Delta p} \qquad (4)$$

kde

Δp jsou změny napětí v elementu kostní tkáně,

η_j jsou objemové změny sledovaných reaktantních složek v elementu kostní tkáně vzniklých účinkem:

a) **chemických látek (které jsou primárně iniciovány mechanickými účinky),** a které vyvolávají objemové změny η_{jm} sledovaných reaktantních složek (například smykovým tokem extracelulární tekutiny v lakunách osteocytů);

b) **chemických látek, transformovaných v buňkách při „vypnutém" genomovém mechanizmu,** které primárně (nebo sekundárně) vyvolávají objemové

změny η_{jchp} sledovaných reaktantních složek ;

c) **chemických látek (vzniklých v součinnosti s genomovým mechanizmem),** vyvolávající objemové změny η_{jchg} sledovaných reaktantních složek;

A_j je součinitel závislý na hodnotě rovnovážného stavu tkáně.

Pro celkovou (výslednou) objemovou změnu η_j sledovaných reaktantních složek (j-té biochemické reakce) a za předpokladu platnosti principu superpozice můžeme psát:

$$\eta_j = \eta_{jm} + \eta_{jchp} + \eta_{jchg} \qquad (5)$$

Sloučením objemových změn η_{jchp} od primárních chemických účinků (bez součinnosti s genomovým mechanizmem) s objemovými změnami η_{jchg} (od chemických účinků iniciovaných v součinnosti s genomovým mechanizmem) v celkové objemové změny od chemických účinků, dostaneme:

$$\eta_j = \eta_{jm} + \eta_{jch} \qquad (6)$$

Rovnici (4) lze pomocí rovnice (6) upravit takto:

$$k_j = A_j\, e^{-(\eta_{jm} + \eta_{jch})\Delta p} \qquad (7)$$

Z rovnice (7) je zřejmé, že rychlost **j-té** biochemické reakce (j = 1, 2, 3) při remodelaci kostní tkáně je ovlivňována změnami napětí Δp a objemovými změnami $h_j = h_{jm} + h_{jch}$.

Vzhledem k tomu, že *rychlost j-té biochemické reakce* (j = 1, 2, 3) *je funkcí objemových změn* η_{jch} *od chemických účinků a objemových změn* η_{jm} *od chemických účinků primárně iniciovaných mechanickými vlivy,* je třeba určit, **jaký vliv na rychlost j-té biochemické reakce mají chemické látky (primárně iniciované chemicky, případně geneticky) a jaký vliv na rychlost j-té biochemické reakce mají chemické látky (vznikající primárně mechanickými účinky).**

Se zřetelem k podrobným analýzám různých vlivů (účinků) na rychlosti biochemických reakcí vyjdeme z rovnice (7), kterou lze upravit na tvar:

$$k_j = A_j\, e^{-\eta_{jch}\Delta p}\, e^{-\eta_{jm}\Delta p} \qquad (8)$$

Označíme-li

$$e^{-\eta_{jch}\Delta p} = k_{jch} \qquad (9)$$

a

$$e^{-\eta_{jm}\Delta p} = k_{jm} \qquad (10)$$

dostaneme pro rychlost j-té biochemické reakce j = 1, 2, 3):

$$k_j = A_j\, k_{jch}\, k_{jm} \qquad (11)$$

Z rovnice (11) je zřejmé, že **rychlost j-té biochemické reakce je úměrná součinu rychlosti k_{jch} od účinků primárně chemických a rychlosti k_{jm} od účinků primárně mechanických.**

Cílem následujících analýz je determinovat, jak výsledná rychlost k_j (j-té biochemické reakce v remodelované tkáni) závisí na velikosti rychlosti k_{jch} ovlivněné primárně chemicky a na velikosti rychlosti k_{jm} ovlivněné primárně mechanicky (tj. sekundárně chemicky).

Vztah mezi k_{jch} a k_{jm} můžeme určit z jejich poměru podle (9) a (10):

$$\frac{k_{jch}}{k_{jm}} = \frac{e^{-\eta_{jch}\Delta p}}{e^{-\eta_{jm}\Delta p}} \qquad (12)$$

Z rovnice (12) dostáváme, že

$$k_{jch} = e^{-(\eta_{jch} - \eta_{jm})\Delta p}\, k_{jm} \qquad (13)$$

Vztah mezi k_{jch} a k_{jm} je modelován exponenciální funkcí.

V kostní tkáni během její remodelace mohou (v průběhu každé j-té biochemické reakce, j = 1, 2, 3) nastat dva fundamentální případy dominance rychlosti, a to dominance rychlosti k_{jch} *(tj. dominance rychlosti od chemických účinků) nebo dominance rychlosti* k_{jm} *(tj. dominance rychlosti od mechanických účinků).*

Protože **objemové změny** η_{jch} **od primárních chemických účinků a objemové změny** η_{jm} **od primárních mechanických účinků** jsou **mimo stacionární stavy ve výrazné nerovnováze,** je v rovnici (13) vždy *jedna z hodnot objemových změn vyšší než hodnota druhé objemové změny* (tzn. $\eta_{jch} << \eta_{jm}$ nebo $\eta_{jch} >> \eta_{jm}$).

Z rovnice (13) je zřejmé, že rovnost mezi rychlostí k_{jch} (od chemických účinků) a rychlostí k_{jm} (od mechanických účinků) nastane, když $\eta_{jch} = \eta_{jm}$ a nebo, když $\Delta p = 0$ (tj. $p = p_e$). Absolutní stacionární stav („zmrtvení" tkáně) vzniká tehdy, když změny napětí jsou nulové (tj. $\Delta p = 0$, tj. $p = p_e$). Pak rychlost biochemické reakce k_{jch} od chemických účinků se rovná rychlosti k_{jm} od mechanických účinků, tj. $k_{jch} = k_{jm}$. Obě rychlosti jsou konstantní.

Signum výsledné objemové změny $\eta_j = \eta_{jm} + \eta_{jch}$ (pro j = 1, 2, 3) **sledovaných reaktantních složek definuje (charakterizuje) houstnutí molekulární směsi a nebo její řídnutí ve tkáni při j-té biochemické reakci.** Řídnutí ve tkáni nastává tehdy, když celková objemová změna η_j, sledované reaktantní složky je kladná ($\eta_j > 0$). **Houstnutí nastane, když objemové změny** η_j **sledovaných reaktantních složek jsou záporné** ($\eta_j < 0$), tj. hmotnostní jednotka uvažované komponenty zaujme menší objem.

Proces houstnutí ve tkáni závisí nejenom na dominanci objemových změn, ale i na změnách napětí v elementu kostní tkáně. Změnou napětí Δp **lze proces houstnutí ve tkáni urychlit nebo zpomalit.** Tímto problémem se budeme zabývat v následujících odstavcích.

3. PODMÍNKY HOUSTNUTÍ V KOSTNÍ TKÁNI

K houstnutí tkáňové substance (při j-té biochemické reakci remodelace kostní tkáně, j = 1, 2, 3) dochází, když výsledná objemová změna η_j sledované tkáňové substance (tj. sledované reaktantní složky) je záporná (tzn., že dochází ke zmenšení objemu její hmotnostní jednotky):

$$\eta_j = \eta_{jm} + \eta_{jch} << 0 \qquad (14)$$

To může být vyvoláno buď:

(1) objemovými změnami: $\eta_{jch} << 0$ (od primárních chemických účinků), nebo

(2) objemovými změnami: $\eta_{jm} >> 0$ (od primárních mechanických účinků).

Vzhledem k tomu, že mimo stacionární stavy jsou objemové změny η_{jch} a η_{jm} v nerovnováze je v případě (1):

$$|\eta_{jch}| \gg |\eta_{jm}| \qquad (15)$$

a v případě (2):

$$|\eta_{jch}| << |\eta_{jm}| \qquad (16)$$

Průběh houstnutí tkáňové substance při j-té biochemické reakci (j = 1, 2, 3), vyvolaný objemovými změnami $\eta_{jch} << 0$ nebo $\eta_{jm} << 0$ je regulován změnami napětí $\Delta p = p - p_e$ (v elementu kostní tkáně). Výše uvedené dva případy (1) a (2), se zřetelem ke změnám napětí $\Delta p = p - p_e$, budeme dále podrobně analyzovat.

3.1 Iniciace houstnutí tkáňové substance objemovými změnami η_{jch} od primárních chemických účinků

Uvažujme případ, kdy při j-té biochemické reakci (j = 1, 2, 3) remodelace v kostní tkáni jsou objemové změny sledovaných reaktantních složek od primárních chemických účinků mnohem menší než nula, tj.:

$$\eta_{jch} << 0 \qquad (17)$$

a současně uvažujeme, že tkáňová substance je v nestacionárním stavu,

$$|\eta_{jch}| >> |\eta_{jm}|$$

Potom celková objemová změna sledovaných reaktantních komponent je:

$$\eta_j = \eta_{jm} + \eta_{jch} << 0 \qquad (18)$$

a také

$$\eta_{jch} - \eta_{jm} << 0 \qquad (19)$$

Výsledná rychlost k_j (j = 1, 2, 3) j-té biochemické reakce dle (8) a (11) je:

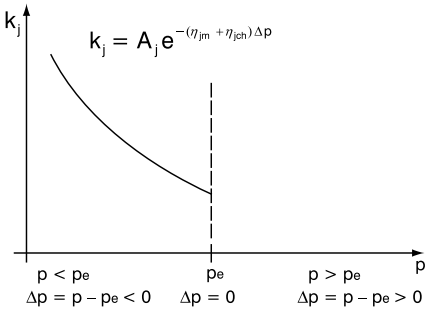
$$k_j = A_j\, k_{jch}\, k_{jm} = A_j\, e^{-(\eta_{jch} + \eta_{jm})\Delta p} \qquad (20)$$

Vlastní průběh houstnutí tkáňové substance je „určován" změnou napětí $\Delta p = p - p_e$. Výsledná rychlost j-té reakce dle rovnice (20) je ovlivněna znaménky změn napětí $\Delta p = p - p_e$.

3.1.1 Vliv záporných změn napětí

Jestliže změny napětí budou záporné, tj. $\Delta p < 0$, tj. $p < p_e$, potom rychlost j-té biochemické reakce (j =1, 2, 3) od chemických účinků bude menší než je rychlost od mechanických účinků (dle rovnice (13) a (19)), tj. $k_{jch} << k_{jm}$, a rychlost k_{jm} (primárně ovlivněná mechanicky) zpomalí (retarduje) rychlost k_{jch} (primárně ovlivněnou chemicky).

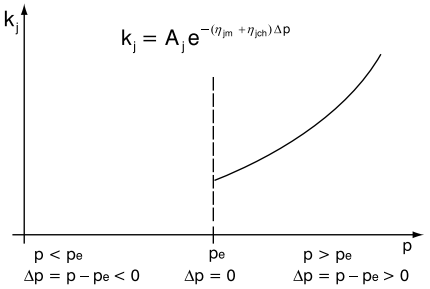
Důsledek je ten, že **výsledná rychlost k_j houstnutí ve tkáni (nastartované primární objemovou změnou $\eta_{jch} << 0$), bude při poklesu napětí p** (vzhledem k p_e, tj. při $\Delta p < 0$) **klesat**, dle (18) a (20), **obr. 3.**



Obr. 3. Průběh funkce výsledné rychlosti k_j biochemické reakce je úměrný exponenciální funkci $e^{-(\eta_{jch} + \eta_{jm})\Delta p}$. V tomto případě je funkce k_j klesající. Houstnutí v kostní tkáni nastartované objemovou změnou η_{jch} se bude při poklesu napětí p (vzhledem k p_e, tj. při $\Delta p < 0$) zpomalovat.

3.1.2 Vliv kladných změn napětí

Jestliže změny napětí budou kladné, tj. $\Delta p > 0$, tj. $p > p_e$, potom rychlost j-té biochemické reakce (j =1, 2, 3) od chemických účinků bude výrazně větší než je rychlost od mechanických účinků (dle rovnice (13) a (19)), tj. $k_{jch} >> k_{jm}, k_{jm}$ podstatně neovlivní k_{jch}. Z toho plyne, že **výsledná rychlost k_j houstnutí ve tkáni, vyvolané primární objemovou změnou $\eta_{jch} << 0$ od chemických účinků bude se při nárůstu napětí p** (vzhledem k p_e, tj. při $\Delta p > 0$) **zvětšovat** (dle rovnice (18) a (20), viz **(obr. 4).**



Obr. 4. Průběh funkce výsledné rychlosti k_j biochemické reakce je úměrný exponenciální funkci $e^{-(\eta_{jch} + \eta_{jm})\Delta p}$. V tomto případě je **funkce** k_j rostoucí. Při účinku chemických látek, na příklad léků nebo hormonů ($\eta_{jch} << 0$), dochází k houstnutí ve tkáni, a to jen tehdy, když tkáň bude zatěžována ($\Delta p > 0$, tj. $p > p_e$), jinak dojde k útlumu houstnutí v kostní tkáni (jak je zřejmé z předchozího odstavce 3.1.1).

3.2 Iniciace houstnutí tkáňové substance objemovými změnami η_{jm} od primárních mechanických účinků

Houstnutí ve tkáni může být též iniciováno objemovými změnami η_{jm} od primárních mechanických účinků. Uvažujme, že objemové změny $\eta_{jm} << 0$, a že tkáň je v nestacionárním stavu, tj. když $|\eta_{jch}| << |\eta_{jm}|$. Potom celková objemová změna:

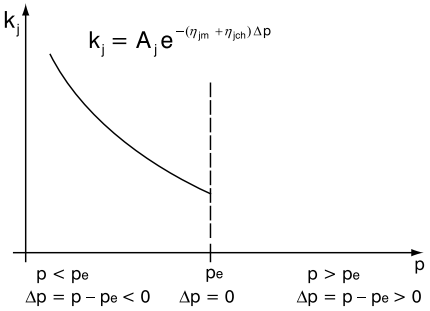
$$\eta_j = \eta_{jm} + \eta_{jch} << 0 \qquad (21)$$

$$\text{a } \eta_{jch} - \eta_{jm} >> 0 \qquad (22)$$

Vlastní průběh houstnutí v kostní tkáni při j-té biochemické reakci (j =1, 2, 3), iniciované objemovou změnou η_{jm} od primárních mechanických účinků, je opět predeterminován (řízen) změnami napětí $\Delta p = p - p_e$ (v elementu kostní tkáně).

3.2.1 Vliv záporných změn napětí

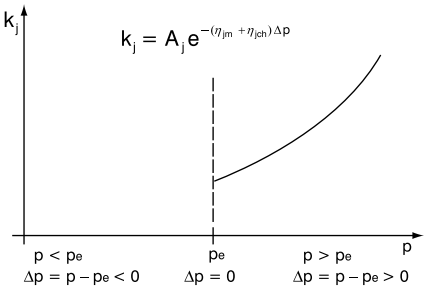
Jestliže $\Delta p < 0$, tj. $p < p_e$, potom dle vztahu (13) a (22) je rychlost $k_{jch} >> k_{jm}$, tedy k_{jch} zpomaluje k_{jm} a zároveň výsledná rychlost k_j (j-té biochemické reakce, pro j = 1, 2, 3) bude zpomalována. Důsledkem je skutečnost, že **houstnutí ve tkáni nastartované objemovými změnami η_{jm} od primárních mechanických účinků, bude při poklesu napětí p** (vzhledem k p_e, tj. při $\Delta p < 0$) **tlumeno.** Rychlost k_j výsledné biochemické reakce bude klesat dle (20) a (21), **obr. 5.**



Obr. 5 Průběh funkce výsledné rychlosti k_j biochemické reakce je úměrný exponenciální funkci $e^{-(\eta_{jch} + \eta_{jm})\Delta p}$. V tomto případě je funkce k_j klesající. Houstnutí v kostní tkáni bude při poklesu napětí p (vzhledem k p_e, tj. při $\Delta p < 0$) tlumeno.

3.2.2 Vliv kladných změn napětí

Jestliže $\Delta p > 0$, tj. $p > p_e$, potom dle vztahu (13) a (22) je rychlost $k_{jch} << k_{jm}$, tedy k_{jch} podstatně neovlivní k_{jm} a výsledná rychlost k_j (j-té biochemické reakce, pro j = 1, 2, 3) bude akcelerována. **Výsledná rychlost k_j houstnutí ve tkáni nastartovaného objemovými změnami $\eta_{jm} << 0$ od primárních mechanických účinků bude při růstu napětí p** (vzhledem k p_e, tj. při $\Delta p > 0$) **narůstat** (akcelerovat), dle vztahu (20) a (21). **obr. 6.**



Obr. 6. Průběh funkce výsledné rychlosti k_j biochemické reakce je úměrný exponenciální funkci $e^{-(\eta_{jch} + \eta_{jm})\Delta p}$. V tomto případě je **funkce** k_j rostoucí. Je zřejmé, že **nutnou podmínkou pro houstnutí ve tkáni je její zatěžování.**

4. ZÁVĚRY

4.1 Principy houstnutí v kosti

(a) Rychlost houstnutí ve tkáni při j-té biochemické reakci (pro j =1, 2, 3), iniciované objemovou změnou $\eta_{jch} << 0$, od primárních chemických účinků nebo $\eta_m << 0$, od primárních mechanických účinků je exponenciální funkcí změn napětí $\Delta p = p - p_e$ ve sledovaném elementu kostní tkáně.
(b) Houstnutí v kosti bude při poklesu napětí (zatížení) $\Delta p < 0$ retardováno (tlumeno), a to jak při iniciaci houstnutí objemovými změnami η_{jch} od primárních chemických účinků, tak i při iniciaci houstnutí objemovými změnami η_{jm} od primárních mechanických účinků.
(c) Při nárůstu napětí (zatížení) $\Delta p > 0$, tj. $p > p_e$ bude houstnutí v kosti akcelerováno, a to jak při iniciaci hhoustnutí objemovými změnami η_{jch} od primárních chemických účinků, tak i při iniciaci houstnutí objemovými změnami η_{jm} od primárních mechanických účinků.

AXIOM I.

(o retardaci houstnutí kosti):

Jestliže změny mechanického napětí ve tkáni jsou *záporné* ($\Delta p < 0$, tzn., že $p < p_e$), dochází k **retardaci houstnutí** ve tkáni.

AXIOM II.

(o akceleraci houstnutí kosti):

Jestliže změny mechanického napětí ve tkáni jsou *kladné* ($\Delta p > 0$, tzn., že $p > p_e$), dochází **k akceleraci houstnutí** ve tkáni.

4.2 Závěry pro klinickou praxi

Z předchozích analýz jsou zřejmé následující nejdůležitější závěry pro klinickou praxi:

I. Při nedostatečném zatěžování skeletu (tj. při minimalizaci nebo při absenci pohybu) bude vnější **aplikace chemických látek** (na příklad léků, hormonů atp.), jejichž cílem bylo iniciovat houst-

nutí tkáně, zcela **neúčinná.** Předpokládané houstnutí bude při nedostatku pohybu retardováno a tlumeno.

II. Při zatěžování skeletu (tj. při maximalizaci pohybu) bude aplikace chemických látek (na příklad léků, hormonů atp.), jejichž cílem bylo iniciovat houstnutí tkáně vysoce účinná. Předpokládané houstnutí bude při dostatečném pohybu akcelerováno.

III. Aplikace léků podporujících houstnutí kostní tkáně je účinná pouze v případě dynamického zatěžování (dynamického namáhání) skeletu (resp. sledované jeho lokality).

IV. Iniciující objemové změny od primárních chemických účinků (aplikace léků, hormonů atp.) a od primárních mechanických účinků jsou zastupitelné. Iniciaci houstnutí kostní tkáně chemickými látkami (léky, hormony) lze nahradit intenzivním dynamickým namáháním skeletu, resp. jejího elementu, a to aktivním pohybem.

V. Dynamické zatěžování kostní tkáně (jako nedílná složka harmonického pohybu) je nezastupitelným faktorem (iniciátorem a vlivem) podmiňujícím remodelaci kostní tkáně.

VI. Dynamickým zatěžováním lidského skeletu je zintenzívněn účinek chemických látek (léků, hormonů), potřebných pro tlumení řídnutí kostní tkáně nebo pro akceleraci jejího houstnutí. Změny mechanického namáhání kostní tkáně mohou, při nedostupnosti léků, tyto léky nahradit. Změny napětí ve tkáni, primárně iniciované pohybem (resp. přetvořením tkáně) mohou druhotně vyvolat vznik chemických látek, které přispívají k retardaci řídnutí kosti.

