THE MECHANISM OF BONE REMODELING AND RESORPTION AROUND PRESS-FITTED THA STEMS

B. VAN RIETBERGEN, R. HUISKES, H. WEINANS,* D. R. SUMNER, T. M. TURNER,* and J. O. GALANTE†

*Biomechanics Section, Institute of Orthopaedics, University of Nijmegen, 6500 HB Nijmegen, The Netherlands, and † Rush-Presbyterian-St. Luke's Medical Center, Department of Orthopedic Surgery, Chicago, Illinois, U.S.A.

Abstract—A major problem threatening the long-term integrity of total hip replacement is the loss of proximal bone often found around noncemented stems in the long term. It is generally accepted that 'stress shielding' is the cause for this problem: after implantation of the prosthesis the surrounding bone is partially 'shielded' from load carrying and starts to resorb. One of the proposed answers to this problem is the application of press-fitted stems. These smooth-surfaced implants are thought to provoke higher proximal bone loading, and, hence, less stress shielding than bonded implants, because they are wedged into the femur every time when loaded. However, in a two-year experiment in dogs, similar amounts of resorption of the proximal cortex were found around press-fitted and bonded implants. The question arises how similar resorption patterns can develop under completely different stress conditions, and whether this phenomenon can be explained by adaptive bone remodeling theories based on Wolff's law.

In the present study an answer was sought for this question. An advanced iterative computer simulation model was used to analyze the remodeling process in the animal experiment. Three-dimensional finite element models were constructed from the animal experimental configuration, in which smooth, press-fitted stems were applied unilaterally in the canine. The FE model was integrated with iterative remodeling procedures, validated in earlier studies. In the model an appropriate non-linear representation of the loose bone-implant interface was realized, also capable of simulating the proximal interface gap that was found around the uncoated implants. The simulation models predicted similar amounts of proximal bone loss and distal bone densification as found in the animal model. Hence, the cortical bone loss could indeed be predicted by the strain-adaptive bone remodeling theory. By unraveling the simulation process, the question stated above could be answered. Densification of the distal bone bed during the initial remodeling process was found to cause reduced axial stem displacement (elastic subsidence), decreasing the wedging effect of the stem and, hence, decreasing the loading of the proximal bone, resulting in proximal bone loss. Hence, whereas in the case of bonded stems the proximal resorption process develops monotonously to a new equilibrium, the process around smooth, press-fitted stems develops nonmonotonously. This is due primarily to the unbonded interface conditions and the development of a proximal fibrous membrane. The remodeling process then gradually causes the stem to be jammed in the distal diaphyses (proximal 'stress bypass').

INTRODUCTION

Bone resorption around femoral hip stems is a disturbing phenomenon, threatening the long-term integrity of stiff, noncemented implants in particular. Resorption, in the sense of reduced cortical thickness and increased porosity, is seen in most patients who have received noncemented total hip arthroplasty (Engh et al., 1987; Galante, 1988; Kiratli et al., 1991; Rosenberg, 1989). It has also been reported in animal experiments (Bobyn et al., 1990; Miller and Kelebay, 1981; Sumner et al., 1992a; Turner et al., 1986). Major clinical problems in patient series caused by this phenomenon have not been reported as yet. However, it is uncertain how long the process will continue late postoperatively, or whether it reaches a status quo at a particular point in time. Late loosening, stem failure or bone fracture are problems which might occur, not to mention a reduced amount of bone stock available for revision surgery. Although the amount of resorption in the mid-long-term in patient series was usually described as moderate (Engh et al., 1987; Rosenberg, 1989), recent studies with more precise X-ray methods such as dual-energy absorptiometry (Kiratli et al., 1991) suggest that 40–60% bone loss in five years time is a rule rather than an exception. They also suggest that the resorption processes have not terminated by this time (Steinberg et al., 1991). Certainly, bone resorption around prostheses is a problem worth avoiding and, hence, worth investigating.

It is generally assumed that the resorptive phenomena are a result of 'stress shielding' and adaptive bone remodeling in accordance with 'Wolff's law'. The stem 'shields' the bone from stress because it shares the load which is normally taken by the bone alone. As a result, the bone stresses are reduced and the bone-remodeling process then reduces its mass accordingly. Because the bone stress and stress-shielding patterns depend on the material, geometrical and bonding characteristics of the implant (Huiskes, 1990; Lewis et al., 1984; Rohmann et al., 1988; Walker et al., 1987), the bone resorption patterns must also depend on these characteristics. For example, when a stem is less stiff, stress shielding is less severe and, hence, the amount of bone resorbed should
be reduced. This was indeed confirmed in patient studies and animal experiments (Bobyn et al., 1990; Engh et al., 1987; Maistrelli et al., 1991). Recently, strain-adaptive bone-remodeling theories have been developed which can be used in combination with finite element models of THA configurations to describe the remodeling process quantitatively and predict the resorption patterns depending on the implant characteristics (Carrier, 1987; Cowin and Hegedus, 1976; Huiskes et al., 1987, 1989; Orr et al., 1990; Weinans et al., 1992). This method was used to simulate animal experiments with hip prostheses and predict the resorption patterns (Weinans et al., 1992). The similarity between the predicted and the experimental results provided confirmation for the mechanical basis of the resorption process and validation of the theory.

