Bone remodeling around implants can be explained as an effect of mechanical adaptation

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Bone Remodeling Around Implants Can Be Explained as an Effect of Mechanical Adaptation

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There is general concern in orthopedics about the long-term effects of adaptive periprosthetic bone remodeling. Long-term resorption may weaken the bone bed and promote bone, implant, or fixation failure. Although functional adaptation of bone may have been the decisive asset in the evolutionary survival of vertebrates (1), it does seem to work against individuals with joint replacements. Particularly hip stems tend to stress shield the femur. They take a share of the load that was formerly carried by the bone alone; hence the bone is subnormally stressed (Fig. 1). According to Wolff's law (2) the adaptive regulation mechanism in the host bone is then led to reduce its mass, unaware of the requirements of the implant, the safety hazards of its interfaces, or conditions required for revision surgery.

Although the association between bone mass and load is generally accepted, Wolff did not give us more than the notion of a relationship and a number of observations in deformed bone specimens as illustrations. Recent clinical and experimental studies with total hip arthroplasty (THA) have definitely suggested that a relationship exists. Bone resorption is more extensive around cementless as compared to cemented stems (3). Because cemented stems are more flexible and create less stress shielding (4,5), it is likely that this difference is governed by mechanics. In animal experiments, hollow stems (6) or composite stems (7) are both less stiff than solid metal stems and provoke much less cortical bone loss. Again the relationship with stress shielding seems obvious (8,9). Proximally coated stems reduce the loss of bone relative to full-coated stems (10). A proximal coating promotes proximal load transfer and hence reduces stress shielding (11); thus, the relationship is again confirmed.

However, controversial results were also reported. A well-known method to increase stresses in the femur and reduce stress shielding is to press a smooth tapered stem in the medullary canal; this method is based on the wedging effect of the stem (4). However, reductions in bone loss around press-fitted stems are not universally seen in the clinic; sometimes dramatic resorption is even reported (12). Sumner et al. (13) found as much bone resorption around their smooth, uncoated stems as around their fully coated stems 2 years postoperative in canine experiments. How can this be explained in terms of stress shielding? Maloney et al. (14) found that

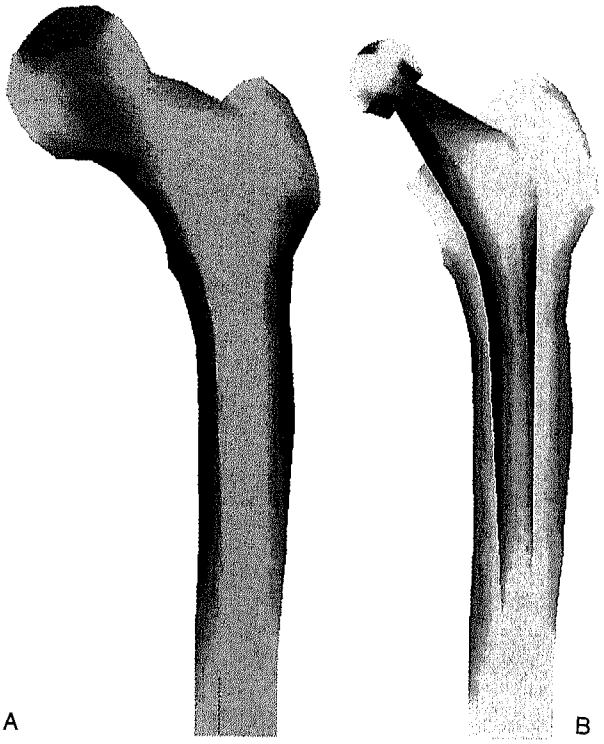

FIG. 1. An example of stress shielding around a femoral stem. Shown are the distributions of the elastic energy (related to stress and strain) **A:** in the intact bone and **B:** after THA for the same external hip joint and muscle loads (midfrontal section through a three-dimensional FE model). Where in the normal bone, most of the load is transferred through the cortices, illustrated by the dark areas, a stem takes a part of the load away.

bone strains did not return to normal after long-term periprosthetic remodeling. They concluded that the concept that stress shielding leads to sufficient bone loss to reestablish a normal strain pattern is not supported by their data, thus casting doubt on the concept of strain-adaptive remodeling around implants. There are other examples in the literature of clinical findings that are believed to contradict Wolff's law. They notoriously appear in cases where innovators present their first clinical results with new prosthetic designs, which claim to show no signs of stress shielding on radiograms. Conversely, clinicians can sometimes be amazed by bone loss around stems for which they believed they had reduced stress shielding, and they suggest that there seems to be no clear relationship between stress and remodeling.

The central thesis of this chapter is that bone remodeling around implants can be explained as an effect of mechanical adaptation. Indeed, it can even be predicted from mechanical loading. I suggest that where clinical results seemingly contradict this thesis, then the load-transfer mechanism was not well understood, Wolff's law was misinterpreted, or measurements were wrong or imprecise.

