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THE INFLUENCE OF THE SCHEME OF LOADING VARIATIONS ON THE RECOVERING OF THE BONE TISSUE ELASTIC MODULUS

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Abstract: The influence of the scheme of therapeutic loading (i.e. duration of the loading stages and therapeutic load values at each stage) on the recovering of the bone tissue elastic modulus in human proximal femur after its long enforced immobilization has been investigated. The computer simulation of the bone recovering process has been performed on the basis of deformation model of bone tissue adaptation. The comparison of two loading schemes has been carried out. The load values at each stage were identical in both schemes, but the change of loading stages took place monthly in one scheme (as it is generally accepted in clinical practice) and every five days in the other one. It has been shown that more frequent change of therapeutic loading stages resulted in total time reduction of bone tissue recovering, however excessive intensification of loads (when the load was increased every five days) caused local collapse in the femur neck. The fracture criterion and the safety factor of bone tissue have been considered. It has been stated that the bone tissue fracture takes place in some bone location when the strain intensity in this location exceeds its critical value. It has been shown that the bone tissue safety factor can be increased by redistribution of the load values at separate stages of recovering process.

Key words: internal remodeling, elastic modulus, bone tissue, strain remodeling stimulus, proximal femur, therapeutic loading

Introduction

The present paper is a continuation of research of the authors on the problem of bone tissue remodeling. In the paper by Akulich and Podgayets [1] the brief review of known equations of adaptive internal remodeling of sponge bone tissue was presented, their numerical analysis was carried out and the following kinetic equation describing adaptive remodeling of bone tissue with local strain stimulus was suggested:

$$dE(\mathbf{x},t)/dt = C\,[\varepsilon_i(\mathbf{x},t) - \varepsilon_i^{hom}(\mathbf{x})], \qquad (1)$$

where $E(\mathbf{x}, t)$, $\varepsilon_i(\mathbf{x}, t)$ were the elastic modulus and the strain intensity at the point $\mathbf{x}$ at the instant of time t, respectively; C was a factor of the remodeling rate; $\varepsilon_i^{hom}(\mathbf{x})$ was the strain intensity at the point $\mathbf{x}$ under conditions of homeostasis, which took place in the bone tissue under physiological load.

In accordance with the kinetic equation (1) the adaptive remodeling of bone tissue runs in the following way. Under the change of external loads, strains in every bone location are changing too. The remodeling stimulus is introduced as a difference between actual and homeostatic local strain intensities. This local strain stimulus results in such a variation of the elastic modulus at the point $\mathbf{x}$ that the actual strain intensity at this point tends to the homeostatic one. For example, the dropped load causes the strain intensity to be immediately decreased. Its subsequent rise to the homeostatic strain intensity runs due to gradual decrease

of the elastic modulus, i.e. the bone weakening is taking place. The load rise, on the contrary, results in the increased strain intensity. The necessity of its diminishing to the homeostatic value causes the gradual increase of the modulus, i.e. the bone is being strengthened.

In the paper by Akulich, Denisov, Podgayets and Akulich [2] an attempt was undertaken to use this kinetic equation in the computer simulation of the history of sponge bone tissue mechanical properties in the hip head and femur neck during the post-operational rehabilitation period. The numerical experiment was carried out with several simplifications. First, the computations of the bone remodeling were performed with some tentative time units. It was stated that for their reference to real time scale, the justified specification of the remodeling rate factors was required and these factors should differ for hardening or weakening of the bone tissue. Second, it was assumed that the bone tissue remodeling began immediately after the joint loading had changed and the current strain intensity at every bone location deviated from its homeostatic value at this location. However, the clinical practice is validating the existence of a certain "dead" zone (Lanyon [3]), within which the bone tissue is irresponsive to variations of load and strain at least for some time. Then, some arbitrary scheme of the load change after the hip joint immobilization was considered. In the present paper we tried to take into account all these particulars and thus approach the solution of the real problem of the patients' rehabilitation after the surgical intervention.

Methods

The investigation of stresses and strains in the proximal femur was carried out by quasi-two-dimensional finite element model with side cortical plates (Svesnsson, Valliapan and Wood [4]). Our model considered two forces: the force $\mathbf{F_1}$ applied to the hip head at angle α with the vertical and the force $\mathbf{F_2}$ applied to the greater trochanter at angle β with the vertical. According to Weinans, Huiskes and Grootenboer [5] three load cases were considered (see Fig. 1).