Because of the relationship between implant characteristics and stress patterns, prosthetic designs can be optimized to produce as little bone loss as possible. It has been proposed, for instance, to fabricate stems out of titanium instead of CoCrMo, making them less stiff, and even to use ‘iso-elastic’ composites with a similar elastic modulus as bone (Bobyn et al., 1990; Maistrelli et al., 1991; Orth et al., 1991; Sumner et al., 1992b). Another measure often applied is the limitation of porous or hydroxyapatite coatings to the proximal part of the stem only, thereby promoting proximal load transfer and limiting the extent of stress shielding (Engh and Bobyn, 1988; Geesink, 1989). A third method often suggested to avoid resorption is the application of smooth press-fitted stems without coating (Huiskes et al., 1989; Walker et al., 1987). The philosophy behind this method of fixation is that the (tapered) stem is wedged into the medullary canal (‘press-fitted’), whereby the endosteal cortex is compressed continuously by the hip-joint force. The endosteal compression then generates hoop stresses in the bone, which cause it to maintain its mass. An additional feature is the reduced rigidity of the stem–bone complex because of the lack of bonding. This will cause more bone deformation in bending and, hence, less stress shielding. These expectations have been confirmed in FE analyses (Huiskes et al., 1989, 1990; Rohlmann et al., 1988) and in laboratory experiments with femur specimens (Fulghum et al., 1991; Walker et al., 1987). In both cases, the bone stresses around bonded and unbonded, press-fitted stems were compared, and it was found that they were higher in the case of the press-fitted prosthesis. In an animal experiment described below, the postoperative morphology around press-fitted (smooth, uncoated) THA stems was investigated and compared with previously studied bonded (porous ingrowth) stems of the same design (Sumner et al., 1992a; Turner et al., 1986). Six months postoperatively, marked cortical resorption around the bonded stems, but no statistically significant bone loss around the press-fitted stems, was noted. Hence, the theory seems to be confirmed. However, at two years, similar amounts of bone loss were observed with the bonded and press-fitted stems.

So the question is, that if the bone-remodeling and resorption phenomena around hip stems can be explained by adaptive bone remodeling, and if the stress patterns around bonded and unbonded stems are so different, how can it be that they produce the same remodeling patterns in the long term? Can an explanation be found which is consistent with adaptive bone remodeling theory and Wolff’s law? And, if so, can this explanation be extrapolated to a better understanding of bone remodeling around unbonded stems in general? These questions were addressed in the present project, using the working hypothesis that the remodeling in the unbonded stem case was influenced by the mechanical effects of a fibrous tissue layer around the stem, which developed gradually postoperatively.

To answer these questions an animal model and a computer simulation model were used in combination. The strain-adaptive bone-remodeling theory was applied in a three-dimensional FE model of the canine THA configuration. Whereas the theory was validated earlier relative to experiments with bonded implants (Weinans et al., 1992), this time it was applied to simulate the bone-remodeling process around the unbonded, smooth implants.

**METHODS**

**Animal experiment**

Eleven adult male mongrel dogs which had a body mass of 26–46 kg underwent left total hip replacement. Each animal received a cemented ultrahigh molecular weight polyethylene acetabular component and a cementless, titanium alloy (Ti6A14V) femoral component (Fig. 1). The prostheses were identical in geometry to circumferentially full-length porous-coated prosthesis reported previously (Sumner et al., 1992a; Turner et al., 1986), but no porous coating was applied. These stems were rectangular in cross section with rounded corners and tapering in the mediolateral direction. The surface was finished by bead blasting.

The surgical procedure and postoperative care have been described in detail elsewhere (Turner et al., 1986). The medullary cavity was prepared in order to obtain an interference fit between the implant and the bone. Radiographs were taken immediately after surgery, and at one, three, six, 12, 18 and 24 months. The animals were killed six months or two years after surgery by a lethal intravenous injection of barbiturates.

Contact radiographs were made of the femora and then the bones were oriented in a standard position in a jig so that comparable sections could be obtained from the operated and contralateral sides (Sumner et al., 1988). The bones were sectioned perpendicular to the long axis of the diaphysis at 1 cm intervals and prepared for undecalcified histology and measurement of the cortical area and medullary bone density following methods outlined previously (Sumner et al., 1992a). The cortical area (CA), the area bounded by the subperiosteal and endocortical surfaces, was measured and the change in cortical area of the operated femur (dCA) was determined from

\[
dCA = \left( \frac{CA_{\text{treated}} - CA_{\text{control}}}{CA_{\text{control}}} \right) \times 100\%,
\]

where \(CA_{\text{treated}}\) is CA for the operated femur and \(CA_{\text{control}}\) is CA for the contralateral femur. The medullary bone area fraction (MBAF) of the treated and contralateral control femur was determined from

\[
\text{MBAF} = \left( \frac{MBA}{MA - PA} \right) \times 100\%,
\]

where MBA represents the area of bone within the medullary canal, MA is the medullary area (i.e. the area bounded by the endocortical surface) and PA is the prosthetic area. For the control side, \(PA = 0\). For the statistical analyses, the difference in MCAF (dMBAF) was calculated in a fashion analogous to the calculation of dCA. For purposes of presentation it is more helpful to present the actual values of MCAF for the control and the treated sides rather than dMBAF. Because of the nature of the host–implant interface was studied using toluidine blue and basic fuchsin surface stained sections. The thickness of the membrane found between the implant and host bone was measured at three equivalent points on each stem face using an eyepiece micrometer. These measurements are reported as the average thickness of the membrane for the proximal (sections B and C), middle (sections D and E) and distal (sections F and G) levels of the stems.

