Stress shielding and bone resorption in THA: clinical versus computer-simulation studies
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“Stress shielding” of bone around noncemented prosthetic hip stems causes long-term adaptive bone resorption which threatens the integrity of the fixation. Recently, computer-simulation models based on adaptive bone-remodeling theory in combination with finite-element methods have been developed. These models can be used to predict the extent of long-term resorption. In this paper a comparison is presented between the results of these predictions and those of precise measurements in retrieval hip-replacement specimens reported by Engh and associates (1992).

It is shown that certainly the predictions and the actual clinical findings are subject to the same trends. Under certain assumptions, these results even match precisely. It is also shown that both clinical results and simulation predictions can be approximated by a simple formula, in which the amount of bone loss is related to the ratio between stem stiffness and preoperative bone density.

**Keywords**: total hip replacement; finite element analysis; biomechanics; stress shielding; bone resorption.

INTRODUCTION

The long-term behavior of Total Hip Arthroplasty (THA) is governed by adaptive bone remodeling. The stresses and strains that occur within a bone depend on its external loads, its shape and its internal structural organization. This implies that when a part of the bone is replaced by an implant of different mechanical properties, the stresses and strains within the remaining bone change, even if the external loads remain the same. In accordance with Wolff’s Law, a process of *strain-adaptive bone remodeling* then emerges, changing its shape and internal structural organization to adapt to the new mechanical requirements. Although this concept of adaptive remodeling is obviously an important asset of biology, it does not necessarily have positive effects when implants are involved. The reason for this apparent contradiction is simply that the implant does not adapt with its host bone.

A notorious adverse effect of adaptive bone remodeling is resorption around femoral hip stems. After a stem is placed in the intramedullary canal of a femur, two important changes occur in the load-transfer mechanism. First of all, the hip-joint load is no longer transferred downwards through the metaphyseal trabecular structures and the cortex, but now involves the implant-bone interface. Secondly, the load, which was earlier carried by the bone alone, is now shared with the stem. This phenomenon, called ‘load sharing’, causes ‘stress shielding’ of the bone; that is to say, the bone is shielded by the stem from the stress it is normally subjected to. As a result, the bone stresses become subnormal and the bone resorbs to adapt to this new situation. Hence, contrary to common suggestions in the way of speech,
'stress shielding' is not synonymous with bone resorption, but rather its cause.

An example of the stress-shielding mechanism, as it can be determined in finite-element models, is shown in fig. 1 (13). The bone stresses are shown here as they occur in reconstructions with a non-cemented femoral stem, in comparison to a cemented stem, relative to what they would have been in the preoperative case for the same external loads. The stress shielding is evident and clearly reduces from proximal to distal. Below the tip of the stem the stresses are again normal. The amount of stress shielding (the difference between pre- and postoperative) is more severe for non-cemented stems as compared to cemented ones, due to the difference in flexibility of the two reconstructions. Because noncemented stems are bulkier, hence stiffer than cemented ones, they take away a larger share of load from the bone and thus create more stress shielding. It is to be expected that, as a consequence, more adaptive bone resorption will also be seen around noncemented stems, and this is indeed generally found in clinical situations (6, 10).

![Fig. 1.](image)

Although very few actual clinical problems due to adaptive bone resorption have been reported in the literature, its potential adverse effects may be stem, bone or interface failure, due to reduced bone stock in combination with impact loading, or that not enough bone stock is available when a revision is required. What is worrisome in particular is that traditional radiograms are unsuitable to monitor the process accurately (32), because of a lack of precision. Clinical and animal experimental studies have revealed relationships with stem stiffness (1, 6, 25) and extent of coating (6, 25). From its assumed relationship with stress shielding it is likely that the factors which govern stress shielding also determine the extent of resorption. These include stem stiffness and coating extent, but also stem shape, fit, bone quality and patient weight (13, 14).

