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THE MODEL OF CORTICAL BONE TISSUE ADAPTIVE REMODELING

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Abstract. The change in mechanical properties of the femoral cortical bone tissue surrounding the stem of hip endoprosthesis is one of the causes of implant's instability, and mathematical description of this phenomenon is the subject of many studies. The kinetic equations of bone remodeling being employed by the authors of these studies contain a constant that characterizes the bone tissue response to changes in the bone loading. To forecast the bone tissue remodeling in a real time scale, we have to know the magnitude of this response factor, but its determination is a very complex problem. In the given paper a new model of bone tissue adaptive remodeling is proposed. In this model the response factor has been calculated by the balance equation of the rates of the bone tissue reproduction and resorption. The model describes as modifications of cortical tissue mechanical properties as changes in the external geometric dimensions of the femur. As a remodeling stimulus, the longitudinal (along the osteon's axis) component of the strain tensor is used. The results of the test calculations of the bone unidirectional extension and contraction are presented.

Key words: adaptive remodeling, cortical bone tissue, activity of bone cells, longitudinal strains

Introduction

The loss of stable fixation of a hip endoprosthesis stem, which is a clinical reason for the second operation, may be induced by different causes. One of these causes is the osteoporosis development around the implant's stem, which leads to decrease of the bone bearing capacity. Moreover, when endoprostheses in use are of some specific design without cement fixation, the bone tissue resorption near the implant's stem distal edge is observed. The given changes in the femur are caused by redistribution of load on the bone tissue under the endoprosthesis installation. The magnitudes of nonspecific loads acting on the bone, which are generated after the endoprosthesis installation, are less than the breaking loads. That is why the standard static failure criteria do not permit to forecast the durability of the bone-implant system. The models of fatigue damage accumulation are also unacceptable for description of the bone failure processes because of incessant replacing of old substance by new one, which proceeds in a living tissue. Therefore, the changes generated in the bone might be considered as an adaptive reaction of a human organism for new functional conditions. The models of the bone tissue adaptive remodeling reflect the relationship between the bone mechanical properties and the level of the load. In the articles [1, 4, 8-10] different models of bone tissue adaptive behavior are presented, but a big number of unknown parameters and the absence of techniques for their determination by experiments make practically impossible their employment for the solution of real problems. In the studies [8-16, 19] the different approach is used. According to this approach the number of unknown parameters in the kinetic equation

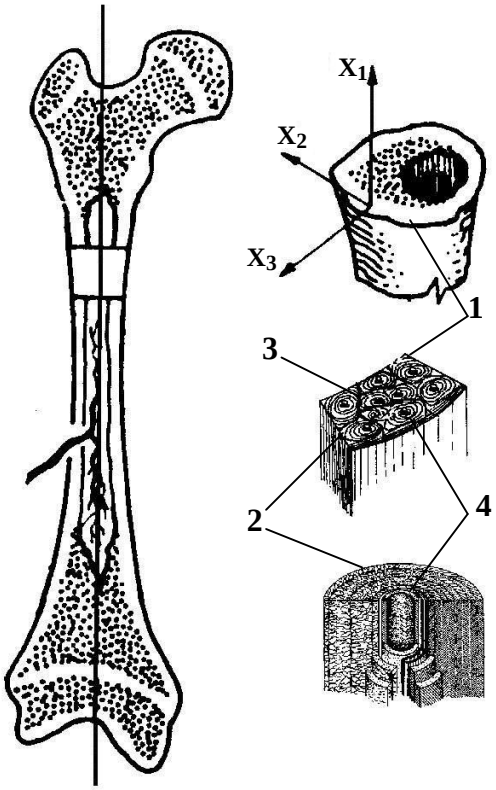

Fig. 1. The structural model of the cortical layer of the femur: 1 – cortical bone tissue, 2 – osteons, 3 – intermediate lamellae, 4 – Haversian canals.

of the bone remodeling is much less, however their determination requires prolonged clinical investigations. The model of adaptive remodeling of the cortical bone tissue that is advanced in the given paper, is based on the physiological features of bone cells activity. At the same time, the employment of known experimental data (see [5, 6]) allows to apply our model for prediction of the time variations of mechanical properties and external geometrical dimensions of the bone under the deviation of the load from the physiological one.