Multivariate analyses of the variance for repeated measures were used to assess the influence of section location.
Fig. 1. The femoral stem was made from Ti6Al4V and was not porous coated.
Fig. 6. Contact radiographs of four sections from the two-year postoperative dog femurs of a typical dog. The operated femur is shown on the left and the contralateral (control) femur on the right.
Fig. 2. Three-dimensional finite element meshes of the operated femur (left) and the contralateral femur (right). The sections B–J, which correspond to the animal experimental ones, are indicated. The stem tip is located near section H.

and time on membrane thickness. Similar analyses, paired t-tests and Student's t-tests were used to analyze dCA and dMBAF. Correlation matrices between these variables were calculated.

Finite element model

Three FE models were created, one representing the direct postoperative femur with prosthesis and direct bone contact, one representing the same with a partial fibrous interface, and a third model representing the contralateral femur of the dog. The geometry of the contralateral model was determined from contact radiographs of the 2 mm slices, together with longitudinal radiographs from a lateral and an anteroposterior view, of one particular dog. The periosteal and endosteal contours of the cortical bone cross sections were digitized from the contact radiographs of the nonoperated femur. The periosteal contours in the longitudinal radiographs of this femur were also digitized to determine the three-dimensional orientations of the cross sections. Using a finite element preprocessor, the shape determined by the cortical contours was divided into a three-dimensional finite element mesh, consisting of isoparametric brick elements with eight nodes. A finite element mesh of the contralateral femur was obtained in this way (Fig. 2).

This model was mirrored to represent the left leg of the dog. In addition, contour measurements of the prosthesis were made from the digitized cross-sectional radiographs and longitudinal radiographs of the treated femur of the dog. These data were used to dimension the elements representing the implant. The elements representing the femoral head in the contralateral model were replaced by elements representing the prosthesis, to create the finite element model for the direct postoperative femur (Fig. 2).

Loads

The orientations and points of application of the loads on both the contralateral and the treated femur were identical and chosen in accordance with Bergman et al. (1984). The force on the femur head was 288 N, 15° medial of the central axis and 30° anterior of the mid-frontal plane (Fig. 2). In the treated femur model, the neck of the prosthesis was modeled in such a way that the point of application of this force is the same in both models. A distributed load of 144 N, 30° medial of the central axis, was applied to the greater trochanter of both models.

Material properties

The material properties of the bone were taken as linear-elastic and isotropic. The Young's modulus of bone was determined from the apparent density values by a cubic relationship (Carter and Hayes, 1977). The maximum value for the Young's modulus of cortical bone was taken as 22,000 MPa. The corresponding maximal apparent density \( \rho_{\text{max}} \) was taken as 1.73 g cm\(^{-3}\); hence, the relationship between modulus and density is given by

\[
E = c \rho^3, \quad (3)
\]

with \( c = 4249 \) (MPa g\(^{-3}\) cm\(^{-3}\)).

The apparent densities in the cross sections C, D, F, H and J were taken from the mean values of the medullary bone area fractions (MBAF) in the control femurs, of 22 dogs used in a previous study (Weinans et al., 1992), to represent the
immediate postoperative density distribution. A MBAF of 100% corresponds to an apparent density of 1.73 g cm\(^{-3}\); hence, the MBAF values were multiplied by 1.73/100% to calculate the apparent density. The unknown densities in the cross sections B, E, G, I and K were calculated from the interpolated MBAF values. In the immediate postoperative situation, the density distribution in each section was assumed uniform. The modulus of the cancellous bone of the femoral head and the greater trochanter were assumed uniform, with a value of 1000 MPa. This value was taken rather arbitrarily, since its effects on the deformations in the rest of the structure are relatively small. The Young’s modulus of the titanium alloy stem was taken as 110,000 MPa.

Interface conditions

No bonding between implant and surrounding bone was assumed. Hence, appropriate nonlinear interface conditions have to be specified in the model of the treated femur. This was achieved by using special nonlinear gap elements at the bone–implant interface (MARC Analysis Corporation, Palo Alto, CA). These elements allow local slip and separation to occur. In the model no friction was assumed at the interface. Hence, only compressive stresses, directed normal to the surfaces, are transferred through the interface. By implementing these nonlinear gap elements, the FEM problem has to be solved in an iterative process to determine the status of the gap elements (i.e. open or closed).

To study the effects of a proximal fibrous tissue interface, relative to a situation with intimate bone–implant contact, two models were constructed. In the first model, intimate bone–implant contact was assumed. In this case the model represents an implant that fits exactly in the reamed canal (cancellous or cortical), \(a(p)=A(p)/V\), where \(V\) is the volume and \(A(p)\) the apparent density of bone. Hence, if the rate of net bone-mass turnover equals \(dM/dt\), then

\[
\frac{dM}{dt} = S - S_{\text{ref}}. \tag{5}
\]

The theory further assumes a threshold level for the remodeling response (Frost, 1964). Hence, when \(|S - S_{\text{ref}}|\) is smaller than the threshold value, no remodeling response occurs. The remodeling objective can, thus, be formulated by

\[
(1 - s)S_{\text{ref}} \leq S \leq (1 + s)S_{\text{ref}}, \tag{6}
\]

with \(s\) a constant. The region between \((1 - s)S_{\text{ref}}\) and \((1 + s)S_{\text{ref}}\) represents the nonresponsive area, or ‘dead zone’. In accordance with the earlier validation study (Weinans et al., 1992), \(s\) is set to 0.35, or 35%.