THE STRAIN-REMODELING RELATIONSHIP

Since Wolff, much has been revealed about strain-adaptive bone remodeling (15–18). Remodeling is a process that is continuously regulated. It is not like the target of a bullet, which is determined by the conditions of the gun when it is fired. The initial stress-shielding pattern of periprosthetic bone (Fig. 1) may be the initial stimulus for remodeling, but, while the remodeling process continues, the stress-

shielding patterns also change due to the changing mechanical relationships in the whole reconstruction. Therefore, we would be naive to assume that eventual resorption patterns could be estimated directly from initial strain patterns. Any paradigm to predict remodeling patterns would have to take this feedback loop into account.

It has become obvious that the relationship between mechanical stimulus and remodeling is a nonlinear one (18–20). Apparently, the remodeling process is triggered only after the strain deviations pass a certain threshold level, what Frost called the *minimum effective strain* (18). An example of a likely paradigm for this nonlinear relationship is illustrated in Fig. 2 (20). It implies that the remodeling process would not continue when the strains are normalized within the threshold region. Hence, the results of Maloney and associates (14), mentioned above, confirmed present concepts of adaptive bone remodeling, rather than creating a controversy.

Wolff's law is a poor vehicle for operational purposes, because it does not specify the relationship between stress and remodeling. However, in recent years, quantitative forms of Wolff's law have been proposed that do (20–22). These *strain-adaptive*

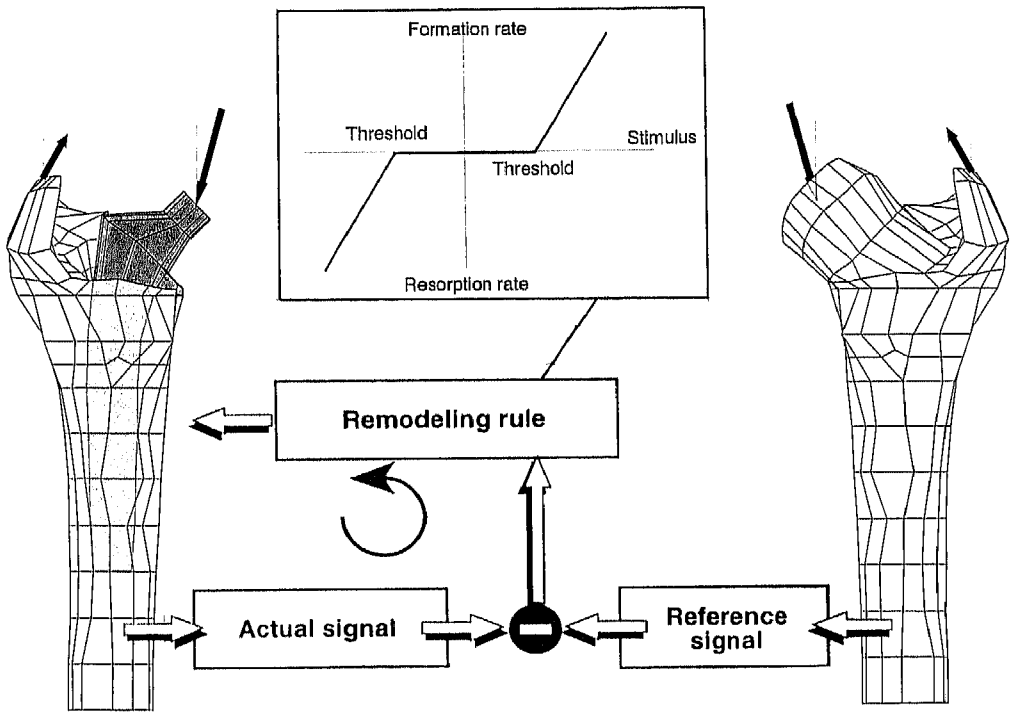

FIG. 2. Illustration of a computer-simulation model for strain-adaptive bone remodeling around implants. The relationship between mechanical stimulus and remodeling—the remodeling rule—is described by a nonlinear function, featuring a threshold dead zone *(inset)*. The stimulus is determined by the difference between the actual strain energy (the signal) in the periprosthetic bone and the reference values in the intact bone. To assess the signal–value distributions, two FE models are applied subject to the same external loading conditions. Bone-mass turnover is effected per elapsed time increment in the THA model. The iterative simulation process continues until a new equilibrium signal distribution is obtained.

