Hypothesis of microfractures by buckling theory of bone's trabeculas from vertebral bodies affected by osteoporosis

NINA IONOVICI1), M. NEGRU2), D. GRECU3), MIRELA VASILESCU4), L. MOGOANTĂ1), ADRIANA BOLD1), RODICA TRĂISTARU5)

1)Department of Histology, Faculty of Medicine, University of Medicine and Pharmacy of Craiova
2)Department of Strength of Materials, Faculty of Mechanics, University of Craiova
3)Department of Orthopedics, Faculty of Medicine, University of Medicine and Pharmacy of Craiova
4)Department of Kinetotherapy, Faculty of Sports, University of Craiova
5)Department of Physiokinetotherapy, Faculty of Medicine, University of Medicine and Pharmacy of Craiova

Abstract
Osteoporosis has become in recent years a public health problem considered a true "silent epidemic", by increasing the number of osteoporosis fractures in the world as a result of increased number of persons 3rd group of age by increasing life expectancy and reducing physical effort and the emergence of sedentary occupations, increasing incidence of obesity, diabetes, liver disease and kidney by applying widely corticosteroid therapy. Starting from the macroscopic and microscopic aspects of the bone spongy tissue affected by osteoporosis, from vertebral bodies, we try to explain the modality of damaging the bone micro-structure by buckling phenomenon, knowing that the bone tissue has at trabecular level, an elasticity degree and supports high levels of mechanical forces.

Keywords: osteoporosis, bone spongy tissue, vertebral body, buckling.

Introduction
Osteoporosis is a generalized bone disease characterized by reduced bone mass and deterioration of micro-architecture leading to bone fragility and increased risk of fracture [1]. Reduced bone mass is due to a deterioration of the process of renewing normal bone tissue, with increased imbalance between the processes of bone formation and resorption.

Osteoporosis is caused by many factors: endocrine dysfunction (hypoestrogenism, Cushing syndrome, hyperparathyroidism, hypogonadism, acromegaly, diabetes mellitus type I), rheumatoid arthritis, chronic liver or kidney disease, nervous anorexia, inflammatory bone disease, chronic treatment (with corticosteroids, heparin, or other cytotoxic cyclosporine) or other factors (early menopause, age, excess alcohol, poor nutrition). Although clinical manifestations are minor, osteoporosis, with bone structure fragility, is responsible for the occurrence of fractures from minor injuries. The most common “osteoporotic” fractures appear at the distal epiphysis of radius, hip, humeral neck, vertebral bodies, ankles and even ribs. Female sex is mainly affected and at an early time, the women/men at 3/1 – 4/1, female menopause is the main element causatives incriminate [2]. Numerous epidemiological data show that about 40% of women over 50 years and 10% of men of the same age are victims of osteoporotic fractures by the end of life [3]. Other authors [4] believe that 51% of fracture in women and 24% of the fracture occurred in men diagnosed with osteoporosis. In the USA are recorded annually around 1.5 million osteoporotic fractures, of which about 250 000 are for hip fractures [5]. Social costs caused by osteoporotic fracture are very high. In the United States the estimates are about 17 billion dollars are spent annually for treatment and recovery of these fractures. Statistical data show that global osteoporosis tends to become a major public health problem. Thus, in Switzerland, the number of days of hospitalization for osteoporosis is much higher than for myocardial infarction, stroke or breast cancer [6].

Material and Methods
The study was performed on the body vertebral collected from 16 patients with osteoporosis (12 women and four men) aged between 60 and 82 years old. Anatomical pieces were collected in the Service of Pathological Anatomy of the Emergency County Hospital of Craiova, and in the Department of Human Anatomy, Faculty of Medicine, University of Medicine and Pharmacy of Craiova. After we obtained the biological material, we fixed them in 10% neutral
formalin for 30 days, cuts with a jigsaw pieces were achieving a thickness of 3–4 mm, which were subsequently decalcified in a 10% aqueous solution of trichloracetic acid and included in paraffin, using the classic histological technique. There were cut in a microtome section of 5 µm thickness, which were then colored with Hematoxilin–Eosin and with green light based trichrome Goldner–Szeckelly.