As it is the objective to simulate external remodeling (or surface modeling) at the peripheral surfaces and internal remodeling in trabecular bone concurrently, a method is needed to connect the two processes in the time domain. For that purpose, the theory of Martin (1972) is applied. According to this theory, it is assumed that bone apposition and resorption can occur only at free bone surfaces and, hence, at the periosteal bone surface (external remodeling or modeling) and inside the bone at the pore surfaces (internal remodeling). Martin (1972) calculated the amount of internal free surface as a function of the porosity characteristics, assuming spherical shaped pores. Using these assumptions, the internal free surface area per unit volume of whole bone (cancellous or cortical), \(a(p)=A(p)/V\), was estimated (Fig. 4). For \(\rho = \rho_{\text{max}} = 1.73\text{ g cm}^{-3}\) it is assumed that

\[
\begin{align*}
\text{Free surface density } a(p) &\text{ in mm}^2/\text{mm}^3 \\
\text{Density } \rho &\text{ in g cm}^{-3}
\end{align*}
\]

Fig. 3. Dimensions of the proximal gap as modeled in one of the finite element models of the operated femur.

Fig. 4. Relationship between the apparent density \(\rho\) in g cm\(^{-3}\) and the free surface area per unit of bone volume \(a(p)=A(p)/V\) in mm\(^2\) mm\(^{-3}\).
\[ a(p) = 0.0; \text{ hence, no remodeling takes place if } p = p_{\text{mar. TO}}. \]

To avoid inaccuracies in the FEM calculation, the minimum value for the apparent density was set to \( p_{\text{min}} = 0.01 \text{ g cm}^{-3} \), which then represents complete resorption.

The adaptive process in the operated femur can then be expressed in terms of the rate of net bone turnover:

\[
\frac{dM}{dt} = tA(p)[S-(1-s)S_{\text{ref}}], \quad \text{if } S < (1-s)S_{\text{ref}},
\]

\[
\frac{dM}{dt} = 0, \quad \text{if } (1-s)S_{\text{ref}} < S < (1+s)S_{\text{ref}},
\]

\[
\frac{dM}{dt} = tA(p)[S-(1+s)S_{\text{ref}}], \quad \text{if } S \geq (1+s)S_{\text{ref}},
\]

where \( T \) is a time constant given in g^2 mm^-2 J^-1 per month, \( A(p) \) is the free surface at the periosteum or in the internal bone structure (Fig. 4), and \( s \) represents the 'dead zone' threshold level. The time \( t \) is given in units of months. With the finite element model of the contralateral femur, the reference signal \( S_{\text{ref}} = [U(p)_{\text{ref}}] \) and with the model of the operated femur the actual signal \( S \) are determined.

The rate of net bone turnover \( \frac{dM}{dt} \) can now be expressed as the rate of change of the external (periosteal) geometry \( \frac{dx}{dt} \):

\[
\frac{dM}{dt} = \rho A \frac{dx}{dt},
\]

with \( A \) the external surface area at which the rate of mass change \( \frac{dM}{dt} \) takes place (the external face of the element concerned) and \( x \) a characteristic surface coordinate, perpendicular to the periosteal surface. For the adaptation of the internal bone mass due to porosity changes we use

\[
\frac{dM}{dt} = \frac{dA}{dt} \frac{dA}{dx}.
\]

with \( V \) the volume in which the bone mass change takes place (the volume of the element concerned) and \( \frac{dA}{dx} \) the rate of change in apparent density. Equation (7) can now be written in terms of \( \frac{dx}{dt} \) for modeling and in terms of \( \frac{dA}{dt} \) for remodeling. Through forward Euler integration, the equations can be solved iteratively to find the new coordinates of the surface nodes and the new apparent density values in the integration points after every iterative step. In the computer program, the integration is carried out in steps of \( t \Delta t \), which represents the proceeding of the modeling and remodeling processes at an arbitrary scale, which can be considered as the simulation time scale. The time constant \( \tau \) was empirically determined in the earlier validation study by comparing the two-year animal and simulation results for bonded implants (Weinans et al., 1992). It was found that the value of \( \tau \) should be set to \( \tau = 130 \text{ g}^2 \text{ mm}^{-2} \text{ J}^{-1} \) per month to have the time \( t \) given in units of one month.

The time step in the integration process is variable and determined in each iterative step such that the maximal density change in the integration point where the maximal rate of density change occurs will not exceed \( \frac{d}{d \text{max}} = 0.865 \text{ g cm}^{-3} \). This can be accomplished by calculating the time step \( \Delta t \) after each iterative step, using the maximal possible value for the expression \( u(p)[S-(1 \pm s)S_{\text{ref}}] \) from all integration points, as

\[
\Delta t = \frac{0.865}{\tau A(p)[S-(1 \pm s)S_{\text{ref}}]_{\text{max}}},
\]

with the positive sign if \( S \geq (1+s)S_{\text{ref}} \) and the negative sign if \( S \leq (1-s)S_{\text{ref}} \).

The scheme of Fig. 5 demonstrates the iterative computer simulation program. The model of the intact contralateral (control) femur provides the reference signal \( S_{\text{ref}} \), which is compared in each integration point (internal remodeling) and in each periosteal nodal point (external modeling) with the actual signal \( S \) in the corresponding point in the model of the operated femur. The differences between \( S \) and \( S_{\text{ref}} \) determine the rates of modeling and remodeling, which occur concurrently. After each iteration a new \( S \) is determined. Due to the nonlinear gap elements used to simulate the unbonded interface, the calculation of the actual stimulus itself requires an iterative process as well.