bone-remodeling theories assume a nonlinear relationship between a local mechanical stimulus and bone-remodeling rate, as shown in Fig. 2. The stimulus is the difference between an actual mechanical signal value and its reference. For example, suppose that the signal is taken as the elastic energy stored in the bone upon loading (8,9,20). Figure 1 shows the value distribution of this signal in an intact femur and after THA for the same external loads. In this case, the stimulus value distribution can be found by subtracting the intact distribution from the THA distribution. This stimulus distribution then determines the initial remodeling rate, bone formation where the stimulus is positive, and resorption where it is negative, taking account of the dead-zone threshold levels (Fig. 2). Of course, this only provides the initial remodeling rate, because, as bone shape and density adapt, the mechanical signal and hence the stimulus change as well. To follow the process in time, an iterative simulation scheme is required, as depicted in Fig. 2. After each small time lapse, the finite element (FE) model determines the actual stimulus distribution, which determines the changes in shape and density for the following time lapse, which are then effected in the FE model. This process continues until the stimulus, throughout the bone, has reached the dead-zone region. The shape and density distribution in that situation represent the prediction of the eventual adaptive remodeling effect of the prosthesis.

If the thesis implied in the title of this chapter is true, then a computer-simulation model of this kind, as a quantitative form of Wolff's law, should be able to predict bone-remodeling patterns that occur around implants in reality. To investigate that hypothesis, a number of animal experiments and clinical cases are discussed in the following pages.

ANIMAL EXPERIMENTS

Methods

Bone remodeling around hip stems was studied experimentally for many years in Chicago (7,13,23,24). The Chicago researchers developed a canine model for hip replacement in which several kinds of stems were tested. Postoperatively, either 6 months or 2 years, the animals were killed, the bones were sectioned, and the periprosthetic bone mass was compared to that of the contralateral, control side. For that purpose, two variables were quantified: One, the cortical bone area (CBA), and the other, the medullary bone area fraction (MBAF). Both were determined per cross-sectional area, as, for example, shown in Fig. 3. Evidently, in this example, the CBA reduced relative to the control side. Hence cortical resorption had occurred in the course of time. Trabecular bone density, however, had increased in this section, as represented by an increase of the MBAF relative to the control side.

In this model, six prosthetic stems were tested in six series. In all the series, the stems were of the same overall shape. What differed were the material stiffness properties and the coating configurations. Because of the differences in stem coating and in material stiffness, differences in stress patterns as well as in remodeling patterns were expected. This present discussion is limited to the first two series of experiments, those with fully coated stems and those with uncoated stems.

The simulation studies of these experiments used two three-dimensional FE models each, one for the reconstructed and one for the control bone (Fig. 2). Hip and muscle loading were identical in both cases, and were based on the in vivo

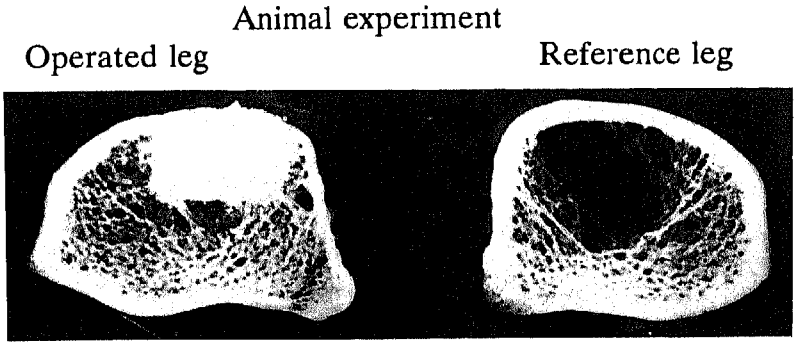

FIG. 3. Examples of cross-sectional morphologies of THA **(left)** and control bone **(right)** in a dog with fully coated stem 2 years postoperative [section C(27)]. Cortical resorption is evident particularly in the medial-anterior region. Trabecular hypertrophy occurs associated with the edges of the stem and, anteriorly, where the trabecular width is small.

telemetric studies of Bergmann et al. (25). The geometry and density distribution of the control-bone model were derived from one experimental dog. For the immediate postoperative configurations, the control model was simply provided with prostheses, of which the coating and material characteristics depended on those in the experimental series to be simulated. It was always assumed that ingrowth occurred where coating was present. That means that in the FE-models the contact between coating and bone was always fully bonded. Where coating was not present, a frictionless (sliding) contact was assumed, which was represented in the model by so-called nonlinear gap elements. In the case of the uncoated stem an interface gap around the proximal part of the stem was assumed, reducing from 1 mm thickness at the resection plane to zero at 40 mm below the plane (26). This gap simulated the soft-tissue membrane seen in the dogs with the uncoated stems (13).

The results of the experimental series with fully coated stems were used to trigger the unknown parameters in the model, such as the width of the dead zone and the time-constant, relating computer time to real time (27). Using the parameter values thus determined, the results of the uncoated series were used to verify the model (26).

