Medical Engineering and Physics 38 (2016) 171–180

Contents lists available at ScienceDirect

Medical Engineering and Physics

journal homepage: www.elsevier.com/locate/medengphy

Long-term response of femoral density to hip implant and bone fracture plate: Computational study using a mechano-biochemical model

Pouria Tavakkoli Avval [a,b], Saeid Samiezadeh [a], Habiba Bougherara [a,*]

[a] Department of Mechanical and Industrial Engineering, Ryerson University, Toronto, Ontario, Canada
[b] Wearable, Interactive and Mobile Technologies Access Centre in Healthcare (WIMTACH), Applied Research and Innovation Centre, Centennial College, Toronto, Ontario, Canada

ARTICLE INFO

Article history:
Received 8 December 2014
Revised 26 October 2015
Accepted 24 November 2015

Keywords:
Bone remodeling
Mechano-biochemical model
Total hip arthroplasty
Bone fracture plate
Finite element method

ABSTRACT

Although bone fracture plates can provide appropriate stability at the fracture site and lead to early patient mobilization, they significantly change the loading pattern in the bone after union (Stress shielding). This phenomenon results in a bone density decrease, which may cause premature failure of the implant. This paper presents the first study that quantifies the long-term response of femoral density to hip implantation and plating (lateral and anterior plating) using a mechano-biochemical model which considers the coupling effect between mechanical loading and biochemical affinities as stimuli for bone remodeling. The results showed that the regions directly beneath the plate experienced severe bone loss (i.e. up to ~ −70%). However, some level of bone formation was observed in the vicinity of the most proximal and distal screw holes in both lateral and anterior plated femurs (i.e. up to ~ +110%). The bone under the plate was divided into six zones. With respect to bone remodeling response, the findings revealed that anterior plating was not superior to lateral plating since the maximum and average bone losses among the zones in the anterior plated femur (i.e. −36% and −24%, respectively) were approximately the same as their corresponding values in the lateral plated femur (i.e. −38% and −24%, respectively).

1. Introduction

Bone fracture plates are used to stabilize diaphyseal fractures when other treatments, such as intramedullary nailing, are impractical or not feasible [1]. For instance, in cases with an extremely narrow canal, or with an existing implant, plates remain an excellent option [2]. Plates can provide appropriate stability at the fracture site, lead to early patient mobilization because of their high stiffness and strength, and are widely used as fracture fixation devices where applicable [1,3]. However, there are some complications that are experienced while healing is in progress, such as malunion, non-union, and fixation device failure [1,2]. Also, post union osteopenia in the vicinity of the plate is undesirable since it can reduce the stiffness and strength of the bone, which can make the structure prone to refracture [1,4-6]. Bone loss is in large part because of the implant high rigidity that causes the bone to sustain a reduced portion of the loads shielding the adjacent bone from the loads it is naturally subjected to [7,8]. This, in turn, can cause the bone to adapt itself by reducing its density [9]. Resorption can also occur as a result of compromising the periosteal blood supply imposed by the plate [2,10].

Although single lateral plating has been successfully used, and has been widely accepted for treating periprosthetic femoral fractures in the last two decades [4,11–14], other treatment options such as using allograft strut alone [15–18], or a combination of lateral fixation plate and cortical allograft have also been considered in the literature [19,20]. Single anterior plating, although not very common due to intraoperative difficulties, can also be used as a means of osteosynthesis. Given that the hip joint and the muscle forces acting on the femur tend to medially bend the intact femur, and given that placing the plate on the lateral side counters this natural deformation, it is worth investigating the long term bone mineral density (BMD) changes in both anterior and lateral plating to determine how altering the plating side can enhance the load sharing behavior of the bone plate.

To investigate peri-implant bone loss, BMD is normally measured using dual X-ray absorptiometry (DEXA), or computed tomography (CT) scans [21]. There are a number of clinical follow-ups that investigated BMD changes following total hip arthroplasty (THA) [22–24] and after plate removal in diaphyseal fractures [5,10,25]. The latter investigations on BMD changes after plate removal in unimplanted bones were done in the 1980s and early 1990s when plating, rather than intramedullary nailing, was considered as the treatment technique for diaphyseal fractures. Although periprosthetic

* Corresponding author. Tel.: +1 416 979 5000 ext. 7092; fax: +1 416 979 5265.
E-mail addresses: ptavakko@ryerson.ca, ptavakkoliavval@centennialcollege.ca (P.T. Avval), saeid.samiezadeh@ryerson.ca (S. Samiezadeh), habiba.bougherara@ryerson.ca, habiba.bougherara@gmail.com (H. Bougherara).