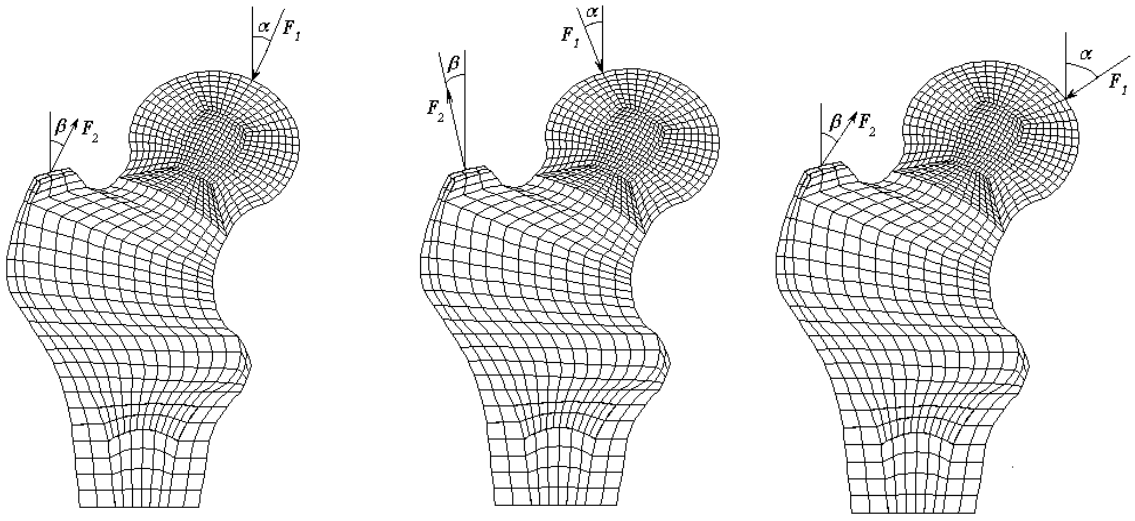


Fig. 1. The central layer of the proximal femur finite element model under three load cases.
a) the first load case: F_1 = 2317 N; α = 24°; F_2 = 702 N; β = 28°;
b) the second load case: F_1 = 1158 N; α = -15°; F_2 = 351 N; β = -8°;
c) the third load case: F_1 = 1548 N; α = 56°; F_2 = 459 N; β = 35°.

We assumed 6000 cycles of load applications for the first load case and 2000 loading cycles for the other cases as a normal physiological load per day. In our model the force $\mathbf{F_1}$ was distributed on an arc about a quarter of the hip head circle by the cosine law and the force $\mathbf{F_2}$ was uniformly distributed on the greater trochanter surface. As the bone was subjected to three alternative loads with different numbers of load cycles, the actual strain intensity $\varepsilon_i(\mathbf{x}, t)$ in the kinetic equation (1) was a result of its averaging over the whole ensemble of loads.

In our computations of the bone rehabilitation process we have considered two schemes of the load time variations (Table 1). For the initial state in both schemes we took the stress, strain and elastic modulus patterns under the normal physiological load mentioned above (Akulich, Denisov, Podgayets and Akulich [2]). The first stage was a model of the joint immobilization after the trauma or surgical intervention; here the load fell abruptly down to 5% of the normal. The duration of this stage was 60 days in both schemes. According to the scheme A in the next stages, from the second to the seventh one, the load has been monthly increasing up to 10%, 25%, 45%, 70%, 95% and 100% of the physiological load, respectively. This scheme of the load time variations was adequate to the generally accepted clinical practice. In comparison with the realistic scheme A, obviously unreal scheme B was considered. According to this scheme the joint load after two-month immobilization was increased up to the same levels step by step, though not monthly, but every five days. The assumption was made that the time variations of therapeutic loading in both schemes consisted only in proportional change of force magnitudes, while their inclinations to the vertical and a number of each type loading cycles remained unchanged.

Table 1. Schemes of the load time variations.

Stage	Load	Stage duration	
		Scheme A	Scheme B
1	5 %	60 days	60 days
2	10 %	30 days	5 days
3	25 %	30 days	5 days
4	45 %	30 days	5 days
5	70 %	30 days	5 days
6	95 %	30 days	5 days
7	100 %	up to a year	

The computer simulation of the bone tissue adaptive remodeling in the proximal femur under time variations of hip joint loading was performed by the above-mentioned kinetic equation (1) with the local strain remodeling stimulus, where special attention was given to remodeling of the hip head and neck bone tissue. In order to take into account the difference between remodeling rate factors in weakened and strengthened bone, the kinetic equation (1) was rewritten in the following form:

$$\begin{aligned} dE(\mathbf{x},t)/dt &= C^{+}\,[\varepsilon_i(\mathbf{x},t) - \varepsilon_i^{hom}(\mathbf{x})] && \text{when } \varepsilon_i(\mathbf{x},t) > \varepsilon_i^{hom}(\mathbf{x}), \\ dE(\mathbf{x},t)/dt &= C^{-}\,[\varepsilon_i(\mathbf{x},t) - \varepsilon_i^{hom}(\mathbf{x})] && \text{when } \varepsilon_i(\mathbf{x},t) < \varepsilon_i^{hom}(\mathbf{x}), \\ dE(\mathbf{x},t)/dt &= 0 && \text{when } \varepsilon_i(\mathbf{x},t) = \varepsilon_i^{hom}(\mathbf{x}). \end{aligned} \qquad (2)$$