Results

The animal results were characterized by trabecular hypertrophy around the stem and by periosteal cortical resorption in all cases. These phenomena were also seen in the simulation of the coated-stem series (Fig. 4). After a simulated 6 months, the proximal MBAF (section B) had already increased by 33% to 54%. Near the distal tip of the stem (section G) the increase was 6% to 35% at this time. Further hypertrophy occurred up to 44% until 2 years postoperatively, particularly in this latter region. Cortical resorption developed slower than trabecular hypertrophy in the simulation. The CBA was reduced by approximately 5% in 6 months, and by approximately 20% in 2 years with a maximum in section F. Comparing the 2-year prediction with the canine measurements (Fig. 4) yields a satisfactory similarity; the predictions are within the 95% confidence range of the experimental results or sufficiently close in

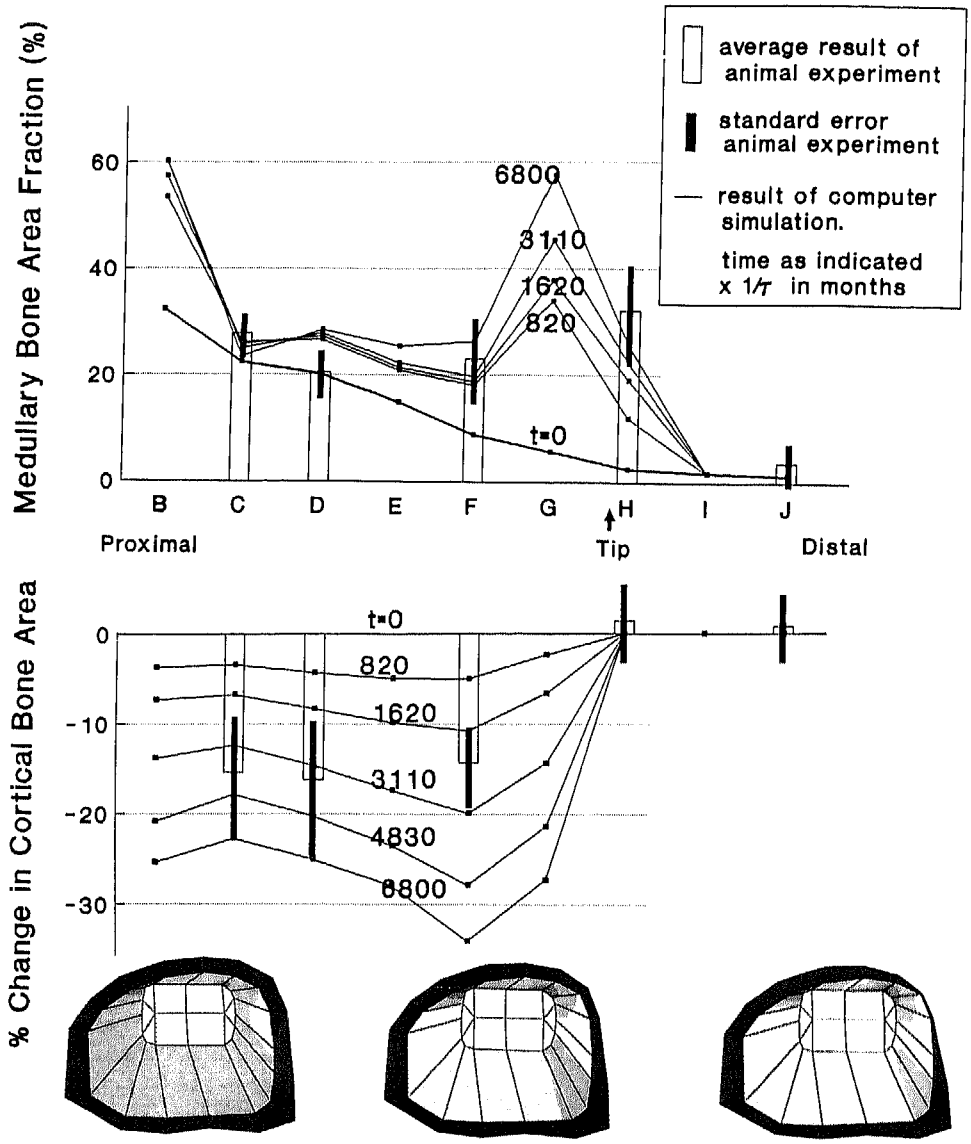

FIG. 4. A comparison between 2-year experimental results in animals and those of the simulation for the fully coated stem series at various time periods (27). The trabecular area fraction (MBAF) is shown in the *top graph* and the decrease in cortical area (CBA) in the *lower graph*. The time, t = 3110 simulation units, was established as representing 2 years; t = 820 is close to 6 months. At 2 years, the simulation results are within the 95% confidence interval of the experimental series. *Below* is shown the development of cortical resorption and trabecular hypertrophy in section C, representing, from left to right, shortly postoperatively, after 6 months, and after 2 years in the simulation model. The patterns seen are very similar to those found experimentally (cf. Fig. 3).

two sections. The simulation model predicted that remodeling would still continue after 2 years (Fig. 4), but no dogs were kept longer.