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ZKOUMÁNÍ TAHOVÉ SÍLY PEDIKULÁRNÍHO ŠROUBU

INVESTIGATION OF PEDICLE SCREW PULL-OUT STRENGTH

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SUMMARY

This study was presented problems with usage of transpedicular spine fixators. The purpose of this work was defined the value of pull-out strength of transpedicular screw from vertebrae. Impact of geometrical parameters of transpedicular screw on value of pull-out strength was analysed. Investigation was carried out on cadaver of lumbar and thoracic vertebrae. The results of investigation showed significance of geometrical parameters of transpedicular screws on durability of screw-vertebrae connection. This study proved also that very important for good performance of the vertebrae and pedicle screw unit is adequate match the screw diameter up with pedicle diameter.

Key words: experimental investigation, transpedicular screw, pull-out test

INTRODUCTION

Transpedicular screws stabilisation is commonly used in the treatment of pathological changes and injuries of spine. Regardless of many years of experience and the knowledge of surgeon teams, the insertion of implants to the spine initiates numerous problems of clinical and biomechanical nature. The complications may arise during the implantation, or in the post operation period. Complications which emerge during the exploitation of stabilisers comprises among others the following: bending or breaking screws, pulling out a screw out of the vertebra, breaking the pediculum, loosening screws at the vertebral pedicle and "ploughing-in" of the screw end in the shaft often accompanied by loosening at the vertebral pedicle (1). Such changes lead to secondary destabilisation and losing spine correction.

Although problems connected with transpendicular stabilisation are numerous, this method still has advantage over other stabilisation techniques. This is the effect of much more beneficial biomechanical conditions of screw anchoring, due to this the achieved stabilisation is shorter (even two-segmental) and more reliable.

The research on the explanation of at least some problems appearing after the implantation of transpedicular stabilisers may turn to be extremely important for the development of the operation technique and the development direction of new transpedicular screws solutions as well as whole stabilisation systems (2).

The research on the parameters which influence the durability of screw-spine connection in transpedicular stabilisation are conducted in way taking into account numerous aspects. Most often the research concerns the definition of force which causes the destabilisation of a screw in the vertebra. There tests which examine maximum force which can lead to pulling out of the screw from bone (they often depend on the way the screw was inserted) (3, 4, 5, 6), or they define the conditions in which the cohesion of the connection is violated in bending attempt (1, 7). The research is carried out for single screws as well as whole stabilisation systems. Various geometric and mechanical parameters of screws are analysed as well as their influence on the obtained values of the pull out force (4, 8).

Regardless, however, of a variety of research there are no analyses which would search for a correlation between the very vertebra pediculum – its geometry (which is one of the most important elements in screw-bone connection durability) and the geometry of the inserted screw in relation to the force which causes pulling out of the screw from bone.

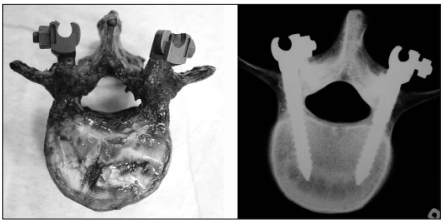
Defining the correlation between screw geometry and vertebra pediculum could make a good "tool" for fast and appropriate screw selection when planning and performing the operation in which transpedicular stabilisation is applied.

In this work experimental research was conducted, the purpose of the research was defining the dependence between selected geometric characteristics of transpedicular screws, vertebra pediculum and the value of force pull-out a screw from a vertebra.

MATERIAL AND METHOD

Materials. The research was conducted on vertebrae section preparation of human spine vertebrae. The analysis was carried out on 20 vertebrae, where 11 vertebrae came from thoracic part of the spine, and 9 vertebrae came from the lumbar part. Trying to obtain repeatable measurements conditions, the vertebrae used in the research came from spines of individuals of similar physical characteristics (average age – 29 years). The quality of bone tissue was additionally assessed in a similar way on the basis of Rtg pictures by densitometry method (2).

The researched material was divided into single vertebrae which were next disseted free of soft tissues. Muscles, ligaments and elements of intervertebral disc were removed. The individual vertebrae were kept in double-bagged at the temperature of –20 °C. Before testing the specimens were thawed at room temperature. Transpedicular screws were implanted in prepared vertebrae **(Fig. 1a).** The implantation was carried out by a physician – a specialist – in accordance with procedures binding for particular screw types.



a) b)

Fig.1. Picture of vertebra with transpedicular screw – a), inferior-superior of a specimen after screw placement – b).

Roentgenographic analysis was conducted for all vertebrae (Rtg pictures – **Fig. 1b**) fore the insertion of transpedicular screws (to eliminate degeneration and pathological changes) and after the screws settlement (to determine implant insertion correctness). On the basis of Rtg pictures the screw diameter ratio to pediculum diameter was determined (d_s/d_p) – **Fig. 2.**

On the basis of Rtg pictures the radiologic density of vertebrae bone tissue was estimated. This parameter created the basis for selection of preparations representing similar density.

Screw Placement. The research was conducted on 7 designs of transpedicular screws differing in geometry and material they were made of.

Screw designation	Length of screw L [mm]	Diameter d_s [mm]	Screw type	Material
W1	40	6,6	cylindrical	austenitic steel
W2	30	4,8	cylindrical	austenitic steel
W3	20	4,8	cylindrical	austenitic steel
W4	20	6,6	cylindrical	austenitic steel
W5	40	6,6	cylindrical	titanium alloy
S1	40	7,6/4,5	conical	titanium alloy
S2	30	5,5/4,0	conical	titanium alloy

Table 1. Parameters of analysed screws

The presented work analyse the influence of the following parameters on connected with transpedicular screws geometry on the value of pull out force:

- Length of the threaded part of transpedicular screw L – **Fig. 2,**
- Shape of screw crew,
- Screw diameter to pediculum diameter ratio d_s/d_p – **Fig. 2.**

Table 1 presents the geometric parameters of analysed transpedicular screws.

Mechanical Testing – Axial Pull-out. Vertebrae with implanted transpedicular screws were settled in a set up specially designed for this purpose. The set up construction assured the consistency of the direction of the determined load with the direction of tarnspedicular screw axis – **Fig. 3.** The research was performed using testing machine (MTS MiniBionix 858).

Pull-out test consisted in loading the implanted transpedicular screw with force acting in the direction consistent with the screw axis. Load velocity made 50 N/s. In all cases the test was continued until the screw was pulled out of the vertebra.

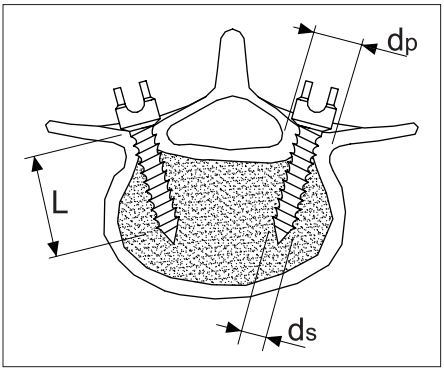


Fig. 2. Schema of inserting screw in vertebra and geometrical parameters.

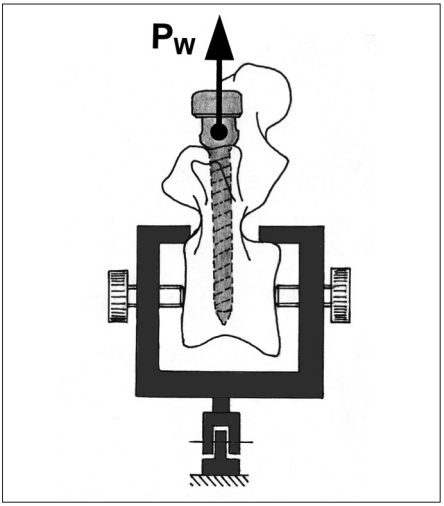


Fig. 3. Schema of loading system.

Screw designation	Among of test N	Average value of pull-out force PW [N] (SD [N])	Average value of ratio d_s/d_p (SD)
W1	6	1218 (192,3)	0,78 (0,11)
W2	6	944 (228,8)	0,71 (0,15)
W3	6	619 (146,8)	0,69 (0,04)
W4	5	762 (233,6)	0,76 (0,08)
W5	6	1487 (128,9)	0,80 (0,03)
S1	5	1565 (181,3)	0,79 (0,06)
S2	6	1365 (230,4)	0,76 (0,09)

Table 2. Statement of average values of pull-out strength PW and ratio d_s/d_p for analysed transpedicular screws

RESULTS AND DISCUSSION

On the basis of the conducted tests, force change course of the force acting on a screw in the function of its displacement was obtained for each of the tested screws. The pulling out force PW was defined as maximum force value registered during the test. **Table 2** presents average pulling out force values for the tested transpedicular screws. The graphic illustration of average values of the pulling out force is shown in **Fig. 4.** The highest value of pulling out force 1565N (SD 181,3N) was obtained in the case of S1 screw, while the lowest values 619 N (SD 146,8N) for W3 screw.

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The performed research showed that the length of the threaded part of the screw allows to obtain material growth in pulling out force value. Comparing average force values for screws W2 and W3 differing in the length of the threaded part, we can claim that the increase of this parameter by $^1/_3$ results in the increase of pulling out force value by 34,4 %. A similar tendency was observed in the comparison of screws W1 and W4. In this case the increase of the threaded part by $^1/_2$ generates the increase of average force value P_W by 37,4 %.

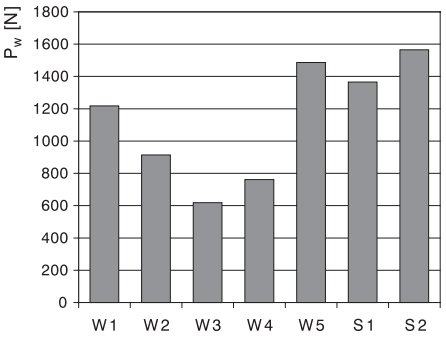

Fig. 4. Average value of pull-out force for investigated screws

The value of the nominal diameter of the thread is connected with another parameter which was also analysed during the research. This parameter is the ratio of the nominal diameter of the transpedicular screw thread to pediculum diameter d_s/d_p. Chart 2 gives average values for d_s/d_p determined for the examined cases and **Fig. 5** makes a graphic presentation of the parameter value.

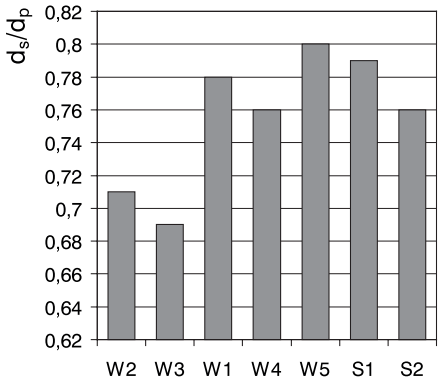

Fig. 5. Average value of ratio dS/dP for investigated screws

Analysing the obtained results one can come to a conclusion that the values of d_s/d_p are lower for screws of d_s = 4,8 mm diameter, i.e. W2 and W3. Analogous values of screw diameters d_s = 6,6 mm (W1, W4, W5) are higher by average of 10,3 %. One can also observe that there is a dependency between the value of d_s/d_p ratio and the value of pulling out force P_W. With the increase of the value of d_s/d_p ratio, the value of pulling out force P_W grows and it is a linear dependency, the matching coefficient makes R_2 = 0,89 **(Fig. 6).** As a result we can conclude that the appropriate selection of nominal diameter of transpedicular screw to the size of pediculum is vital for the durability of the connection between the screw and the vertebra. This is connected with the fact that 90-95 % of the force pulling out the screw settled in a vertebra is transferred by pediculum (9). The word „appropriately" has been used here on purpose as the increase of d_s/d_p coefficient value above 0,8 may result in the decrease of pulling out force, which is the effect of excessive weakening of pediculum in compact bone tissue. This may also lead to micro-cracks of pediculum at the screw implantation stage (10), as a result during the exploitation of the stabiliser leads to system destabilisation. During the research, the results of which are presented in this work in three cases (1 in the case of screw W5 and 2 in the case of screw S1), the effect of pediculum lengthwise crack (parallel to the screw axis) was registered during an attempt of pulling out. It is worth mentioning that the average values of d_s/d_p for these screws reached the highest values: 0,80 and 0,79 respectively. To be able to exactly predict the value of d_s/d_p ratio of the pediculum before it generates a crack it is necessary to conduct extra research of this question.

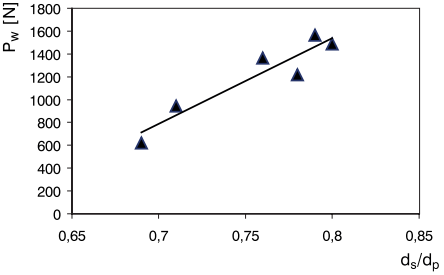

Fig. 6. The changes in pull-out strength value in relation to dS/dP ratio

The last parameter analysed in the research was the shape of a screw core. The obtained results show that for a screw with a conic core (case S1) the average pulling out force value is 22,1 % higher than in the case of a screw with a cylindrical core (case W1). In the case of shorter screws, of 30 mm length (S2,W2) the difference of the value of pulling out force makes 30 % to the benefit of the screw with a conic core. Higher values of pulling out force for screws with conic core have several reasons. The first one is the above discussed question of d_s/d_p ratio. Conic screws are characterised by substantial diameter d_s in the part co-operating with pediculum. In the case of screw S1 d_s = 7,6 mm, for comparison in the case of screw W1 d_s = 6,6 mm (**table 1**). The second reason is connected with a parameter we have not mentioned so far, namely the pitch of thread. For all tested cylindrical screws the pitch of thread made 3 mm, while for conic screws the pitch of thread was lower and made 2 mm. We need to emphasise here that generally in the offered conic screws the pitch of the screw of the majority of them makes 2mm and is characterised by a different profile. Certainly a lower value of the pitch of the thread results in the increase of the area of screw action on the vertebra

structure, in effect the stresses in bone tissue decrease (10). This makes it possible to transfer force of higher value.

CONCLUSIONS

The conducted research indicate that the parameters characterising transpedicular screw geometry, i.e. threaded part length, nominal diameter of the screw thread and the shape of screw thread have essential influence on the value of the force pulling them out of vertebrae. Defining the durability of transpedicular screw connection with a vertebra makes it possible to select appropriate mechanical parameters (rigidity in particular) and other elements which create the construction of transpedicular stabiliser. Taking into consideration the values of force which cause pull out of the screw, the constructor may select the rigidity of other construction elements of the stabiliser in such a way so as not to exceed these forces in exploitation conditions, i.e. everyday activity.

The presented research analyses only the reaction of a few selected parameters. When the assumed programme for the research was performed new suggestions concerning other parameters arose, their influence on the value of the force pulling out a transpedicular screw of the vertebra is interesting and it has not been analysed so far. One of such questions is the definition of optimum value for d_s/d_p ratio to assure the highest ability to transfer the axis force and not to generate pediculum cracking simultaneously. Another issue is answering the question of the influence of chemical properties (composition) and mechanical properties of the material and in particular the microstructure of surface layer which is characteristic of a given material and of the technology of its manufacturing on the durability of the connection between a screw and bone.

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ÚČINNOST KOLAGENNÍCH PEPTIDŮ VS. DICLOFENAC U KOSTNÍ FORMY OSTEOARTHRITIDY

COMPARISON OF THE EFFICACY THREE DIFFERENT THERAPY KINDS IN OSTEOARTHRITIS

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SUMMARY

This is a double-blind randomized, parallel group study. Patients with either knee and/or hip osteoarthritis were supplemented with one of three therapies for 12 weeks: pharmaceutical grade of collagen hydrolysate (PCH)(10 g/day), diclofenac sodium (DCF) (75 mg/day), or the combination of PCH and DCF. The primary efficacy was the Lequesne's Index. The study was completed with 15, 16, and 13 patients in the three treatment groups. All patient suffered from bone form of osteoartrhritis (OA) 3rd or 4th stage according to the nomenclature of Kellgren and Lawrence.

Lequesne's Index showed some improvement during the therapy, which however, for the DCF monotreatment disappeared until the follow up. Based on Index score there were no recognizable disadvantages of PCH to DCF. On the other hand, early responses were noted in 85 % of patients under DCF monotreatment and in case for any on the patients under PCH monotreatment. Urinary pyridinolines and bone alkaline phosphatase did not exhibit any recognizable trends. At the end of the three month treatment phase, the chondrex score showed a definite improvement in PCH group.

Key words: osteoarthritis, pharmaceutical grade collagen hydrolysate,m diclofenc, pyridinoline, chondrex, bone alkaline phosphatase

INTRODUCTION

In the last decades interest is paid to nutritional supplements (Nutraceuticals) especially to collagen, both as symptomatic relieving agents, and agents which may have a specific effect on disease pathophysiology and pathologic structural changes. Hydrolyzed gelatine derivatives, which are manufactured from bovine or porcine hide or bones, have been used over many years. Oesser et al. using radioactively labelled collagen peptides showed their passing through gastrontestinal wall (**12**). Clinical studies with these derivatives showed efficacious symptomatic effect in osteoarthritis (**1**). Pharmaceutical grade collagen hydrolysate (PCH) is a soluble powder obtained using an enzymatic digestion with a food aproved enzyme. The everage molecular mass ranges from 500 to 6000. In our previous studies we did not observed any side effects (**1, 3, 4**). Acute, subacute, teratogenic and mutagenic testing of gelatin hydrolysates have not revealed any health risk.

The aim of this study was to objectify the therapeutic effect of PCH over a three months treatment in the bone form of osteoarthritis (BOA) by means of clinical outcome criterion (Lequesne's Index) as well as specialised laboratory tests – urinary pyridinoline, urinary deoxypyridinoline (UPD, UDPD), serum bone alkaline phosphatase (BAP), and serum chondrex and to compare its the therapeutical effect with that of diclofenac.

STUDY DESIGN

The study was randomized, double-blind clinical trial comparing the efficacy and tolerability of oral treatment with PCH versus diclophenac in three age-matched groups of patients.

Patients: A total of 46 patients of both sexes (25 females, 19 males) suffering from OA bone form (radiologic severity Kellgren-Lawrence (**7**), grade 3 or 4) of hip or knee joints were enrolled in this study. Patients suffering from neoplasia, bone metabolic diseases and/or other metabolic diseases, secondary OA of any kind, inflammatory joint disease, intraarticular corticosteroids in the preceding six months, genu valgum or varum exceeding 8^0, any SYSADOA in the preceding 6 months were excluded. Only two drop-outs (both on DCF) occured during the study due to cause not related to the therapy administered. No patient dropped out of the study because of side-effects, pain increasing, inefficacy, and/or compliance.

Radiological evaluations were performed on anteroposterior X-rays in a weight-bearing monopodal position upon entry.

Demographic and baseline characteristics (Tabs. 1 and 2). A comparison of the demographic data upon entry showed only negligible differences under studied groups. Patients groups are comparable with respect to demographic characteristics and also with respect to disease characteristics except for a slight underrepre-

	Age [years]	**Height** [cm]	**Weight** [kg]
PCH	66.27±8.13	170.93±7.44	82.27±12.06
DCF	67.52±8.05	164.16±6.17	75.15±9.32
PCH+DCF	66.50±7.72	166.56±6.99	76.75±9.82

Tab. 1. Demographic Characteristics

sentation of patients with gonarthrosis in PCH group and an overrepresentation of patients with more than 12 month of previous therapy in DCF group (**Tab. 3**).

Medication. Patients were randomly allocated to either pharmaceutical collagen hydrolysate (PCH) (10 g a day) – 15 patients, or to diclofenac (DFC) (75 mg a day) – 13 patients, or to the combination of both the substances i.e. PCH (10 g a day) and DFC (75 mg a day) – 16 patients. PCH was product of DGF Stoess, Eberbach, Germany. Double-blind treatment was carried out for 12 weeks from visit 2 to visit 5 with one sachet containing either from the three tested medications daily for 12 months and followed by an eight week post-treatment washout.

Treatment efficacy assessment occured at screening, baseline (visit 1), at weeks 4 (visit 2), weeks 8 (visit 3), 12 (visit 4), followed by assessments at week 20 (visit 5) representing post-treatment follow-up. The efficacy of treatment was evaluated according to the functional status of the patients in their daily lives using *Lequesne's Index* in the three point scale (none, moderate, severe) and according to specialised biochemical tests (**8, 9**). *Pyridinoline (UPD) and deoxypyridinoline (UDPD)* were measured in the fasting urine by HPLC, *bone alkaline phosphatase (BAP)* in serum by ELISA, and the serum levels of *chondrex (YLK40)* also by ELISA (**Tab. 4**). The normal values see in **Tab. 5.**

Specialised biochemical test: both the pyridinolines were determined in fasting urine after hydrolysis and microgranular cellulose CC31 prefractionation and fluorescence detection after strong cation HPLC (**13**). Serum bone alkaline and chondrex were measured using antibodies Metra (CA,USA) by ELISA.