http://dx.doi.org/10.1016/j.medengphy.2015.11.013
1350-4533/

fractures with stable hip prosthesis are commonly treated with a bone plate, limited data is available regarding the long-term BMD changes in femurs implanted with both hip prosthesis and bone plate in the case of Vancouver B1 periprosthetic fractures, which are quite common. As long-term follow-ups are usually costly and time consuming, using a realistic validated model to predict the post-union BMD changes in periprosthetic femoral fractures is essential, and could provide clinicians with the long-term response of such implantation.

Among existing bone remodeling models, several are mechanical-based [26–28] which consider stress, strain, strain energy, or mechanical damage as mechanical stimuli for bone remodeling but fail to take into account the underlying biological mechanisms that control this process. Contrariwise, mechano-biochemical models [29–34] consider biological mechanisms as well as mechanical loading for regulating bone remodeling, and therefore present a more realistic predictive model.

In this study, femoral density changes in response to a bone fracture plate and a hip implant were investigated using a validated mechano-biochemical model (thermodynamic-based model) recently developed by the authors [29,33,34]. BMD changes were evaluated separately for lateral plating and anterior plating and the results were compared thereafter. As plate removal is being limited to symptomatic patients [1], having an understanding of the long-term behavior of plated bones can help clinicians choose proper plating techniques, and help bioengineers design more effective bone plates.

2. Materials and methods

2.1. Mechano-biochemical model (thermodynamic-based model)

In the mechano-biochemical model (thermodynamic-based model) that was developed and validated in previous studies [29–31,33,34], bone was hypothesized as a thermodynamic system that exchanges energy, matter and entropy with its surroundings. In this model, it is assumed that the bone remodeling mechanisms are executed by bone resorption and bone formation phases through five biochemical reactions, i.e., formation of multinucleated osteoclasts, old bone decomposition, production of osteoblast activator, osteoid production, and calcification. These reactions are in the form of Michaelis–Menten enzyme reaction (Eq. (1)) [35]:

$$E + S \underset{k_{-\alpha}}{\overset{k_{+\alpha}}{\rightleftarrows}} SE \rightarrow E + P, \tag{1}$$

where enzyme (E) retroactively binds to substrate (S) to form the complex of SE. Then, the complex decomposes into the enzyme and a product (P). Aforementioned biochemical reactions in bone remodeling mechanisms have been detailed in Appendix, along with our previous studies [29,33,34]. As shown in the Appendix, equations (A. 1) to (A. 5) contain 15 substances (N_1, $MCELL$, $MNOC$, N_4, Old_B, … and N_{15}) whose molar concentrations will be denoted by $[N_1]$, $[MCELL]$, $[MNOC]$, $[N_4]$, $[Old\_B]$, … and $[N_{15}]$, respectively.

In the mechano-biochemical model (thermodynamic-based model), it is assumed that the coupling between mechanical and biochemical fluxes, or forces, drives the bone remodeling mechanisms. Although mechano-biochemical coupling is in the early stages of characterization, there are experimental findings confirming this phenomenon [36]. Including the effect of mechanical factors on biochemical reactions is a crucial component in modeling bone adaptation processes. To achieve this consideration, the standard law of mass action was replaced by a modified version, represented by Eq. (2), which takes the coupling between the applied mechanical load and biochemical affinity of the reactions (or concentration of substances in the biochemical reactions) into account [29–31,33,34]:

$$r_\alpha = l_{\alpha\alpha}A_\alpha + l_{\alpha\upsilon}d_{(1)} = k_{+\alpha}\prod_{i=1}^{n}[N_i]^{\upsilon_{\alpha i}} - k_{-\alpha}\prod_{i=1}^{n}[N_i]^{\upsilon'_{\alpha i}} + l_{\alpha\upsilon}d_{(1)}, \tag{2}$$

where the rate and affinity of the αth biochemical reaction (α is the reaction number) are denoted by r_α and A_α, respectively. $d_{(1)}$ denotes the first invariant of the strain rate tensor, representing the rate of volume change. Phenomenological and reaction rate coefficients are shown by l and $k_{\pm\alpha}$, respectively. $\upsilon_{\alpha i}$ and $\upsilon'_{\alpha i}$ are the stoichiometric coefficients of the mixture of N_i entering and leaving the αth reaction, respectively.