It is quite interesting to regard the distribution of remodeling patterns in the individual cross sections, which are really very similar in experiment and simulation. In both cases, trabecular hypertrophy was associated predominantly with the edges of the stem and in regions of narrow trabecular width, which act as stress risers. Cortical resorption occurred mostly medial-anteriorly (compare Figs. 3 and 4).

The animal results of the uncoated stems gave rise to controversy. After 6 months, cortical resorption was significantly less in comparison with the fully coated stems (28). This was expected, in view of the bone stress-increasing wedging effect of the unbonded stem. However, after 2 years, the amount of cortical resorption appeared even higher in the second series, which seemed to defy Wolff's law (13). The simulation study provided the explanation (26), which was related to gradual changes in load-transfer, discussed below. Again the 6-month and 2-year predictions of CBA and MBAF were satisfactorily close to the experimental ones, but only after the FE-model was adapted to account for interface membrane formation (Fig. 5). Cortical resorption was similar, but trabecular hypertrophy was higher distally when compared to the fully bonded stem series after 2 years.

The simulation revealed that, initially, cortical stress shielding is reduced around the unbonded stem relative to the bonded one, and proximal trabecular interface compression is increased, just as expected. Remodeling then develops accordingly in the initial stage. However, in the course of time, the stem sinks in the medullary canal, thus increasing distal interface compression and trabecular hypertrophy. As a result, a stress bypass develops, which emphasizes increasingly distal versus proximal load transfer, distal hypertrophy, and proximal atrophy in a self-perpetuating cycle. This phenomenon is amplified by the gradual development of a fibrous mem-

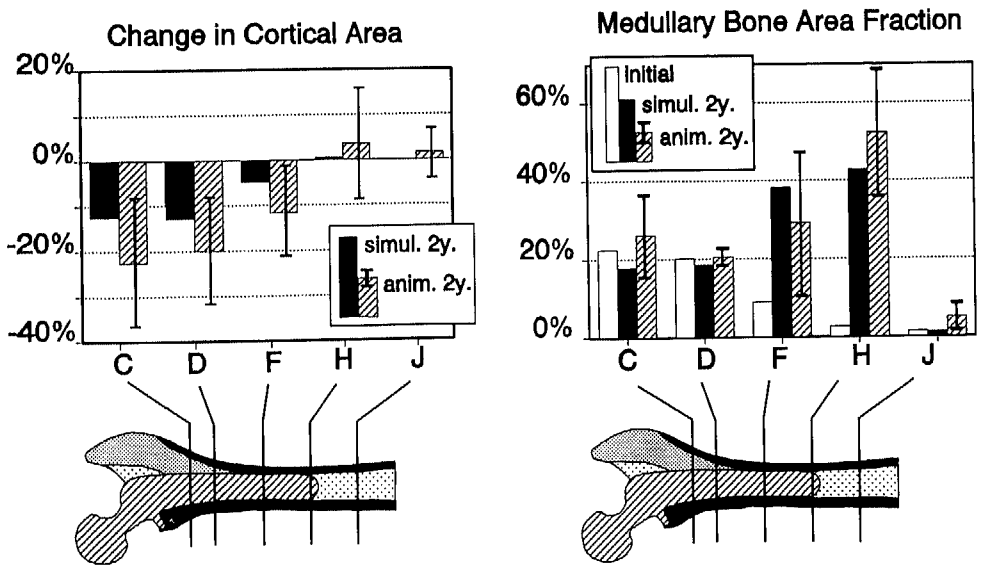

FIG. 5. A comparison between 2-year experimental results in animals and those of the simulation for the uncoated-stem series (26). Again the predicted CBA and MBAF are within the 95% confidence interval of the experimental values.

brane at the proximal interface, probably as an effect of relative motion, which reduces proximal load transfer even more. After this membrane was accounted for in the model, the similarity between 2 years predicted and experimental values improved, indicating its mechanical significance (26).

In summary, the coated stems are grown in and develop a stable position. High interface stresses cause trabecular hypertrophy, particularly proximally and distally, and cortical stress shielding causes periosteal resorption. These processes develop slowly in time. Conversely, the uncoated stems do not reach a stable position, but move and gradually subside, develop a proximal interface gap, and are eventually clamped in the distal diaphysis. The resulting stress bypass causes cortical resorption to approximately the same extent as the initial stress shielding does in the bonded-stem series. Therefore, in retrospect, the remodeling phenomena around the unbonded stems are easily explained in terms of Wolff's law, once the nonlinear, time-dependent load-transfer mechanism is understood.