Paracetamol consumption: during the 16 months study patients were allowed to take paracetamol as rescue medication. Patients with PCH consumpted paracetamol exceptional only, those treated with DCF did not used it at all.

	sex		**Disease Duration**				**Previous therapy**			
	F	M	less than 1 year	1–2 years	2–5 years	more than 5 years	none	in last 6 months	6–12 months ago	13 and more m. ago
Substance	N	N	N	N	N	N	N	N	N	N
PCH	15	10	0	1	3	11	7	5	0	3
PCH+DCF	13	3	1	2	1	12	5	1	5	5
DCF	7	6	0	0	2	11	2	0	1	10

Tab. 2. Baseline Characteristics

	Total	**OA of knee joints**	**OA of hip joints**
Substance	N	N	A
PCH	15	5	11
PCH+DCF	16	9	9
DCF	13	9	6

Tab. 3. Frequency Distribution of OA

		Visit 1	Visit 2	Visit 3
Chondrex	PCH	131.72±75.78	94.23±51.56	125.01±77.68
	PCH+DCP	87.44±71.03	71.79±65.00	88.13±70.47
	DCF	112.66±66.58	96.10±61.55	100.40±51.29
UPD	PCH	42.21±14.6	134.83±11.13	32.79±12.51
	PCH+DCP	43.33±16.01	40.11±12.20	51.99±12.40
	DCF	50.13±35.82	39.38±17.09	40.92±14.37
UPDP	PCH	8.25±2.85	6.17±2.89	6.38±2.52
	PCH+DCP	7.56±3.21	5.98±2.71	9.83±3.17
	DCF	10.32±7.29	7.53±3.65	8.12±3.48
BAP	PCH	19.75±7.36	20.43±3.65	21.11±10.45
	PCH+DCP	22.38±7.16	21.41±6.44	21.23±6.16
	DCF	19.98±5.04	21.39±4.21	20.05±6.20

PHC – Pharmaceutical Grade Collagen Hydrolysate
DCF – Diclofenac

Treatment:
Collagen Peptides 10g/day
Collagen Peptides 10g/day + Diclofenac 75 mg/day
Diclofenac 75mg/day

Tab. 4. Specialized Biochemical Tests (Means and Standard Deviations)

UPD (n = 26)	41.6 ± 10.6 (nmol/mmol cr.)
UPDP (n = 26)	8.1 ± 2.8 (nmol/mmol cr.)
BAP (n = 10)	12.2 ± 2.7 (U/L)
Chondrex (n = 39)	43.2 ± 14.5 (ng/ml)

UPD – urinary pyridinoline
UDPD – urinary deoxypyridinoline
BAP - serum bone alkaline phosphatase

Tab. 5. Control values

	total	**Doctors' Evaluation**			**Patients' Evaluation**		
		poor	moderate	very good	poor	moderate	very good
	N	N	N	N	A	N	A
PCH	15	0	8	7	0	7	8
PCH+DCF	16	1	6	9	1	5	10
DCF	13	1	8	4	0	6	7

Tab. 6. Doctors' and Patients' Evaluation at the End of Therapy

Statistical evaluation of the data was performed by an independent AAI Deutschland GmbH, Neu Ulm, Germany in compliance with Good Statistical Practices

RESULTS

Whereas treatment groups showed definite differences with respect to the frequency of early reaction according to the Lequesne's Index (none in PCH group, 5 in the group combined treatment, and 11 in DCF monotreatment), they were not relevantly different with respect to the doctor's and patient's evaluation (**Tab. 6**).

Lequesne's Index scores are showed in **Fig. 1.** All the three treatments caused a decrease in this score after one month therapy, with a far greater decrease observed in the DCF group (to 29.74 % of the baseline value). However, in the PCH group the score droppped down to 66.42 % of baseline score only. In both the groups treated with PCH scores improved definitely during the trial. The scores in the follow-up examination (visit 5) increased for the DCF monotreatment, where they reached values close by those obtained at visit 1 (16.31 versus 15.31)

With a fairly large spread of results under patients, there were no clearly recognizable trends for changes in most of specialized biochemical tests. There was a marginal tendency for an increase in BAP during the treatment periods. **Tab. 4** sum-

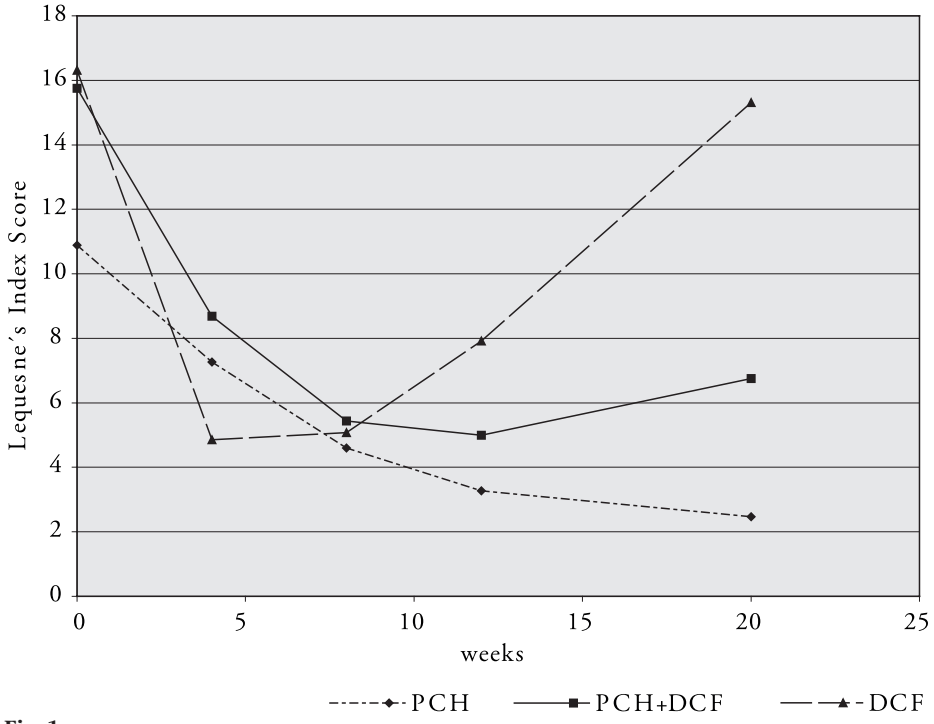

Fig. 1.

marizes statistical results. At the end of the three months treatment phase (visit 4), the chondrex main values showed a definite improvement PCH group but not for the other treatment. UPD and UDPD detected an down regulation mainly in PCH and DCF group, and two month later (visit 5) this tendency still was recognizable.

DISCUSSION

OA are overlaping disorders in which articular cartilage and subchondral bone are disturbed and synovial membrane often infammed. From the etiopathogenetical point of view OA of hip joint seems to be different from that of knee joint. It cannot be excluded that some

immune processes may play an important role especially in the etiopathogenesis of coxarthrosis. We namely found in patients with OA high collagen type I, II, III antibodies titers OA resembling those in rheumatoid arthritis (**11**).

It is necessary to stress that the presented trial was performed on patients with bone form (grade 3 or 4) OA , i.e with bone deterioration, which is connected with new tissue formation, characterized by the presence of collagen type III (**2**). In such a newly formed tissue activity of metaloproteinases is according to our findings upregulated and breakdown of newly formed collagen may by also a source for urinary pyridinolines – cross links of bone and cartilage collagen. When evaluating UPD, UDPD, and chondrex values it should be kept in mind of what processes they are markers. UPD as well as UDPD are crosslinks of bone and cartilage collagen and they are hardly present in other tissues, Moreover, after breakdown of collagen structure pyridinolines are not further metabolized. Therefore their urinary concentration is a good marker of collagen breakdown. Because bone collagen represents about 50 % of body collagen and is permanently metabolized both these pyridinolines are mainly bone derived. According to the presented results all three types of treatment suppressed collagen breakdown in the joint compartment.

Chondrex, what is a chitinase according its primary stru cture but posses a proteolytic activity, is synthesised by chondrocytes and synoviocytes both the cells of joint compartment, but also by neutrophiles and macrophages. This may be an explanation for its rapid downregulation in corticosteroid therapy of RA. From this reason it is reflexing not only procedures in joint compartment and its real significance must be elucidated (**6**). On the other hand BAP is a marker of bone formation (**14**). An increase of its level after PCH treatment may show its positive effect on bone formation.

The effect of chondroitin sulfate in OA which was similar to that presented in this paper with PCH published Morreale et al. (**10**). Both the substances diminished patients troubles connected with OA. The effect of PCH like that of chondroitin sulfate similarly appeared later on but lasted up to two months after the end of treatment. Contrariwise DCF caused prompt and plain reduction of clinical symptoms, which reapeared after the end of treatment.

Big advantage of PCH treatment is its very good tolerability. In contrast to DCF it does not present problems of tolerability, especially at the gastrointestinal level. Therefore DCF should not be used for prolonged treatment especially in the elderly. Our experience on many patients treated

with PCH (with postmenopausal osteoporosis) for three years (Adam et al., in preparation) showed that it is not causing any gastrointestinal troubles. This is certainly an advantage of PCH to substances of NSAID group, which are known to cause gastric ulcers. They are namely potent inhibitors of protein synthesis and are in this way harmful in respect to gastric mucous membrane. Bečvář et al. showed in cultures of chondrocytes and fibroblasts an inhibitory effect of phenylbutazone and naproxen on their proliferation (**5**). When glycosaminoglycan-peptides complexes were added to the culture medium the inhibition of proliferation and also of synthetic activity of cultured cells by naproxen was blocked. Treatment of OA with PCH according to our results is effective and without dangerous complications and it is also not harmful on cells of joint compartment. Naproxen was blocked. According to our results such a counteraction to DCF on cell proliferation and turnover posses also collagen peptides.

According to the mentioned effect PCH may be classified as SYSADOA (symptomatic slow-acting drugs for the treatment of OA) (**8**). Nowaday good evidence is available that SYSADOA are valuable for OA treatment. Also in this study PCH showed good tolerance and no substantial adverse event was assesed as related to the study medication. Treatment of OA with PCH according to our results is effective and without dangerous complications and it is also not harmful on cells of joint compartment.

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OSTEOLOGIE 2003, GÖTTINGEN, 26. – 29. MÄRZ 2003

KUKLÍK M.

Genetická ambulance, Ambulantní centrum pro vady pohybového aparátu v Praze 3, Olšanská 7
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Ve dnech 26 – 29. 3. 2003 se, konal sjezd německých osteologických společností, tzv. Gemeinschaftsveranstaltung der CRHUKS – DGO – OGEKM – OGO za spolupráce dalších členů a hostů v Göttingenu. Kongres byl velmi dobře organizován a sponzorován řadou význačných farmakologických společností, zúčastnila se jej též celá řada svépomocných organizací pacientů s chorobami pohybového aparátu.

Vzhledem k tomu, že kongres proběhl za nezájmu českých odborných a osteologických společností, rádi bychom upozornili na tento kongres vybranými abstrakty.

Uvedený výběr je nutně neúplný a subjektivní. Vzhledem k rozsahu sympozia a počtu referátů v něm přednesených je nemožné postihnout reprezentativní souhrny referátů. Nedílnou součástí sympozia je účast svépomocných skupin pacientů a prezentace nabídky farmaceutických a zdravotnických firem. U nepublikovaných referátů anebo přednesených v němčině je uveden překlad v češtině.

SENILNÍ OSTEOPORÓZA

J. D. Ringe, Leverkusen

Osteoporóza není pouze čistě ženská choroba. 15 až 20 % případů je u mužů. Osteoporóza starých lidí, tzv. senilní osteoporóza, není ostře vymezena od tzv. postmenopauzální osteoporózy, popř. primární osteoporózy u mužů. Senilní osteoporóza má však důležité patogenetické a diagnostické zvláštnosti, které je nutno zohlednit. Od 70 let by se mělo podávat z důvodu substituce 1000 mg kalcia a 1000 mezinárodních jednotek vitaminu E.

ZMĚNY V LÉČBĚ OSTEOPORÓZY – osteoporotická péče, srovnání Německa a ostatního světa

F. Jakob, Würzburg

V Německu je postiženo osteoporózou 4 – 6 milionů lidí, každá 3 postmenopauzální žena a každý pátý muž nad 50 let onemocní osteoporózou. Onemocnění se rozvíjí razantně, exponenciální řadou a lze je hodnotit podle vertebrálních kompresivních fraktur. Za rok vznikne v Německu 200 000 fraktur obratlů, tzn. každé 2,5 minuty nová fraktura. To představuje nepřehlédnutelnou socioekonomickou zátěž. Pacienti jsou postiženi další zvýšenou morbiditou, zhoršenou kvalitou života a vyšší úmrtností.

Základní péče o pacienty je zajištěna kalciem a vitaminem D, v dubnu 2002 tomu tak bylo u většiny pacientů. Slabší polovina pacientů nad 55 let je léčena bisfosfonáty, malá část pouze fluoridy, vitaminem D, preparáty řady SERM a vyjímečně kalcitoninem. Ve Velké Británii je podíl bisfosfonátů v léčbě nesrovnatelně vyšší, jen malá část pacientů – asi osmina je léčena kombinací vit. D a kalcia, nepatrná část preparáty SERM.

Mírná převaha bisfosfonátů je v léčbě osteoporózy ve Francii. Následuje pak kombinace kalcia a vitaminu D, preparáty řady SERM a kalcitonin jsou jen vyjímečně používány. Více než 50 % nemocných s osteoporózou nejsou diagnosticky rozpoznáni a z těchto je zase pouze polovina léčena. V celku pak 77 % pacientů není léčeno.

Nejčastější a nejčasnější manifestací jsou fraktury obratlů.

Ženy s radiologicky zjištěnými frakturami obratlů mají 5× větší riziko nové fraktury, nezávisle na denzitometrické tloušťce kosti. Kvalita života se výrazně mění s přibývajícím počtem fraktur obratlů k horšímu. Nejzávažnější frakturou působenou osteoporózou je fraktura kyčelního kloubu. Za 12 měsíců po této fraktuře umírá 20 % pacientů, dalších 22 % musí zůstat v pečovatelské službě a pouze 24 % pacientů dosáhne předcházející mobility jako před úrazem.

OSTEOPORÓZA

F. Raue, Heidelberg

Osteoporóza nepřešla ještě zcela do povědomí lékařů, např. ve srovnání s diabetem mellitem nebo hypertenzí. V Německu se počítá s 4 – 6 miliony nemocných, tj. 30 % postmenopausálních žen a 20 % mužů nad 50 let. Netýká se to jen důchodců, ale i aktivně pracujících lidí.

V Evropě jsou prováděny studie nově vznikajících fraktur obratlů. Od 50 do 79 let včetně to znamená přes 160 zlomenin za hodinu, tj. 3 fraktury za minutu.

Jak ukazují studie z roku 2001, u žen které mají frakturu obratle, lze očekávat u 20-25 % další frakturu i přes léčbu vitaminem D a kalciem.

Zůstává otázkou, proč je ze skutečného počtu nemocných diagnostikována jen polovina, chybí snad symptomatologie? Zlomeniny obratlů zůstávají ve dvou třetinách nerozpoznány. Na možnost osteoporózy ukazuje především snížení výšky těl obratlů a tělesné výšky o více než 4 cm. Pouze 18 % pacientů, u kterých byla diagnostikována osteoporóza je léčeno! Ne vždy rentgenolog ve své zprávě (popisu) frakturu uvádí, ne vždy je uvedena v lékařské zprávě, jejíž součástí je rentgenologický nález.

Osteoporózu lze léčit rychle, není to žádný osudový chronický proces stáří.

OSTEOPOROSE IST NICHT NUR NIEDRIGE KNOCHENDICHTE

M. A. Dambacher et al.,Curych

Studium postmenopauzálních žen s čerstvými frakturami obratlů a průběh jejich léčby byl studován s využitím třídimenzionální techniky, zobrazující mikroarchitekturu kostí. V 80. letech se mylně předpokládalo, že tloušťka kortikalis a denzita kosti jsou jediným kriteriem úspěšnosti léčby. Tomu však odporovaly některé studie s fluoridy, které naopak při doplnění těchto kriterií ukazovaly zvýšenou fragilitu kostí. S využitím vysoce rozlišující, účinné computerové tomografie lze nyní sledovat v bioptických vzorcích mikroarchitekturu

skeletu a lze očekávat, že tato metoda bude v nejbližších letech zavedena do praxe. Metoda také přispívá k objasnění antiresorptivního mechanismu Risendronátu.

Metoda umožňuje stanovit pevnost kosti na základě pomocných parametrů jako je kostní kvantita a kostní kvalita.

DIE ROLLE DES VITAMIN D FÜR DIE MUSKELFUNKTION

A. Lorani, Konstanz

Vitamin D deficiency is common among older people. 57 % subjects of an unselected hospitalized group showed a relative vitamin D deficiency, 22 % had a clear deficiency, and even 80 % of beddridden older people had a clear vitamin D – deficiency. Vitamin D deficiency may by associated to muscle weakness 25 OH D3 under 20 mmol/l with an increased risk of falls and bone fractures. Receptors for 1,25 /OH/2 D3 have been deceted in human muscle tissue. Growing evidence suggest an interaction between intracellular vitamin D receptors and muscle function. Studies have shown dependency between muscle strength, coordination and daily activities. Bone mineral density and reduced muscle strength are predictors for an elevated risk of falls and falls-related fractures. However, it is still controversial discussed, whether reduced muscle strength increases disability in older subjects, which may be improved by vitamin D supplementation in vitamin D – deficient subjects.

Key words: – vitamin D deficency – muscle strength – vitamin D receptor – risk for falls – vitamin D-supplementation

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MUSKELSCHMERZEN BEI METABOLISCHEN MYOPATHIEN

J. Schafer, H. Reichmann, Dresden

Muscle pain may be caused by intrinsic muscular factors including inflammatory, mechanical, ischaemic, metabolic and membrane disorders or may be secondary to peripheral (neuropathy) or central nervous system disease. Primary metabolic myopathies are generally disorders of energy metabolism. Typically, they led to exercise induced myalgias which are almost always associated with some degree of muscle weakness – in contrast to the non specific exertional muscle pain syndromes which do not present with signifikant weakness, if followed by rhabdomyolysis, exercise induced muscle pain is likely to be caused by a metabolic myopathy and warrants further investigation. Muscle pain in metabolic myopathies has been attributed to a number of chemical mediators (inosinmonophosphate, adenosin, acidosis, biogenic amines and lactate), but there is not definite proof for the involvement of any of these substances in the development of metabolic muscle pain. Common metabolic myopathies leading to muscle pain are defects of glycogen and glucose metabolism, defects of muscular fatty acid oxidation, defects of mitochondrial oxidative phosphorylation and defects of the purine nucleotide cycle (myoadenylate deaminase deficiency). Besides non-specific measures (physiotherapy, non steroidal analgesics, chinine, L carnitine) some metabolic myopathies can be treated succesfully with the appropriate diet and dietary supplements. The most important aspects for the prevention of muscle pains is however, adequate and selective physical exercise.

NEW DATA ABOUT BONE STRUCTURES (MICROARCHITECTURE) IN VIVO AND IN VITRO IN ANIMALS AND IN HUMANS

M. A. Dambacher et al., Zurich

Osteoporosis is a skeletal disease characterized by low bone mass and a deteriored microarchitecture of bone which increases the fragility of the bone and increases the risk of fracture (Consensus Development Konference, JAMA 2001, 285, 785-795). The evaluation of 3D bone structures in vivo and in vitro in animals and in humans, give new insights into the pathogenesis and treatment of osteoporossis. The increase of bone density is not parallel to the reduction of fracture risc. The stability of bone depends not only on bone mass density, but on microarchitecture too (bone quality). The evaluation of 3D bone structures in vivo and in vitro in animals and in humans, give new insights into the pathogenesis and treatment of osteoporosis.

GENOME WIDE EXPRESSION ANALYSIS DURING LATE OSTEOBLAST DIFFERENTIATION

Schinke, T. et al., Hamburg

It is well documented that bone remodeling relies on the balanced activity of osteoblasts and osteclasts. In contrast, the function of osteocytes, representing the most abudant cell type in bone, is still unclear. Osteocytes are terminally differentiated osteoblasts that are completely surrounded by mineralised matrix. Their stellate – shaped morphology and the existency of interconnections between them have raised the hypothesis that the osteocytes play crucial roles in mechanotransduction and thereby bone remodeling. To date only few genes have been found which are specifically expressed in terminaly differentiated osteoblasts or osteocytes. In an attempt to identify more of such genes we performed a genome-wide expression analysis during late stages of osteoblasts differentiation using Micro-Array technology.