The concept of strain (or stress) adaptive bone remodeling was first emphasized in the literature of the last century, a development which culminated in what we now know as 'Wolff's Law' (33). This is not a law in the sense of a quantitative, falsifiable statement in line with the tradition of the physical sciences, but rather a series of observations, based in particular on the work of the anatomist Meyer and the engineer Culmann, who discovered a remarkable similarity between the trabecular structure of the proximal femur and the patterns of stress trajectories calculated in a mathematical model of this structure, using the new theory of 'graphic statics', developed by Culmann (21, 22). One of Wolff's hypotheses, concerning adaptive remodeling, had been discussed extensively earlier in the writings of Roux (23). Roux suggested the adaptive remodeling process...
to be governed by a “quantitative self-regulating mechanism”, nothing else “but what nowadays would be described as a biological control process” (22). Although attempts were made to describe this process mathematically in the course of this century (8, 20), it was only in the mid-seventies when a first quantitative form of Wolff’s law emerged (4, 12). Particularly later forms, which combined mathematical remodeling rules with finite element models, have enabled practical applications of ‘strain-adaptive bone-remodeling analysis’ to orthopedic problems (2, 11, 15).

Strain-adaptive bone-remodeling analysis takes the concept of the ‘quantitative self-regulating mechanism’ — or biological control process — of Roux (23) as a basis, according to which bone cells locally appraise loads and mediate bone formation and resorption. A schematic representation of this model is shown in fig. 2. The sensor cells, we assume, are the osteocytes (5), and the actors the osteoclasts and osteoblasts, although these assumptions are not critical for the remodeling theory. The sensors measure a strain-related mechanical signal and compare that with a normal reference value. If the signal is too high, the sensor mediates the actors to form bone; when it is too low, bone is resorbed. This process continues until the mechanical signal is again normalized. The values of the signal (or stimulus) in each location of the bone depend on the external loads and the mechanical properties of the bone; that is to say, on its shape or geometry and on its internal structural organization or architecture. While the remodeling process is enacted, and shape and architecture are changing, the signal values change as well, providing the feed-back control loop for the sensors which govern the process.

This process is simulated in a computer model. Stresses and strains are determined in a finite element model of the bone, representing its shape and architecture (i.e. density patterns), and simulating its external loads. The model is then used iteratively. During each iteration the mechanical signals are determined from the stresses, strains, volumes and masses in each element and compared to natural reference values. Using a mathematical remodeling rule, the local amounts of bone mass per element to be formed or removed are calculated and adjusted in the finite element model by changing the element volumes or densities. This process continues until a new equilibrium between bone mass and load is obtained (homeostasis).

![Fig. 2. — Adaptive bone remodeling can be considered as a local biological control process, governed by a mechanical signal, appraised by sensors (osteocytes), which mediate actors (osteoclasts and osteoblasts) to regulate bone mass. The scheme depicts, in essence, the hypotheses of Roux (23).](image)

We have developed our computer-simulation model particularly to study bone remodeling around joint replacements, for instance to evaluate the relationships between prosthetic characteristics and the extent of resorption (15, 16). Of course, the values of many of the parameters needed in such a model are unknown or uncertain. Of some quantities and relationships, as for instance the remodeling signal and the mathematical remodeling rule, we do not even know the character. This problem is approached in a way typical for modeling in the physical sciences, by trial-and-error. First sensible assumptions are made for quantities, values and relationships. These are then tried in the computer-simulation model relative to remodeling configurations of which the solution is known. The theoretical and real solutions are compared and if they do not match, the model parameters are adjusted accordingly, until we are satisfied that its predictions are valid. In this way we have triggered and verified our model and its parameters relative to the density distribution of the normal femur (16, 29), and three series of canine experiments with different types of hip
prostheses (26, 27, 31). We are confident that the model and its predictions make sense, and we have used the method to predict the effects of human THA stem stiffness (17, 30), bone quality (17), stem fit, coating placement and bonding characteristics (28).