In order to reach convergence, the process must be continued until no more density or geometric changes occur. This means that all points fulfill equation (6), or have reached the maximal or minimal values for the apparent density tolerated (1.73 or 0.01 g cm\(^{-3}\)), respectively. To monitor the convergence rate, object functions were defined according to

\[
F = \sum_{i=1}^{n} |S_i-(1 \pm s)S_{\text{ref}}|.
\]

The summation takes place over a number of (sensor) points, which are represented in the iterative remodeling process by the element integration points for internal remodeling and the periosteal nodal points for external modeling. Internal points with a maximal or minimal value for the apparent density tolerated (1.73 or 0.01 g cm\(^{-3}\)) or points having a signal within the dead zone, take no part in the summation of equation (11). The object functions for internal and external remodeling are determined after each iteration.

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Fig. 5. Schematic representation of the iterative computer simulation model.
Representation of data

For all cross sections B–J the medullary bone area fraction [MBAF, equation (1)] and the changes in cortical area [equation (2)] are determined in the simulation after each time step. The MBAF is determined within the FE code, by averaging the extrapolated volume fraction at the nodal points in the cross sections. The volume fraction is determined as \( p / p_{\text{max}} \). The cortical area (CA) is determined within the FE code by calculating the cross-sectional areas of the cortical elements directly from the coordinate data.

The density distributions as predicted by the simulation model are compared per cross section to the cross-sectional radiographs of the treated femur in the two-year animal experiment. In addition, the results of the simulation in terms of medullary bone area fraction and changes in cortical area are compared with the averaged experimental results over the whole animal experimental group.

RESULTS

Animal experiment

All of the animals bore weight on the operated limb within one week. The animals had a normal range of motion and appeared to have normal function within two weeks postoperatively and throughout the course of the study. At sacrifice all hips were stable and all acetabular and femoral components were securely fixed to the host bone. Figure 6 shows the two-year remodeling patterns in radiographs from four sections of a dog of the experimental group. The contralateral (untreated) control sections are shown as well.

The host–implant interface included, in order of most common occurrence, a membrane, intimate bone contact and marrow. The membrane consisted of collagen fibers and fibrocytes oriented parallel to the implant. The average thickness of the membrane increased from six months to two years proximally, but not distally (Fig. 7). In most places the membrane was separated from the surrounding marrow by a trabecular shell of bone. At two years, the proximal membrane was usually continuous with rare areas of bone or marrow in direct contact with the implant. Distally substantial portions of the interface included intimate bone contact at both six months and two years.

At two years there was a 20–23% reduction in cortical bone area proximally (Table 1). At six months the magnitude and extent of the proximal bone loss was not as great as at two years and the treated vs control side differences were more variable. Thus, at six months the differences between the treated and control femurs were not statistically significant, whereas at two years these differences were significant proximally \( (p<0.05) \). The thickness of the proximal bone–implant membrane was inversely related to the dCA at section C \((r = -0.69, p < 0.05)\) and section D \((r = -0.88, p < 0.001)\), indicating that a thicker proximal membrane was associated with a greater loss of proximal cortical bone.

There were 15-fold increases in the amount of medullary bone in both the six-month and the two-year animals near the tip of the stem (Table 2). Elsewhere, changes in the density of bone within the medullary cavity were less dramatic, although proximally in the six-month group there was an increase compared to the control \((p < 0.03)\). Distal to the prosthesis, there were no significant differences between the treated and control sides.

![Fig. 7. The average membrane thickness at six months and at two years (mean and 95% confidence interval).](image)

<table>
<thead>
<tr>
<th>Section</th>
<th>C</th>
<th>D</th>
<th>F</th>
<th>H</th>
<th>J</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>28.9</td>
<td>6.3</td>
<td>12.7</td>
<td>3.6</td>
<td>52.7</td>
</tr>
<tr>
<td>95% CI</td>
<td>25.3/32.5</td>
<td>5.8/19.5</td>
<td>11.8/16.7</td>
<td>2.9/6.3</td>
<td>49.9/56.7</td>
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<tr>
<td>Proximal</td>
<td>24.1</td>
<td>10.2</td>
<td>5.2</td>
<td>3.6</td>
<td>3.9</td>
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<tr>
<td>Middle</td>
<td>20.2</td>
<td>6.2</td>
<td>1.2/2.6</td>
<td>3.4</td>
<td>3.9</td>
</tr>
<tr>
<td>Distal</td>
<td>17.5</td>
<td>13.9</td>
<td>2.6</td>
<td>1.1/8.9</td>
<td>54.0</td>
</tr>
<tr>
<td>Membrane</td>
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<td>6.3</td>
<td>12.7</td>
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<tr>
<td>Thickness</td>
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<td>49.9/56.7</td>
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Table 1. Percentage change in cortical bone area measured in the six-month and the two-year animal experiment

<table>
<thead>
<tr>
<th>Section</th>
<th>Percentage change in cortical area</th>
<th>Six months</th>
<th>Two years</th>
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<tbody>
<tr>
<td>C</td>
<td>-16.1</td>
<td>-48.6/16.4</td>
<td>-22.7*</td>
</tr>
<tr>
<td>D</td>
<td>-1.6</td>
<td>-16.2/13.1</td>
<td>-20.0†</td>
</tr>
<tr>
<td>F</td>
<td>-5.8</td>
<td>-21.1/9.6</td>
<td>-11.6*</td>
</tr>
<tr>
<td>H</td>
<td>3.2</td>
<td>-7.8/18.3</td>
<td>3.5</td>
</tr>
<tr>
<td>J</td>
<td>3.6</td>
<td>-5.5/12.6</td>
<td>1.5</td>
</tr>
</tbody>
</table>

* Different from control, \( p < 0.05 \), paired t-test.
† Different from control, \( p < 0.01 \), paired t-test.
‡ Different from control, \( p < 0.001 \), paired t-test.