Primary osteoblasts cultures were isolated from newborn mouse calvariae and mineralised ex vivo using ascorbic acid and beta glycerophosphate. cDNA was preparated from these cultures after 5 and 25 days and used for the generation of biotinyled cRNA probes. Both probes were used to hybridise Affymetrix 35K chips according to the manufacters protocol. The analysis of the hybridisation patterns was performed using the Affymetrix GENECHIP software. The expression patterns of relevant genes was subsequently determined by Reverse Transcriptase PCR.

From 30 000 genes present on Affymetrix chips we found 52 genes that were up regulated more than 20 fold upon mineralisation of the cultures. Among these genes were osteocalcin, bone sialoprotein and PHEX, three genes that are known to be specifically expressed in late stages of osteoblast differentiation. Surprisingly, in addition to these expected findings, we also observed a more than 50 fold up-regulation of a few well-known genes such as hepatic lipase, CD53 or cathepsin S. Most importantly however, the majority of the 52 genes mentioned above represented non characterised expressed sequence tags, i.e. novel genes. In an initial screen by RT – PCR we found that some of these genes are specifically expressed in bone, but not in any other tissue, suggesting a putative role in bone formation or remodelling.

To identify genes, that are specifically expresed in terminally differentiated

osteoblasts, we have compared the expression pattern of non mineralised and mineralised osteoblast primary cultures. We have focussed our subsequent analysis on these 52 genes that are up regulated upon mineralisation more than 20 fold. The fact that osteocalcin, bone sialoprotein and PHEX were found among these genes demonstrates that our screening system was effective. For that reason, we believe that few strongly up regulated genes that have well defined roles in other cell types should be analysed for their ability to influence bone formation. However, the most important result of this study is that we identified sever novel bone-specific genes whose expression is strongly activated upon mineralisation. As cell specific gene expression is often a predictor for an important cell-specific function, we believe that the further analysis of these genes will provide important new insights into the process of bone remodeling and thereby into the pathogenesis of osteoporosis.

SYNOVITIS SCORE: A NEW HISTOPATHOLOGICAL GRADING SYSTEM FOR DEGENERATIVE AND INFLAMMATORY JOINT DISEASES

L. Moravietz et al., Hamburg, Berlin

As in OA synovitis is regarded as a result of degenerative cartilage destruction whereas in inflammatory joint diseases (reumatoid arthritis, psoriatic arthritis and reactive arthritis) synovitis is considered to be the cause of cartilage destruction, it can be concluded that scores with reasonably high values indicate the pathogenetic potential of synovitis at the same time. In experimental pathology this score could provide standardised information on molecular synovial tissue analyses where a correlation of molecular with morphologic data is essential. In diagnostic pathology this score (in combination with other scorring systems) could provide basic and standardised information concerning the degree of inflammatory alterations in synovial tissue. The feasibility of a computer – assisted morphometric analysis of synovial tissues according to this score is presently under investigation.

THE ORTHOSIS SPINOMED IMPROVES POSTURE, TRUNK MUSCLE STRENGTH, AND QUALITY OF LIFE IN WOMEN WITH SPINAL OSTEOPOROSIS: RESULTS OF A PROSPECTIVE, RANDOMIZED, CROSS OVER STUDY

B. Begerow et al. Bad Pyrmont

Spinomed improves posture, trunk muscle strength, lung function, body sway, and quality of life in postmenopausal women with osteoporosis and may therefore play an integral part in the rehabilitation process of osteoporotic patients. The efficacy of thoracolumbal braces needs to be further investigated in prospective, randomized and controlled clinical trials.

Key words: spinal osteoporis, orthosis, evidence based medicine

COMPENSATORY Na EXCESS IN DAHL RATS: A LINK BETWEEN OSTEOPENIA, Na RETENTION AND HYPERTENSION

J. Titze et al. Erlangen, Berlin, Kulmbach

We conclude that Ca^{2+} abd Na balance are integratively balanced. Na retention in SS rats (and thus salt sensitive hypertension) on a high Na diet does not occur because of an inability to excrete Na. Instead, we suggest that Na retention is

a compensatory mechanism to maintain Ca balance. This trade off occurs art the expense of volume excess and hypertension. Such a mechanism may provide a link between osteoporosis and essential hypertension.

Key words: osteoporosis, hypertension, renal Ca leak, Na intake electrolyte balance

ESTROGEN AND TESTOSTERONE ENHANCE AND ACCELERATE FRACTURE HEALING

M. Haberland

Fractures of the skeleton belong to the most common medical problems. Although most fractures heal appropriately, they still lead to pain, disability or confinement of patients, which in turn leads to a tremendous loss of productivity and income. Recent years have shown rapid progress in the molecular understanding of fracture healing, leading to advanced experimental therapies including the application of bone morphogenetic proteins and growth factors. However, till today, no therapy exists that could rutinely be given to patients suffering from fractures in order to enhance or accelerate fracture healing. As these drugs have been safely administered to patients in the past, e.g. in hormone replacement therapy, they may be a therapeutical option for enhancing and accelerating fracture healing for a broad group of patients, thereby reducing the amount of patients days lost due to incapacity, and may be used for the prevention and therapy of disturbed fracture healing seen in delayed and non union.

The results of this study show, that estrogen is essential for fracture healing in mice, estrogen and testosterone enhance the biomechanical stability of fractures in eugonadal mice, short term estrogen application can overcome the deleterious effects of estrogen deficiency on fracture healing and that estrogen accelerates fracture healing by approx. 25 %.

Key words: fracture – estrogen – testosterone – biomechanics

MUTATION DES KATHEPSIN K GENS FUEHRT ZUR EINER VERMINDERTEN KNOCHENQUALITAET IN PYKNODYSOSTOSEPATIENTEN

A. Valenta et al. Leoben, Wien, New York

Kathepsin K je cystein proteináza, která je velice silně exprimována v osteoklastech a je důležitá pro odbourávání kosti. Diskutuje se potenciální přínos antagonistů katepsinu K jako blokátorů osteoresorpce. Vyšetření katepsinu K u „knock – outovaných“ myší ukázalo, že osteoblasty bez katepsinu K odbourávají pouze kostní minerál, nikoli však organickou matrix. Mutace lidského genu pro kathepsin K vedou k pyknodysostóze, vzácnému autosomálně recesivnímu syndromu, který je charakterizován sníženou kostní resorpcí, a tudíž zvýšeným objemem kosti, tj. osteosklerózou. Ta je mj. charakterizována zmnoženým množstvím fraktur. Jak velká porucha funkce osteoklastů tím může být způsobena, není toho času známo. Dále je málo známo o materiálových vlastnostech kostních tkání u pacientů s pyknodysostózou. Biopsie z lopaty kosti kyčelní u dvou pacientů s pyknodysostózou ve věku 5 a 21 let byla nejprve histologicky a histomorfometricky vyšetřena. Pomocí řádkovacího a rastrovacího elektronového mikroskopu byl stanoven průměrný obsah kalcia. Dále byla stanovena střední tloušťka krystalů

a orientace krystalů v kostní matrix pomocí rtg.

U obou pacientů byl zjištěn silně zvýšený objem kosti, se ztluštěním kortikalis, stejně tak ztluštění trabekul a větší tvorba sítě trabekul. Barvení zlatem umožnilo identifikovat na povrchu kosti aktivní mnohojaderné osteoklasty. Resorpční lakuny byly demineralizovány a barveny na kolagen. Histomorfometrické vyšetření ukázalo zvýšený počet osteoklastů u mladšího pacienta, počet osteoblastů byl však ve srovnání s normou snížen na polovinu. Střední obsah kalcia byl snížen. Naproti tomu u dospělého pacienta obsah kalcia byl v normálním rozsahu. U dospělého nebyly žádné aktivní osteoblasty a málo aktivních osteoklastů. Obraz biopsie v polarizovaném světle ukázal odchylné uspořádání kolagenních fibril od dlouhé osy trabekul. Vyšetření nanostruktur kostního materiálu ukázalo signifikantní zvýšení střední vrstvy minerálních částeček u obou pacientů. Krystaly jsou relativně velké. Krystalické částečky jsou chaoticky uspořádány.

Kost není optimalizována pro biomechanickou zátěž. Snížená kvalita materiálu vede k zvýšené incidenci zlomenin u pyknodysostózy.

WELCHEN EINFLUSS HAT DIE OSTEOGENE DIFFERENZIERUNG MESENCHYMALER VORLAUFERZELLEN IN VITRO AUF DIE HEILUNG VON KNOCHENDEFEKTEN IN VIVO

K. H. Frosch et al. Goettingen

Tkáňové inženýrství mesenchymálních prekurzorových buněk a kmenových buněk u defektů hojení kostí získává stále na významu.

Buněčná terapie se stále rozšiřuje a její hranice nejsou ještě zcela stanoveny. Dosud nebylo popsáno, jakou roli má diferenciace prekurzorových mesenchymálních buněk. Osteogenní stimulace mesenchymálních prekurzorových buněk, které byly použity v rámci tkáňového inženýrství u kostních defektů nemají žádný pozitivní efekt na hojení kosti in vivo, je to možná způsobeno stresem buněk při osteogenní stimulaci in vitro, ztrátou vitality, která se projeví in vivo. Mesenchymální prekursorové buňky mohou nalézt při hojení kostí uplatnění, pokud se však nepoužije k jejich stimulaci in vitro vitamin D3 nebo dexamethason.

FABRICATION OF CARTILAGE POLYMER CONSTRUCTS USING MULTIPOTENT HUMAN TRABECULAR BONE DERIVED MESENCHYMAL STEM CELLS

U. Noeth et al. Würzburg, Bethesda

The use of multipotent human mesenchymal stem cells has opened new therapeutical approaches for the reconstruction of articular cartilage defects. These cells can be isolated from bone marrow, fat, muscle and skin. We have previously shown that multipotent human mesenchymal cells can be isolated from human trabecular bone fragments. In this study we tested the hypothesis whether these trabecular bone derived cells can be used for cartilage tissue engineering.

The study shows that mutipotent mesenchymal cells derived from human trabecular bone fragments can be isolated from the iliac crest by harvesting small trabecular bone cylinders. When coated on biodegradable polymer surfaces these cells underwent chondrogenic differentiation and showed a similar chondrogenic dif-

ferentiation pattern over the three week culture period as previously described for mesenchymal stem cells derived from human bone marrow.

Key words: mesenchymal stem cells, trabecular bone, chondrogenic differentiation, polymer

FUNCTIONAL DISTURBANCES OF THE CALCIUM RECEPTOR BY SOMATIC AND GRMLINE MUTATIONS: CLINICAL IMPLICATIONS

D. Riccardi, Manchester

Ca^{2+} concentration in the extracellular fluids (Ca^{2+}) is essential for a number of vital processes from bone formation to blood clotting. For this reason, it is necessary that (Ca^{2+}) must be strictly controlled. Mammalian species have developed a complex homeostatic system that includes parathyroid glands, kidney and bone. The extracellular Ca^{2+} sensing receptor is an essential component of this system, regulating parathyroid hormone secretion, calcium excretion by the kidney and bone remodeling. Inherited mutations in the Ca^R gene present on chromosome 3 are responsible for hypercalcemia or hypocalcemia depending upon whether they are inactivating or activating, respectively. Heterozygous loss of function mutations cause familial hypocalciuric hypercalcemie (FHH), in which the mild hypercalcemia is usually asymptomatic. When the mutation is present on both alleles this causes neonatal severe hyperparathyreoidism, a disorder characterized by extreme hypercalcemia with severe bone and neurological phenotypes. The latter is normaly lethal unless the parathyroid glands are removed early in life. The mirror image of FHH is represented by autosomal dominant hypocalcemia (ADH), which is due to gain – of – function mutations in the Ca^R gene. Several polymorphisms have also been identified and one of them, A986S, has been suggested as candidate locus for genetic predisposition to various bone and mineral disorders in which extracellular calcium concentrations play a predominant part.

Pozn. V této plenární přednášce bylo upozorněno na senzory a efektory v homeostatických dějích, bylo prezentováno schéma receptoru Ca^{2+}, zejména jeho ECT oblast, aktivace receptoru probíhá např. sperminem, gentamicinem, hořečnatými ionty Mg^{2+}, beta amyloidními peptidy, polyargininem, modulátory tzv. kalcimimetik.

Choroba z poruchy kalciových iontů, tzv. hypokalciurická hyperkalcémie je autozomnálně dominantní nozologická jednotka s vyjímkou homozygotního stavu, jedná se o abnormální citlivost ledvin na Ca^{2+}. Často je to spojeno s hyperparathyreoidismem, případně pankreatitidou, žlučníkovými kameny a chondrokalcinózou. Z genetického hlediska jde o typický případ haploinsuficience s efektem genové dávky. Je známa i genová lokalizace defektu (3q2 a 19q13). Může se jednat o redukci exprese určitých proteinů v proximálních ledvinových kanálcích anebo dědičnou resistenci k vnímavosti ke kalciovým iontům.

MULTIPOTENT MESENCHYMAL STEM CELLS DERIVED FROM HUMAN TRABECULAR BONE

Noeth, U., Wuerzburg

The identification and isolation of multipotent progenitor cells in different mesenchymal tissues (e.g. bone marrow, fat and muscle) has opened new avenues for

tissue engineering applications. These cells are characterized by the ability to differentiate into a variety of mesenchymal tissues such as bone, cartilage, tendon, ligament etc. In contrast, cells derived from human trabecular bone fragments are known to undergo osteoblastic gene expression and matrix mineralization, and are thus considered as human osteoblastic cells (hOBs). Similar to mesenchymal stem cells derived from marrow, fat or muscle, these cells are able to differentiate into osteogenic, adipogenic and chondrogenic cell lineages. The finding, that hOBs derived from human trabecular bone, traditionally considered as osteoblastic cells, have mesenchymal stem cell characteristics raises interesting questions on the plasticity of cells normally residing within mineralized matrix of mature bone. These cells might play an important role in pathological bone processes and fracture healing and can be used for tissue engineering applications to restore damaged tissue of mesenchymal origin.

TISSUE ENGINEERING VON KNOCHENERSATZGEWEBE MIT EINER INJEZIERBAREN TRAEGERSUBSTANZ

Schaefer, D. J., Basilej

Osteogenní buňky se uplatňují in vivo a in vitro a jejich klinické perspektivy jsou v krytí defektů lbi, po resekcích tumorů a u fraktur distálního radia. V jejich využití v rámci tkáňového inženýrství se využívá jejich osteogenního potenciálu a angiogeneze. In vitro se používají lidské periostální buňky enzymaticky izolované. Osteogenní buňky se dají odlišit od fibroblastů z hlediska jejich fenotypu. Z biomechanických vlastností jsou důležité schopnosti adheze na povrch. Kost je injikována do cyst, stabilizována kalciofosfátovým cementem. Velikost granulí cementu činí 200 až 400 mikrometrů. Byl vypracován specielní aplikátor pro injikování kosti. Po aplikaci u pacientů jsou sledovány metabolické vztahy. V experimentu jsou využívány „nahé myši". Biomechanické studie ukazují na stabilitu v tlaku, stimulaci cévního zásobení, schopnost integrace a remodelace u injikované tkáně.

RECONSTRUCTION OF SEGMENTAL BONE DEFECTS IN ANIMALS AND HUMANS

Quarto, R., Genova

Rekonstrukce ve své kvalitě závisí na různých vlivech, v neposlední řadě na fyzikálních a biomechanických. Referát se zabývá problematikou aplikace porézní biokeramiky. Tento děj je ovlivňován FGF2 – formace kosti a chrupavky je též ovlivněna IGF1 a IGF2. FGF2 ovlivňuje též délku telomér. Kostní formace je funkcí implantace biomateriálu. Makrostruktury jsou ovlivňovány porozitou aplikovaného materiálu, existuje kritická velikost defektu. Při autologní aplikaci jsou neúspěchy 1:4 při řešení segmentálních defektů dlouhých kostí. Výsledky nejsou ještě zcela optimální.

IDENTIFIKATION ZWEI NEUEN TUMOR – SUPRESSOR – GENLOCI BEIM OSTOSARKOM DURCH EINE VERGLEICHENDE ALLELVERLUST-UNTERSUCHUNG AN MURINEN UND HUMANEN OSTOSARKOMEN

Nanrath, M., Neuherberg

V sekci věnované novým molekulárně biologickým a prognostickým aspektům u osteosarkomu se jednalo o identifikaci dvou nových tumor supresorických lokusů. Jedná se o ztrátu dvou alel v tumoru

a lokalizaci ztrátových mutací v určitých lokusech. Bylo studováno 42 lidských osteosarkomů s využitím fluorescenčních DNA prób. Bylo využito PCR netody. Studovány lokusy na 16p, 9p21, 11p, 10q chromozomech a dalších. V 54 % byla zjištěna mutace na 16q21+, mutace tzv. retinoblastomového genu byla prokázána v 65 %. Mutace na lokusech 17p131–p53 byla prokázána v 83 %. Hovoří se o tzv. dvouzásahové teorii.

Osteosarkomy byly sledovány též u myší, zejména myší 9. chromozóm a byla prokázána určitá genová shoda mezi 9. myším a 15. lidským chromozomem. Pro stanovení delecí se osvědčila metoda FISH.

Byly studovány též tzv. kandidátní geny jež se mohou podílet na vzniku osteosarkomu – vliv genové dávky je u nich nesporný, jako např. u TBX 5 genu, který souvisí se vznikem srdečních a končetinových vad, dále TBX 2 a 3. Zvláštní pozornost zasluhuje TBX 18, jehož větší exprese je zaznamenávána v mezenchymálních tkáních než u osteosarkomu. Studuje se též efekt metylace, haploinsuficience. Žádná mutace TBX 18 v souvislosti s osteosarkomem zatím nebyla nalezena. Genový produkt TBX 18 sledovaný pomocí protilátek by event. mohl sloužit jako marker. Geny na chromozomu 15 se jeví též perspektivní pro další studium.

OSTEOBLASTIC AND NON OSTEOBLASTIC EFFECTS OF LEPTIN ON BONE METABOLISM

T. Thomas, St. Etienne

Osteoblastic and non osteoblastic effects of leptin on bone metabolism – stejně jako předchozí referát nebyla tato přednáška publikována. Autor sledoval expresi v osteoblastech a proliferaci v chondrocytech osteoblastických linií. Leptin má vliv na apoptozu lidských osteoblastů. Má vliv na expresi markerů osteoblastické diferenciace, včetně syntézy kolagenu, dále na mineralizaci stromatu, ovlivňuje expresi osteoprotegerinu a RANKL ve studii in vitro, inhibuje formaci osteoklastů, tlumí expresi interleukinu IL-1RA v lidských monocytech. Leptinové receptory jsou exprimovány v skeletálních růstových centrech a leptin stimuluje chondrogenezi. Leptin má nezastupitelnou roli v enchondrální osifikaci, má vliv na proliferaci buněk a je nezávislým prediktorem BMC a BMD. Leptin má preventivní vliv na ztrátu kosti, zvyšuje mechanickou odolnost kosti. U vyvíjejících se myší zvyšuje délku femuru (periferní aplikace).

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MEZINÁRODNÍ ANTROPOLOGICKÝ KONGRES S NÁZVEM „ANTROPOLOGIE A SPOLEČNOST“ POŘÁDANÝ U PŘÍLEŽITOSTI 60. VÝROČÍ ÚMRTÍ DR. ALEŠE HRDLIČKY

SEDLAK P.

Katedra antropologie a genetiky člověka, PřF UK Praha, Viničná 7, 120 00 Praha 2

Mezinárodní antropologický kongres proběhl ve dnech **22. až 24. května 2003 v Praze a Humpolci.**

Kongres byl věnován vzpomínce 60. výročí úmrtí dr. Aleše Hrdličky, antropologa světového formátu, humpoleckého rodáka, spoluzakladatele a mecenáše české antropologie počátku 20. století.

Organizace kongresu se ujala Česká společnost antropologická ve spolupráci s Katedrou antropologie a genetiky člověka Přírodovědecké fakulty Univerzity Karlovy v Praze, Národním muzeem v Praze, městem Humpolec, Evropskou antropologickou asociací a městem Mělník, pod záštitou rektora Karlovy univerzity, děkana PřF UK a generálního ředitele Národního muzea. Výraznou vědeckou a organizační podporu poskytlo kongresu Evropské centrum pro medicínskou informatiku, statistiku a epidemiologii – Kardio, které organizovalo sekci Bioinformatiky.

Kongresu se zúčastnilo cca 260 antropologů, lékařů a humánních genetiků z 22 zemí. I když převažovalo zastoupení evropské antropologie, mohli organizátoři přivítat i účastníky z Jihoafrické republiky, USA a Austrálie. Jednacím jazykem kongresu byla již tradičně angličtina.

