REVIEW ARTICLE

Changes in bone mechanical strength in response to physical therapy

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KEY WORDS

bone mechanical strength, osteoporosis, rehabilitation

ABSTRACT

Numerous unfavorable factors that disturb the balance between resorption and bone formation affect bone mechanical strength. Also mechanical loading (body mass and muscle tension) and risk factors for osteoporosis affect bone strength.

Loading that can be applied to the bone is limited by bone structure. At the same time, loading applied to the bone may condition its structure. This is possible thanks to the cellular mechanisms of bone modeling and remodeling called adaptation.

Physical therapy affects bone mechanical strength by stimulating bone cells. Movement increases muscle strength and restores muscle cooperation, thus improving functional state and movement activity, which, in turn, indirectly increase bone strength. The analysis of the studies on bone mechanical strength confirms the efficacy of physical exercise, which is an important element of comprehensive management. A lower risk of falls and fractures has been observed in patients who had received physical therapy apart from pharmacological treatment. It has also been observed that an increase in bone mechanical strength is not necessarily reflected in densitometric evaluation.

The skeleton has a number of functions in the body: it supports, moves, protects (vital organs), and provides a supply of minerals necessary to maintain mineral homeostasis. The human skeleton consists of 213 properly formed bones, which, in order to fulfill their role, have to be light, rigid, elastic, and durable.

Modeling processes are essential in bone formation. Each bone undergoes a constant renewal with a major contribution of reconstruction processes. Remodeling is necessary to maintain the mechanical properties of the bone through replacement of an old, worn bone with a new, resistant one. Remodeling is initiated and regulated by osteocytes within the remodeling units with a major impact of sequentially functioning osteoclasts and osteoblasts.[1]

Bone remodeling takes place in a cell cluster termed the bone remodeling unit. The process can be divided into 4 phases: activation, resorption, reversal, and formation. It is thought that mechanical loading regulates the mechanism of negative feedback between bone formation and resorption. Osteoclasts are activated in response to lower loading, while osteoblasts are stimulated by increased loading.[1-3]

Numerous unfavorable factors that disturb the balance between bone resorption and formation, or inefficient osteoid mineralization, influence the mechanical strength of bones. Bone strength is also affected by mechanical loading (body mass and muscle tension) and risk factors for osteoporosis.

Loading that can be applied to the bone is limited by its structure. At the same time, loading that is applied to the bone conditions its structure. Cellular modeling and remodeling mechanisms, termed adaptation, account for this phenomenon. The shape and size of the bone are adapted in response to mechanical loading. Mechanical bone adaptation is observed in a modeling process. Bone modeling involves deposition of bone tissue without earlier resorption, while bone remodeling involves resorption and subsequent bone formation.[4-6]

Recently, it has been hypothesized that structural strength of the bone (starting from the growth period, through adolescence, to aging)

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Received: March 2, 2010.
Revision accepted: April 30, 2010.
Conflict of interests: none declared.
Pol Arch Med Wewn. 2010; 120 (9): 368-373
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is conditioned mainly by effective mechanisms of structural modeling and remodeling.

During the growth period and adulthood, the bone adapts to mechanical loading that is applied to it. Mainly modeling processes are observed, by which the shape of the bone is changed.

In the adult body, as a result of repeated load-bearing, the energy is accumulated and microfractures occur, thus reducing bone strength. Detection and removal of damage is vital for bone strength, which is maintained by the proper function of osteocytes.[7]

Osteoclasts, osteoblasts, and osteocytes participate in bone remodeling. It is hypothesized that osteocytes are the "brain" of the bone, and thanks to their numerous processes, they form a communication network. In this way, information about the surrounding environment is conveyed.

Osteocyte-canalicular system helps initiate repair remodeling through adaptative remodeling and removal of damage. Bone microfractures, which occur as part of normal functioning, if accumulated, can decrease bone strength and must be removed. Osteocytes detect damage, trigger modeling and remodeling, and adapt the bone to mechanical loading. Adaptation may be thus perceived as a mechanism that prevents damage.[8,9]

Osteocytes also participate in bone adaptation to loading by changing the size and shape of bones, and by adequate deposition of bone mass.[10]

In a cross section of long bones at different levels, different diameter of the marrow canal and different thickness of the cortical bone (caused by irregular deposition of bone mass) can be observed. Such bone structure shows that modeling and remodeling processes are diversified, depending on the needs. Deposition of bone mass is not only conditioned genetically, but also depends on dominating stress, which accounts for varied shape and structure of the bone in the human body.[11,12]