One problem we have met is the difficulty to validate biological parameters we use in our model with experimental human data. Traditional radiograms are virtually useless for longitudinal studies of bone remodeling (32). However, recently dual-energy x-ray absorptiometry became available for precise in vivo measurements of bone density (18, 19, 24). In our recent article on THA bone resorption analysis in humans (17), we have used the information provided by these recent studies as a guideline. This is a shaky basis, admittedly, but reasonable when the simulation model is applied in a relative sense only. A new opportunity arose after Engh and associates (7) reported a study on long-term bone resorption around the stem of five postmortem THA specimens, using precise dual-energy x-ray absorptiometric measurements, and a contralateral control model to estimate initial preoperative bone density (18, 19, 24). In our recent article on THA bone resorption analysis in humans (17), we have used the information provided by these recent studies as a guideline. This is a shaky basis, admittedly, but reasonable when the simulation model is applied in a relative sense only. A new opportunity arose after Engh and associates (7) reported a study on long-term bone resorption around the stem of five postmortem THA specimens, using precise dual-energy x-ray absorptiometric measurements, and a contralateral control model to estimate initial preoperative bone density. The purpose of the present study was to test the validity of our earlier theoretical predictions relative to the clinical results reported by Engh et al. (17). The idea for this study actually emerged because Engh and associates found a strong inverse relationship between preoperative bone density and long-term resorption, a relationship also predicted by our simulation model (17).

### METHODS

**Simulation study**

The details of our earlier study can be found in Huiskes et al. (17). We use the elastic energy stored per unit of mass in the bone by the external loads as the remodeling signal, calculated as the product of the stress and strain tensors determined in the finite-element procedure, divided by the actual density. This quantity gives an excellent representation of local bone loading, to the extent that normal bone density patterns can be predicted when used in bone growth and maintenance analyses (16, 29). The mathematical remodeling rule, in which the signal values are compared to their normal references, is a linear one, in the sense that a threshold level, or 'dead zone', for bone reactions to abnormal loads is adopted (15), as illustrated in fig. 3. This implies that, locally, bone must be under or over-loaded by at least a certain percentage, before it reacts. This percentage depends on bone reactivity, and was established at average values of 35 for dogs (24, 31), by a comparison of theoretical and animal-experimental results, and 75 for humans (17), by a rough general comparison with the measurements of Steinberg et al. (24) and Kiratli et al. (18). This ‘dead zone’ represents in fact the ‘Mean Effective Strain’ (MES) concept of Frost (9).

![Fig. 3. The relationship assumed in the computer-simulation models between mechanical stimulus S (elastic energy per unit of mass) and the bone-remodeling rate. S_ref is the normal (natural) stimulus value in a particular region. As suggested by Frost (9) it is assumed that bone only reacts to abnormal mechanical signals when these pass a certain threshold level, here denoted by ± s, which thus represents the ‘reactivity’ of the bone. The region of 2s around the natural stimulus value is called the ‘dead zone’. Stimulus values in that zone will not prompt a reaction.](image-url)

For the natural reference signal values we use the distribution of elastic energy per unit of mass as it occurs in a normal bone, subjected to a typical daily loading cycle (2). The procedure for simulating bone remodeling around hip stems is then illustrated in fig. 4: finite-element models are made of the intact femur and the same femur with a prosthesis, which are subjected to the same external — hip and muscle — loading cycles. The model with the implant is subjected to the remodeling simulation procedure, whereby the element signal values after each time step are compared to those in the intact model, and the element-density values are adjusted accordingly for the next time step. This process continues until the signal values in the replacement model are again equal to those in the intact
one, minus the threshold level. Some elements will not
reach that stage, because they have either resorbed
completely in the process, or reached the maximal
density value of cortical bone. An example of such an
end-stage density configuration is shown in fig. 5 (17).
The resorption patterns, particularly at the proximal
side around this fully bonded, titanium prosthesis, are
evident.