The structural model of the bone

Let us consider a compact bone tissue as a composite material and distinguish its main structural components: Haversian canals, osteons, and intermediate lamellae (see Fig. 1). We suppose the longitudinal (along the axis X_1) elastic modulus of cortical bone tissue being dependent on the mechanical properties of these structural elements as

$$E_1 = \lambda_{hav} E_{hav} + (\lambda_{ost} - \lambda_{hav}) E_{ost} + \lambda_{lam} E_{lam}, \quad (1)$$

$$\lambda_{ost} + \lambda_{lam} = 1, \quad (2)$$

where λ_{hav} is the relative content of Haversian canals (in the bone cross section), λ_{ost} is the relative content of osteons (together with Haversian canals), and λ_{lam} is the relative content of intermediate lamellae.

The consideration of osteons and Haversian canals as ideal cylinders yields

$$\lambda_{hav} = n \pi (r_{hav})^2, \quad (3)$$

where r_{hav} is a radius of the Haversian canal, n is a number of osteons per mm^2 in the bone cross section.

In accord with the paper [6]: λ_{hav}=0.037, λ_{ost}=0.460, λ_{lam}=0.540, r_{hav}=0.03 mm, and n=13.2.

The assumption that the Haversian canals are empty (i.e. E_{hav}=0) gives:

$$E_1 = (\lambda_{ost} - \lambda_{hav}) E_{ost} + \lambda_{lam} E_{lam}. \quad (4)$$

The model of change in the cortical bone mechanical properties

The bone tissue growth or destruction (resorption) proceeds in very parts of a bone being in contact with osteogenic cells. For the cortical bone the areas of remodeling are the periosteum (a fibrous membrane surrounding the bone surface), the endosteum (a membrane lining bone cavities), and internal surface of Haversian canals. Thus, the change of Haversian canals radii results in changing the mechanical properties and porosity of the bone tissue, and the kinetic equation describing the rate of elastic modulus change may be written in the following form:

$$\frac{dE_1}{dt} = \frac{\partial E_1}{\partial r_{hav}} \frac{dr_{hav}}{dt}. \quad (5)$$

Supposing that the parameters λ_{ost}, λ_{lam}, n, E_{ost} and E_{lam} in Eq. (4) are constants and do not depend on time, we have

$$\frac{dE_1}{dt} = -2n\pi \cdot r_{hav} \cdot E_{ost} \frac{dr_{hav}}{dt}. \quad (6)$$

If we consider osteons and Haversian canals as the cylinders oriented along the longitudinal axis of the femur, the change rate of the Haversian canal internal radius may be determined, following to [16], by the balance equation of the rates of the bone tissue reproduction and resorption:

$$\frac{dr_{hav}}{dt} = -(a_b \lambda_b \delta_b - a_c \lambda_c \delta_c), \quad (7)$$

where a is the bone thickness resorbed or formed by cells per unit time, λ is the fraction of the total surface area of all the femur Haversian canals that is available for resorption / formation cell activity, δ is the density (per unit area of λ) of bone cells at these sites of active remodeling. The index b denotes osteoblasts and the bone reproduction, and the index c denotes osteoclasts and the bone tissue resorption.

According to the data of paper [5], in the human bone λ_b =0.89, λ_c =0.11. With the

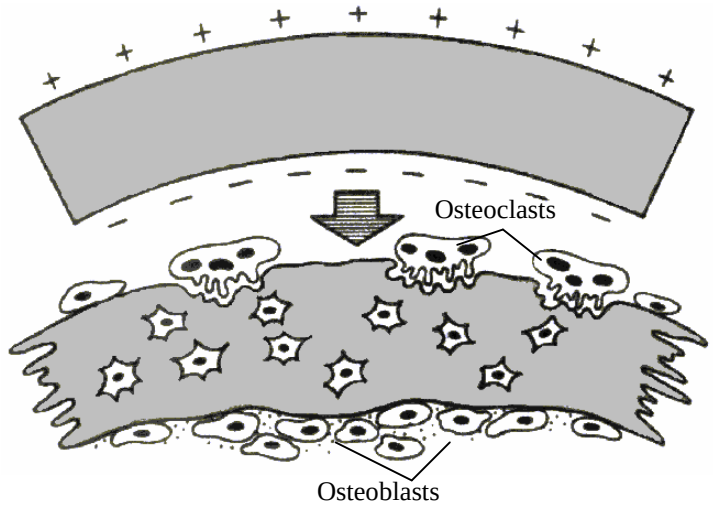

Fig.2. The effect of strain on the activity of bone cells [2].