Results

Macroscopic appearance of vertebral body evidenced the different stages of osteoporosis, made evident by the deformation of vertebral body, but also by modifying trabecula appearance of spongy bone. In the section on the vertebral body, there was reduced trabecular density, mainly in the central area of the vertebral body, especially in the horizontal trabeculae considered the link trabeculas. Vertical trabeculas considered resistance trabeculae were rarefied, deformed, some with less thickness, others non-homogenously hypertrophied. These changes of the trabeculae have led to the appearance of areola spaces that increased in volume, sometimes taking the appearance of cystic cavity with totally irregular contour prevailing trabeculae’s scrap (Figures 1 and 2).

All these changes explain the deformation of vertebral body, which sometimes had the appearance of “fish vertebra” by increasing vertebral plate’s concavity due to decreased resistance, which in turn is dependent on mass marrow, the thickness of cortical bone, the spongy bone architecture and vertebral body dimensions. Vertebral bodies with advanced osteoporosis in the thoracic region had changes of shape and sizes having a cuneiform aspect. Destruction and spongy bone tissue remodeling led some bone cavity to suffer large deformation and damage bone trabeculae. Damage of bone trabeculae did not affect only the central portion of the vertebral body, but has also radially spread to the edges of vertebral body. By deterioration of link trabeculae, some resistance were curved, limited large areola spaces between them, compensatory thickened or, on the contrary, thinned.
Hypothesis of microfractures by buckling theory of bone’s trabeculas from vertebral bodies affected by osteoporosis

It is possible that the processes of reshuffling and remodeling to be dominated by processes that affect progressive resorption and cortical tissue from the periphery of vertebrates, including the shift towards cartilage tissue of intervertebral disc, with increasing porosity and reducing vertebral mechanical resistance, which explains deformation vertebral body under the bodyweight.

Microscopic study confirmed the macroscopic observations. In histological preparations colored in classical spongy bone tissue appeared thin, composed of the trabeculas with more reduced thickness, with areolare wide cavity filled mostly with yellow bone marrow. The link trabeculaes were some microscopic fields absent and others were thin, with non-homogenously width or “disrupted” (Figures 5 and 6).

Some resistance trabeculae occurred thinned other non-homogenously thickened, evidence of inefficient marrow remodeling processes. In the trabecula, there were rarefied, smaller osteocytes with pycnotic nucleus and reduced cytoplasm, a clear evidence of the reduction of the synthesis of bone matrix. The osteoprogenitors cells of endostium were rarely emphasized, so that in some areas of trabecular bone the endost seemed absent. This microscopic appearance reduced bone forming of osteoporotic tissue (Figures 7 and 8).

Starting from these macro-and microscopic aspects and taking into account the mechanic support function of the bones, we tried to find a mechanical explanation of the bone fracture process. Of the studied mechanical theories, the most plausible theory was the buckling theory of resistance trabeculae.

In the images taken of the microscopic samples we noticed the slight bending of the bone crossbeams, bending which led to the trabecular bone fracture, and also the crack by chips of the trabecular bone.

From a strictly mechanical point of view, we considered the spongy bone as being a spatial structure of beams with variable widths and directions, known in engineering as “braced girder”.

According to the classical mechanics theory, the structure has a good behavior in time, as the solicitation of the component beams is extension-compression. After the osteoporotic damage a part of the crossbeams of link are affected by absorption, losing the material continuity and, by consequence, the mechanical role. The crossbeams not affected will also be axially loaded, but their length between two successive transversal bone links, will increase.

We consider that on these areas the bone crossbeams crack due to the loss of the elastic equilibrium stability,
phenomenon known by the strength of materials experts as buckling.

Buckling is the denomination given in Strength of Materials to the bending phenomenon of an axial beam loaded to compression. According to Figure 11, the axial beam loaded with force \( F \) should remain straight; the only deformation should be only the shortening of the beam.

![Figure 11 - Illustration of the buckling.](image)

The beam damage should be produced only when the mechanical crack stress is attained due to compression; this fact is valid in practice only if the compression force \( F \) is relatively small. It was noticed that if the beam is long enough, the beam bends and cracks when a critical force \( F_c \) is achieved, even if the compression mechanical stress is much smaller than the one which will produce the crack by compression; bending and crack are owed to buckling.