Jednání kongresu byla rozdělena do **10 odborných sekcí,** které tématicky pokrývaly celé spektrum antropologických a hraničních oborů:

sekce 1: Růst a vývoj; funkční antropologie
sekce 2: Klinická antropologie; obezitologie; endokrinologie
sekce 3: Variabilita lidského genomu
sekce 4: Ekologie člověka a epidemiologie
sekce 5: Pohybový systém
sekce 6: Evoluční antropologie a primatologie
sekce 7: Historická antropologie a paleopatologie; forenzní antropologie
sekce 8: Bioinformatika
sekce 9: Sociální a kulturní antropologie
sekce 10: Sociální historie a antropologie

V předsednictví jednotlivých sekcí se střídali významní představitelé příslušných oborů z České republiky a ze zahraničí.

Vědecká jednání kongresu probíhala v prostorách Národního muzea v Praze a Přírodovědecké fakulty UK ve Viničné ulici a byla rozdělena do tří půldenních bloků. První den kongresu (čtvrtek 22. 5.) proběhlo ve večerních hodninách slavnostní zahájení v historické aule Karolina. Účastníky přivítali předseda České společnosti antropologické a prezident organizačního výboru kongresu doc. Pavel Bláha, prorektorka UK doc. Jaroslava Svobodová, sekretář Evropské antropologické asociace prof. Charles Susanne, děkan Přírodovědecké fakulty UK prof. Pavel Kovář a v neposlední řadě i čestný host ČSA, prezident kongresu prof. Phillip V. Tobias z University Witwatersrand v Johanesburgu v Jihoafrické republice. Při této příležitosti předal děkan PřF UK zlatou pamětní medaili fakulty za zásluhy o českou antropologii prof. Charlesu Susannovi a stříbrnou medaili bývalému starostovi Humpolce Janu Kotenovi.

Druhý den kongresu (pátek 23. 5.) po dopoledních jednáních odjeli účastníci na „Memoriál Aleše Hrdličky", který v rámci kongresu uspořádalo město Humpolec. Hlavním bodem bylo udělení pamětních medailí dr. Aleše Hrdličky za rozvoj a propagaci české antropologie. Toto ocenění převzali z rukou starosty města Humpolce mgr. Jiřího Kučery a předsedy ČSA doc. Pavla Bláhy dr. Ladislava Horáčková, doc. Jan Šteigl (oba Česká republika), prof. Janusz Piontek (Polsko), prof. Charles Susanne (Belgie), prof. Eva Bodzár (Maďarsko), prof. Uwe Jaeger (Německo) a dr. Eva Neščáková (Slovenská republika). Medaili dr. A. Hrdličky v občanské rovině za zásluhy o rozvoj města získal spisovatel, básník a sochař František Brzoň, autor Hrdličkova životopisu „Chlapec s arnikou". Po cca dvouhodinovém volném programu, ve kterém mohli účastníci dle vlastní volby zhlédnout expozici Hrdličkova muzea v Humpolci, rodný dům Gustava Mahlera v Kališti nebo podniknout organizovaný krátký výlet na hrad Orlík, byli všichni pozváni starostou města k malému pohoštění.

Třetí a poslední den kongresu (sobota 24. 5.) proběhla v dopoledních hodinách další jednání v odborných sekcích. Ve 14 hod. v přednáškové místnosti Národního muzea v Praze kongres oficiálně zakončil prezident organizačního výboru doc. Pavel Bláha, tajemník EAA prof. Charles Susanne a prezident kongresu prof. Phillip V. Tobias, který poděkoval organizátorům za důstojný a úspěšný průběh tohoto setkání světových antropologů.

Součástí kongresu byly i posterové prezentace. Instalováno bylo celkem 98 posterů, ke kterým probíhaly individuální i kolektivní diskuse v rámci harmonogramů jednání jednotlivých vědeckých sekcí. Každý z účastníků kongresu obdržel 250 stránkovou knihu abstrakt s harmonogramem kongresu a programem jednotlivých sekcí. Jednotlivé referáty jsou, dle individuálního zájmu, publikovány v časopise „Anthropologie", č. 1–2 a 3 za rok 2003, vydávaným Moravským muzeem, Anthropos Institute v Brně.

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Připojena jsou abstrakta prací prezentovaných 24. 5. 2003 v Národním muzeu v sekci 5: Pohybový systém a některá další vybraná abstrakta.

ORTHOPROSTHESIS FITTING AT SOME SYSTEMIC BONE DISEASES AND CONGENITAL LIMB DEFECTS

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The authors present technical solution of lower extremities shortening by orthoprosteses and some clinical applications. Plastic materials are the basis of individually made orthoprostheses. Orthoprosthesis (early called prosthesis device) should fulfil as function of prosthesis – it means to substitute a part of extremity and its motion function as orthosis that acts on deformities, supports joint movement and corrects axis of limbs (in frontal, sagital and axial planes) during growing period. Orthoprostheses are indicated and applied in cases with severe deformities and shortening of legs e.g. in longitudinal defects.

Skeleton of orthoprostheses are made from thermoplast, polyethylene or polypropylene that are connected with a suitable lengthening-piece to enable gait of patient. According to magnitude of shortening, the othoprostheses are fitted with prosthesis sole, tubular part or extension of constructional sponge polyuretan that is armoured (reinforced) by iron pieces, cosmetic roofing and contact gum elastic frame. Election of single components is from aspects of leg shortening, weight and height of patient, presumed mechanical stress and use of aid. Orthoprostehesis is constructed with respect to the leg deformity, possibility of corrective promotion (function) and anticipated therapeutic course (e.g. corrective osteotomy, surgical reconstruction or amputation).

Recent possibilities of orthoprosthesis fitting of biomechanical severe congenital defects and leg shortenings are demonstrated as short case reports of patients with neurofibromatosis von Recklinghausen (pseudoarthroses of shanks), osteogenesis imperfecta (type III according to Sillence), enchondromatosis (M. Ollier), proximal femoral focal deficiency and complex femur-fibula.

SPINE STRESS STATE UNDER BRACE EFFECT

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Introduction

Corrective braces are used for the treatment of spine scoliosis of children (deformation of chest curve). The brace pushes on child trunk and after a long time using it corrects pathologic spine curve. The brace is worked at this manner: it is made a plaster negative and then a positive form of child trunk. The orthotic according to his and orthopaedist experience deeps the plaster positive form at the place where the brace has to push on the child trunk. The plastic brace is then made according to this plaster form. The brace after its application pushes at the places, where the form has been deepen (the small shoe principle). The brace force effect is result of orthopaedist experiences only. The paper shows algorithms and computer programs, which are able to determine the stress state at vertebras and intervertebrae discs for a con-

crete brace using. The remodelling of spine curvature depends spinal stress state in time and form of the brace application and it can be simulated on computer. The theoretic conclusions are verified with many treatment courses.

Methods

The finite elements method (deformation variant according to the Lagrange principle) is used for the stress state solving. It is supposed that the vertebrates have no deformation. The potential energy is calculated for the inter-vertebrae discs volume and for the pressed soft tissue region of the trunk. The inertia moment has to be determined for an inter-vertebrae disc cross-section and lignums. The cross-section is divided to triangles and the third parts of areas are concentrated to the side centres. Because it is no deformation between vertebrae centre and inter-vertebrae disc bounder, the central spine line is at this part straight. The follow algorithm is valid for the frontal and medial plane, the planes will not be indicated by the plane index.

The difference between displacements and turning measured on the X-rays without and with a brace are the deformation of spine under brace force effect. The deformation is given by displacements and turning at the vertebrae centres

$$r^T = [w_i, \varphi_i, w_{i+1}, \varphi_{i+1}] \tag{1}$$

and the node forces and moments at the vertebrae centres are

$$R^T = [R_i, M_i, R_{i+1}, M_{i+1}] \tag{2}$$

It is valid

$$K_r = R \tag{3}$$

where K – stiffness matrix for the spine part between centres of neighbouring vertebrae is

$$K = \begin{bmatrix} \frac{6k}{l^2} & -\frac{3k}{l}\left(\frac{2a}{l}+1\right) & -\frac{6k}{l^2} & -\frac{3k}{l}\left(\frac{2a}{l}+1\right) \\ -\frac{3k}{l}\left(\frac{2a}{l}+1\right) & k\left[2+\frac{3a}{l}\left(\frac{2a}{l}+1\right)\right] & \frac{3k}{l}\left(\frac{2a}{l}+1\right) & k\left[1+\frac{3a}{l}\left(\frac{2a}{l}+1\right)\right] \\ -\frac{6k}{l^2} & \frac{3k}{l}\left(\frac{2a}{l}+1\right) & \frac{6k}{l^2} & \frac{3k}{l}\left(\frac{2a}{l}+1\right) \\ -\frac{3k}{l}\left(\frac{2a}{l}+1\right) & k\left[2+\frac{3a}{l}\left(\frac{2a}{l}+1\right)\right] & \frac{3k}{l}\left(\frac{2a}{l}+1\right) & k\left[2+\frac{3a}{l}\left(\frac{2a}{l}+1\right)\right] \end{bmatrix} \tag{4}$$

where the beam stiffness is k = (2EI)/l, E,I are the module of elasticity and the moment of inertia of a cross-section at the inter-vertebrae disc and lignums place, l is thick of disc. The node force and moments R can be calculated from (3), the stress state at inter-vertebrae parts and vertebra can be now calculated.

If it is completed the vectors r and R for whole spine and the equation (3), can be calculated vector R. The elements of the vector R are spine load because the inner forces and moments are according to the action and reaction principle eliminated. The load of spine is at appositive direction load from spine to the trunk soft tissue.

The pressed soft tissue can be considered as an elastic grunt. The brace pushes at a child trunk at the place, where the plaster positive form has been deepen; it means that the trunk surface (soft tissue) has at these places the non-zero prescribed displacements. The prescribed displacement is supposed above for lying patient. The compression of the soft tissue part up the spine is $w_0 - w$ and below it is w (for $w_0 > w > 0$), where w is a spine displacement and w_0 is a trunk surface displacement. Let the matrixes K_{above}, K_{below} be calculated for trunk part above and below the spine according to formulas for elastic grunt. The variation of potential energy of soft tissue part is

$$\delta E_p = \delta r^T [-K_{above} (r_0 - r) + K_{below} r] = r^T [-K_{above} r_0 + (K_{below} + K_{above})] r. \quad (5)$$

The vector r_0 can be calculated from (5), the r_0 elements are needed deepen distances at perpendicular direction to spine for the trunk plaster form according which is the plastic brace made. The analogical formulas are valid for medial and frontal planes.

Results and discussion

If the brace is put of the child trunk after some time of application, then the spine does not return to previous position but the pathologic spine form is partly corrected. The part of spinal deformation (the difference between spine curves without and with brace) is permanent and the spinal curve has a new form. The permanent part of deformation depends on spinal curve type according to King. If the computer calculation is repeated at the time periods then the scoliosis treatment can be simulated.

Conclusion

The effect of cure will be compared with computer model behaviour and the model and parameters will be changed to be the same behaviour of the model and reality. The computer simulation model and its parameters are verified to be the behaviour of the model same that the child treatment course. Because the treatment takes a long time the theoretical conclusion could be determined after sufficient number of verifications between observed treatment courses and their computer simulations. Many child patients are observed at this grant and the dependence between the spine curve correction and the spine stress state and a time interval of brace application are studied and the theoretical conclusions about the spine remodelling are searched.

If the computer model will be verified, it will be used for cure prognosis and searching of optimal brace form variant.

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A CASE OF PELVIC SCARS: PARTURITION, LOCOMOTION OR TRAUMA?

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Sao Cucufate, located near Vidigueira (Southeast of Portugal), is one of the most important roman site in Iberian Peninsula. During Roman Empire, Sao Cucufate occupied an important place since it was near *Pax Iulia* (Beja). An osteological sample was studied in Departamento de Antropologia, were we examined some interesting morphological alterations and pathological conditions. In this study, we report a case of pelvic scars in an almost complete and well-preserved female skeleton.

Some researchers use the occurrence of pelvic scars as an indicator of parturition. Others believe that it can be a consequence of excessive movement of the bone pelvis or trauma.

The present case aetiology is here discussed.

PERPENDICULAR DIMENSIONS OF THORACOLUMBAL SPINE IN STANDING AND SITTING POSITIONS

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The paper deals with the differences in length of thoracolumbal spine and her particular sections in 100 young adult women in standing and sitting positions. To measurements were used the Columnometer.

The mean value of investigated section of spine between the points Cervivale (C) and Lumbale (Lu) is 444.76 mm in standing position and 429.82 mm in sitting position. Then in sitting there exists in average to shorten of 14.94 mm, it is 3.36 % of the mean length in standing position. The distance of peaks of thoracal kyphosis (point T) and lumbal lordosis (point L) is in sitting position shorten about 68.86 mm as in standing position (15.47 % of total length C-Lu in standing). The point T at the same time shifts in comparison with the standing position more caudal, point L contrary ascent more cranial.

Like the position for the judgement of distances both of peaks of the thoracolumbal curves was constructed the point x. It lies at lateral norm on the point of intersection of the curve which connects the most dorsal location borders of processus spinosus thoracal and lumbal verterbrae and of the verticale in the centre of the projective distance of points T and L. From the calculated values it is perceptible, the peaks of both investigated anterposterior curves have the same distance from the point x in both standing and sitting positions. In standing position it is 117 and 120 mm (26.3 % and 27 % of the total length of C-Lu). In sitting position it is 83.5 mm and 84 mm (19.4 % and 19.5 % of the total length of C-Lu). The both sections of thoracolumbal spine in the sitting position are then shorted uniformly.

HYPERHOMOCYSTEINEMIA IN A BOY WITH JUVENILE OSTEOPOROSIS

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Juvenile idiopathic osteoporosis (JIO) is a heterogenous multifactorial disease with clinical manifestation between 5^{th} and 17^{th} year of life (abnormal gait, pains in the feet, spine, hips, or elsewhere; progressive deformities of spine and extremities and/or metaphyseal fractures of long bones). Toxic effect of increased levels of plasmatic homocysteine (Hcy) -Hyperhomocysteinemia (HHC) was proved outside endothelial cells and coagulation proteins also in other tissues of mesenchymal origine. Although the direct relation between HHC and tromboembolic events were intensively studied, the correlation between HHC and the risk of development of connective tissue disorders has been till now only experimentally followed (Griffits 1976, Lindberg, 1976, Lubec1996, Siegel 1975). The toxic mechanism of elevated Hcy was explained by its very active -SH groups interfering with crosslinking of collagen mediated by aldehydic groups. In similar way might increased Hcy interfere with elastin structures, too (Lubec 1996, Mudd 1995). In our previous pilot study the higher levels of Hcy were observed in population of children suffering from different bone dysplasias: 6,6 +/-3,6 μmol versus 5,4 +/-1.3 μmol/l found in healthy children (Hyánek 2002). That's why the Hcy estimation was included among routine monitoring analyses and enabled us to detect and present a boy suffering from JIO where severe HHC was observed.

Case Report: 16 years old boy was referred to osteological examination because of repeated fractures of upper extremities; from 11 to 16 years he susteined 7 fractures (both forearms and wrists, fingers and patella). Proband is a Czech boy born after a physiological pregnancy of healthy mother, normal delivery at 40 weeks uneventfull gestation.

Status praesens at 16 years: The boy of asthenic habitus, height 186 cm/weight 64 kg; interested in athletics. A mild sinistroconvex scoliosis of thoracic spine, accentation of thoracis kyphosis and lubar lordosis was observed. Mobility of spine not restricted. X-rays of spine a mild thoracic dextro-convex and sinistro-convex scoliosis and lumbar dextro-scoliosis. Thoracic kyphosis due to wedge shaped vertebral bodies (T5,T7 and T8). Osteoporosis-abnormal pattern of bone structure of thoracic and lumbar vertebral bodies (vertical striation), that framed up. X-ray of left hand shows „marfanoid" shape, abnormal pattern of bone texture of distal radius, ulna and carpal bones.

Biochemical and metabolic examinations: mineral, protein, lipid markers within normal limits. Markers of osteosynthesis (S-osteocalcin –46.9 μg/l; S-bone alkaline phosphatase 42.9 U/l) and osteoresorption (U-pyrridinoline 91.8 μmol/mmol creat.,U-deoxypyrridinoline 10.4 μmol/mmol creat) were elevated S-parahormon (26 pg/ml),vitamin D_3 (63.8) μmol/l, FT_4 16.8 pmol/l). Hcy at admission 43.8 μmol/l, repeteated 57,8 μmol/l . L-methionine loading test: fasting 39.8 μmol/l and 77.4 μmol/l 6 hrs after load. Critical remethylation vitamins: folate: 8.3 nmol/l (!); B12: 154 pmol/l; B6: 16.9 μg/l. Creatinine 79.0 μmol/l, Glucose 5.2 mmol/l. Methylenetetrahydrofolate reductase 677 C > T genotype proved homozygote status (TT).

According to radioclinical and biochemical examination of bone turnover we conluded diagnosis as idiopathic juvenile osteoporosis and the treatment with Cholecalciferolum and Calcium was introduced accompanied with folic acid 5 mg/d and

cyanocobalamin 300 μg/weekly i.m.that normalised HHC and markers of bone metabolism within one month. The estimation of cystathione-beta synthase activity in fibroblast (to exclude homocystinuria) is under investigation.

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TREATMENT WITH BRACE, FROM OUR FIRST STEP UP TO NOW

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Braces formerly consisted in two rings, (respectively around shoulders and pelvis), so called „reference lines", joined together with „vertical lines", and in hump pressing parts. In 1987 we quitted the concept of reference lines. Conscious, that shoulders and pelvis are also deformed, we submitted them, too, to the action of pressure-expansion systems. The wide and deep concave sided expansion volumes remain the main idea of the concept.

The body has been divided into 54 zones. Brace is acting by means of nine mechanisms. They are the cherry-stone effect; the elective convex-concave tissue transfer system; growth; breathing; movements; reduction in „clamp" of the greater diameter of thorax; the use of a paradox secondary pressure around right breast; bending; anti-gravitation effect. Four mechanisms are active and five passive. During the use of brace, the shape of body changes. Those changes must be previewed in a certain measure during manufacturing of brace. Regular adjustments have to be done all along the duration of the brace, taking those changes in account.

The scoliotic patients have been divided into two classes, so-called three and four curved. Intermediary cases are well known and well managed now.

Gymnastics is most important. It must be adapted to the brace concept, and accompanied by elective breathing.

Braces are efficient. We give proofs of it. They correct, not only the curvatures, but also rotation, rib state and wedged vertebrae. In a further study we calculate those facts in a small series.

Short time results have been calculated among others in Belarus and long time results in Austria.

SCOLIOSIS TREATING BRACE: EVOLUTION OF OUR BRACE SINCE 1970. AN EVALUATION OF THE NORMALISATION OF ROTATION, RIB STATIC, AND OF THE WEDGE SHAPED VERTEBRAE

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In 1970, the so-called Cheneau brace was built after Abbott. It consisted not in plaster, like those of Abbott but in polyester and glass wool. Then it has been made with polyethylene after 1976, according to Professor Matthiass of Münster, Germany.

Strong idea has always consisted in managing huge hollow spaces in the places where the body is concave, corresponding with concavities of curvatures. These spaces should allow tissue wandering, growth, breathing and some correcting

movements. Nine correcting mechanisms combine their action: the so-called „cherry stone effect"; convex-concave tissue transfers; growth; breathing; movements; paradoxical pressure under right breast; reduction of the greater diameter of thorax; concave side bending; anti gravitational effect. Five of them act an active way and the other four a passive one.

The brace of today does not look like a human shape. Its shape is the one which the brace maker will give to the patient's body one year later. Since 1987, the clavicular parts have been suppressed, replaced by a higher anterior brace wall. Bracing needs a huge knowledge of scoliosis and much care, not only when manufacturing brace, but also when making the first essay on patient and all along the duration of treatment. Regular adjustments have to be made, consisting in raising pressure parts and expanding concave sided hollow spaces. Neglecting the last but very important adjustment leads to a severe narrowing of the trunk.

The brace reaches a Cobb's angle correction identical to the one reached by the best other schools. The main supplementary advantages expected are the preservation of the trunk width without narrowing and a normal breathing balance. For some months, we study the effect of bracing on three rarely or not at all studied features: rotation, rib static and wedge shaped vertebrae. Rotation and wedged vertebrae are bettered or at least not worsened in all patients. Rib static was worsened in two patients, and we discuss why. The three kinds of deformation show a clear average bettering after one year of treatment.

To conclude, braces are effective, not only Cobb's angle, but also on rotation, rib static and wedge shape of vertebrae. All those three deformations tend to be normalized by brace treatment.