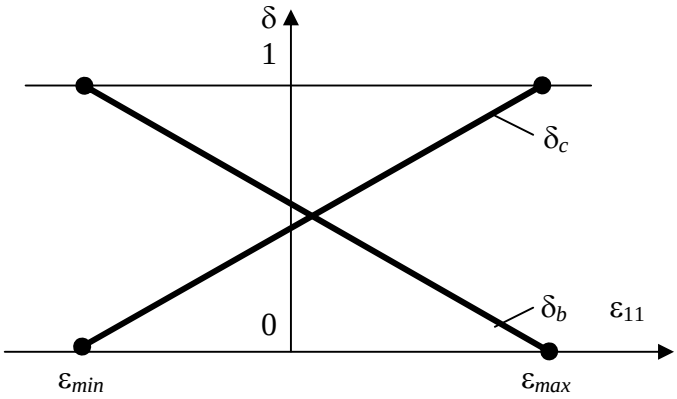

Fig. 3. The dependence of δ_c and δ_b on longitudinal strain.

condition that in the homeostasis $\frac{dr_{hav}}{dt} = 0$, the equality (7) yields:

$$\delta_b^{hom} = \frac{a_c \lambda_c}{a_b \lambda_b} \delta_c^{hom}. \tag{8}$$

If we assume [5] a_c=10 a_b, under the condition $\frac{dr_{hav}}{dt} = 0$ we have a relationship δ_b=1.24 δ_c.

The strain mechanism of the bone adaptation is validated by the studies of piezoelectric [1, 8-10] and biochemical [14] effects in the bone, therefore the stimulus of internal and external remodeling is taken as the deviation of the longitudinal component of the strain tensor ε_{11} from its homeostatic value ε_{hom}. Moreover, in the study by [2] it has been stated that the bone cells are activated under the effect of deformation. Therefore, the values δ_b and δ_c in Eq. (7) are dependent on the strain. We suppose that δ_c and δ_b have a linear dependence of longitudinal strain ε_{11} (see Fig. 3):

$$\delta_b = \frac{\varepsilon_{max} - \varepsilon_{11}}{\varepsilon_{max} - \varepsilon_{min}}, \tag{9}$$

$$\delta_c = \frac{\varepsilon_{11} - \varepsilon_{min}}{\varepsilon_{max} - \varepsilon_{min}}. \tag{10}$$

As the maximal strain ε_{max}, we shall use the breaking strain of tension, it is corroborated by numerous experimental studies [3, 6, 18, 19]. According to [3], we specify the maximal strain ε_{max} =1.54%.

The scope of experimental data on compressive strain is insufficient to assess the accuracy of studies by separate authors, therefore we calculate the minimal strain ε_{min} from the following considerations. As the normal physiological compressive stress in a cortical bone is σ_{hom}=2 kg/mm^2, and the elastic modulus in longitudinal direction is E_1^{hom} =1872 kg/mm^2, the homeostatic strain is ε_{hom} = -0.11%, and under the condition ε_{11} =ε_{hom} Eqs. (8) and (9) yield the minimal strain ε_{min}= -1.44%.

With the homeostatic strain, Eqs. (8) and (9) may be rewritten in the following form:

$$\delta_b = \frac{-(\varepsilon_{11} - \varepsilon_{hom})}{\varepsilon_{max} - \varepsilon_{min}} + \delta_b^{hom}, \tag{11}$$

$$\delta_c = \frac{(\varepsilon_{11} - \varepsilon_{hom})}{\varepsilon_{max} - \varepsilon_{min}} + \delta_c^{hom}. \tag{12}$$

Taking into account Eqs. (8), (11) and (12), Eq. (7) may be written as

$$\frac{dr_{hav}}{dt} = \frac{(\lambda_b + 10\lambda_c)a_b}{\varepsilon_{max} - \varepsilon_{min}}(\varepsilon_{11} - \varepsilon_{hom}). \quad (13)$$