We consider that the fiber load is an elastic domain and the critical force of buckling can be calculated with Euler relation:

\[
F_c = \frac{\pi^2 E \cdot I}{l_f^2}
\]

where: \( I \) – axial inertia moment of the transversal section; \( l_f \) – length of buckling (depends on real length and a constant \( l_f = l \times k_f \)); \( l \) – real length; \( k_f \) – buckling constant; \( E \) – longitudinal elasticity modulus of the bone material.

For a better understanding of the mechanical phenomenon of buckling, we will define a very simplified bone structure in Figure 12a. If the compression mechanical load is produced, the fibers collinear with the load direction are shrinking, resisting well at load (according to Figure 12b). After absorption, a part of lateral links are breaking, appearing long fibers of resistance without lateral support, according to Figure 12c. When this damaged structure is normally loaded, on the long parts (without lateral links), the fibers bend and crack (Figure 12d).

Even at less advanced absorption, when the lateral crossbeams are broken after a decalcification less quantitatively extended, there collapses the lateral support for the resistance fibers on areas relatively long, a fact which leads to the mechanical state of instability, that is buckling.

Thus, we can emit the hypothesis that some of the bone crossbeams (those which have sufficient length) break through buckling; the breaks through buckling are not generalized, thus the local breaks, only of several crossbeams, do not instantly lead to the fracture of the respective bone. The local breaks, only of several crossbeams, are manifesting locally, at microscopic level and random, accumulate in time, diminishing the resistance of the entire bone, fact which has the fracture as the final consequence.

![Figure 12 - The schematization of the bone structure: (a) Normal structure not loaded; (b) Normal structure loaded (deformed); (c) Structure with absorptions not loaded; (d) Structure with absorptions loaded, with local buckling.](image)
Therefore, we can say that the fracture of the bone affected by osteoporosis suffers a rheologic damage of the microstructure; the damage is cumulative and has as consequence the appearing of the macro-fissure and the final fracture, fracture that is produced even at a normal mechanical solicitation of that bone. We can say that, probably, the bone affected by osteoporosis cumulates micro-defects during the normal functioning of the skeleton, inevitably leading to the fracture (fracture is not due to mechanical overload). This damaging in time is called the fatigue of the spongy structure of the bone affected by osteoporosis. The cause of damaging by fatigue is the osteoporotic decalcification of the spongy bone crossbeams at microstructure level, in different zones and moments, which will break through the buckling phenomenon; the breaks have a cumulative character and finally lead to the macroscopic fracture.

**Discussion**

Osteoporosis is the most common metabolic bone affection, considered as a true “silent epidemic” by long asymptomatic evolution, becoming most frequently clinical evident by the emergence of bone fractures from minor injuries.

In a report to the Department of Health and Humanitarian Services of the USA in 2004, osteoporosis is defined as “a public health problem undiagnosed and untreated and many patients are predisposed to an increased risk of fracture” [7]. After some research [8] each year approximately 2 million people worldwide suffered a fracture associated with osteoporosis. In 1990, there were was recorded 1.6 million hip fractures in the world and their number is estimated to reach about 6 million in 2050. Regarding vertebral fracture determined by osteoporosis, it is believed that of all osteoporotic fractures, 30% are located at the spine, but many of them remain undiscovered [9] of numerous micro-fractures of vertebral trabecular bone. The most frequent spontaneous fractures occur in the dorsal spine and the dorsal-lumbar junction [10].

Vertebral osteoporosis affects both sexes, most of all women, but its incidence increases with aging. Yoshimura N [11], pursuing a group of 400 people from rural areas in Japan for 10 years found that the prevalence of vertebral fractures was different according to sex and increased with the age from 2.9% to 25% for men aged between 50 and 80 years old and from 2.1% to 54% for women between 50 and 80 years old. The first bone change in the vertebral structure diagnosed by laboratory methods seems to occur around the age of 50 years, but microscopic and metabolic changes of the trabecular bone that start before that age are represented by the reduction in bone formation at the cells level [12].