The model was applied to study the effects of prosth­
etic stiffness, bone stiffness and bone reactivity, the
latter as represented by the extent of the 'dead zone'
(s, see fig. 3). For that purpose, parameters were varied
relative to a reference configuration of a (fully bonded)
titanium stem (elastic modulus 110,000 MPa), a 'dead
zone' threshold level $s = 0.75$ (75% of the natural,
preoperative value), and a standard bone, of which
shape and density distribution were based on CT­
measurements of a typical femur specimen. Variations
of parameters implied reducing the stem modulus to
20,000 MPa, to simulate a stem material with a stiffness
similar to cortical bone ('iso-elastic'), reducing the
threshold level to $s = 0.375$ (37.5% of the preoperative
value), to simulate a bone twice as biologically reactive,
and increasing bone density at large by a factor of two,
to simulate a stiffer bone. All variations were applied
in separate simulations, hence, there were four simu­
lations altogether, including the reference configura­
tion (17).

Fig. 4. — In the analyses presented here, two finite-element models are applied. One, of the intact femur, provides for the
reference values of the mechanical signal. In the other, with the prosthesis, bone density is gradually adapted to equali­
the actual signal values to the reference ones. When this is accomplished, a new equilibrium has been established (17).

Retrieval study

The details of the study can be found in Engh et al. (7). Five bilateral pairs of retrieved specimens were
studied, of which each nontreated contralateral served
as the control for the treated one, to estimate preop­
erative bone density. It is, of course, not entirely certain
that the density distribution of the nonoperated femur
represents that of the one to be operated upon so many
years ago, but the authors provided enough arguments
and test results to make this a reasonable assumption.
The subject ages at the time of the operation ranged
from 61 to 87 years. An AML hip replacement had
been performed. The AML has a porous coated stem,
made out of CoCrMo alloy (elastic modulus about
215,000 MPa). the prostheses had been in situ for 17,
84, 77, 72 and 76 months in specimens 1 through 5,
respectively, and the stem diameters were 12.0, 13.5,
13.5, 15.0, and 13.5 mm, respectively. Subject weights
varied between 53.5 and 86 kg. Dual-energy x-ray
absorptiometric (DEXA) scans were made along the
lengths of the treated and control femurs, anteropos­
eriorly and mediolaterally, on both sides of the stem.
The results were reported as grams of mineral content
per section, level, or whole bone, representing sums
of values measured by the DEXA system. Comparing
left to right, estimated percentages of bone loss were
also reported (7).
Two examples of results are shown here. One represents the correlation between overall bone loss in the five specimens and the overall 'preoperative' mineral content (fig. 6). Apparently, two of the specimens (2 and 3) had initial mineral contents about twice those of two others (4 and 5). Linear-regression analysis provided a strong correlation coefficient of $r^2 = 0.94$ (fig. 6). The second example represents the percentages bone loss along the length of the stem (fig. 7). Engh et al. (7) presented these for each specimen separately, but in fig. 7 the specimens 2 and 3, and 4 and 5, representing the ones with the highest overall mineral contents and the lowest, respectively, were averaged. Apart from the differences in bone losses in these two categories (correlated with preoperative density, fig. 6), Engh et al. (7) noted the differences in gradients of bone loss along the stem, clearly visible in fig. 7.

A first comparison

In comparison, the results of the simulation studies (17) are very similar (fig. 8) to those of the retrieval study (fig. 7). As fig. 7, fig. 8 also shows the amount of long-term bone loss along the stem, but this time as resulted from the simulation study. The different percentages in bone loss for the dense bones, as compared to the less dense bones (fig. 7), is also found in the simulation study, as curve 4 versus curve 1 in fig. 8. In both cases, more bone resorbed in the retrieved specimens than had been predicted in the simulations. Of course, these differences can be caused by several factors. The elastic modulus of the AML stem is almost twice that of the titanium stem in the simulation model, not to speak of differences in stem and bone geometries, or bone reactivity. As fig. 8

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**Fig. 5.** — Immediate postoperative density distribution, as based on CT-scan, on the left, and density distribution after long-term remodeling simulation on the right, corresponding with the reference configuration of titanium stem and 'normal' bone. The shades represent density values in g/cm³ (17).
indicates, higher bone reactivity (a threshold level of 37.5 versus 75%) produces much more bone resorption in the same prosthetic configuration. However, these different factors notwithstanding, the predictions are quite similar to the actual findings in the retrieved specimens. The different gradients in the curves of bone loss along the stem, noted by Engh et al. (7), are also found in the simulation results (fig. 8). When more bone is resorbed, the curves gradually shift from a concave to a convex course.