HUMAN RETRIEVALS

Methods

Validation of strain-adaptive bone-remodeling theory relative to human cases is more complicated. Radiographic measurements of bone density are notoriously imprecise (29) and postmortem evaluation is not common. Recently, Engh and associates (30) published a study of femoral specimens, retrieved postmortem from patients who had received anatomic medullary locking (AML) hip replacements. Five bilateral pairs of specimens were studied, of which each nontreated contralateral served as the control for the treated one, to estimate preoperative bone density. Of course, it is not entirely certain that the density distribution of the nonoperated femur represents that of the one which was to be operated upon so many years earlier, 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 of cobalt–chromium–molybdenum alloy (elastic modulus approximately 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 absorptiometry (DEXA) scans were made along the lengths of the treated and control femurs, anteroposteriorly 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.

An obvious study for the purpose of verification is to use the computer-simulation model to investigate these retrieved specimens, in the same way as for the animal series discussed above, and that is precisely what is now being done. For the moment, however, I want to discuss a preliminary, generalized comparison between the results of the retrieval study and those of simulation studies of human THA (31). The simulation study, reported previously (8), used a similar model to the one discussed above. The model was applied to study the effects of prosthetic stiffness, bone stiffness, and bone reactivity—the latter represented by the extent of the dead zone (Fig. 2).

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 75% of the natural preoperative value, and a standard bone, shape and density distribution of which were based on computed tomography 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 (isoelastic), reducing the threshold level to 37.5% 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 models altogether, including the reference configuration (8).

Results

Engh and associates (30) reported overall bone mineral content in their retrieved specimens—treated and contralateral controls, as well as mineral contents in regions along the length of the stem. Not surprisingly in retrospect, they found vastly different bone mass in the controls, between 25 and 50 g overall mineral content. They also saw that these preoperative values correlated nicely with those of overall postoperative bone loss, in the sense that denser (hence stiffer) bones had lost less bone mass than the less dense ones. That is precisely what one would expect if remodeling is related to the mechanics, and it is also precisely what we predicted from our simulation study in which a variation of bone density was included (8). The stiffer the bone, the less the stress shielding. Engh et al. (30) presented a linear correlation between postoperative bone loss and preoperative mineral content. However, the relationship between stress shielding and bone stiffness is in fact nonlinear (31). I reinterpolated the data with an analytical function that takes this nonlinear stress-shielding relationship into account and found a nice correlation, as shown in Fig. 6 (31). This clearly confirms that mechanics is the driving force for the remodeling process, and it also confirms, in a qualitative sense, our earlier simulation results of human THA (8).

Also, in other aspects, there were similar results in the retrieval and the simulation studies, when regarding bone loss along the length of the stem (31). Engh et al. (30)

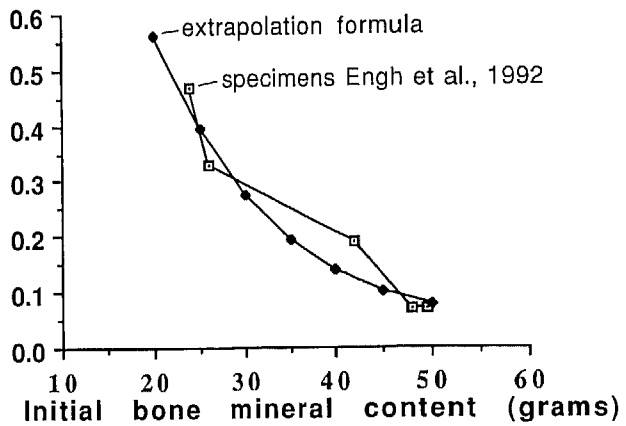

FIG. 6. Interpolation of the data from Engh and associates (30), relating preoperative bone mass to postoperative bone loss in five specimens. The data was reinterpolated with an analytical function that accounts for the nonlinear relationship between bone stiffness and stress shielding (Adapted from [31].)

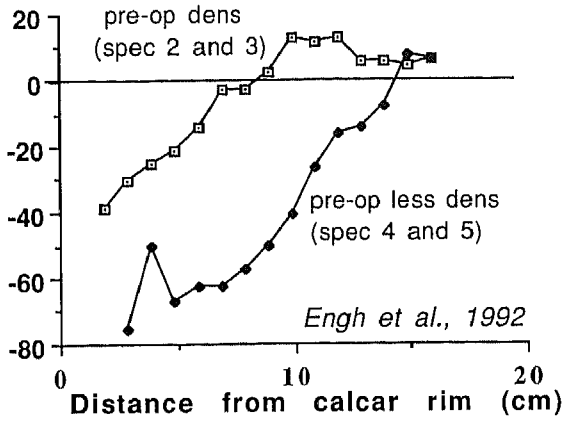

FIG. 7A. Percentages of bone mass increases and decreases along the stems as reported by Engh et al. (30), shown here averaged for the specimens 2 and 3 (which were relatively dense preoperatively, cf. Fig. 6) and the specimens 4 and 5 (which were less dense preoperatively). (Adapted from [31].)