It was assumed that the remodeling rate factor C^{-} under weakening (resorption) of bone tissue was 1.33 times larger than the factor C^{+} under bone strengthening (reposition). To bring the remodeling rate factors to the real time scale, their specification was carried out, where the time unit was one day. The calculations were performed with the following values

of the remodeling rate factors: $C^+ = 960$, $C^- = 1280$. With the aim of reflecting the fact that the bone remodeling does not start immediately after the load has been changed, both remodeling rate factors on the first day of the load change were equal to 1/64 of their nominal values. Then during the first eight days after the load change they were gradually rising until their full values. In the case of scheme B, with 5-day stage duration, this rise of remodeling factors to their full values has not been completed at the stages from two to six.

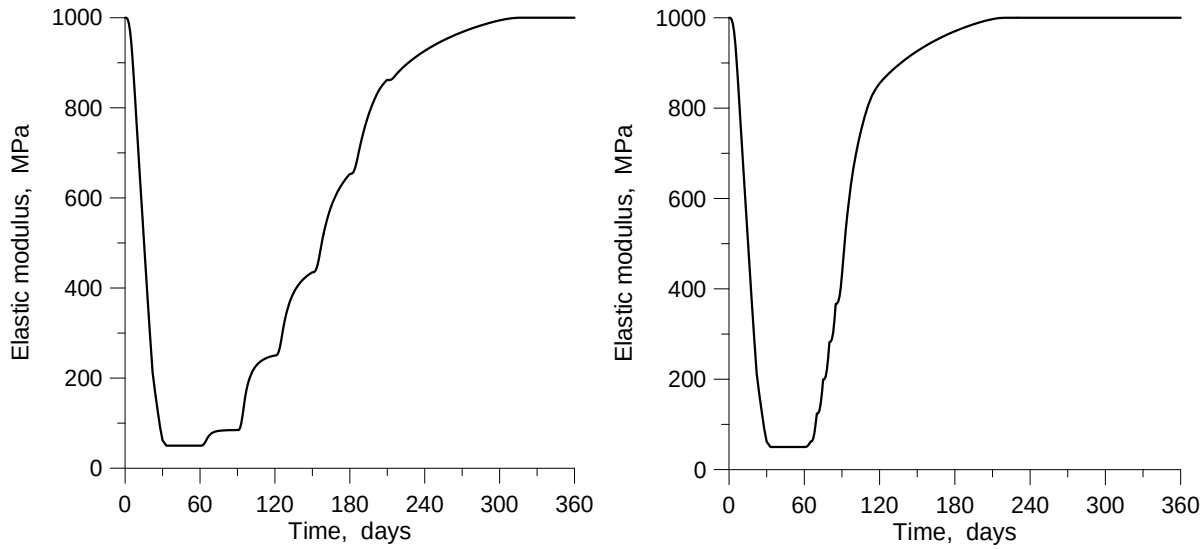
Results and Discussion

The time variations of bone tissue elastic modulus in the same location at the lateral side of femur neck for both load change schemes examined are plotted in Fig. 2. We can see that formally the bone tissue modulus has been recovered until its initial value in both schemes and this process was more rapid at more frequent increase of joint load (scheme B). However, it contradicts the clinical data, which testify that application of a high load to bone not reinforced enough, does not cause strengthening of the bone tissue, but its collapse.

To resolve this contradiction it is necessary to introduce a bone tissue fracture criterion. Brown, Baker and Brand [6] presume that such a criterion is stress-to-strength ratio (SSR) or safety factor (reciprocal of SSR). At approach SSR to unity, probability of bone tissue collapse critically increases. We accepted the stress intensity as a measure of a stress state and used a hypothesis that bone tissue collapse occurred in a location corresponding to some finite element, when the stress intensity in this element reached the strength limit.

It was shown in the experiments by Brown, Way and Ferguson [7] that the strength limits of bone tissue samples from different parts of proximal femur were proportional to their local elastic moduli. We supposed this proportion remained valid also under variations of local elastic modulus in consequence of bone tissue remodeling. The proportionality factor was found from the condition that average sponge bone elastic modulus of 500 MPa corresponded to strength limit of 5.5 MPa (Ueo, Tsutsumi et al [8]).