![Bone loss (percent)](image1)

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**Fig. 6.** — Illustration of results obtained by Engh and co-workers (7) from precise radiodensity measurements in five postmortem specimens (1 through 5). An inverse correlation was found between postoperative bone loss around noncemented femoral stems and preoperative bone density, estimated by measurements of the contralateral bone.

![Bone mass increase (percent)](image2)

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**Fig. 7.** — Percentages of bone mass increases and decreases along the stems as reported by Engh et al. (7), shown here averaged for the specimens 2 and 3 (which were relatively dense preoperatively). Compare fig. 6 and the specimens 4 and 5 (which were less dense preoperatively).

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**Fig. 8.** — Percentages of bone mass increases and decreases along the stems as found in the computer simulations of the four cases studied (17).

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**Interpolation and extrapolation**

In a further comparison between clinical results and the predictions of the simulation studies, we will focus on the data presented in fig. 6, in relation to the four cases simulated: the reference configuration, the 'isodense' stem configuration, the bone with twice the initial bone density of the reference configuration, and the bone with twice the reactivity. In comparing these results, a problem emerges, because no two cases are the same. The AML prosthesis has a different shape than the one in the simulation studies, and is made out of the stiffer CoCrMo material as compared to titanium. The five retrieved specimens feature three different stem diameters, different bone dimensions and different preoperative bone stiffnesses (fig. 6). All these parameters, we know, have an effect on the extent of stress shielding, hence also on adaptive bone loss. The four simulation studies represent different configurations as well, either different in stem modulus, bone stiffness or assumed bone reactivity, and none of them correlates precisely with any of the retrieved specimens. Hence, in order to allow for comparisons, extrapolation of results is required. For that purpose an analytically-based extrapolation formula is proposed here.

Engh et al. (7) performed linear-regression analysis on their correlation between bone loss and preoperative mineral content (fig. 6). However, if one expects bone loss to be related to stress shielding — we do, and so do Engh and associates — a linear relationship is not likely. In a first approximation, the amount of stress shielding is a nonlinear function of the ratio in
stress and bone (flexural and compressive) rigidities which, in their turn, are directly proportional to their elastic moduli (14). The nonlinear relationship between stress shielding and stem modulus was documented by Huiskes et al. (17). The relationship between stress shielding and bone mineral content (or elastic modulus) has not been studied explicitly, but will probably not be linear either. Hence, instead of the linear function used by Engh et al. (17), we propose a nonlinear extrapolation function, which also accounts for other parameters, of the form

\[ m_r = \frac{\mu}{1 + \mu} \]

where \( m_r \) is the overall fraction of bone loss, and \( \mu \) is a function of the ratio between stem and bone rigidities, and the "dead zone" threshold level \( s \), of the form

\[ \mu = c (1 - s) \frac{E_s}{\rho_b} \]

where \( c \) is an unknown constant, \( E_s \) (MPa) is the stem modulus, and \( \rho_b \) (g/cm³) is the average, preoperative apparent density of the bone which surrounds the stem after it is inserted. Its cubic power is directly proportional to the average elastic modulus of the bone (3). Any dimensional discrepancy in the bone or stem rigidities is accounted for by the empirical constant \( c \).

The average density is measured as the bone mass \( M \) (g) in a volume \( V \) (cm³). In the evaluation of the stimulation results this volume is taken as the total of the trochanter region and the regions 1, 2 and 3 of the finite element model (17), which is almost all the bone around the stem. In the retrieval analysis, the bone mineral content was measured in a sample volume \( V_0 \) (7). The magnitude of this volume was not reported; hence, the bone masses in the retrieved specimens cannot be directly compared to those in the simulation study.