The reaction rate calculated by Eq. (2) was incorporated into Eq. (3), which describes the time changes of molar concentration ($[\dot{N}_i]$) in terms of stoichiometric coefficients ($\upsilon_{\alpha i}$ and $\upsilon'_{\alpha i}$), reaction rate (r_α) and substrate flux (j_i).

$$[\dot{N}_i] = \sum_{\alpha=1}^{5}\left(\upsilon'_{\alpha i} - \upsilon_{\alpha i}\right).r_\alpha + j_i\,. \tag{3}$$

The aforementioned substitution provides a system of differential equations describing the mechanism of bone remodeling. In other words, using the modified version of the law of mass action, the time evolution of the concentration of biochemical substances involved in the bone remodeling mechanisms was described by a set of differential equations. Assuming the fluxes are constant in time, and the forward reactions are dominant in all considered biochemical reactions, the system of differential equations, and subsequently their stationary solutions, for five independent variables were obtained. The differential equations are presented in Eqs. (4) to (8) (in dimensionless/normalized form) [29], and their stationary solutions are given in Eqs. (9) to (13) (in dimensionless/normalized form) [29,33,34]:

$$\dot{\overline{[MCELL]}} = -\delta_1(\beta_1 + \overline{[MCELL]})\,\overline{[MCELL]} + J_3 + J_{14} - D_1\,, \tag{4}$$

$$\begin{aligned}\dot{\overline{[Old\_B]}} = &-(\beta_3 - \overline{[MCELL]} + \overline{[Old\_B]} + \overline{[Activ\_OB]} + \overline{[Osteoid]} + \overline{[New\_B]})\\ &\cdot\overline{[Old\_B]} - \delta_3(\beta_7 - \overline{[Old\_B]} - 2(\overline{[Activ\_OB]} + \overline{[Osteoid]} + \overline{[New\_B]}))\\ &\cdot\overline{[Old\_B]} + 2J_{14} - D_2 - D_3,\end{aligned} \tag{5}$$

$$\begin{aligned}\dot{\overline{[Activ\_OB]}} = &\delta_3(\beta_7 - \overline{[Old\_B]} - 2(\overline{[Activ\_OB]} + \overline{[Osteoid]} + \overline{[New\_B]}))\overline{[Old\_B]}\\ &-\delta_4(\beta_{10} - \overline{[Osteoid]} - \overline{[New\_B]})\overline{[Activ\_OB]} + D_3 - D_4\,,\end{aligned} \tag{6}$$

$$\begin{aligned}\dot{\overline{[Osteoid]}} = &\delta_4(\beta_{10} - \overline{[Osteoid]} - \overline{[New\_B]})\overline{[Activ\_OB]}\\ &-\delta_5(\beta_{13} - \overline{[New\_B]})\overline{[Osteoid]} + D_4 - D_5\,,\end{aligned} \tag{7}$$

$$\dot{\overline{[New\_B]}} = \delta_5\left(\beta_{13} - \overline{[New\_B]}\right)\overline{[Osteoid]} - J_{14} + D_5\,, \tag{8}$$

$$\overline{[MCELL]} = \frac{1}{2}\left(-\beta_1 + \sqrt{{\beta_1}^2 + 4\frac{-D_1 + J_3 + J_{14}}{\delta_1}}\right), \tag{9}$$

$$\overline{[Old\_B]} = \frac{1}{2}\left(\begin{array}{l}-\left(\beta_7 + 2\beta_3 - 2\overline{[MCELL]}\right) +\\ \sqrt{\left(\beta_7 + 2\beta_3 - 2\overline{[MCELL]}\right)^2 + 4\left(\frac{J_{14}-D_3}{\delta_3} + 2J_{14} - 2D_2\right)}\end{array}\right), \tag{10}$$

$$\overline{[Activ\_OB]} = \frac{1}{2}\left(\begin{array}{l}-\left(\beta_{10} + \frac{1}{2}\left(\overline{[Old\_B]} - \beta_7 + \frac{J_{14}-D_3}{\delta_3\overline{[Old\_B]}}\right)\right) +\\ \sqrt{\left(\beta_{10} + \frac{1}{2}\left(\overline{[Old\_B]} - \beta_7 + \frac{J_{14}-D_3}{\delta_3\overline{[Old\_B]}}\right)\right)^2 + 4\frac{J_{14}-D_4}{\delta_4}}\end{array}\right), \tag{11}$$