In Fig. 3 the development in time of stress intensity (solid line) and strength limit (dotted line) at the same location at the lateral side of femur neck is shown for both schemes of load change. We can see that at monthly load increase (scheme A) the stress intensity



scheme A scheme B

Fig. 2. Time dependence of elastic modulus of the bone tissue at the lateral side of femur neck for two schemes of hip joint loading variations. The scheme A corresponds to monthly load change and the scheme B provides rise of the hip joint load every five days after two-month immobilization.

nowhere exceeds strength limit and only once comes nearer to it, in the beginning of the third stage. At the same time when the load increases every five days (scheme B), the stress intensity in this location exceeds strength limit at each step of load. For the first time this excess occurs in the beginning of the third load stage on the 66th day of the rehabilitation (or the 6th day of load rise after immobilization).

Fig. 4 shows time development of stress-to-strength ratio in the same element. When the joint load is monthly increased (scheme A), the SSR nowhere exceeds 1 (only once comes nearer to unity in the beginning of the third stage). But under load increase every five days (scheme B) SSR becomes more than one at each load change, it means the collapse of the bone tissue.

Within the framework of the linear theory of elasticity the stress intensity is proportional to both elastic modulus and strain intensity. At the same time the strength limit according to Brown, Way and Ferguson [7] as well as our hypothesis is proportional only to the elastic modulus. Thus, the ratio of stress intensity to strength limit in each finite element does not depend on the local magnitude of the elastic modulus and is determined solely by strain intensity in the given element. The stress intensity exceeds strength limit, which means the collapse of the bone tissue, when the strain intensity reaches a magnitude exceeding some limiting value (the critical strain intensity), in our case this value is equal to 0.00953.

Thus, the most fracture dangerous zones in a proximal femur are those where the strain intensities are the greatest. In the paper by Akulich, Denisov, Podgayets and Akulich [2] the distribution of the homeostatic strain intensity in proximal femur was obtained (Fig. 5). It is quite visible that such most dangerous zones are located on a surface of the femur neck (at its medial and especially at its lateral side). The above and the subsequent diagrams correspond to suchlike critical bone location at the lateral side of the neck.

The change of strain intensity in time for both schemes of hip joint loading after long immobilization is plotted in Fig. 6. We can see that in the case of slower load increase (scheme A) the strain intensity does not surpass the critical value and only once comes nearer to it. Fast increase of joint load (scheme B), when the bone tissue is insufficiently strengthened, brings in the strain intensity exceeding the critical magnitude at each load step (first it happens on the 66th day), i.e. the collapse of bone tissue really occurs in the given

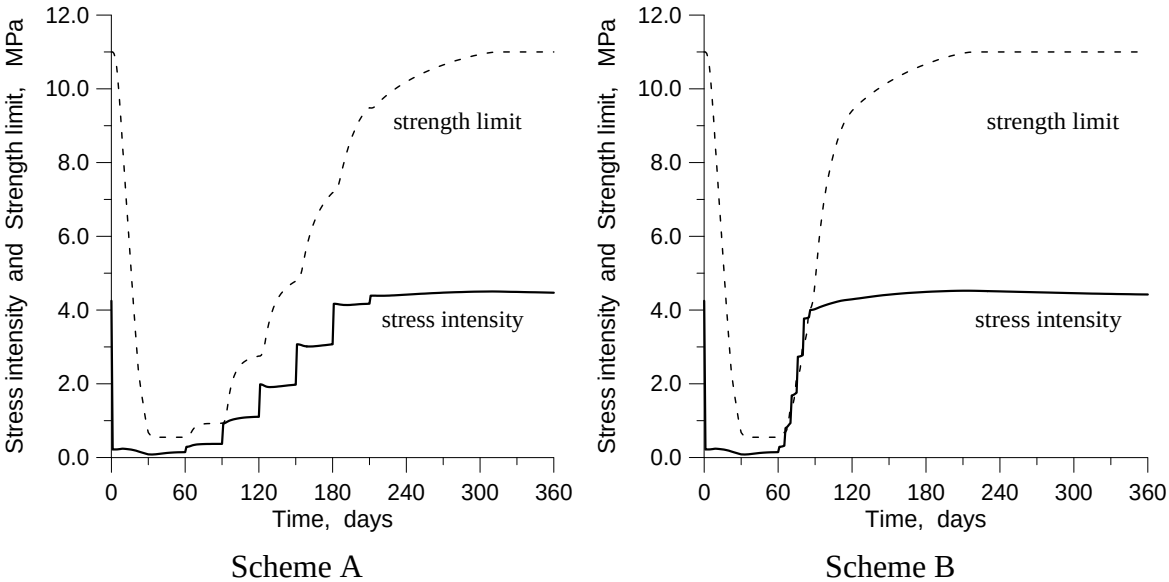


Fig. 3. Time dependence of stress intensity (solid line) and strength limit (dotted line) at the lateral side of femur neck for two schemes of hip joint load change. The scheme A corresponds to monthly rise of joint load after two-month immobilization and the scheme B provides load change every five days.

bone location.