Table 2. Medullary bone area fraction measured in the six-month and the two-year animal experiment

<table>
<thead>
<tr>
<th>Section</th>
<th>Control</th>
<th>Medullary bone area fraction</th>
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</thead>
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<tr>
<td>C</td>
<td>24.7</td>
<td>38.2†</td>
</tr>
<tr>
<td>D</td>
<td>20.2</td>
<td>22.1</td>
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<tr>
<td>F</td>
<td>10.1</td>
<td>32.7‡</td>
</tr>
<tr>
<td>H</td>
<td>3.4</td>
<td>54.6</td>
</tr>
<tr>
<td>J</td>
<td>3.9</td>
<td>-1.1/8.9</td>
</tr>
</tbody>
</table>

* Combined data from the six-month and the two-year time periods.
† Different from control, \( p < 0.05 \), paired t-test.
‡ Different from control, \( p < 0.01 \), paired t-test.
§ Different from control, \( p < 0.001 \), paired t-test.
* Different from the six-month group, \( p < 0.05 \), t-test.
Computer simulation

In the immediate postoperative situation, we find high stresses (hence, high values of the remodeling signal $\Delta S$) in the bone surrounding the implant, particularly at the proximal side. This is illustrated for section C in Fig. 8. Particularly in the case of the perfectly fitting implant, the stem taper in combination with the loose interface generated interface compressive and hoop stresses which cause high values of the strain energy per unit of mass. In the model including the proximal gap (Fig. 8), the high-stress regions are at the medial side only; laterally, the gap is too wide for contact during loading.

As $\Delta S - S_{\text{ref}}$ is the stimulus for the bone-remodeling process, one would expect both densification of trabecular bone and bone apposition at the periosteal surface (Fig. 8). However, when we consider the two-year results of the simulation (Fig. 9), we find quite the opposite. In fact, proximally, in the antero-medial region in particular, we find cortical resorption in both models, with and without interface gap (sections C and D). In the middle region (section F) we also find some cortical resorption medially and laterally, very similar to what was found in the animal experiments (Fig. 6). The trabecular morphology is also very similar. Trabecular densification occurs adjacent to the implant and particularly distally. At the proximal side, the model with interface gap generated more bone loss than the model of the perfectly fitting stem (Fig. 9). On the distal side, the morphology is almost equal in both models.

The explanation for this seemingly inconsistent remodeling behavior, whereby the two-year results defy the immediate postoperative distribution of the remodeling signal, is found when the iterative process in the simulations is regarded more closely. In order to understand the course of affairs, it is important to note that the high stress values generated in the bone initially (Fig. 8) are caused by a wedging effect of the tapered implant, whereby loading the stem causes high interface compression through axial relative displacement (or elastic subsidence). Then, initially, densification of trabecular bone, adjacent to the whole length of the stem, indeed occurs during the first iterations. Proximally, the cortex increases in thickness, just as expected from the initial remodeling signal (Fig. 8). As an effect of the trabecular densification, the bone becomes gradually stiffer, particularly distally, where the trabecular width between the stem and the endosteal surface is small. After a few iterations, the distal densified bone bed begins to carry the stem, and the axial relative displacement upon loading reduces. Hence, also proximal interface loading reduces. So, while this process continues during the first iterations, the distal bone gradually takes over the load transfer from the proximal part, which starts resorbing. Figure 10 shows the distribution of the remodeling signal in section C of the model, with a proximal interface gap, right after this 'distal stem jamming' effect has occurred. As evident when comparing these values to those immediately postoperatively (Fig. 8), the proximal femur is now 'stress bypassed'. This mechanism occurs in both models, but is more pronounced and faster in the model with the proximal interface gap as compared to the one with the perfectly fitting stem.

The convergence of the iterative process according to equation (11) is illustrated in Fig. 11, showing the values of the object functions for internal and external remodeling as found in the model with the proximal gap. As can be seen in this figure, the simulation was continued until 53.5 months postoperatively. The internal remodeling process occurs much faster than the external (surface) process because there is more free surface available in the trabecular bone. In fact, it is predicted that the cortical resorption process continues until far beyond the two-year period of the animal experiment. It is not impossible that in time the whole proximal cortex would disappear.

![Remodeling signal](image-url)
Density distribution

Fig. 9. The density distribution and cortical morphology as predicted after a period of two years by the computer simulation for the model with a proximal interface gap (left) and for the model without this gap (middle). The density distribution of the contralateral control femur, which is also taken as the initial distribution for the treated femur, is shown on the right side.

Animal experiments versus computer simulation

Comparing Figs 6 and 9 shows quite a reasonable similarity between the two-year postoperative animal experimental results and the results of the simulation, where it concerns both trabecular densification patterns and cortical resorption. The most important trends shown in the animal results are reproduced in the simulation, particularly with the model in which the proximal gap is accounted for: the proximal anteromedial cortex reduction, the lateral and medial cortex reduction in the mid-stem region, the proximal trabecular densification adjacent to the stem and resorption on the periphery, and the trabecular densification patterns in the mid-stem and distal region, predominantly associated with the edges of the stem.