MULTIPLE CARTILAGINOUS EXOSTOSES , ENCHONDROMATOSES AND ENCHONDROMATOSES WITH VERTERAL INVOLVEMENT

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These groups of skeletal disorders are relatively common and occur in all ethnics, they belong to benign tumours of skeleton with a high risk of malignant degeneration. Clinical features consist primarily of bony lumps and bumps (enchondroma and exostoses) that first appear in early childhood. The lumps can cause problems by pressing on adjacent nerves, vessels or tendons. The exostoses occur most commonly at the ends of long bones (from the epiphyses). They lead to deformities and disproportionate shortening of long bones, eg. ulna and fibula resulting in Madelung deformity of forearm. Exostoses of various size also occur on the ribs and on both the pectoral and pelvic girdles. *Inheritance* is autosomal dominant with chromosomal location 8q24.11-q24.13 (type I) or 11p11.2-p12 (type II) and 19p (type III). *Mutation spectrum* is a broad, missense, nonsense, frameshift and splice site point mutation (and others). All mutations are with a loss of function effect. *Molecular pathogenesis* is very important – proteins EXT 1 and EXT 2 form an oligomeric complex that acts as

a glycosyltransferase in the polymerization of heparan sulfate (co-factors in signal transduction). Signal transduction summarized tendency to grow, differentiation and migration. *Combined protein product EXT 1 and EXT 2* heterocomplex (localized to the endoplasmic reticulum – as the transmembrane glycoproteins) act as tumor – supressors. It has a negative regulatory role on cell turnover. Multiple exostoses are a well recognized diagnostic feature in *Langer – Giedion syndrome (LGSy).* Deletion of 11p11.2-p12 also leads to the development of multiple exostoses and mental retardation with microcephaly and biparietal foramina. LGSy is type II of the trichorhinophalangeal syndrome with characteristic facial appearance (bulbous nasal tip, long philtrum and large protruding ears). The fingers are short and often show angulation at the interfalangeal joints. The other features include short angulated fingers, short stature, deformities of the hips, cleft palate, etc.

We studied a group of patients with hereditary multiple exostoses, which have a loss of hetero-zygosity for EXT 1. Loss of the normal allele give rise clonal osteochondroma with only inherited mutant allele (so-called multistep concept of carcinogenesis). Due to this aspect long-term follow up of patients is necessary. Growing bony lumps and bumps are indicated to surgical intervention according to *X-ray examination. Histological investigation* differentiates grading of malignancy (chondrosarcoma). The markers with tendency to malignancy were examined in a group of our patients, too. Enchondromatosis and other atypical forms (e.g. Maffuci syndrome) we determined as fresh (postzygotic) autosomal dominant mutation (blastopathy) without genetic risk for siblings. *Biochemical investigation* proved high bone turnover. Both markers of bone resorption (serum acid phosphatase, urine pyridinoline and deoxypyridinoline) and markers of osteosynthesis (calcitonine and bone isoenzyme of alkaline phosphatase) were elevated.

Tissue cultures of patients with this diagnosis have been studied, too. We suppose on the basis of known chondrocyte culture studies of patients with multiple cartilaginous exostoses and/or enchon-dromatosis similar cell changes in tissue cultures with abnormal shape and division. It could be a result od defect regulation of cell proliferation due to mutations of oncogenes, antioncogenes and other regulatory genes. Their products ere were monitored – some were elevated.

Specific mutation analysis is not routinely available at multiple cartilaginous exostoses. This analysis is not possible at enchondromatosis. Recently, several disorders with the characteristic localisation of chondromatosis have been investigated. Genetic counselling is based on the mode of inheritance. The expression is variable.

ORTHOTIC REMODELLING CONTROL AT SYSTEMIC BONE DISORDERS

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Orthotic treatment is generally based on remodelling control of connective tissue. The conservative treatment of genu valgum or genu varum in children is based on step by step straightening of deformities by orthoses. The corrective forces of orthosis act on the basis of the three points principal.

There was experimentally verified that the shortening/extension of bone tissue at intermittent loading is followed by apposition of bone tissue. The apposition increases with the intermittent loading of bones. In the course of growth, the remodelling is considerably affected by the epiphyseal plates. According to Hüter-Volkmann law, the overload imposes the limitation on the growth. At the oblique loading, epiphysis plates regulate the growth of long bone into the direction of the pressure resultant. The periosteum affects in an important manner the remodelling of long bones. The push of periosteum against the bone surface involves the resorption of bone tissue whereas its take up is a cause of apposition. The convex surface of the bone inclines to resorption and the concave one to apposition to maintain the overall bone shape (so-called lateral drift).

In 1997, the authors developed and introduced for treatment a new type of orthosis with dynamic bending pre-stressing that is suitable for correction of varus or valgus deformities of legs (in various shank level and supracondylar region of femur) as in children suffering from so-called idiopathic deformities of lower extremities so in children with inborn bone dysplasias and/or acquired disorders of the skeleton. Step by step correction of bone deformities is based on remodelling control of growth epiphyses and bones. The orthoses consist of two parts that are jointed with hinge (on lateral or medial side) and screw on the opposite side. Orthoses are applied through night and time of bending force deformation is noted. In the last year the special screws with spring were developed with possibility to measure and keep steady prestressing.

In the whole group of patients the bone turnover was evaluated using some selected markers of bone metabolism. The increased bone metabolism in the investigated group of children was the indication for treatment with calciotropic drugs.

Degree of deformity and efficacy of orthotic treatment was evaluated according to correction of tibio-femoral angle measured at X-rays of lower limbs in standing patient. There is possibility to calculate the tibio-femoral angle according to measurement of intermalleolar or intercondylar distance in standing child and anthropological estimation.

The new developed limb orthoses with high bending pre-stressing appear as a perspective therapeutic method for some congenital and acquired lower limb deformities especially in pre-school age. Encouraging results were achieved in a few children at the age of 10 years or more and even in a few children suffering from bone dysplasias, which are documented.

PROGRESSIVE PSEUDORHEUMATOID CHONDRODYSPLASIA COMBINED WITH BRACHYMETATARSALIA – A NEW NOSOLOGICAL UNIT?

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In Czech Republic patients with rare bone dysplasias and severe biomechanical involvement of locomotor apparatus are centralized in the Ambulant Centre for Defects of Locomotor Apparatus in Prague. The authors would like to share with presumably a new nosological unit „Progressive pseudo-rheumatoid dysplasia with brachymetatarsalia" (PPDB). Clinical and radiological manifestations: Onset 3 – 8 years, progressive walking difficulties, waddling gait, easy fatigability, muscular weakness, joint stiffness and prominence, large joint contractures, decreased cervical spine mobility, kyphoscoliosis, flection deformities of fingers. X-rays shaw platyspondyly, round upper and lower margins, anterior end-plate erosions and Scheurmann-like defects, narrowed joint spaces of small, big and other joints. Widened metaphyses, flattened epiphyses and enlarged femoral heads, chondrocalcinosis. Erythrocyte sedimentation rate, C – reactive protein are normal and rheumatoid factor and HLA B27 is negative. Described clinical and radiological manifestations are pathognomonic for progressive pseudorheumatoid arthritis (arthropathy, chondrodysplasia) that is also called as spondylo-epiphyseal dysplasia tarda with progressive arthropathy in literature. In addition to these features we diagnosed brachymetatarsalia of both feet (the 3rd and 4th rays) in examined 4 members of one family. Pedigree comprises 8 patients suffering from the same bone dysplasia. Autosomal dominant inheritance was proved into the 4th generation. (Transfer of defective gene from father to son differentiate possibility of XD inheritance). We present four case reports that depict the development and severity of involvement from preschool age till adulthood.

STRUCTURAL FEATURES OF THE CELOIDAL SCARS

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The process of formation of the celoidal scar is manifestation of reparativ regeneration; it results in formation of scar's connective tissue. The objective of present work was to research the composition of the cells in the structure of the celoidal scars. The basic methods we used are the qualitative and quantitative evaluation of fibrocyts using the data of light microscoping.

It was discovered that the thin epidermal layer covers celoidal scars without invasion into the underlying dermal layers. Thick epidermal layer of the scar keep the equal structure of all layers. The growth layer consists of the big cells. Presence of the immature connective tissue allowed distinguishing the «growth zones». The friable filament tissue of the «zones» consists of the great number of the fibroblasts; there are many big and gigantic cells among them. Morphometrical count showed that the number of the fibroblasts in the «growth zone» is about 60 – 80 and even 120 – 150 in the sight-field of the microscope. It's 2 – 3 times more than in the hypertrophied and 3 – 5 times more than in the usual scars. Among the fibroblasts were revealed such ones, which structure shows graduate transition from immature to mature cells with functional activity. The prevalence of gigantic and active fibroblasts, which synthesizes proteins and carbohydrates among the cells of connective tissues, is revealed. The presence of imma-

ture connective tissue, which forms the «growth zone», is the basic attribute of the growing celoid.

Immature character of the connective tissue is determined by the condition of the fibroblasts and fibrous elements; among the fibroblasts prevails the cells with functional activity. Fibrocyts can be found only in deep layers. Presence of the gialuronical acid and unripe fibrils testifies to continuation and development of collagen fibers.

Thus, the presence of special forms of the fibroblasts, which doesn't synthesize elastical fibers in «growth zone» is one of the proofs of immaturity of celoidal scars' connective tissue.

EXTERNAL AND INTERNAL GOVERNING OF BONE REMODELLING

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The bone tissue remodelling is a cyclic process involving the replacement of an old tissue by the new one (in the volume micro/mezo unit of this tissue – BMU). Each remodelling limit cycle consists of five principal *intensive biochemical reactions* (intensive metabolic processes), which are defined by five stoichiometric equations, and *periods of weakly steady states* (i.e. periods in which the biochemical processes are relatively calm). The weakly steady states always arise (and last for a certain period) between the intensive biochemical reactions. In normal physiological conditions, and within the extent of each remodelling limit cycle, the activities (i.e. intensive molecular activities) alternate with the periods of relative passivity (i.e. the periods of „calm“ biochemical processes). Supposing that one remodelling limit cycle lasts approximately 6 – 8 years (in the considered BMU), the life of a human is then comprised of approximately 10 – 12 remodelling limit cycles in the BMU.

The limit cycles are controlled (governed) genetically and biomechanically. The genetic (internal) control and biomechanical (external) control initiate biochemical (metabolic) processes. The genetic factors (under normal physiological conditions) not only establish the precise time rhythm of the intensive biological activities and the rhythm of the weakly steady states, but they also „start“ the remodelling limit cycles.

The remodelling of a bone tissue results from very complex metabolic processes. On the basis of the up-to-now recognized and available knowledge of biochemical processes related to the creation of a new bone, the **kinetics of chemical substances (molar mixtures)** can be expressed in the following **five global stoichiometric equations:**

$$D_1 + D_2 \Rightarrow D_3 + D_4 , \quad (1)$$

$$D_3 + D_5 \Rightarrow D_6 , \quad (2)$$

$$D_2 + D_3 \Rightarrow D_7 + D_8 , \quad (3)$$

$$D_9 + D_7 \Rightarrow D_{10} + D_{11} , \quad (4)$$

$$D_{12} + D_{10} \Rightarrow D_{13} + D_{14} , \text{ in which} \quad (5)$$

The first stoichiometric equation indicates the process of osteoclast propagation (D_3 mixture) by merging from mononuclear cells (D_2 mixture, the fuse).

The second stoichiometric equation indicates biochemical processes that result in the activity of osteoclasts (D_3 mixture) after they have adhered on the surface of

a bone tissue (D_5 mixture). Once the enzymatic system of the osteoclasts has been activated, the inorganic component of a bone is degraded. The equation (2) defines the process of degeneration in the bone tissue.

The third stoichiometric equation indicates the production of refuse substrates (D_8) within the process of resorption of the bone tissue when the mononuclear cells are activated (D_2). The equation also indicates the arising of molar substances (D_7) that subsequently participate in the activity of osteoblasts while they produce the osteoid (see the fourth stoichiometric equation).

The fourth stoichiometric equation indicates the creation of the osteoid (D_{10}), i.e. the non-mineralized matrix, being accompanied by the arising of a refuse substrate (D_{11}).

The fifth stoichiometric equation *indicates the creation of a new bone tissue (D_{13}), i.e. the mineralized osteoid, being accompanied by the arising of a refuse substrate (D_{14}).*

The overall volume changes in molecular mixtures in the BMU are generally given by the sum of volume changes in molecular mixtures, which are the product of biochemical processes (genetically initiated), and the volume changes in molecular mixtures, which are the product of biochemical reactions as well, being, however, initiated by mechanical/biomechanical effects. The general equation is as follows:

$$\eta_i = \eta_{i,g} + \eta_{i,m}, \qquad (6)$$

in which $\eta_{i,g}$ is the volume change resulting from biochemical reactions (metabolic processes) **initiated genetically;**

$\eta_{i,m}$ is the volume change of molecular mixtures resulting from biochemical reactions **initiated mechanically.**

The volume changes (in four primary molecular mixtures) **have the dominant and absolutely decisive influence on the process of remodelling limit cycle.** The molecular mixtures include the following:

- η_1 a volume change in the molecular mixture of osteoclasts,
- η_2 a volume change in the molecular mixture of osteoblasts,
- η_3 a volume change in the osteoid molecular mixture, and
- η_4 a volume change in the molecular mixture of a mineralized osteoid.

The speed of biochemical reactions can be generally expressed as follows:

$$k_j = C_j\, e^{\upsilon j(p\text{-}pe)} \qquad (7)$$

in which υ_j (j = 1, 3, 4, 5) are volume changes of the determined components of remodelling processes in the bone tissue, i.e.:

$$\upsilon_1 = \eta_1, \qquad (8)$$
$$\upsilon_5 = \eta_5, \qquad (9)$$
$$\upsilon_4 = \eta_4 + \eta_5, \qquad (10)$$
$$\upsilon_3 = \eta_3 + \eta_5, \qquad (11)$$

Each volume change υ_i (j = 1, 3, 4, 5) equals the sum of the volume changes resulting from the proceeding biochemical reactions, and the volume changes resulting from the proceeding biochemical reactions initiated mechanically. Thus,

$$\upsilon_i = \upsilon_{i,g} + \upsilon_{i,m} \qquad (12)$$

For the speeds of biochemical reactions, according to expression (7), then applies:

$$k_j = C_j \, e^{-(\upsilon j,g + \upsilon j,m)(p-pe)}, \qquad (13)$$

respectively:

$$k_j = C_j \, e^{-\upsilon j,g \, \Delta p} \, e^{-\upsilon j,m \, \Delta p}, \qquad (14)$$

Provided that $k_{j,g} = e^{-\upsilon j,g \ \Delta p}$ and $k_{j,m} = e^{-\upsilon j,m \, \Delta p}$, for the speed of the j^{-th} biochemical reaction, the following general expression is obtained:

$$k_j = C_j \, k_{j,g} \cdot k_{j,m} \qquad (15)$$

Then, it is obvious from the expression (15) that the **resultant speed of the j^{-th} biochemical reaction,** which forms part of biochemical (metabolic) processes in the bone tissue (in the remodelling limit cycle) **is dependent on the product of speeds of the biochemical reaction *initiated genetically* and on the speed of chemical reaction *initiated biomechanically.*** Thus, the j^{-th} biochemical reaction is influenced by the **internal – genetic effects and the external – biomechanical effects.** The external effects and the internal influence on the speed of remodelling processes in the bone tissue are completely coordinated by the live environment.

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FIFTEEN YEAR FOLLOW-UP OF PATIENTS WITH FAMILIAL CALCIUM PYROPHOSPHATE DEPOSITION DISEASE

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Chondrocalcinosis (CCA) has been defined as a spectrum of connective tissue disorders resulting from the accumulation of calcium pyrophosphate dihydrate (CPPD) crystals in the tissues. The components of the syndrome include acute arthritis, chronic inflammatory joint disease, osteoarthritis- and rheumatoid arthritis-like changes, chondrocalcinosis of the spinal joints and of the ligaments, tendons and menisci (1). The familial occurence of articular chondrocalcinosis was recognized by Žitňan and Siťaj (2).

We evaluated genetic, clinical features, radiographic findings, transmission electron microscopy and the quality of life in 14 patients suffering from familial chondrocalcinosis for 10 – 40 years.

Genetics. Genetic analysis was performed in 8 families (28 persons). The analysis confirmed familial occurrance CCA probably with autosomal recesive inheritance.

Clinical feature. Deformities of hands were not so pronounced. The function of the hands was preserved (grip and dexterity), the patients were able to perform limited personal care. More severe alterations concerned joints of the lower limbs, espe-

cially of feet. Frontal desaxations of ankles and subtalar joints were apparent. Small joints of the feet showed severe peroneal deviations of MTP joints, including subluxations and extremely valgous halluces, resulting in severe functional damage of foot in majority of the patients. Crural lymphedema and lymphedema of the dorsal side of the feet was observed in three patients. In one female patient, the lymphedema was as pronounced as to remind of elephantiasis. In the older patients inspection of feet reminded the rheumatoid arthritic foot.

Radiology. Radiographs showed calcifications in triangular articular discs of wrists. In PIP and DIP joints calcifications were localized in cartilage and capsule. The first carpo-metacarpal joint was often involved by severe form of rhisarthrosis.

Pronounced alterations in the lower limbs involved hip joints, knees, ankles and small joints of the feet. In the hip joints strip-like calcifications lined cartilage surface of the femoral head. Radiographs showed also narrowing of joint space, osteophytes, cysts and periostosis of the femoral neck. In 3 patients the protrusion of femoral head was found. In the knee joints thin stripe-like calcifications of articular cartilages and punctate calcifications of menisci were found. Moreover, findings characteristic of severe gonarthrosis and patello-femoral arthrosis were observed.

Radiographs of the spine showed signs of calcification of intervetebral discs. In some patients radiographs of the sacroiliac region showed changes imitating ankylosis of sacroiliac joints, subchondral sclerotizations, pseudocysts, and osteophytes.

Transmission electron microscopy. In two patients, deposits of CPPD crystals were identified by transmission electron microscopy in articular cartilage, synovial membrane and tendon obtained during surgery.

Quality of life. Quality of life was assessed using questionnary (MOS Short Form-36). The patients showed reduced mobility of the thoracic and lumbosacral spine. In spite of reduced mobility of spine the patients were able particularly to compensate the functional loss. The quality of life of the patients was markedly reduced. They could carry out but small works in their households. Advanced alterations, in particular those involving the lower extremities, resulted in patients physical disability.

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POSSIBILITIES OF 3D EVALUATING OF BACK SURFACE AND SPINE SHAPE

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Introduction. Idiopathic scoliosis is a three-dimensional change of spine. Scoliosis is usually first observed as a change in back shape and so back surface evaluating is important in clinical assessement of various spinal disorders. There is no direct determination between surface shape and skeletal anatomy, but changes of the spine curvature are manifested by back shape change (asymmetry). There is no conventional method of description of back surface shape in clinical practise. Standard X-ray diagnostics gives information in 2D only (Cobb) and is not possible to repeat it often

(radiation). The aim of our study is to show possibilities of quantification of spine and back surface shape in 3D. We have used two non-invasive diagnostics methods to monitor spinal deformities.

Experiment. Our work is a pilot study. For detection of back shape optical moire topography and stereometry were used. A shadow moire method works on principle of contour lines, which are formed on the back surface and give us 3D information about shape. A stereometry uses at least two cameras to obtain spatial co-ordinates of an object. We have followed the influence of muscle activity on surface and spine shape changes, relationship between lateral and axial spine deviation. We scanned individuals in stand with and without muscle activity of upper limbs and then compared changes in shape. Before scanning we marked prominent bones as an orientation about internal skeletal structures. We evaluated parametres of the spine by distance and angles of structures in frontal, sagittal and transversal plane. We have also constructed reliefs in definited torso cross sections.

Outcomes. We have found 3D changes of spine and back shape in various situations, but reactions of individuals were different. For example by influence of muscle activity the spine became straighter in sagittal plane (14° change), the asymmetry of paraspinal muscles increased (rotation), but there was a small change in frontal plane only. The maximum of rotation was always in apical points of curvature in frontal plane.

Conclusion. Moire and stereometry are usable methods for quantification of various spinal disorders. 3D data can give more information about axial system pathology. Because of possibilities of often repeating and movement recording, dynamics of curvature development can be followed. Advantage of these methods is evaluating of spine flexibility. This complete information about shape of trunk is available in diagnostics and therapy of scoliosis. We will continue in formulation and verification of methodology of scanning and data processing, elimination of technical and methodical errors and verification on a larger specimen. A comparing with other diagnostics method is necessary (Quantec, scoliometer, X-ray, CT). Present outcomes show a promising prospective diagnostic possibilities. We plan 3D model of classification to be form into axial deformity diagnostics.