The elastic modulus and the strength limit in a damaged bone tissue are much below their values in a healthy bone. Therefore, since the strain intensity in any finite element goes over the critical level, it is necessary to consider the given element elastic modulus value being equal to the modulus of the damaged bone tissue. As our calculations did not include such procedure of modulus change, they did not agree with the real situation after the instant of time when the strain intensity excess of its critical value first happens. Thus, the recovering of bone tissue elastic modulus (Fig. 2, scheme B) actually cannot take place in the case of 5-day load rise because of the local bone tissue collapse. At the same time these results signal

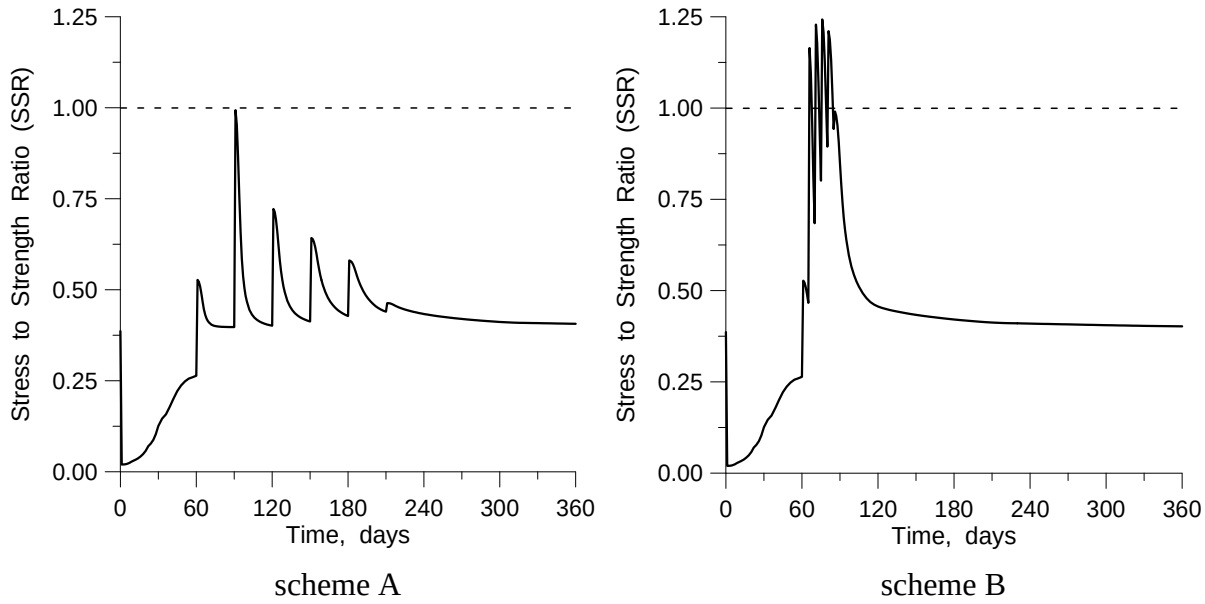


Fig. 4. Time dependence of the SSR at the lateral side of femur neck for two schemes of hip joint load change. The dotted line (SSR=1) denotes the danger of collapse.

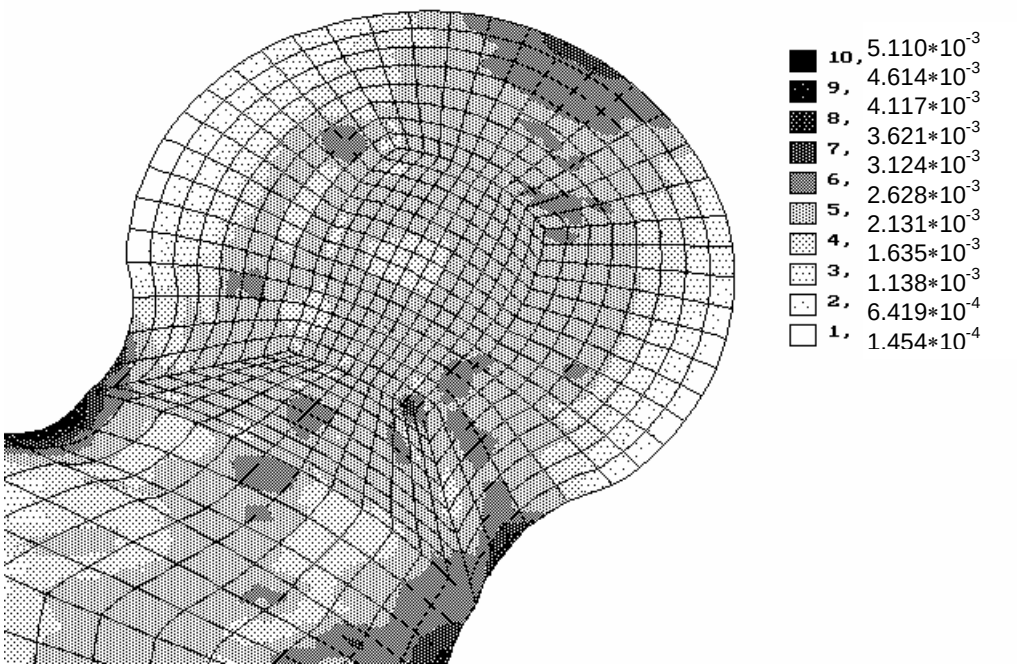


Fig. 5. The homeostatic strain intensity pattern in the hip head and femur neck.