Thus, with the equality (12), the relationship (6) gives us the kinetic equation of internal remodeling:

$$\frac{dE_1}{dt} = -C(\varepsilon_{11} - \varepsilon_{hom}), \quad (14)$$

where C is the adaptive response of the cortical bone tissue on the variation of the longitudinal strain:

$$C = \frac{2n\pi \cdot r_{hav} E_{ost}(\lambda_b + 10\lambda_c)a_b}{\varepsilon_{max} - \varepsilon_{min}}. \quad (15)$$

According to the data [5], the osteoblasts increase the bone thickness by 12 microns over the period of 8 or 10 days, i.e. a_b=(0.0012÷0.0015) mm per day ≈ 0.00135 mm per day.

The changes in geometric dimensions of the bone

We suppose the diaphysis of the femur has a form of a hollow cylinder with external and internal radii R_{ext} and R_{int} respectively. Taking into account that periosteum and endosteum surfaces are the areas of high activity of osteogenic cells, we may write the following equations that describe the changes of the femoral surface geometric dimensions:

$$\frac{dR_{int}}{dt} = \frac{(\lambda_b + 10\lambda_c)a_b}{\varepsilon_{max} - \varepsilon_{min}}(\varepsilon_{11} - \varepsilon_{hom}), \quad (16)$$

$$\frac{dR_{ext}}{dt} = -\frac{(\lambda_b + 10\lambda_c)a_b}{\varepsilon_{max} - \varepsilon_{min}}(\varepsilon_{11} - \varepsilon_{hom}). \quad (17)$$

Results and discussion

In order to verify the technique described above, the calculations were performed for the cylindrical specimen from the femoral diaphisis under unidirectional contraction. It was assumed that the minimal radius of the Haversian canal is r_{min}=0.015 mm (according to experimental study [6]), osteon's radius is r_{max} =0.11 mm, and r_{hom}=0.03 mm.

Fig. 4 shows the variations in the Haversian canal's radius r_{hav}, the longitudinal component of strain tensor ε_{11}, the elastic modulus E_1 and porosity of the cortical bone P under the decrease of the physiological load by 30% (solid line) and by 70% (dotted line) from the homeostatic one. The calculations were performed without consideration of external remodeling. The curves of the longitudinal strain ε_{11} (Fig. 4d) show that the time of the homeostatic strain restoration practically does not depend on the level of the load and is equal to 3 – 4.5 years. Under the 70% underloading the elastic modulus (Fig. 4c) is nearly halved. The porosity (Fig. 4b) is growing due to the rise of the Haversian canals radii (Fig. 4a). During the underloading of 70% the osteons became destroyed (i.e. r_{hav}= r_{max}).

The variations in the same mechanical characteristics of the cortical bone tissue under the load increase by 5% and 20% are presented in Fig. 5, in the second case the calculations were performed taking into account the external remodeling. Under the femur overloading the Haversian canals radii (Fig. 5a) and the porosity (Fig. 5b) became decreased. The elastic modulus is slightly increased, in particular 4.5% under the load increase of 20% (Fig. 5c).

Under the 5% overloading the homeostatic strain has been completely restored only in 24 years (Fig. 5d). However, under the 20% overloading the homeostatic strain restoration proceeds very slowly, because the internal remodeling has reached its limit (the radius of

Haversian canal has been decreased until its ultimate value), and the effectiveness of the external remodeling is very small. Therefore, the critical overloading has to be less than 20%.

The weak effectiveness of both external and internal remodeling, and limited possibilities of internal remodeling displayed under the 70% underloading, too. The osteons have been completely absorbed by osteoclasts (Fig. 6a). The porosity became 10 times more than the normal value of 5% (Fig. 6b), and the bone has been degraded within 3 years. Further, the external diameter of the bone is slowly decreased, and internal diameter increases. Fig. 6 shows the variations in the bone characteristics under the physiological load decrease by 85% calculated with and without change of femoral geometric dimensions.

It can be seen from the presented graphs that the adaptive changes in microstructure of the cortical bone tissue generated by the load variations have an effect on the mechanical characteristics of the femur. After the radius of the Haversian canal reaches the threshold level, the strain aspiration for its homeostatic value is realized by the change of femoral external dimensions. The resorption time of bone tissue in load reducing case is equal to 3÷3.5 years. There is the consensus of these data and the Roentgen rays search results of the tissue resorption around the hip endoprosthesis stem [7] (4÷5 years).