SCAPHOID NONUNION FROM THE MEDIEVAL SITE KLADRUBY

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At the medieval burial site Kladruby, scaphoid nonunion was identified in two graves: No 4019 (man in the age of 40 – 50 years) and No 4025 (admixture to the main finding, undefined sex and age).

Comparison with a model created by Hidaki and Nakamura (1998) using three-dimensional computed tomography allowed, on the basis of a clinical set consisting of three skiascopically checked patients (5 months, 7 years and 19 years after injury), to set up the chronological succession of the development of degenerative changes in not united scaphoid bone fractures.

Between the 4^{th} – 7^{th} year, onset of the development of degenerative changes on the distal scaphoid fragment takes place. From the 7^{th} to 10^{th} year tapering of the sty-

loid process of the radial bone occurs, and cysts may be visible on X-ray films. As a rule, enlargement of the distal fragment osteophyte occurs after the 10th year.

Using this classification it will be possible to determine the time of injury prior to death if the distal fragment of the scaphoid, and the radial bone are preserved at least (maybe also the contralateral – for comparison).

In the 40 to 50 years old man from the grave No 4019 this was 7 – 10 years prior to death, in the person from the grave No 4025 the estimate amounts to 4 – 7 years prior to death.

BIOMECHANICAL ASPECTS OF PODOGRAMS IN CHILDREN SUFFERING FROM SOME BONE DYSPLASIAS AND CONGENITAL LIMB DEFECTS

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The paper is concerned with dependence of representative podographic measurements of children suffering from bone dysplasias and/or congenital limb defects.

The last research project was focused on podograms of children with flat foot and pes excavatus that were compared with normal podograms of a group of Czech children at the age 5 – 17 years. In three types of podograms were analysed 3 exactly defined parameters of foot: width of forefoot, width of fornix and width of heel. The forefoot width at podograms of pes excavatus and normal (healthy) foot correlates the best with body height and weight. There was established instable dependence of the forefoot width of flat foot on body weight. Among others the results lead to hypothesis that the same nosologic units of bone dysplasias should show similar extraordinary features and on the other hand similar podograms could be present at various congenital disorders.

That is why we have studied podograms of children suffering from achondroplasia (hypo-chondroplasia), osteogenesis imperfecta, vitamin D resistant rickets, multiple cartilaginous exostoses, some chromosomal aberrations and a few other bone dysplasias. The next group we have studied were foot deformities of children with cerebral palsy and further neurogenic deformities. The last group that was studied contained some congenital foot and/or limb deformities e.g. pes equinovarus, complex femur-fibula-ulna, etc. The results are documented in graphs. Atypical podograms at foot deformities of children are demonstrated. Some podograms can be used for identification of person especially these of asymmetric (unilateral) limb defects.

ANTROPOMETRY UTILISATION AT MONITORING OF CHILDREN WITH JUVENILE IDIOPATHIC ARTHRITIS

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Introduction: Juvenile idiopathic arthritis (JIA) is the most common rheumatic disease of children's age. During the course of the disease total and local defects of growth can appear. Anthropologic exami-

nation is a part of comprehensive care provided to these children. It helps to objective assessment of their clinical status and has also its place at the control of the therapy.

Patients and methods: 95 children with juvenile idiopathic arthritis aged from 3 years 5 months to 16 years 10 months (10.10 ± 4 years) were divided into 4 groups according to the form of disease. 11 children were affected by systemic arthritis, 31 by oligo-arthritis, 45 by poly-arthritis (together seropositive and seronegative), 8 by arthritis with entezithis.

Anthropometric examination took place in the years 1998 – 2002. It was focused on growth disorders, proportionality and leg length discrepancy. Anthropometric dimensions of patients were expressed in SD-score according to National anthropological survey 1991 and own reference data.

Results: Severe growth failure (body height – 2.5 SD ± 2.2 SD) was found in the patients affected by systemic form, below – 2 SD were 45 % of patients. Average height of patients with other forms was in norm, at polyarthritis (– 0.4 SD ± 1.4 SD) and at oligo-arthritis (– 0.1 SD ± 1.2 SD), nevertheless growth retardation may occur. Body height below – 2 SD was in 11 % of patients with polyarthritis and 10 % of patients with oligo-arthritis. At patients with poly-arthritis and oligo-arthritis relatively shorter upper body segment was detected. Leg lengths discrepancy (overgrowths) was found at all forms of disease except the systemic form. Biomechanically serious overgrowths (from 1 cm to 3 cm) appeared at 21 % patients with oligo-arthritis and at 11 % with poly-arthritis. All these discrepancies were treated conservatively.

Conclusions: The most significant deviation from the norm at JIA is the growth retardation, which affects mostly the patients with systemic form, nevertheless in a lesser degree it appears also at other forms. The next important problem is occurrence of biomechanically serious overgrowth over 1 cm. Dependence of growth disorders on the disease activity and therapy are a subject of our further investigation.

DETAILED STATISTICAL MODEL FOR MONITORING INDIVIDUAL CHILDREN GROWTH

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We will present a flexible statistical model, useful for monitoring children growth individually. That is, to test whether a particular child conforms with typical growth form and to detect quickly if it begins to show unusual growth-curve patterns and/or starts to lag behind a population standard. Results serve as a screening tool and detected suspicious cases can be sent to a pediatric examination in order to determine whether any particular medical action is warranted. The model is based on state-space modeling and subsequent Kalman filtering of individual child measurements series. We will discuss some attractive features of model-implied variability decomposition into inter-individual growth curve shape (i.e. structural) variability and measurement error variability. Rather detailed covariance structure modeling yields also some interesting biological insight. Testing for possible covariates effects (like various proxies of socio-economic status)

can be accommodated and its examples will be discussed. The model is designed in such a way that it can deal with short time series of individual measurements as they occur in pediatric practice. This is achieved by „borrowing information“ across individuals when estimating certain state-space model parameters. Model is „trained“ on a relatively large dataset, but once its parameters are estimated, it can be applied on individual level. We will demonstrate utility of this model for short-term growth prediction and its sensitivity when it is used for monitoring based on detection of certain unusual growth curve patterns, lagging behind growth standards, etc. To illustrate the model's real-life data performance, we will analyze some measurements obtained from a recent Czech semi-longitudinal anthropometric study.

This research is supported through the grant of the Czech Ministry of Health, IGA NJ/6792-3.

6th NATIONWIDE ANTHROPOLOGICAL SURVEY OF CHILDREN AND ADOLESCENTS 2001 (AGE 0 TO 18 YEARS)

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In 1895 the Czech physician and anthropologist Prof. Matiegka made anthropological measu-rements of 100 000 schoolchildren in Bohemia and Moravia. Since 1951 every 10 years a similar investigation is made, in 2001 already the sixth survey was implemented. Part of these surveys was also assessment of the bodily dimensions of parents and some socio-economic characteristics of the child's family.

Material

Nationwide survey in 2001

- Number of children: 55 000 in age 0 to 18 years
- Preschool children: measured in clinics, by instructed health professionals, mainly paeditricians
- School children: measured by teachers, provided with detailed written instructions
- Number of clinics: 186
- Number of schools: 310
- Anthropological measurements: height, body weight, circumferences of the head, arm, waist and hip – Martin Saller method
- Questionnaires for children: dietary habits, sports activity, TV watching and PC games
- Questionnaires for parents: parental height, weight, educational level, number of children in the family, breastfeeding and health status of their child

Objective

1. To assess secular trend of the height. 2. To assess the prevalence of overweight and obesity in the child and adolescents population, 3. To assess the association between height and weight (BMI) of children on the one hand, the size of the community where the child lives and the parents' education on the other.

Methods

The analysis was based on data assembled within the framework of the 6th Nationwide Anthro-pological Survey of Children and Adolescents in 2001. The ratios of overweight and obese children were compared – cut-off points according a) to Czech standards, b) to Cole et al. BMJ 320:1 – 6, 2000. As socio-economics groups size of community (5 categories) and the parents' education (3 categories) were used. For statistical processing of means and for calculating χ^2 tests EpiInfo software was used. χ^2 tests were made separately for every investigated factor.

Results

The mean height of twelve-year old boys increased between 1895 and 2001 by 17.3 cm, the height of girls by 18.9 cm. As assumed, in recent years the secular trend of height was arrested. Between 1991 to 2001 the mean height of twelve-year-old boys increased by 2.1 cm, in girls only by 1 cm.

The ratio of obese children (> 97^{th} centile), as compared with Cole's reference data, is 2.4 % boys and 1.6 % girls. The proportion of overweight children (90^{th} – 97^{th} centile) is 12.1 % in boys and 9.8 % in girls, in age 6.00 to 17.99 years. The ratio of obese and overweight children rises significantly with the declining size of the community and lower education of the parents. Comparing with Czech reference data assembled in 1991, the ratio of obese is 4.7 % boys and 3.8 % girls. The ratio of overweight is 7.4 % and 6.5 % resp. The ratio of overweight and obese declines with age of the child. The χ^2 tests confirmed the association between BMI values of the child on the one hand and education of the parents, size of the community and number of children in the family on the other.

This research is supported by the Internal Grant Agency MofH CR, grant no. NJ/6792-3.

TISKOVÉ CHYBY

ERRATA

1. Redakce časopisu se omlouvá autorům (M. Adam, H. Hulejová, P. Špaček) práce **„Comparison of the efficacy three different therapy kinds in osteoarthritis“,** která byla omylem uveřejněna v PÚ 9, 2002, č. 3 – 4, str. 61 v neúplném znění. Původní práce je zde uveřejněna v plném rozsahu včetně tabulek a obrázků.

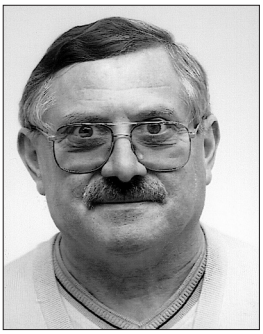
2. Redakce časopisu se omlouvá za záměnu jmen autorů referátů ze sjezdů E. Strouhal a M. Kuklík v PÚ, 9, 2002, č. 3-4, s. 121. Správně:

10TH INTERNATIONAL CONGRESS OF HUMAN GENETICS AUSTRIA CENTRE VIENNA, AUSTRIA, MAY 15 – 19, 2001 ORGANISED ON BEHALF OF THE INTERNATIONAL FEDERATION OF HUMAN GENETICS SOCIETIES BY THE EUROPEAN SOCIETY OF HUMAN GENETICS (ECHG)

M. KUKLÍK

Institute of Rheumatology, Prague, Czechia

ŽIVOTNÍ JUBILEA
ANNIVERSARIES



Doc. RNDr Pavel Bláha, CSc. – šedesátiletý

Pavel Bláha je Jihočech. Narodil se v Červeném Újezdě u Písku 20. 11. 1943. Středoškolské vzdělání absolvoval v Písku. V letech 1961–1966 studoval na Přírodovědecké fakultě UK v Praze obor biologie a chemie se specializací v antropologii. Jeho první zaměstnání v oboru bylo v Kriminalistickém ústavu, které mu nebránilo zvýšit svou kvalifikaci na základě rigorózních zkoušek titulem RNDr. v r. 1970. V práci si vedl zdatně, avšak z místa musel v r. 1974 odejít pro údajnou politickou nezpůsobilost (po roce 1991 byl plně rehabilitován). V oboru počal znovu pracovat od roku 1976 v Ústavu sportovní medicíny MZ v Praze (původně Odd. zdravotního zajištění vrcholového sportu a poté ÚNZ pro vrcholový sport) do r. 1998. Během tohoto zaměstnání pokračoval v odborném studiu na Katedře antropologie UK jako aspirant (1986–1989) a získal titul CSc. Od podzimu r. 1994 působí na Katedře antropologie a genetiky člověka na Přírodovědecké fakultě UK. Na základě habilitačního řízení na Katedře antropologie Masarykovy university v Brně získal docenturu v březnu 1997, když tam již po dva roky zajišťoval blokovou výuku fyzické antropologie (1995 a 1996).

Jako pedagog působil v kurzech a odborných stážích na subkatedře tělovýchovného lékařství 1. LF UK a v Ústavu tělovýchovného lékařství 3. LF v letech 1980 až 1989 a jako instruktor antropometrie pro hygienickou službu Středočeského kraje od r. 1991 do r. 1995.

Těžiště jeho práce bylo ve výzkumu, kde organizoval sběr dat a jejich zpracování při Čs. Spartakiádách v letech 1980, 1985 a Pražských tělovýchovných slavnostech 1990. V témž roce provedl výzkum tělesných rozměrů dětí od 3 do 7 let. Tehdy jsem ho přizval k spolupráci na 5. Celostátním výzkumu dětí a mládeže v r. 1991, při čemž osvědčil své zkušenosti a kontakty. Od té doby se datuje jeho spolupráce s mou dlouholetou spolupracovnicí Ing. J. Vignerovou, CSc. na řadě publikací a výzkumných projektů, sledujících vliv různých faktorů na růst a dětskou obezitu, zakončená 6. Celostátním výzkumem dětí a mládeže v r. 2001. Kromě toho se věnoval s jinými spolupracovníky a kolektivy výzkumu dalších problémů (objasnění náhlých změn ve tvaru hlavy u naší populace, sledování intenzivního tréninku vrcholových gymnastek na jejich tělesný rozvoj, prevence a hodnocení dětské obezity.

Hlavními výsledky vědecké a výzkumné práce doc. Bláhy jsou normy dětí i dospělých, které dovedl do stadia publikací dostupných všem zájemcům a vypracování počítačových programů několika generací, které vedou k automatickému zpracování antropometrických dat (včetně složitých výpočtů jako např. složení těla podle Matiegky aj.).

Doc. Bláha je vynikající organizátor a nikdy neváhal vzít na sebe zodpovědnost za akce nejen národního, ale i mezinárodního významu, které dovedl do zdárného konce. Svědčí o tom uspořádání kongresu k 50. výročí úmrtí Dr. A. Hrdličky v Praze 1993, antropologických dnů v Liblicích v r. 1996, 4. Mezinárodního kongresu Aleš Hrdlička v r. 1999, Workshop European Child Obesity Group (ECOG) v r. 2002 nebo Mezinárodního antropologického kongresu „Antropologie a společnost" v r. 2003.

Od r. 1998 stojí v čele České společnosti antropologické jako její předseda, je členem řady českých i zahraničních vědeckých společností (mj. Evropské antropologické společnosti – EAA, kde je voleným členem rady, Gesellschaft für Anthropologie, European Childhood Obesity aj.). Od roku 2001 se stal členem redakční rady mezioborového odborné časopisu Pohybové ústrojí – pokroky ve výzkumu, diagnostice a terapii.

Ocenění se dostalo doc. Bláhovi za zásluhy o Čs. Spartakiádu 1985 (stříbrná medaile) a za zásluhy o českou antropologii udělením medaile Dr. Aleše Hrdličky v r.1995.

V r. 1999 přednášel na kongresu ve Vancouveru, British Columbia, Canada, kde též absolvoval stáž, účastnil se přednáškou na Winklerově memoriálu v Xanthi v Řecku v r. 1995, na škole „Anthropologie a zdraví" na ostrově Hvaru v Chorvatsku v r. 1999 a 2000 a 2001 a semináře v Aschauhofu v Německu v r. 2000 a 2001. V r. 2000 přednášel na kongresu Německé antropologické společnosti v Potsdamu a v letech 2001 a 2003 absolvoval stáž na Fridrich-Schiller Universitaet v Jeně.

Každý český antropolog zná a užívá Bláhovy knihy – manuály rozměrů dětí a dospělých (1986). V zahraničí jsou známy jeho publikace se spoluautory v angličtině: The growth of the Czech child during the past 40 years (Budapest, 1998), Risk Factors of Obese Czech Children (Göttingen, 2001), Bodily characteristics and Lifestyle of Czech Children Aged 7.00 to 10.99 years, Incidence of Childhood Obesity (Central Eur. J. publ. Health, 2002) a další. S J. Vignerovou Bláha redigoval publikaci: Investigation of the Growth of Czech Children and Adolescents – Normal, underweight, overweight (SZÚ, Praha 2002) s příspěvky pediatrů, endokrinologů a výživářů.

Za dobu svého působení v Ústavu sportovní medicíny se stal z doc. Bláhy aktivní sportovec a dodnes, pokud mu čas dovolí, si rád zalyžuje nebo si zahraje tenis. Pro svou práci nachází pochopení u své dcery z prvního manželství, která mu i vypomůže, když potřebuje. O svého synáčka z druhého manželství se vzorně stará. Je pro něho příznačná velká pracovní kapacita (je ranní ptáče), pečlivost a schopnost každou akci do detailu promýšlet a zajišťovat. Počáteční nezaviněný neúspěch v povolání ho nezlomil, ale zvýšil jeho průbojnost. Má schopnost si vytvářet a udržovat dobré vztahy se spolupracovníky a osobnostmi, významnými pro hladký průběh připravovaných akcí, jako např. představitelé města Humpolce či evropské asociace, vedoucí antropologové našich i zahraničních pracovišť aj.

Doc. Bláha se těší dobrému zdraví, je odborníkem ve svém oboru a šedesátka, na kterou nevypadá, mu dává dostatečný prostor pro jeho další aktivity ve prospěch české antropologie.

Miroslav Prokopec

Ke gratulaci k významnému životnímu jubileu milého kolegy pana docenta Pavla Bláhy, CSc. se připojují **členové redakční rady** s přáním stálého zdraví a tvůrčích sil do dalších let, jakož i pro vědce nezbytné podpory celé rodiny, aby úspěšně řešil další interdisciplinární výzkumné úkoly.

PROF. MUDR. MILAN ADAM, DRSC., 75 LET

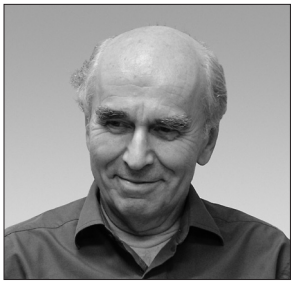
25. května 2004 oslavil profesor Adam své 75. narozeniny. Není skutečně jednoduché postihnout jeho osobnost. Profesor Adam pracoval od roku 1954 v Revmatologickém ústavu v Praze Na Slupi. Od roku 1959 podnikl řadu studijních pobytů, z nich bych uvedl alespoň časté pobyty v Max Planck Institutu v Mnichově a Martinsriedu, dále ve finském Oulu a na Universitě v Remeši. V roce 1982 byl jmenován docentem a od roku 1987 profesorem. Kromě klinických aktivit a pedagogické činnosti věnoval svou pozornost hlavně pracím z oblasti pojivových tkání, převážně kolagenu. Je autorem více nežli 280 vědeckých prací a spoluautorem řady monografií. V Revmatologickém ústavu vybudoval centrum věnované výzkumu pojiva a vychoval celou generaci revmatologů i vědeckých pracovníků. Účastnil se velkého počtu symposií a kongresů prakticky ve všech světadílech a jeho práce byla oceněna řadou cen, vyznamenání, čestných členství v domácích i zahraničních společnostech a čestného doktorátu university v Remeši. U nás založil profesor Adam v sedmdesátých letech sekci pojiva při Společnosti klinické biochemie, která se v roce 1992 transformovala do samostatné Společnosti pro výzkum a využití pojivových tkání a byl od počátku jejím předsedou. Kromě těchto aktivit nelze nezmínit jeho společenskou angažovanost. Chtěl bych uvést alespoň jeho jmenování ministrem školství v polistopadové vládě v letech 1989 až 1990.

S Milanem Adamem jsem se seznámil v roce 1972 při symposiu věnovaném kostnímu výzkumu. Milan Adam využíval při svých vědeckých pracích v oblasti peptidů a bílkovin chromatografické metody, a tak se pravidelně zúčastňoval symposií o aplikacích chromatografie v klinické chemii a biochemii, které jsem organizoval doma i v zahraničí. Z původního seznámení vzniklo naše přátelství, které trvá do dnešních dnů. Vedle společných zájmů vědeckých a organizování konferencí nás spojují i zájmy kulturní, cestovatelské a musím zde zmínit i radosti kulinářské.

Pětasedmdesáté narozeniny jsou dobrou příležitostí, abychom poděkovali Milanu Adamovi za všechno, co vykonal v oblasti revmatologie a výzkumu pojiva. Chtěl bych Ti, milý Milane, popřát stálou životní pohodu a ještě mnoho let aktivní činnosti v oblasti pojiva.

Karel Macek

Ke gratulaci k tak významnému životnímu jubileu pana profesora Adama se připojují **členové redakční rady** s přáním pevného zdraví do dalších let, úspěchů při řešení výzkumných úkolů a spokojenosti v kruhu rodiny.