In our study about showing the distinction between the microscopic changes of induced osteoporosis, we used vertebral bodies on persons over 50 years old. Microscopic and macroscopic changes were more intense as the patient was older. They have also been prevailing in women. Note that the shape and size of bone pieces are genetically determined. Moreover, vertebrae, the other bone pieces, are subject to processes of formation, remodeling and rehabilitation of the microscopic structure of the mechanical requirements to which they are subject. For vertebrae, increased bone mass is via periosteal apposition. The number of trabeculae during the development of vertebral body does not increase with age [13]. At puberty, increased mineral density of trabecular bone is achieved by increasing the length and thickness trabeculae, both in boys and girls. So, women and men present about the same number trabecule and the same thickness during pre-puberty growth, puberty and young adult periods [14]. Until the age of 35–40 years old, there is a balance between bone formation and resorption in the process of bone remodeling parts. After that age, the formation of bone tissue is progressively reduced due to decreased osteoblastic cells function, while bone resorption processes are maintained at the same level or stress. This leads to both sexes, a decrease in bone mass, more or less accelerated according to sex and bone mass existing. Until the age of 50–55 years, the rate of bone loss is smaller and affects almost exclusively the trabecular bone, because, after that age, osteoclastic resorption becomes more intense and affects the cortical bone. After menopause, in vertebra, the periosteal apposition is modest, while the endocortical resorption is intense [15].

In the histological preparations made by us, we showed that, in the first instance, there is a thinning and destruction of link trabeculae with the emergence of small geodes in spongy structure of the vertebral body. The bone resistance trabeculae behave as a “girder” with certain elasticity [16]. The disappearance of link trabeculae generates the buckling phenomenon, which is the consequence, in the first instance, of a compensatory thickening, a non-homogenous link of trabeculae, coming to a hypertrophic aspect of the atrophied bone, in the first stages of osteoporosis. This thickening is actually the trabeculae’s response to mechanical forces that apply on their side that leads to the maintenance of bone density but not to a full restoration of bone resistance [17].

With the advancement of osteoporosis, there is exceeded the compensatory bone capacity and breaks follow and resolve the resistance trabeculae. So appear some wider geodes in the spongy bone structure. Areolas arising in the process of marrow resorption cannot be reduced through the process of bone remodeling, resulting in areas of low resistance in the vertebral body, even if there are no significant changes in the bone mass, and leading to the bone fracture even at a less than normal mechanical load [18]. In addition, we note, as other authors [19], that reducing vertebral bone tissue at the start of bone marrow cavities and progressing towards cortex produces the so-called eccentric bone atrophy. Under the body weight, the vertebrae change their shape, the thoracic region being especially settling previously, resulting in “arrow-headed vertebrae, and the lumbar region taking part in “fish vertebra” by settling and by emphasizing vertebral plates’ concavity. Sometimes, because of the cortical bone tissue thinning from the plate it becomes the inter-
vertebral disc protrusion into the rarefied vertebral body [20], resulting in intra-spongist hernias visible on radiological images.

There are two main types of osteoporosis: primary osteoporosis, senile or involution, and secondary osteoporosis. Differences between the two are difficult and hazardous to be made in people over 50 years old [21]. In women aged between 50 and 65 years old of the rapidly developing osteoporosis. Trabecular bone resorption due to estrogen deficiency is accelerated in women of the past 2–4 Petru Rareş Street, 200349 Craiova, Romania; Phone +40251-523 654, e-mail: mogoanta@umfcv.ro

References

Corresponding author
Nina Ionovici, Assistant, MD, PhD, Department of Histology, University of Medicine and Pharmacy of Craiova, 2–4 Petru Rareș Street, 200349 Craiova, Romania; Phone +40251-523 654, e-mail: mogoanta@umfcv.ro

Accepted: October 20th, 2008

5 Conclusions
This phenomenon explains the buckling of the mechanical process of deterioration of trabecular architecture of spongy bone tissue, as confirmed by the microscopic and macroscopic level seen in vertebral body.


DUAN Y., TURNER C. H., KIM B. T., SEEMAN E., Sexual dimorphism in vertebral fragility is more the result of gender differences in age-related bone gain than bone loss, J Bone Miner Res, 2001, 16(12):2267–2275.