Conclusions

In the given paper a new model of cortical bone tissue adaptive remodeling based on the relationship between the bone cells activity and the longitudinal component of strain tensor has been proposed. The analysis of performed calculations proves that the given phenomenological model allows to describe variations in the mechanical properties and the geometric dimensions of the cortical bone.

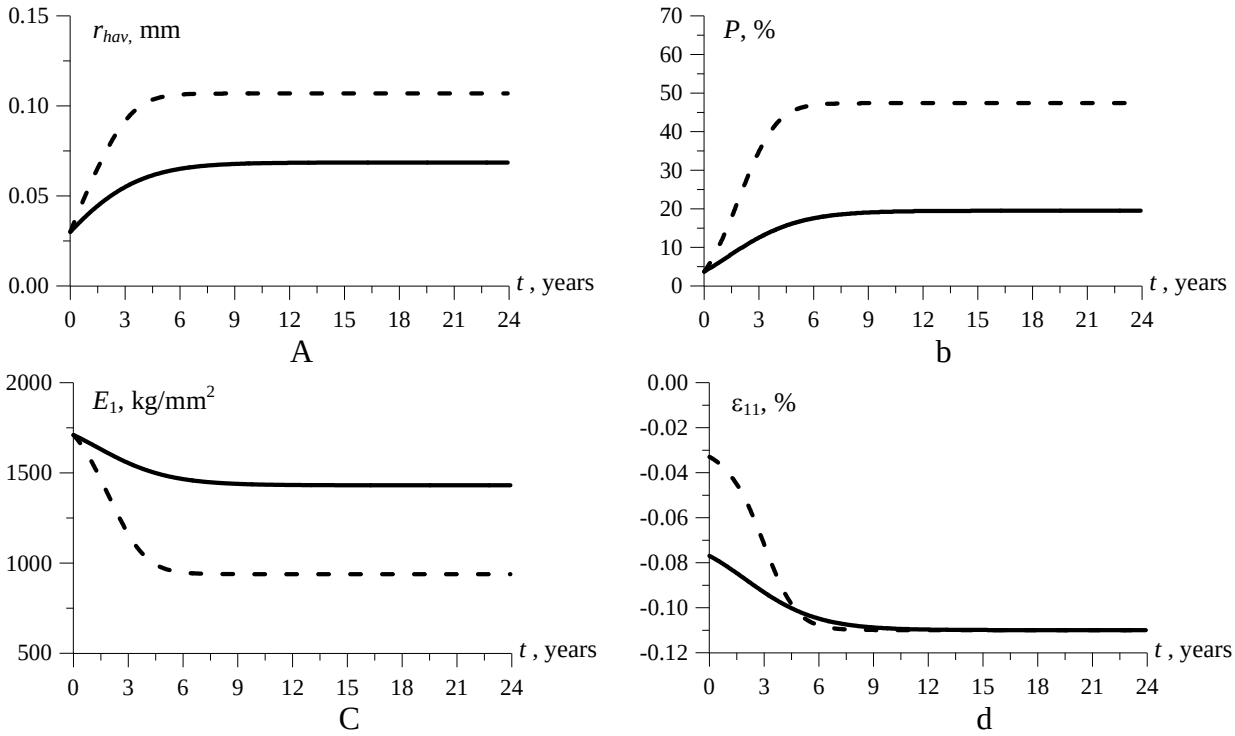

Fig. 4. The time variations in geometric and mechanical parameters of the cortical bone tissue under the load decrease by 30% (solid line) and by 70% (dotted line). a – radius of Haversian canal; b – porosity of the cortical bone; c – elastic modulus of the cortical bone tissue; d – longitudinal component of the strain tensor ε_{11}.