DOC. RNDr. Karel Macek, DrSc. – 75 let

Pan docent Karel Macek se narodil 31. 10. 1928 v Praze. Absolvoval obor biochemie na Přírodovědecké fakultě UK v r. 1951, o deset let později obhájil titul kandidáta chemických věd. Již v době studií pracoval jako demonstrátor na I. ústavu lékařské chemie FVL UK v Praze.

Po studiích pracoval v Biochemickém výzkumném ústavu, od r. 1951 transformovaném do nově vzniklého Výzkumného ústavu pro biochemii a farmacii. Zde byl jako vedoucí vědecký pracovník až do r. 1968. V letech 1969 – 70 byl vedoucím biochemických laboratoří na interním oddělení FN Praha - Strahov, v letech 1971 – 1977 působil jako vedoucí biochemických laboratoří III. interní kliniky FVL UK a od r. 1977 jako vědecký pracovník Fyziologického ústavu ČSAV v Praze.

Ze zahraničních pobytů nutno vzpomenout postgraduální studium v Göttingen v r. 1957. V r. 1966 byl hostujícím profesorem nejprve na univerzitě v Mnichově, v letním semestru 1968 na Chelsea College v Londýně a v letech 1968 až 1969 na univerzitě v Římě. Zaměřil se na studium chromatografických metod, které aplikoval v analýze léčiv a v lékařství vůbec. Studoval zákonitosti planární chromatografie, navázal pak na tyto znalosti v oblasti aplikace papírové chromatografie v toxikologii, klinické biochemii, farmakokinetice a základním výzkumu. Chromatografie jej přivedla ke studiu pojivové tkáně.

Je autorem a spoluautorem desítek vědeckých prací, několika desítek monografií, několika patentů. Jeho klasické a stěžejní dílo je „Papírová chromatografie“ z r. 1954, přeložená nejen do světových jazyků angličtiny, ruštiny a němčiny, ale i do rumunštiny a maďarštiny. Nutno připomenout jeho redakční činnost (od r. 1961) v Journal of Chromatography (B). V r. 1960 založil Československou chromatografickou společnost a do roku 1990 byl jejím předsedou. Organizoval řadu sympozií, sám pak přednášel téměř na celém světě.

Za jeho zásluhy mu byla udělena Cvětova medaile akademie věd SSSR v r.1978 a Hanušova medaile Čs. společnosti chemické a v r. 1985 americká Tswettova medaile.

Jeho vystupování je vždy vysoce kulturní a decentní, má široké zájmy i uměleckého charakteru. Zde kombinoval profesionální zájem se zájmem uměleckým: zajímal se o chemickou analýzu barev obrazů, s možností určení stáří, oprav a přemalování. Patnáct let studoval operní zpěv, v některých rolích pak v divadle uplatnil svůj tenor.

V posledních dvanácti letech pracoval mimo jiné jako místopředseda Společnosti pro výzkum a využití pojiva, k jejímž zakládajícím členům v r. 1992 patřil. Při organizování odborných akcích Společnosti, přednáškách a zasedáních výboru jsme měli možnost se blíže seznámit s jeho nevšední osobností, vyznačující se přesností, smyslem pro přátelství i s mladšími kolegy a vždy charakterním noblesním jednáním. Jeden z jeho dvou synů se věnuje též biochemii.

Velmi si vážíme možnosti podílet se s jeho zkušenostmi při publikační činnosti i organizování odborných akcí Společnosti pro pojivové tkáně. Jubilantovi upřímně přejeme mnoho zdraví a splnění jeho plánů do budoucna.

Za redakční radu
MUDr Miloslav Kuklík, CSc. a MUDr Ivo Mařík, CSc.
Ambulantní centrum pro vady pohybového aparátu
Olšanská 7, 130 00 Praha 3

PROF. MUDR. JOSEF HYÁNEK, DRSC. – 70 LET

V roce 2003 oslavil své sedmdesátiny náš milý a vážený kolega pan profesor MUDr. J. Hyánek, DrSc.

Narodil se v roce 1933 na Valašsku, gymnasium navštěvoval ve Zlíně. 1957 promoval na Fakultě dětského lékařství UK v Praze, atestace z pediatrie a klinické biochemie skládal v letech 1960-66; 1975 obhájil disertaci s tématikou dědičných poruch metabolismu aminokyselin, 1980 habilitoval na docenta pro obor klinická biochemie a profesuru pro tento obor získal 1985 po obhajobě doktorské disertace z biochemie na ČSAV.

Profesně působil jako sekundární a později samostatně pracující odborný lékař na dětském a infekčním oddělení nemocnice v Uherském Hradišti a později ve Fakultní nemocnici II v Praze. Od roku 1968 do 1984 pracuje jako odborný asistent a později docent Oddělení klinické biochemie FVL UK v Praze. Přednostou na stejném oddělení působí v letech 1984–1992, poté odchází a vede nově otevřené Oddělení klinické biochemie, hematologie a imunologie Nemocnice Na Homolce, kde působí dodnes.

Byl zakládajícím členem čs. společnosti klinické biochemie a čs. spol. lékařské genetiky a dlouholetý člen jejich výborů, člen čs. pediatrické společnosti, kde je aktivní v její Komisi pro prevenci aterosklerózy u dětí a adolescentů. Člen Society for Inborn Errors of Metabolism (Liverpool), IFCC (Washington), AACC (Washington), ISNS (Tokyo), Čs. a Evrop. společnost pro aterosklerózu; short term consultant of WHO; člen redakčních rad časopisů: Klin. Biochemie – Metabolismus, J. Inher. Metabol. Diseases, Screening, Pohybové ústrojí.

Vydal 2 knihy o dědičných metabolických poruchách, je spoluautorem 5 dalších monografií, uveřejnil v odborných lékařských časopisech více jak 200 publikací, přednesl více než 300 přednášek na domácím i zahraničním fóru. Organizoval 19 národních a 6 mezinárodních symposií, kongresů a seminářů. Byl úspěšným řešitelem 12 grantových úkolů.

Odborný zájem v posledních letech věnuje primární prevenci kardiovaskulárních onemocnění u dětí, především hypercholesterolemií. Tato onemocnění se po zavedení povinného selektivního screeningu v naší rizikové dětské populaci stala velkým diferenciálně diagnostickým problémem. Dále se věnuje diferenciaci hyperhomocysteinemií, protože problematika hypomethylace, jejíž důsledkem hyperhomocysteinemie vznikají – získává stále více na významu ve spojení s nutrigenomikou hlavně v prevenci cévních a pojivových onemocnění.

Vedení komise pro dědičné metabolické poruchy v ČR předal už svým mladším následovníkům, ponechal si jenom narůstající problematiku mateřské hyperfenylalaninemie. Byl zvolen předsedou Sdružení chronicky nemocných dětí v ČR.

Stále je žádaným, uznávaným i obávaným oponentem disertačních prací, členem nebo předsedou habilitačních komisí nejen na lékařských fakultách, ale i např. na Přírodovědecké fakultě UK v Praze.

Rádi bychom uveřejněním tohoto krátkého neúplného přehledu činností poděkovali našemu milému kolegovi za vše co pro dětské a dospělé pacienty vykonal, za jeho rozsáhlou

odbornou a vědeckou práci a přínos jeho osobnosti pro studenty, kolegy, českou medicínu, ale i za reprezentaci našeho státu v době minulé i současné.

Členové redakční rady upřímně přejí panu profesorovi pevné zdraví, spokojenost v kruhu své rodiny a další úspěchy na poli medicíny a vědy.

OZNÁMENÍ ÚMRTÍ

OBITUARY

VZPOMÍNKA NA PROF. PHDR. ET PAEDR. VLADIMÍRA KARASE, DRSC.

Ve čtvrtek 19. 6. 2003 náhle zemřel prof. PhDr. et PaeDr. Vladimír Karas, jeden ze zakladatelů české biomechaniky. Ti kteří ho znali, věděli, že zemřel člověk s nesmírnou houževnatostí, invencí a inteligencí v různých činnostech, kterým se věnoval. Zemřel moudrý učitel, kolega a kamarád, který byl vzorem, kritikem a rádcem. Jeho sarkastický humor, zarputilost v hledání řešení a pedagogická laskavost a liberální postoje v názorové rozmanitosti byly pro něj typické.

Pan profesor Karas se narodil 22. 5. l927, maturoval na reálném gymnáziu 1946 a v roce 1950 absolvoval Přírodovědeckou fakultu UK v Praze a Vysokou školu pedagogickou v Praze. Na VŠP působil jako asistent a v roce 1953 zde získal titul PaeDr., později i PhDr. Vysoká škola pedagogická byla sloučena s Fakultou tělesné výchovy a sportu UK, po sloučení pracoval nejprve na katedře gymnastiky, později přešel na oddělení biomechaniky.

Zpočátku se věnoval sportovní gymnastice, byl olympijským reprezentantem na olympiádě v Londýně v r. 1948. Později se změnil v předního evropského biomechanika, tento přerod však nebyl snadný. V biomechanice se zabýval nejdříve sportovní biomechanikou, především závažnému tématu analýzy labilních stavů ve sportovní gymnastice. Propracoval velmi originální problematiku metody měření momentů setrvačnosti segmentů lidského těla. Těsně spolupracoval se strojní fakultou ČVUT na katedře mechaniky a velmi dobře zvládl technickou mechaniku v aplikacích pro pohyb lidského těla. Titul CSc. získal v oblasti pedagogických věd v r. 1963. Potom se věnoval biomechanice jak pedagogicky, tak i vědecky. Na l. mezinárodní sympozium o biomechanice v Curychu v r. 1964 vycestoval pololegálně, vystoupení bylo utajené, protože oficiálně ze socialistické vlasti vycestoval jako expert na lanovky. Ohlas jeho vystoupení byl mimořádný a rychle se zařadil mezi přední evropské experty v biomechanice a u nás se stal jedním ze zakladatelů české biomechanické školy. Jeho biomechanické analýzy sportovně gymnastických prvků jsou dodnes velmi populární nejen doma, ale i v zahraničí. Složitá mechanická řešení zjednodušoval do sdělitelné praktické instrukce. Brzy zjistil, že přístup pouze z pozic technické mechaniky není sám o sobě schopen odhalit důležité aspekty příčin a vnitřních důsledků pohybu živého organismu.

V letech 1971–72 obhájil habilitační docentskou práci na téma „Teoretické základy biomechaniky lidského svalu“. V r. 1980 obhájil doktorskou disertaci na téma „Biomechanika struktury a chování pohybového systému člověka při volní motorické činnosti“ , která již obsahovala i kriminalistický aspekt při analýze bipedální lokomoce.

Získal titul DrSc. v oblasti biologických věd pro obor biofyzika. V r. 1983 byl jmenován profesorem pro obor „Teorie tělesných cvičení se zřetelem na biomechaniku".

V letech 1980 až 1993 byl vedoucím katedry anatomie a biomechaniky FTVS UK. Byl spoluzakladatelem Československé společnosti pro biomechaniku, v letech 1991 až 1994 jejím předsedou. Byl členem řady národních a mezinárodních vědeckých společností: Československá společnost pro mechaniku, Československá fyziologická společnost, Československá kybernetická společnost, International Society of Biomechanics, International Society of Electrophysiological Kinesiology. Jeho publikační činnost byla mimořádně rozsáhlá. Původní vědecká sdělení publikoval v zahraničních a národních vědeckých časopisech s významným citačním indexem.

Profesor Karas se stal zakladatelem forenzní biomechaniky, neboť se zajímal o biomechaniku pohybového systému člověka a měl vždy smysl pro řešení „záhad". V 80. letech tak začal řešit praktické otázky předpovědi a kvantifikace mechanického zatížení organismu a definice hranic tolerance organismu na vnější dynamickou zátěž. Postupem času získal i v této oblasti mezinárodní prestiž a stal se zakladatelem forenzní biomechaniky v Čechách. Ve forenzní biomechanice působil jako soudní znalec i jinak velmi aktivně až do konce svých dnů, v době, kdy pro zrak lékaře znatelně přibývalo projevů jeho fyzických zdravotních obtíží.

Na Ústavu soudního inženýrství VUT v Brně pan profesor Karas přednášel řadu let soudním znalcům – analytikům silničních nehod vybrané statě z biomechaniky, předmět, který v tomto kurzu sám založil a jenž znalcům přináší zcela odlišné pohledy na aplikace mechaniky. Také na konferencích byly jeho přednášky velkým objevem pro řadu znalců, jak analytiků silničních nehod, tak i soudních lékařů.

Celoživotní úsilí pana profesora Karase bylo věnováno rozvoji biomechaniky jak v oblasti teoretické, tak aplikační, zejména v oblasti tělesné výchovy, sportu, rehabilitace a kriminalistiky. Jeho přístup zahrnoval jak studium vnějšího pohybového projevu, tak i činnost vlastního pohybového ústrojí a podmínky vnějšího prostředí. Dovedl získat pro biomechaniku i lékaře, což vedlo k přímým aplikacím nejnovějších poznatků biologie, a k syntéze lékařských technických oborů v biomechanice. Řada z nás, kteří jsme se podíleli společně s ním v osmdesátých letech na tzv. Státním plánu základního výzkumu ČSAV (SPZV) v oblasti interakcí člověka a prostředí považovali jsme si za čest s ním spolupracovat.

Nelze opomenout ani skutečnost s jakým úsilím a houževnatostí prosazoval uznání biomechaniky v tehdejší ČSAV v oblasti lékařských věd v jednání u akademika Houšťka.

Celkový přínos profesora Karase je obtížné ve stručnosti uvést, měl široký záběr odborného a společenského působení včetně kriminalisticko – soudních aplikací. Přispěl k budování nových moderních základů kriminalistiky stop a identifikace, včetně trasologie. Významně se podílel na vzniku a tvorbě identifikačních systémů.

Profesor Karas miloval akademické debaty, měl nekompromisní intelekt. Smysl pro humor a zarputilost mu význačně v těchto debatách i v životě pomáhaly. Jeho humor přecházel často v sarkasmus, uměl se vysmívat naivnímu optimismu, kterému řada z nás podléhala. Sám se řadil mezi tzv. konstruktivní pesimisty, jeho kritika skutečně nebyla nikdy destruktivní.

Pro všechny z nás zanechal výzvu k řešení otázek lidského pohybu, navíc určité detaily životního údělu zůstávají v paměti těch, kteří ho nějakým způsobem znali, mnozí si určitě občas vzpomenou.

Dr. h.c. Prof. JUDr. Ing. Viktor Porada, DrSc.
Prof. PhDr. Jiří Straus, DrSc.
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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. Časopis má zájem otiskovat články kvalitní, vysoké odborné úrovně, které přinášejí něco nového a jsou zajímavé z hlediska aplikací a nebyly dosud nikde uveřejněny s výjimkou ve zkrácené formě.

Redakce přijímá původní práce a kasuistiky, souborné články, které informují o současném stavu v příslušných oblastech souvisících s pohybovým ústrojím a abstrakty příspěvků z národních a mezinárodních konferencí, věnovaných hlavně pohybovému ústrojí. Původní práce a kasuistiky doporučuje publikovat v anglickém jazyce. Rukopisy jsou posuzovány dvěma (někdy i třemi) oponenty redakční rady.

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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, diagnostical methods, orthopaedic and traumatological problems, rehabilitation as well as the medical treatment and preventive care of skeletal diseases. The object of interest are interdisciplinary papers of paediatric orthopaedics and osteology, further object of interest are problems of biomechanics, pathobiomechanics and biorheology. 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.

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Manuscripts should be submitted in original (we recommend to the authors to keep one copy for eventual corrections), printed double-spaced on one side of the page of size A4 with wide margins. The contributions (including Illustrations and Tables) has to be submitted in the well-known computer programs on disk.

While no maximum length of contributions is prescripted, 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 author should be indicated who is to receive correspondence and proofs for correction. Papers are reviewed by two (and/or three) opponents.

The second page should contain a short abstract about 100 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 layout depends on the character of communication. The paragraphs should begin five free spaces from the left margin and contain at least four rows.

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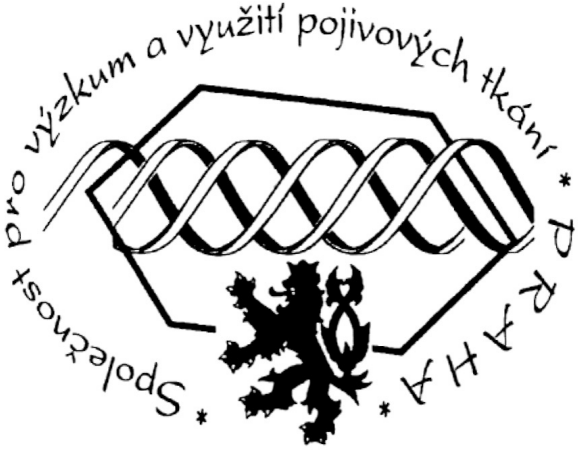
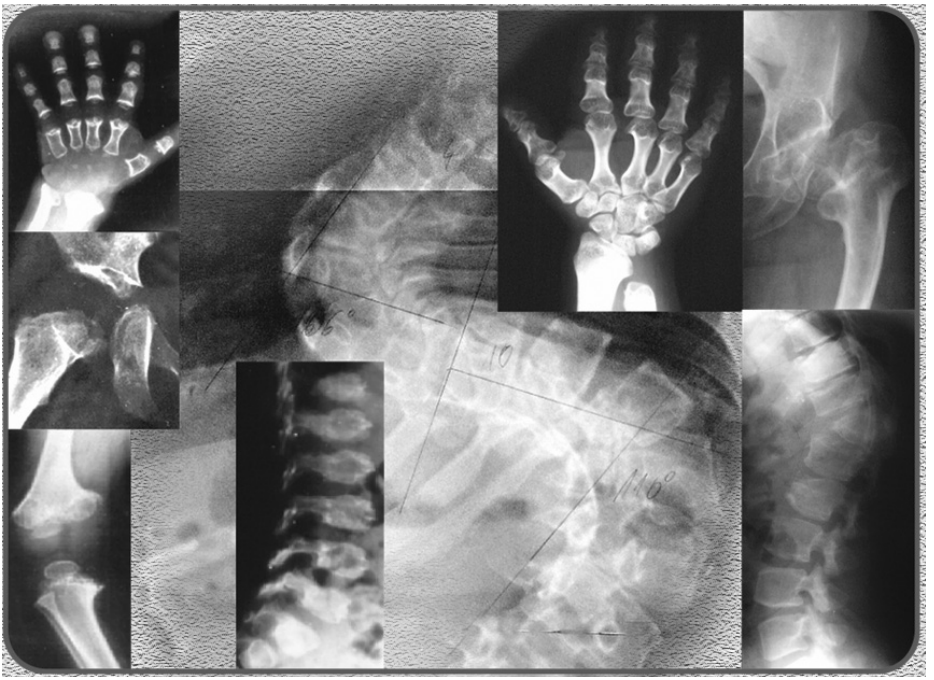


Společnost pro výzkum a využití pojivových tkání je od roku 2003 vydavatelem časopisu Pohybové ústrojí.

OBRÁZEK NA TITULNÍ STRANĚ ČASOPISU



Charakteristické plně vyjádřené RTG příznaky **Pseudoachondroplazie,** biomechanicky závažné, zpravidla autosomálně dominantně dědičné, spondyloepi(meta)fyzární kostní dysplazie (KD). Jedná se o raritně se vyskytující (6 : 1 000 000) KD s krátkými končetinami a relativně dlouhým trupem, kterou P. Maroteaux a M. Lamy vyčlenili ze skupiny spondyloepifyzárních dysplazií v roce 1959.

Vlevo na obrázku je rentgenogram ruky, kyčelního, kolenního kloubu a bederní páteře dítěte předškolního věku s nápadnou dysplastickou osifikací hlavic femurů, retardací osifikace, krátkými metakarpy a falangami a těžkými dysplastickými změnami obratlových těl, epifýz a metafýz - tyto změny je možné hodnotit pouze v období růstu, kdy jsou epifýzy a metafýzy dlouhých kostí odděleny růstovými chrupavkami.

Uprostřed a vpravo na obrázku jsou velmi těžké deformity páteře (skolióza 4. stupně dle Cobba a gibbus v důsledku klínovitého těla obratle Th 12), Madelungova deformita distálního předloktí a subluxace kyčelního kloubu s defektním vývojem krčku i hlavice femuru a acetabula dospělého pacienta. U všech spondyloepi(meta)fyzárních KD je nutno cíleným radiologickým vyšetřením zhodnotit vývoj dens epistrophei, protože jeho možná hypo- nebo aplazie může spolu s hyperlaxicitou ligament být příčinou atlantoaxilní instability s možným důsledkem neúplné či úplné příčné léze míšní.

MALÍK a spol. s.r.o.

Technicko-protetická péče

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