By modifying the bone structure, loading may contribute to differences in size, shape, and bone mass deposition without changing bone mass, as observed in tennis players within the humerus bone of the active hand. If no loading is applied to the skeleton, a rapid bone loss occurs, as observed in bed-ridden patients or astronauts during spaceflight.[13-15] This proves that bone strength does not have to be associated with increased bone mass, but rather results from the modification of the size and shape of the skeleton and the distribution of bone mass under the influence of loading, at the minimal growth of a new bone.[16] Significance of osteocytes in the process of bone adaptation is associated with the release of sclerostin, produced by the *Sost* gene. Increased bone loading enhances the expression of *Sost* in a given site, which influences osteogenesis. Bone formation is inhibited in the sites where no loading is applied.[1]

Excess apoptosis of osteocytes caused by estrogen deficiency or corticosteroid treatment accelerates the rate of remodeling. Apoptosis occurs during aging. The conditions for bone modeling and remodeling deteriorate, which negatively affects the bone's architecture. In postmenopausal women, the rate of bone remodeling increases as a result of constant resorption and decreased bone formation. Shorter life of osteoblasts and apoptosis of osteocytes impair the ability to repair microdamage. This leads to bone loss and deterioration of bone structure, which, in turn, reduces its mechanical strength.[17,18]

Accumulation of microdamage significantly weakens the bone, leading to fractures even in the case of minor injuries.[19,20]

Activation of bone cells (osteocytes, osteoblasts, and osteoclasts) depends on the presence or lack of loading. Progression of remodeling is also regulated by loading that results from physical activity.

To maintain proper bone structure, loading and movement should be applied with caution. Loading is adequate when it does not exceed bone strength, i.e., when the acting forces are lower than the strength of structures. If the forces are equal to or exceed the strength of anatomical and functional structures, overload occurs, which leads to bone damage.[21]

Every day, the skeleton is subjected to compressive, tensile, shearing, and torsion forces, which are part of the normal functioning of the human body. If the limit values of these forces are exceeded, fatigue fractures of the trabecular bone and disorders of the compact bone structure occur, and as a result, the bone's internal structure is impaired. If bone metabolism is normal, such fractures are efficiently removed. Reconstruction is triggered in normal bone, and the system of bone trabeculae is oriented according to the direction of the acting forces.[20]

The composition and structure of bone are adapted to loading. Bone brittleness is a result of material failure and/or structural adaptation. Not all fractures have the same etiopathogenesis and manifest with the same disturbances of bone structure, and not all occur with decreased bone mineral density (BMD). Similarly, fractures occur in people with different bone turnover, whether normal, high, or low. This proves that there are numerous factors that reduce bone mechanical strength, and therefore different methods of pharmacological treatment are suggested.

If bone loss is associated with higher bone resorption, antiresorptive drugs are indicated (e.g., bisphosphonates). In patients with inhibited bone formation, anabolic drugs are used (e.g., parathormone used cyclically), and in those with decreased metabolism, drugs with both anabolic and antiresorptive properties should be used (e.g., strontium).[22,23]

Supplementation with vitamin D (acts on muscles by increasing the number and strength of

muscle fiber) and calcium prevents a decrease in bone mechanical strength.[23]

Recently, the role of comprehensive treatment of osteoporosis has been stressed in patients, in whom the use of effective drugs in combination with calcium, vitamin D, and physical exercise promotes an increase in BMD and gives hope for a reduced risk of fractures.[24,25]

Primary muscle atrophy (sarcopenia) or secondary muscle atrophy (caused by lack of exercise) results in reduced muscle and bone mass.[26] It has been proved that muscle atrophy reduces muscle strength, but, what is even more important, it inhibits periosteal bone growth. Because women have lower bone mass than men, it may be one of the reasons why fractures are more common in women.[27] Muscle atrophy and decreased muscle strength disturb the dynamic and static balance of the body.[28] Sarcopenia reduces muscle mass and strength in elderly people, leading to balance and gait disorders.

Efficient muscle system, controlled by the central and peripheral nervous system, protects bones and joints. If bone structure is weak, lack of muscle protection can cause pain and microfractures.

Falls, which usually occur because of balance disorder, result in fractures at the sites typically associated with osteoporosis. Rizolli et al.[23] analyzed the studies published between 1985 and 2009 and suggested that the balance between bone strength and disposition to falls that end in fracture is a combination of bone disease and central nervous system disorders.

Disturbed stimulus transmission between the nerves and muscles (resulting from involution of the central nervous system), muscle failure, and structural changes in the vertebral column (birth defects, degenerative disease) lead to the development of bad posture, including more pronounced curvatures of the vertebral column. Thoracic kyphosis triggers forces that lead to even greater kyphosis. Thus, an abnormal movement pattern is formed, which affects trunk, upper and lower limb muscles, and may lead to slow fractures of the vertebral bodies.