Equations (1) and (2) are intuitive empirical expressions, based only on the expectation that the overall fraction of long-term bone loss is related to the extent of direct postoperative stress shielding which, in its turn, is a nonlinear function of the rigidity of the stem relative to that of the bone. All the parameters of the different cases can be substituted in the extrapolation formula — \( s, E_s, \rho_b \) — but the constant \( c \), the empirical extrapolation parameter, depends on shape and dimensions of stem and bone in an unknown way. It will be calculated by applying the formula to one single case, that of the reference configuration in our simulation studies. We will then keep that same value for \( c \) when applying it to the other cases, to interpolate the results of the retrieval studies and extrapolate the theoretical predictions. We also assume that the bone reactivity of the retrieval cases can be represented by \( s = 0.75 \), as in the simulation study of the reference case. In short, all differences between the reference case of the simulation study and the specimens of the retrieval study are neglected, except those concerning the stem moduli and the average, preoperative bone densities.

**RESULTS**

For the reference configuration in our simulation studies (17) the overall bone loss determined was 23%; hence we have \( m_r = 0.23 \). Titanium was the stem material; hence we have \( E_s = 110,000 \) (MPa), and a "dead zone" threshold level of 75% percent was applied, giving \( s = 0.75 \). The initial bone mass in the volume \( V \) concerned for this case was \( M = 125 \) grams. With this information the interpolation constant can be calculated at \( c = 21.3/V^3 \) from formulas (1) and (2), and using \( \rho_a = M/V \).

We now extrapolate overall bone loss for our reference configuration, using formulas (1) and (2), and the above value for \( cv^3 \), to bone losses as functions of initial (preoperative) bone mass (fig. 9a), stem elastic modulus (fig. 9b) and the bone-reactivity threshold level (fig. 9c). Surprisingly, we find that this extrapolation, using this relatively simple formula, predicts the simulation results of the other case quite nicely. This is surprising particularly because the bone-remodeling simulation model describes a rather complex, nonlinear process.

We then use the formulas (1) and (2) to reinterpolate the results found in the retrieved specimens by Engh et al. (7). For that purpose the interpolation parameter \( c \) was recalculated to provide for the best fit at \( c = 0.19/(V_0)^3 \), where \( V_0 \) is the (unknown) sample volume in which mineral content was measured. The extrapolation can then be expressed as a function of \( M_0 \) and the sample value of the total preoperative mineral mass, as in fig. 6. The result is shown in fig. 10. Again, the interpolation produces excellent results, confirming the assumed parametric relationships represented by formulas (1) and (2).
Finally, we extrapolate the simulation results of the reference configuration to encompass the retrieval measurements. For that purpose, the extrapolation parameter is again taken as \( c = 21.3/V_s \), as in fig. 9. To include the measured results, it is assumed that \( V_s/V = 0.2 \) in order to obtain the best fit. The results are shown in fig. 11. The two curves represent Formula (1) for the elastic modulus values of titanium and CoCrMo stem materials, using the above value of \( c \) (derived from the reference simulation case), and expressed as functions of the total preoperative bone masses \( M \). So in fact the 'titanium' curve in fig. 11 extrapolates the results of the reference simulation study (with one particular preoperative bone-density value) to the whole range of preoperative bone-density values, and the 'CoCrMo' curve in fig. 11 extrapolates these results to what they would have been in the case that the stem material in the simulation study would have been CoCrMo, instead of titanium. The curves are compared to the findings of the computer simulations (as in fig. 9a) and those of the retrieval studies, for which purpose the preoperative bone masses were re-evaluated by using the empirical relationship shown above, between the sample volume in the bone measurements \( V_s \) and the total bone volume \( M \) in the simulation studies. Again, the similarity is excellent, suggesting that the differences between clinical and experimental results could, in principle, be fully explained by the difference in stem stiffnesses.
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Fig. 10. — The results of the retrieval study (fig. 6) interpolated with the extrapolation formula, after a value for the extrapolation parameter c was calculated which provided the best fit.