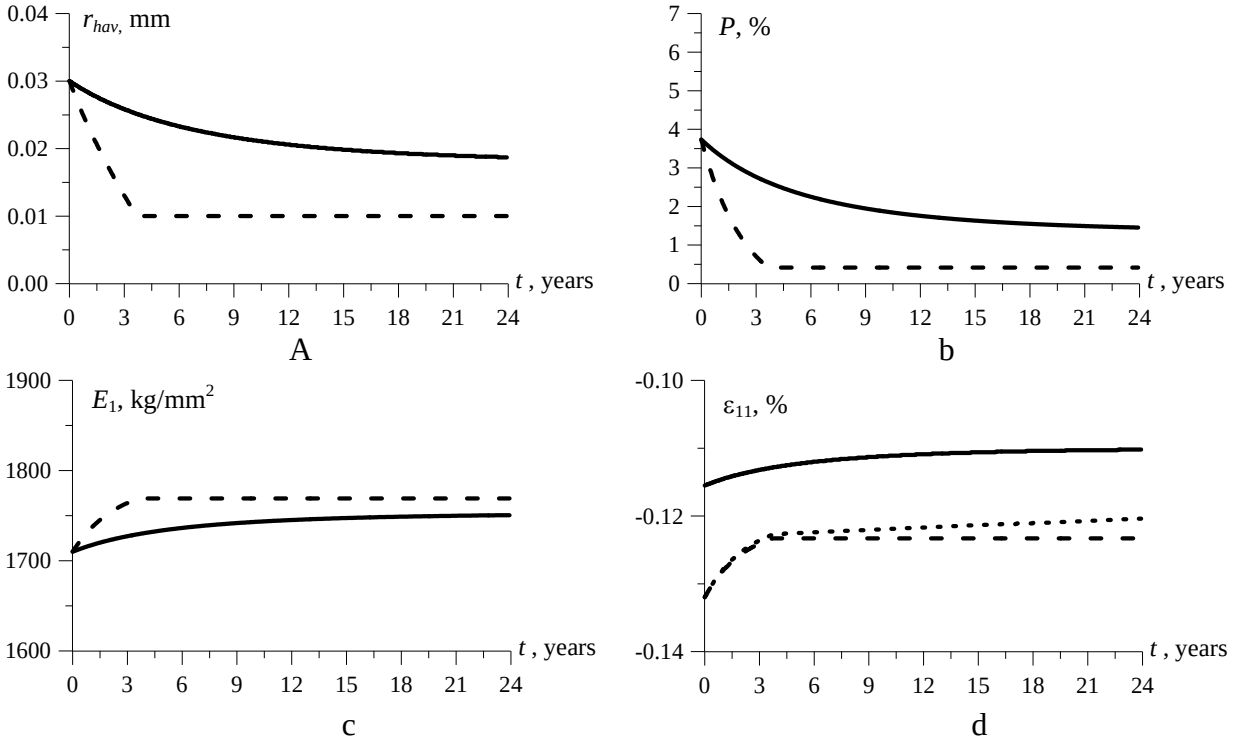

Fig. 5. The time variations in geometric and mechanical parameters of the cortical bone tissue under the load increase by 5% (solid line) and by 20% (without consideration of external remodeling – point line, with consideration of external remodeling – dotted line). a – radius of Haversian canal; b – porosity of the cortical bone; c – elastic modulus of the cortical bone tissue; d – longitudinal component of the strain tensor ε_{11}.