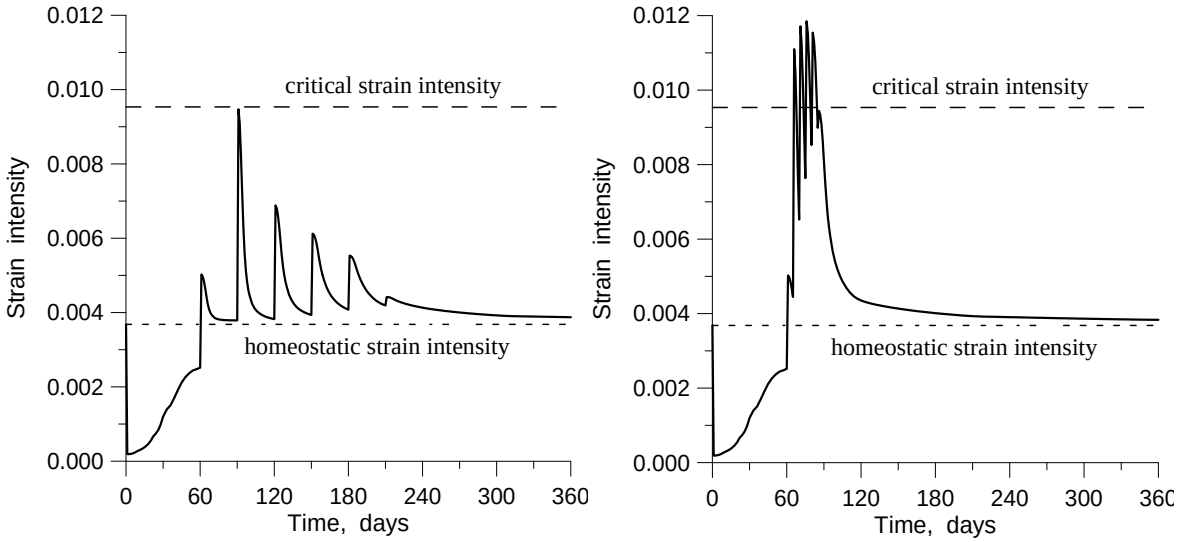


Fig. 6. The dependence of strain intensity on time in the same finite element at the lateral side of femur neck for two schemes of load change. The scheme A corresponds to monthly change of hip joint load and the scheme B provides load rise every five days after two-month immobilization.

the existence of some minimal duration of the load stages, at which the rehabilitation process runs without bone tissue collapse.

It was indicated above that under the monthly joint load increase the strain intensity in the beginning of the third stage of process did come nearer to the critical one (see Fig. 6, scheme A). The corresponding ratio of stress intensity to strength limit became close to unity also in the beginning of the third stage (Fig. 4, scheme A). To remove this risk we have considered a modified scheme of load variations (see Table 2). According to this scheme the load level at the third stage was reduced to 20 % from physiological load instead of 25 % and at the fourth stage it was reduced to 40 % instead of 45 %, while the loads at the subsequent stages were left unchanged.

Table 2. The modified scheme of the load time variations.

Stage	Load	Stage duration
1	5 %	60 days
2	10 %	30 days
3	20 %	30 days
4	40 %	30 days
5	70 %	30 days
6	95 %	30 days
7	100 %	up to a year

The below diagrams illustrate time dependence of strain intensity (Fig. 7), stress intensity and strength limit (Fig. 8) for the modified scheme of therapeutic loading. We can see that at all the stages of the bone tissue remodeling process the strain intensity is much below its critical magnitude and the stress intensity is less than strength limit. The peak value of the SSR magnitude at the third stage is 0.795 (i.e. the minimal safety factor is 1.26). Therefore the considered modified scheme of therapeutic loading is more preferable than the scheme A, where the maximal magnitude SSR = 0.993 is also observed at the third stage and the corresponding minimal safety factor is 1.007.

