The effect of functional overloading on crestal bone loss around a dental implant

At the end of the bone remodeling process, frequently funnel-like collapses of the cortical bone (crestal bone losses) are clinically registered around the neck of dental implants. It is generally suspected that either inflammations or mechanical “overloading” of the bone during the clinical loading phase are responsible for this phenomenon. From the results of this numerical simulation we conclude that overloading might indeed be a major cause for these bone losses.

1. Bone reossification process and Finite Element (FE) model

Due to mechanical and thermal damage of the bone during the insertion (drilling) process, a necrotic zone, the so-called repair zone, containing demineralized bone matrix and coagulum, is created around the implant. The reossification of this necrotic zone is the result of two processes: 1) A purely biological process triggered immediately after insertion of the implant which, in absence of external stimuli, would only lead to an insufficient wound healing, and 2) a mechanobiological process induced with a retardation of several days which, under the effect of external mechanical stimuli leading to stresses in the bone, is indispensable for a (at least) partial reossification of the repair zone. Nevertheless, quite often a funnel-like crestal bone loss is clinically registered around the neck of implants (Fig. 1).

The (conventional) two-stage implantation method consists of a (primary) healing phase where the implant remains hidden underneath the mucosa for several months thus not being exposed to direct loads and the surrounding bone experiencing only stresses due to functional deformations of the mandible, and a (secondary) functional phase when, after a second surgical step, the implant is connected with a suprastructure (crown, bridge, prosthesis) thus being exposed to direct loads from now on.

In the reossification model [1] the equivalent (von Mises) stress \( \sigma_{eq} \) is taken as the reference quantity for the bone response. This model contains the following characteristics:

1. Bone apposition, characterized by an incremental increase \( \Delta E_c \) and \( \Delta E_s \) of Young’s modulus in an element of the cortical and spongy section of the repair zone, is allowed if the response \( \sigma_{eq} \) falls into a given “response window” \([\sigma_{eq,lower}, \sigma_{eq,upper}]\).

2. Bone resorption, characterized by an incremental decrease of Young’s modulus, occurs if the response falls out of the response window, i.e. either by “underloading” or by “overloading”.

3. Revascularization: Bone apposition has to follow the reconstruction of blood vessels, i.e. through “bridging” from the intact bone surface in direction towards the implant.

This simulation used a realistic mandible characterized by an anatomically realistic distribution of cortical (crestal) and spongy (trabecular) bone in the cross-section, with a cylindrical titanium implant (diameter: 4 mm, insertion depth: 16 mm) at the site of a premolar. The mandible was discretized into 40,023 elements with trilinear shape functions corresponding to 11,560 nodes. An especially fine mesh (5 layers) was chosen for the repair zone which was assumed to have a width of 500 \( \mu \)m. A perfect bond between the tissue of the repair zone and the implant was presumed. The Young’s moduli (t: titanium, c: cortical bone, s: spongy bone) were chosen as \( E_t = 110 \) GPa, \( E_c = 18 \) GPa, \( E_s = 2 \) GPa, and the Poisson’s ratios as \( \nu_t = 0.33, \nu_c = \nu_s = 0.3 \).

The periodic stimuli (loads) were produced by a distribution of muscle (m.) forces in the m. temporalis, m. mas- seter and m. pterygoideus medialis such that a (moderate) chewing force consisting of an axial force of 100 N and alternating lateral forces of 30 N (in lingual and buccal direction) were created. In the healing phase the force system was applied to the contralateral side of the mandible (no direct loading of the implant), and in the functional phase on the ipsilateral side (direct loading). The periodic loading (iteration) was continued until a stable final configuration was obtained in each phase. The degree of ossification was defined as \( \kappa_c(n) = (\Sigma E_i(n)V_i)/E_cV_c \) and the results of this numerical simulation we conclude that overloading might indeed be a major cause for these bone losses.
\( \kappa_s(n) = \frac{\sum \kappa_i(n) \cdot V_i}{E_s \cdot V_s} \), where \( \kappa_i(n) \): Young's modulus of the \( i \)-th element after the \( n \)-th load (iteration) step, \( V_i \): volume of the element, \( E_c \) and \( E_s \): target values of Young's modulus as mentioned above, \( V_c \) and \( V_s \): volume of the cortical and spongy section, respectively, in the repair zone.

**2. Results of a special simulation**

The following results (Fig. 2) are based on a special simulation with the initial value \( E_0 = 500 \) MPa for the repair zone, the response window \([0.3 \) MPa, \( 5.0 \) MPa\]), and the incremental increases and decreases \( \Delta E_c = \pm 100 \) MPa, \( \Delta E_s = \pm 200 \) MPa. Special attention is directed to the following 3 (of the 5) layers of the repair zone: layer adjacent to the intact bone surface (itb), central layer (rpz), and layer adjacent to the implant (imp).

According to this simulation, a rather homogeneous reossification of the spongy section of \( \kappa_s \approx 89\% \) is achieved in the final configuration. The cortical section, however, as shown in Fig. 2, after also having reached a nearly homogeneous reossification of \( \kappa_c \approx 85\% \) at the end of the healing phase (grey region), experiences a drastic decrease of \( \kappa_c \) during the functional (direct loading) phase (white region), this decrease being most pronounced for the layer adjacent to the implant \( (\kappa_c \approx 15\%). \)

**3. Discussion and conclusion**

On the basis of the presented reossification model, the two-stage implantation method reveals a drastic loss of cortical bone, especially around the neck of the implant, during the functional phase of the mechanobiological repair process. If it is assumed in the simulation model that elements in the cortical section of the repair zone with \( E_c < 2 \) GPa have not been reossified and elements with \( E_c > 2 \) GPa have undergone full reossification, the funnel-like crestal bone loss as sketched in Fig. 3, can be reproduced which resembles much the clinical situation shown in Fig. 1.

It may therefore be concluded that, besides inflammations, mechanical overloading might indeed be responsible for the cortical bone loss around the neck of implants.

**4. References**


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