Figures 12 and 13 summarize the mean densities and morphological results of the animal experiments and the simulations. Figure 12 shows the mean area fractions (related to the densities) for the six experimental dogs per section (two-year results), in the sections C, D, F, H and J. Also shown are the mean area fractions of the contralateral control femur, which are equal to the initial mean area fractions for the simulations. Furthermore, the simulation results are shown for a six-month and a two-year follow-up period, also as means per section, for the perfectly fitting model (left part of Fig. 12) and the model with the proximal interface gap (right part of Fig. 12). It should be noted that whereas the animal experimental results are shown as averages for six dogs, the simulation results concerned the geometry of one particular dog. The two-year simulation
Remodeling signal

**Fig. 10.** Distribution of the remodeling signal $S$ in cross section C in the model with a proximal interface gap, just after distal jamming of the stem has occurred (left). The distribution in the contralateral control femur is drawn on the right side (compare Fig. 8).

**Fig. 11.** Convergence of the object functions as defined in equation (11) after the distal jamming of the stem has occurred. For the internal and the external remodeling processes, the values of these functions before distal jamming are too far out of range to draw in this graph. [Note that according to equation (9) the time step is variable; hence, the initial gradient is simulated with many small time steps].

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results fit well within the 95% confidence intervals of the experimental results, particularly for the model with the proximal interface gap. On the proximal side (sections C and D) this model predicts hardly any increase of the mean trabecular density, as was also found in the experiments. As discussed above, density changes do occur here, as densification adjacent to the implant and resorption near the endosteal periphery, but the average values in the sections remain almost the same. In sections F and H severe densification occurs in simulation as well as in experiments to about the same values, from about 8-30% and from about 2-45%, respectively. The results of the simulation model with the perfectly fitting implant (left part of Fig. 12) do not nearly agree as well with the animal experimental results as those of the interface-gap model. In all regions but section H, near the tip of the stem, we find much more densification. This trend is reversed in section H.

Figure 13 shows the mean changes in cortical bone area per section for the two-year animal experiment compared to the simulation results for six months and two years, in both models. In this case, the results of the model with a perfect fitting implant and the one with the proximal interface gap are not very different. In simulations and experiments we see decreasing amounts of cortical resorption from proximal to distal. The agreement of the model predictions with the animal values is adequate, between the 95% confidence interval levels, but less close to the mean values than in Fig. 12, relative to the trabecular densities. The agreement with the six-month animal results is also within the 95% confidence interval levels.

**DISCUSSION**

A three-dimensional FEM computer simulation model was employed in conjunction with animal experiments, in order to find an explanation for the bone-remodeling behavior around smooth, press-fitted femoral hip prostheses. The simulation model, including the quantitative formulation of the time-dependent remodeling process, was validated earlier relative to bonded prostheses in the same animal model (Weinans et al., 1992). The FE model used is quite advanced, featuring, apart from its three-dimensional characteristics, nonlinear interface conditions to describe the unbonded interface behavior and the interface fibrous tissue gap. In the computer simulation process, this implies that an interface setting iteration had to be performed within the remodeling iterations. Hence, a large amount of computer capacity was required. Nevertheless, the computer simulation model remains what it is, a model and, hence, a simplified representation of reality. First of all, its limitations must be briefly discussed.

In view of the computer capacity required, the FE mesh was less refined than one would ideally wish for. However, it was adequate. The eight-node brick elements used adequately describe the cross-sectional contours of the bone. The elements can also degenerate into a tetrahedron without causing problems (MARC Analysis Corporations, Palo Alto, CA). To improve precision, the strain-energy density and the apparent density values were determined per integration point rather than per element. Mesh refinement was tested in one calculation with 20-node brick elements, assuming a bonded stem-bone interface. No important differences were found in the strain-energy density distribution in comparison with the eight-node element.

The loads were taken according to in vivo experiments performed by Bergmann et al. (1984). In reality, of course, the loads are highly variable. However, when using the site-specific remodeling theory, whereby the actual bone deformations in the treated femur are always related to those in the natural bone under the same loading conditions, the precise loading characteristics are of lesser importance, as long as they represent a typical load in which axial, bending and torsional components are included (Huiskes et al., 1987).

No friction was assumed at the implant-bone interface. In the area where the soft tissue layer is present, this assumption
is certainly legitimate (Hori and Lewis, 1982). For areas in which intimate bone–implant contact is present, friction will occur. However, in view of the biological environment and the smoothness of the stem, it will probably be very low. The proximal gap in the animal experiments was found to be occupied by fibrous tissue. This membrane is known to have very low initial stiffness in compression, negligible tensile stiffness, and negligible stiffness in shear (Hori and Lewis, 1982). If this layer is modeled as in the first FE model, with no proximal gap, the initial stiffness in compression is too high. Weinans et al. (1991) have shown that the stress distribution in this situation may differ significantly from the distribution found when more realistic conditions are assumed at the interface. In the second FE model, a better representation of the interface material properties is obtained by taking the gap into account.