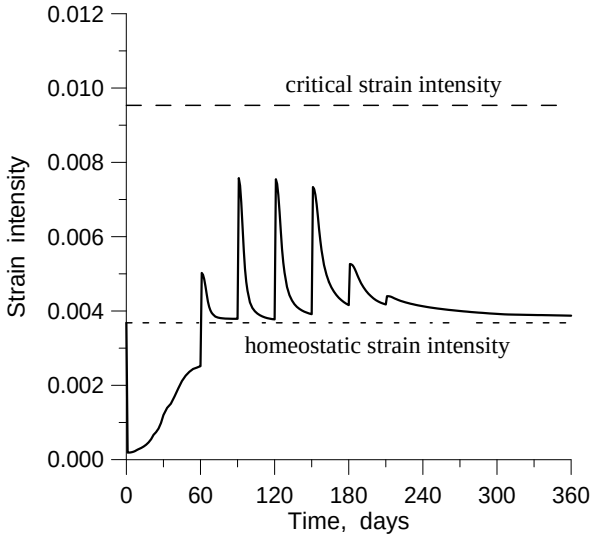


Fig. 7. Time dependence of strain intensity at the lateral side of femur neck for modified scheme of load rise after two-month immobilization.

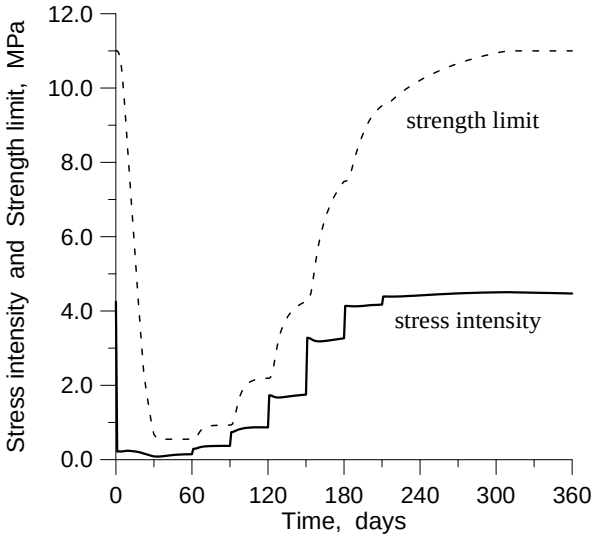


Fig. 8. Time dependence of stress intensity (solid line) and strength limit (dotted line) at the lateral side of femur neck for modified scheme of hip joint load change.

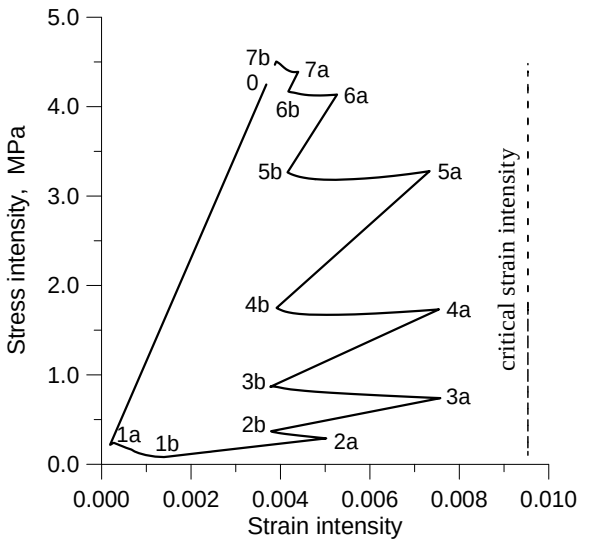


Fig. 9. The image of the process of bone tissue remodeling for the modified scheme of therapeutic load change plotted as a graph of stress intensity versus strain intensity of deformations (see explanation in the text).

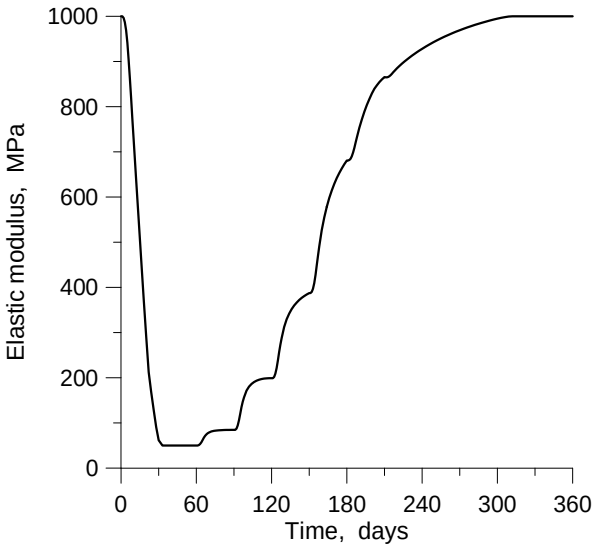


Fig. 10. Time dependence of elastic modulus of the bone tissue at the lateral side of femur neck for the modified scheme of hip joint loading variations.