Bad movement patterns result in balance disorders and falls, which, in turn, will cause osteoporotic fractures, if bone mechanical strength is reduced.

Reduced bone strength leads to fractures, and osteoporotic fractures significantly increase the risk of future fractures. Therefore, apart from pharmacological treatment, proper management should also involve physical activity and rehabilitation.

There is ample evidence to show that lack of movement (e.g., astronauts, bed-ridden patients) reduces bone mass and weakens muscles, and as a result, bone mechanical strength is reduced.[27,29]

Nowadays, it cannot be questioned that patients have to take regular, properly selected physical exercises. The selection depends on age, general fitness, and coexistent diseases that affect the musculoskeletal system.

In patients with reduced bone strength, the management that aims to improve the general fitness is both prophylactic and therapeutic, and involves various forms of bone stimulation.[30-32]

Prophylaxis should involve daily physical activity and instructions on how to properly perform everyday tasks.[30] Physical activity includes various forms of gymnastics. The following types of exercise are recommended: dynamic, weight-bearing exercises, which stimulate bone cells (e.g., walking, running, jumping, weight-lifting, dancing); less dynamic exercises without mechanical loading, which restore the balance of muscle tension, increase muscle strength, improve balance and general condition (e.g., isometric exercises, cycling, swimming).[33]

Physical activity and mechanical loading regulate the activity of bone cells, thus stimulating bone formation. An increase in bone mechanical strength does not have to be reflected in increased BMD. Studies on the effect of different forms of exercises promoting bone strength do not definitively determine the amount of loading that is beneficial for the musculoskeletal system.[34]

Already during the growth period, physical activity that involves resistance and aerobic exercise helps achieve high peak bone mass, which was demonstrated by comparing the BMD of physically active children with those who are not active.[35-37]

In adults, resistance and aerobic exercise does not always increases the bone mass, but it prevents bone mass loss.[38]

In postmenopausal women, bone mass can be maintained by weight-bearing exercise with additional resistance, as well as aerobic exercise.

In the case of osteoarthritis associated with aging, marching with the elimination of the weight of lower limbs is recommended.

It has been noted that simultaneous application of resistance exercise and walking for 1 hour 4 times a week facilitates bone turnover and an increase in bone mass in the lumbar area of the vertebral column.[39]

Stengel et al.[40] reported that comprehensive physical training for 2 years led to an increase in BMD in postmenopausal women. The authors assessed the effect of loading on BMD in postmenopausal women with osteopenia. Only very intensive exercise with high mechanical loading increased BMD in the lumbar area of the vertebral column and proximal epiphysis of the femoral bone.

One of the forms of exercise, especially in people who experience fatigue and lack of motivation, are vibrations of the whole body on a special platform. They increase muscle strength and increase BMD in the neck of the femoral bone. The method has been observed to improve balance and body position, which reduced the risk of a fall and was more acceptable by older people than the conventional methods.[41,42]

Physical exercises increase muscle strength and mass, which positively affects bone strength. They also help maintain the function of muscles that are responsible for body balance and movement coordination.[38,39]

Weak muscles and lack of muscle cooperation may cause falls that result in fractures, especially in people aged 65 years or older. Rizolli et al.[23] conducted the analysis of 44 studies including 9603 participants, which showed a significantly lower ratio of falls in people who did various types of physical exercise compared with people who were not physically active. Intensive exercises (about 50 hours during the observation period) as well as balance exercises were particularly important. In people aged 65 years or older, walking as a recommended form of physical activity may be a risk factor for falling. In postmenopausal women, walking prevents bone mass loss, but also improves cardiovascular function, the disturbance of which is a risk factor for falling.

Tai chi is one of the types of balance exercise used in patients. Li et al.[43] observed a significantly lower number of falls in people at the mean age of 77.5 years, who had practiced tai chi for 6 months, compared with those who practiced only stretching.

Balance disorder is often a cause of falls and fractures in people with osteoporosis. Madureira et al.[44] evaluated the effect of balance exercises on the static and dynamic balance, ability to move, and the rate of falls in patients with osteoporosis. After 12 months of regular exercise (once a week assisted by a physical therapist, and 3 times a week alone at home according to an established training program), the authors observed improved static and dynamic balance and a reduced number of falls compared with controls who did not practice.