The trigger between the external and internal processes in the simulation model is determined by the relationship between apparent density and free surface area in the trabecular bone (Fig. 4), which is based on an analytical model and not necessarily equal in the experiment. In addition, no porosity was assumed to develop in cortical bone. As a consequence of these two aspects, external modeling developed more slowly than internal remodeling. It may be that the relative velocities of the two processes, as predicted in the model, are not entirely realistic.

The ‘dead zone’ threshold levels, set at ±35% of the reference signal to obtain the best conformity with the experimental results, have important influences on the amount of bone eventually resorbed or added. Maloney et al. (1989) found in autopsy-retrieved human femoral specimens with cemented hip prosthesis 50% reduced cortical strain values up to 17 years postoperatively, suggesting a threshold value of ±50% in this case. Frost (1990) assumes that these threshold values may have considerable interindividual variations, and even that they may vary throughout life in the same person.

The FE model was reconstructed from contact radiographs of a typical dog, out of the experimental group. Ideally, one would wish to have separate models for each experimental animal for a quantitative comparison. It should be emphasized that the predicted results for one particular dog were compared with the average results of animal experiments. Furthermore, the medullary bone area fraction and the changes in cortical area are the average values of a cross section. Hence, serious cortical bone loss on one side of a cross section and simultaneous bone apposition on the other side can result in a zero change in cortical bone area for this section.

Notwithstanding all these simplifications and limitations of the models, the agreement between the simulation predictions and the experimental findings after six months and two

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**Fig. 12. Medullary bone area fractions predicted by the computer simulation at six months and at two years, and the two-year animal experiment results (mean and 95% confidence interval). The initial value is the same for both the animal experiments and the simulation, and is drawn at each cross section at the left.**

**Fig. 13. Change in cortical bone area predicted by the computer simulation at six months and at two years, and the two-year animal experiment results (mean and 95% confidence interval).**
Fig. 14. Axial relative stem displacement, strain-energy density, and bone apposition around a bonded stem. The postoperative bone apposition, predicted initially, turns into bone loss after the axial stem displacement is reduced due to distal jamming of the stem.

years postoperatively is quite satisfactory. Hence, the first conclusion can be that this study provided a validation of the adaptive-remodeling simulation model, in addition to the validation study performed earlier relative to bonded (in-grown) prostheses (Weinans et al., 1992). Using the present simulation results as a descriptive analytical basis for the animal experimental findings, the questions put forward in the introduction can be answered. Indeed, it was confirmed that the smooth press-fitted stems produce high proximal bone stresses due to its wedging effects in the bone, as was earlier reported as a result from analytical studies (Huiskes et al., 1989, 1990; Rohllmann et al., 1988) and laboratory experiments (Fulghum et al., 1991; Walker et al., 1987). However, this is only true in the immediate postoperative configuration. With time, a process evolves whereby the stress patterns gradually change, due to bone adaptations. This process is illustrated in Fig. 14, which is a conceptual interpretation of the simulation findings. Initially, the bone stresses (hence, also the remodeling signal) are high compared to normal. This is mainly a result of the wedging effect of the stem, pressed into the bone. Hence, the axial relative displacement of the stem (the elastic subsidence) is also high (Fig. 14). As a result, trabecular bone densification takes place. This stiffens the bone bed, particularly at the distal tip, so the axial relative displacements reduce. Hence, also the bone stresses in the proximal bone reduce. This process continues until the stem is jammed distally in the intramedullary canal, and the proximal bone is bypassed, and, hence, there is no adaptive-remodeling process. This process occurs in the model with a perfectly fitting stem and when a proximal interface gap is assumed, but is much more prominent in the latter case. Proximal interface gaps were found in all experimental animals, and the simulation results better agree with the experimental ones if this interface gap was accounted for. Thus, the present experimental results with smooth stems underscore the importance of time as a variable in situations where the histology, characteristics of the interface changes. With identical implants fixed by bone ingrowth, the amount of cortical remodeling had reached a steady state within six months (Sumner et al., 1992a; Turner et al., 1986), but in the present study it is not known if a steady state was reached within the two-year course of the experiment. Given the evidence of a thickening proximal membrane, it is entirely possible that a continued loss of cortical bone could occur.

In short, the finding that proximal bone resorption is similar in bonded and un-bonded, press-fitted implants alike in the long term can be explained consistently with adaptive bone remodeling theory and Wolf's law. Whereas the bonded stem generates monotonous resorption until a new homeostatic equilibrium is found, the resorptive process around the unbonded, smooth stems is nonmonotonic. This is caused by the axial displacement characteristics of the stem in relation to the adapting stiffness characteristics of the bone, and enhanced by the formation of a proximal interface gap. The present results also suggest that proximal cortical resorption may continue far beyond the two-year term until the proximal cortex is completely gone. Although such a phenomenon has indeed been reported in the literature (Miller and Kelebay, 1981; Tronzo, 1989), it has not been confirmed as yet in the present canine model. In extrapolating the present results to press-fitted, un-bonded stems in general, the third question posed in the introduction, one should be careful. It must be appreciated that the mechanism unraveled here and illustrated in Fig. 14 depends on the surface contour of the prosthesis, in relation to the envelope of the endosteal bone, and on the smoothness of the implant surface. Hence, it can be more or less pronounced if the taper angle is smaller or bigger, or when the implant surface is more or less structured to increase friction and reduce elastic subsidence. However, if a prosthesis, whatever its shape or surface, is allowed to gradually subside and find support distally in the relatively stiff diaphysis, a 'stress bypass' will develop, and the same phenomenon as shown here is bound to occur in the longer term.

REFERENCES