The complete recovering of the bone tissue elastic modulus and the stress and strain state take place in the examined remodeling process. The history of stresses and strains at the lateral side of femur neck during this process is visualized as a graph of stress intensity versus strain intensity (Fig. 9) where the stages of the remodeling process are designated in figures and letters. In comparison, the time dependence of the bone tissue modulus is shown in Fig.10 where the first stage lasts 60 days, the subsequent stages change every 30 days and the seventh stage lasts up to one year (see Table 2).

The initial stress and strain state of the bone tissue is designated in Fig. 9 by 0. The straight-line segment from 0 to 1a corresponds to load decrease down to 5 % from normal at the originally high elastic modulus and the portion of a curve from 1a to 1b represents weakening of the bone tissue at the first stage of the process (two-month immobilization). The reduction of the elastic modulus at this stage (see Fig. 10) is accompanied by increase of the

strain intensity and the stress intensity decrease. On the second stage of the process the rectilinear segment from 1b to 2a meets the load increase from 5% to 10% at the minimal elastic modulus. Further, the portion of a curve from 2a to 2b represents strengthening of the bone tissue, where the elastic modulus grows during a month (Fig. 10), which causes decrease of the strain intensity and increase of the stress intensity.

At the following stages the process runs similarly, each new load rise occurs with a higher magnitude of the elastic modulus, it is visible by growing the inclination angle of the corresponding strait-line segments. The curvilinear segment from 7a to 7b represents bone tissue remodeling at the seventh stage of process and the point 7b which corresponds to the final stress and strain state, practically coincides with the initial point 0. This fact is an evidence of complete recovering of homeostatic stresses and strains in the bone tissue. In Fig. 9 it is shown that at all the stages of the recovering process the strain intensity does not exceed its critical value, i.e. the studied modified scheme of therapeutic loading is safe.

Conclusions

The analysis of computer simulations of the bone tissue recovering process proves that the phenomenological model used in the present study describes the clinical facts qualitatively correct. In comparison with the results presented in the paper by Akulich, Denisov, Podgayets and Akulich [2], a step in the direction of a more exact quantitative correspondence with the real rehabilitation process has been made. The presented mathematical model of the rehabilitation process takes into consideration the different rates of bone resorption and reposition, the relationship between strength limit and elastic modulus and the existence of the zone of adaptive insensitivity (the "dead zone"). These improvements allowed us to state that the bone tissue fracture took place at some bone location, when the strain intensity at this location exceeded its critical value. Thus, the most fracture dangerous zones in a proximal femur are the medial and lateral sides of the neck, where the strain intensities are the greatest. It was shown the existence of the minimum duration of therapeutic load stages, at which the bone recovering occurs without risk of its destruction. It was also demonstrated the possibility of essential reduction of the risk of bone tissue fracture by a redistribution of the levels of therapeutic loading at different stages of treatment. However, to solve the real problem of bone rehabilitation after surgical intervention, additional investigations of the bone remodeling are required. It is necessary to define more exactly the remodeling rate factors by clinical experiments and determine the optimal safe scheme of the therapeutic load rise, during which the complete recovery of the bone tissue properties would be achieved within the shortest possible time.

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ВЛИЯНИЕ РЕЖИМА ИЗМЕНЕНИЯ НАГРУЗКИ НА ВОССТАНОВЛЕНИЕ МОДУЛЯ УПРУГОСТИ КОСТНОЙ ТКАНИ

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С помощью двумерной конечноэлементной модели проксимального отдела бедра, учитывающей адаптацию губчатой костной ткани с использованием деформационного стимула внутренней перестройки, исследуется влияние параметров режима лечебных нагрузок (величина нагрузки и продолжительность стадий нагружения) на процесс восстановления модуля упругости костной ткани после вынужденной иммобилизации бедра. Проведено сравнение двух схем повышения нагрузки. В обеих схемах величина нагрузки на каждой стадии была одинаковой, но в одной схеме смена стадий нагрузки проходила ежемесячно (что соответствует установившейся медицинской практике), а во второй - каждые пять дней. Показано, что более частая смена стадий лечебных нагрузок ведет к уменьшению общего времени восстановления кости, однако чрезмерная интенсификация нагрузок (увеличение нагрузки через каждые пять дней) приводит к местным разрушениям в шейке бедра. Рассмотрен вопрос о критерии разрушения и запасе прочности костной ткани. Установлено, что разрушение костной ткани в некоторой области кости происходит, когда интенсивность деформаций в этой области превышает критическое значение. Показано, что запас прочности кости можно повысить путем перераспределения уровня нагрузок на отдельных стадиях процесса восстановления. Библ. 8.

Ключевые слова: костная ткань, головка бедра, модуль упругости, внутренняя перестройка, деформационный стимул, лечебная нагрузка

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