Fig. 11. — Quantitative comparison between the simulation predictions and the retrieval specimen measurements. For that purpose the extrapolation formula is evaluated as a function of the preoperative bone mass, for both a titanium (simulation) and a CoCrMo (retrievals) stem. The value of the extrapolation parameter c was based on the reference simulation case (hence fits the curve exactly). The results of the retrieval specimens are added by assuming that the DEXA measurements represented 20% of total bone mass, which provides for the best fit.

DISCUSSION

The simulation model we developed is nothing more than a simple mathematical description of the concept of bone as a "quantitative self-regulating mechanism", suggested by Roux (23) in 1881, combined with finite-element analysis to make it applicable to the morphological complexities of bone structures. Although the finite-element models are still crude relative to these structures, and the remodeling model features a number of assumptions, the results it produces proved to be quite realistic in simulations of animal experiments. The morphological adaptations around canine prostheses in a number of experimental series could be predicted in detail (26, 27, 31). The purpose of the present paper was to evaluate the validity of predictions made for human cases (17).

Ideally, such a validation requires an actual simulation study of the specimens investigated by Engh et al. (7), with models representing the bone and prosthetic characteristics of those specimens in the immediate postoperative situation. This information was not available at the time. As a consequence, an extrapolation formula had to be developed to permit comparisons between predictions and measurements. In addition, quite a few assumptions had to be made and quite some mathematical manipulation was performed. In summary, actual and potential differences in shapes and dimensions of stem and bone were neglected, and the relationship between the preoperative densities of the retrieval bones and the simulation models was assumed. Hence, the conclusions must be weighed very carefully.

First of all, the extrapolation (or interpolation) formula proposed performed extremely well, for both the simulation results and the retrieved specimens. By evaluating the extrapolation parameter c for one single case from the simulation study, the results of the other three cases could be approximated quite nicely. It is in fact surprising that the results of such a complex control process can be predicted by a relatively simple formula, based on the overall stiffness ratios between stem and bone. When applied to the retrieval series, a c-value could be found such that an excellent interpolation of the experimental results emerged in the correlation between preoperative mineral content and long-term postoperative bone loss. This at least suggests that the clinical specimens had been subject to similar parametric relation-
ships as assumed in the computer-simulation model.

It is tempting to apply the extrapolation formula found here to predict long-term bone resorption for a given prosthetic design, without relying on computer-simulation models. Although it seems that the formula can give a reasonable indication, a word of caution is required. The extrapolation parameter c lumps a number of factors in an unknown way. As long as prosthetic shape and bonding conditions are similar to the ones studied here, the value of c will probably not be very different. However, particularly when variable bonding conditions play a role, its value may change significantly, as indicated by studies of bone resorption around press-fitted stems in dogs (26).

About the precision of the predictions relative to the retrieval findings, only preliminary conclusions can be drawn, because in the comparison the dimensional differences in bone and stem dimensions had to be neglected, and the assumption had to be made that the DEXA values represented bone mass in 20% of the bone volume. If these assumptions are justified, the similarity between predictions and retrieval findings is indeed extremely precise (fig. 11).

The third conclusion however is that in a qualitative sense, not regarding the precise mathematical evaluation, the results of the retrieval studies are quite similar to those of the computer simulations. The orders of magnitude of overall bone loss and the patterns of bone loss along the stem compare very well, as do the trends of the effects of preoperative bone mass. So even if the predictions of the simulation model are not very precise, they are certainly not unrealistic.

So the retrieval study and the simulations both show the same trends, and may even be very close quantitatively. These results indicate that bone resorption around noncemented stems is a grave long-term problem indeed, producing much more bone loss than often assumed based on conventional radiograms. The analytical results presented proved that this phenomenon is directly proportional to the extent of stress shielding, and is subject to direct (nonlinear) relationships with the relative stiffness of stem and bone. The results also show that the extent of bone resorption can be predicted in computer-simulation studies, and approximated by a simple, analytical formula.

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REFERENCES


* Translated as ‘The law of bone remodeling’.