presented these for each specimen separately, but specimens 2 and 3, representing the ones with the highest, and 4 and 5, those with the lowest, overall mineral content were averaged, as shown in Fig. 7A. Apart from the differences in bone losses in these two categories (correlated with preoperative density as in Fig. 6), Engh et al. (30) noted the differences in gradients of bone loss along the stem, clearly visible in Fig. 7A. In comparison, the results of the simulation studies are very similar to those of the retrieval study. Figure 7B shows the amount of long-term bone loss along the stem from the simulation study. The different percentages in bone loss for the dense, as compared to the less dense bones, are also found in the simulation results (curve 4 versus curve 1 in Fig. 7B). In both cases, more bone was resorbed in the retrieved specimens than predicted in the simulations. These differences are caused by several factors, because the simulation concerned a generic THA model, not the bones nor the prostheses from the retrieval study. The elastic modulus of the AML stem is almost twice that of the titanium stem in the simulation model and hence provokes more stress shielding. There are differences in stem and bone geometries as well. However, these different factors notwithstanding, the predictions are quite consistent with the actual findings in the retrieved specimens. The different gradients in the curves of bone loss along the stem (Fig. 7A) are also found in the simulation results

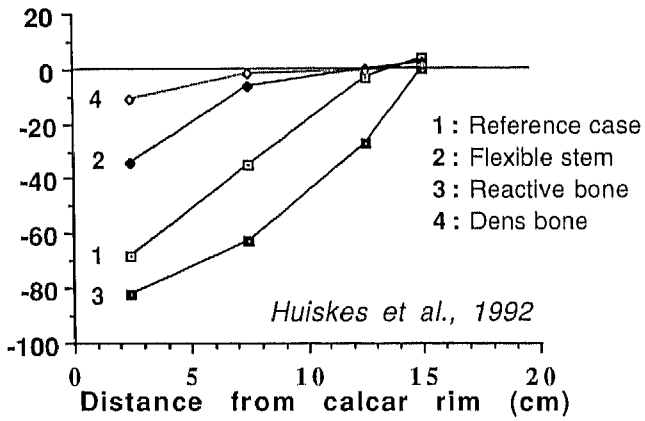

FIG. 7B. Percentages of bone mass increases and decreases along the stems as found in the computer simulations of the four cases studied by Huiskes et al. (8). (Adapted from [31].)

(Fig. 7B). When more bone is resorbed, the curves gradually shift from a concave to a convex course.

In short, all the qualitative effects or trends found in the simulation study were reproduced by the retrieval study. In addition, the orders of magnitude of the amounts of bone lost postoperatively were very similar indeed. Originally, we were worried about the numbers we found (8), because in clinical studies much less long-term bone loss was usually reported than we predicted. Obviously, however, the notorious imprecision of traditional radiographs in this respect (29) has caused the problem to be underestimated. Precise measurements, as for instance using DEXA, do produce values that are quite close to what mechanics would predict.

DISCUSSION

Sumner et al. (7), in a discussion of their canine THA studies, ask themselves whether the relationships implied by Wolff's law, which they call the "stress-shielding paradigm," are useful. Their answer, however, is more concerned with the question of whether the paradigm is true. In fact, both issues, validity and applicability, are equally important. There is no doubt that a relationship between mechanics and bone remodeling exists; the question, rather, is how it affects postoperative bone behavior in THA. It is intuitively obvious that the remodeling process, like any biological process in living tissues, only works normally in healthy bone. Although it is not believed that remodeling is proportional to the amount of vascular supply, normal supply is obviously required. Without vascularity, there is no remodeling. The process is also probably disturbed by drugs, infections, or reactive interface processes, which implies that the mechanical paradigm will only show its true colors in a situation of biological and mechanical homeostasis. It is also obvious that other processes than strain-adaptive remodeling can affect the turnover of bone mass. For instance, interface reactions to wear particles in bone lysis, tissue-repair processes of the traumatized endosteum, or ingrowth of implant coatings. This does not make the mechanical paradigm less valid; it only shows that its applicability is limited to those situations where it is not overshadowed by other mechanisms.

In all investigations where the above conditions were met and where dependable measurements of bone mass were performed, the relationship between mechanics and periprosthetic remodeling was confirmed, as mentioned above. Measures that obviously reduce stress shielding also reduced bone loss. The similarity between actual postoperative bone remodeling patterns and those predicted with a mathematical model representing the mechanical paradigm, as discussed above, makes the validity of the paradigm likely. Where long-term postoperative remodeling patterns contradict expectations based on assumed strain patterns, it is likely that the ideas about the strain patterns are not correct.