The necessity of supervising patients while doing the exercise is stressed. A therapist needs to provide instructions on how to do a particular exercise and explain its purpose, so that patients are able to continue on their own at home. Adjusting the level of difficulty and type of exercise to individual needs may bring positive effects. Physical therapy aims to prevent deformities by improving balance, strengthening muscles, teaching new movement patterns, and, if necessary, relieving pain.[31,45]

After the analysis of muscle strength and activity in the musculoskeletal system, a therapist should aim to restore proper muscle tension and muscle cooperation rather than just work to strengthen individual muscles. Proper muscle elasticity helps achieve proper muscle tonus, i.e., muscle tension at rest. We should restore muscle elasticity, without disturbing their length or rigidity, to avoid contractions. Contractions during everyday tasks may cause unfavorable levers, balance disorder, falls, and fractures.

Comprehensive rehabilitation therapy should include strengthening, coordination, balance, flexibility, and breathing exercises.

Progressive thoracic kyphosis caused by compressive fractures of the vertebral bodies, reduces the general fitness. Prolonged lack of physical activity reduces BMD and muscle strength. In such cases, aerobic exercises are an inevitable part of a comprehensive rehabilitation program.[46]

Exercises that prevent or reduce thoracic kyphosis are a typical element of the management plan. In the case of compression fractures of the vertebral bodies, exercises for kyphosis should be performed in a horizontal, not vertical, position.[47]

In individual patient management, methods of manual therapy (postisometric relaxation, soft tissues technique) and neurostimulation should also be considered.

Patients with osteoporotic fractures pose a significant challenge. Currently, all methods of orthopedic treatment aim at early mobilization of the patient. These include stabilization of peripheral bone fractures, hip replacement, or orthopedic corsets in vertebral fractures.[40]

Early mobilization of the patient will facilitate bone healing and prevent muscle atrophy. The next stage is a precise functional analysis of the patient in order to restore proper movement patterns and teach proper behavior in everyday life. Such management will reduce forces that exceed bone mechanical strength, and thereby, the risk of future fractures.

To summarize, reduced bone mechanical strength results in osteoporotic fractures. Movement, axial load, and proper muscle activity increase bone mechanical strength. Mechanical loading applied to bones maintains their strength and reduces the risk of fractures. An increase in bone mechanical strength does not have to be reflected in densitometric evaluation.

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ARTYKUŁ POGLĄDOWY

Zmiany wytrzymałości mechanicznej kości w terapii ruchem

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SŁOWA KLUCZOWE

osteoporoza,
rehabilitacja,
wytrzymałość
mechaniczna kości

STRESZCZENIE

Na wytrzymałość mechaniczną kości ma wpływ wiele niekorzystnych zdarzeń prowadzących do zaburzeń między procesem resorpcji i tworzenia kości. Na wytrzymałość kości oddziałują także obciążenia mechaniczne (masa ciała i napięcia mięśni) oraz czynniki predysponujące do wystąpienia osteoporozy.

Struktura kości ogranicza obciążenia, jakie mogą wytrzymać kości, a jednocześnie obciążenia działające na kość warunkują jej strukturę. Jest to możliwe dzięki komórkowym mechanizmom modelowania i przebudowy kości, zwanymi adaptacją.

Terapia ruchem ma wpływ na wytrzymałość mechaniczną kości poprzez stymulację komórek kostnych. Ruch zwiększa siłę i przywraca współpracę mięśni, przez co poprawia się stan funkcjonalny i zwiększa się aktywność ruchowa, co w sposób pośredni działa na zwiększenie wytrzymałości kości.

Analiza opublikowanych prac na temat wytrzymałości mechanicznej kości potwierdza skuteczność ćwiczeń jako ważnego elementu kompleksowego postępowania. Obserwowano zmniejszenie ryzyka upadków i złamań u osób, u których obok leczenia farmakologicznego stosowano terapię ruchem. Zaobserwowano także, że zwiększenie wytrzymałości mechanicznej kości nie musi mieć odzwierciedlenia w ocenie densytometrycznej.

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Praca wpłynęła: 02.03.2010.
Przyjęta do druku: 30.04.2010.
Nie zgłoszono sprzeczności interesów.
Pol Arch Med Wewn. 2010; 120 (9): 368-373

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Erratum

Wrigley BJ, Tapp LD, Shantsila E, Lip GYH. Antithrombotic therapy in anticoagulated patients with atrial fibrillation presenting with acute coronary syndromes and/or undergoing percutaneous coronary intervention/stenting. Pol Arch Med Wewn. 2010; 120 (7-8): 290-293.

In the Table on page 291, the dose of acetylsalicylic acid should be stated as “ASA ≤100 mg/day”.