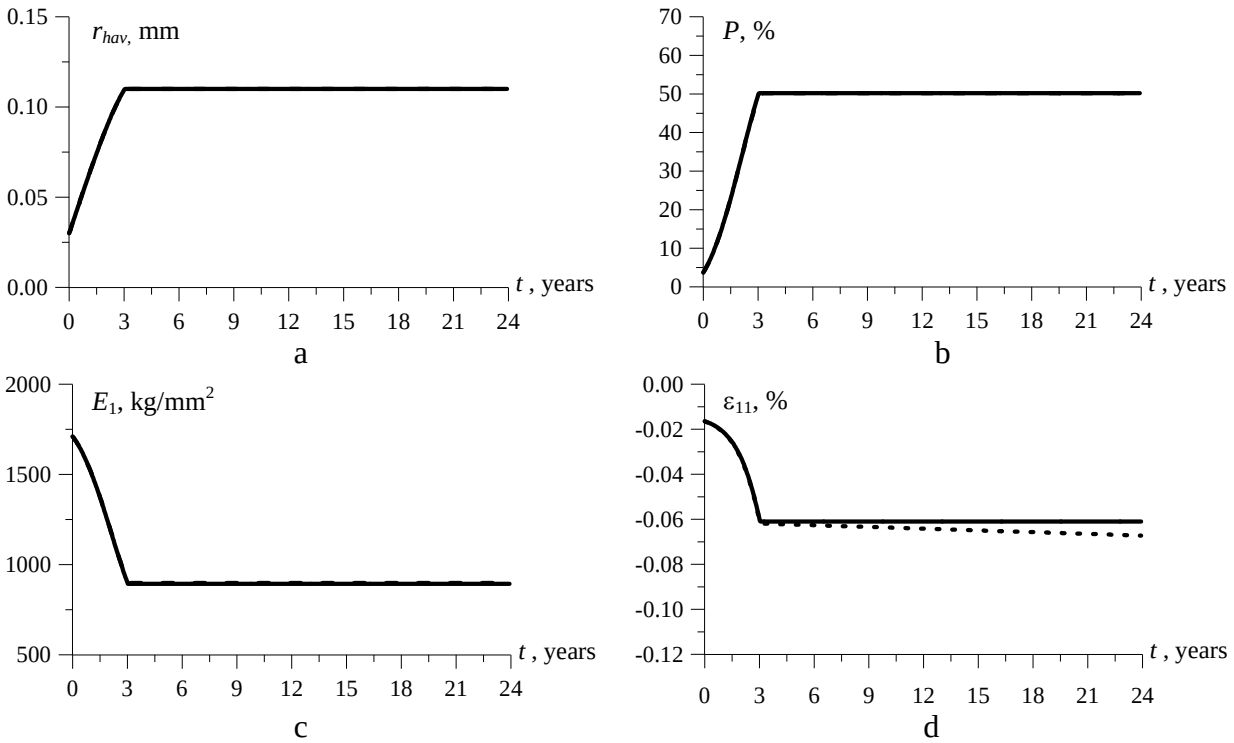

Fig. 6. The time variations in geometric and mechanical parameters of the cortical bone tissue under load decrease by 85%, without consideration of external remodeling (solid line) and with consideration of external remodeling (dotted line). a – radius of Haversian canal; b – porosity of the cortical bone; c – elastic modulus of the cortical bone tissue; d – longitudinal component of the strain tensor ε_{11}.

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МОДЕЛЬ АДАПТИВНОЙ ПЕРЕСТРОЙКИ КОРТИКАЛЬНОЙ КОСТНОЙ ТКАНИ

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Потеря стабильности фиксации ножки бедренной компоненты эндопротеза тазобедренного сустава ведет к повторной операции и может быть вызвана различными причинами. Одной из главных причин является уменьшение упругих свойств кости вокруг ножки имплантата. Кроме того, при использовании некоторых моделей эндопротезов с бесцементной фиксацией наблюдается резорбция костной ткани возле дистального края ножки имплантата. Данные изменения в бедре вызваны перераспределением нагрузки на костную ткань при установке эндопротеза. Величина неспецифических нагрузок на кость, возникающих после установки эндопротеза, меньше экспериментально измеренных разрушающих значений, поэтому для расчета долговечности конструкции кость-имплантат нельзя использовать критерии статической прочности. Модели накопления усталостных повреждений также неприемлемы для описания процессов разрушения кости из-за происходящего в живых тканях непрерывного замещения старого вещества новым. Поэтому изменения, возникающие в кости, рассматриваются как приспособительная реакция организма к новым функциональным условиям. Адаптационные модели перестройки костной ткани отражают взаимосвязь между механическими свойствами кости и уровнем нагрузки. В работах [1, 4, 8-10] представлены теоретические модели адаптационного поведения костной ткани, однако наличие большого числа неизвестных параметров и отсутствие методик их экспериментального определения не позволяют использовать их в реальных задачах. В исследованиях [8-16, 19] отражен другой подход, при котором кинетическое уравнение перестройки содержит существенно меньшее число неизвестных параметров, для определения которых, однако, необходимы длительные клинические исследования. Предложенная в данной работе модель адаптивной перестройки кортикальной костной ткани основана на особенностях физиологической активности костных клеток. Применение известных экспериментальных данных [5, 6] для количественного её описания позволяет проследить изменение во времени механических свойств и внешних геометрических размеров кости при отклонении нагрузки от физиологической. Библ. 20.

Ключевые слова: адаптивная перестройка, кортикальная костная ткань, активность костных клеток, продольные деформации

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