The answer to the second question, whether the paradigm is useful, depends on what one wants to use it for. As illustrated in the discussions of the animal experiments and the retrieval studies, the paradigm and the predictive model are very useful to explain phenomena seen clinically and to reveal their underlying mechanisms. A good example is the explanation for the remodeling patterns around the unbonded stems in the canine study, which revealed the importance of prosthetic instability and interface mechanics. Without analytical tools such as the simulation model, explanations may be confused by anecdotal or intuitive arguments.

Whether the mechanical paradigm provides a useful vehicle for the preclinical prediction of bone-remodeling patterns for a given THA configuration is quite another matter. As shown repeatedly in FE analyses (4,11,32,33), and again in the above example of the uncoated canine stems, prosthetic load transfer is very sensitive to interface conditions. These can also be subject to gradual changes, due to ingrowth, migration, or local loosening processes where the predictability is, as yet, limited (34). For the purpose of preclinical testing, this implies that the simulation models must rely on particular assumptions about the interface bonding conditions. However, if we see preclinical testing methods as tools to estimate the sensitivity of new THA designs or materials to particular failure scenarios (35), then this restriction does not influence their applicability. The ultimate proof of safety and efficacy of THA designs cannot be provided by computer-simulation studies. However, they do provide useful preclinical checks, or guidelines for animal studies, at very low cost.

REFERENCES

1. Vogel S. Copying nature: a biologist's cautionary comment. *Biomimetics* 1992;1:63–79.
2. Wolff J. *Das Gesetz der Transformation der Knochen*. Berlin: Kirchwald; 1892 (translated by Maquet P, Furlong R. *The law of bone remodeling*. Berlin: Springer-Verlag; 1986).
3. Harris WH. Will stress shielding limit the longevity of cemented femoral components of total hip replacement? *Clin Orthop* 1992;274:120–123.
4. Huiskes R. The various stress patterns of press-fit, ingrown and cemented femoral stems. *Clin Orthop* 1990;261:27–38.
5. Weinans H, Huiskes R, Grootenboer HJ. Effects of material properties of femoral hip components on bone remodeling and interface stresses. *J Orthop Res* 1992;10:845–853.
6. Bobyn JD, Mortimer ES, Glassman AH, Engh CA, Miller JE, Brooks CE. Producing and avoiding stress shielding. Laboratory and clinical observations of noncemented total hip arthroplasty. *Clin Orthop* 1992;274:79–96.
7. Sumner DR, Turner TM, Urban RM, Galante JO. Experimental studies of bone remodeling in total hip arthroplasty. *Clin Orthop* 1992;276:83–90.
8. Huiskes R, Weinans H, van Rietbergen B. The relationship between stress shielding and bone resorption around total hip stems and the effects of flexible materials. *Clin Orthop* 1992;274:124–134.
9. Huiskes R, Weinans H, Dalstra M. Adaptive bone remodeling and biomechanical design considerations for noncemented total hip arthroplasty. *Orthopedics* 1989;12:1255–1267.
10. Engh CA, Bobyn JD. The influence of stem size and extent of porous coating on femoral bone resorption after primary cementless hip arthroplasty. *Clin Orthop* 1988;231:7–28.
11. Weinans H, Huiskes R, Grootenboer HJ. Effects of fit and bonding characteristics of femoral stems on adaptive bone remodeling. *J Biomech Eng* 1994;116:*[in press]*.
12. Menge M, Maaz B, Textor U. Design of femoral stem and early results of cementless hip replacements: comparison of different principles of fixation. In: Buchhorn GH, Willert HG, eds. *Technical principles, design and safety of joint implants*. Seattle: Hogrefe & Huber; 1992:142–148.
13. Sumner DR, Turner TM, Urban RM, Galante JO. Long-term femoral remodeling as a function of the presence, type and location of the porous coating in cementless THA. *Transactions of the 34th Meeting of the Orthopedic Research Society*, Palatine, IL. 1988;13:310.
14. Maloney WJ, Jasty M, Burke DW, O'Conner DO, Zalenski EB, Bragdon C, Harris WH. Biomechanical and histological investigation of cemented total hip arthroplasties. *Clin Orthop* 1989;249:129–140.
15. Treharne RW. Review of Wolff's law and its proposed means of operation. *Orthop Rev* 1981;10: 35–47.
16. Currey J. *The mechanical adaptations of bone*. Guilford, UK: Princeton University Press; 1984.
17. Roesler H. The history of some fundamental concepts in bone biomechanics. *J Biomech* 1987;20: 1025–1034.
18. Frost HM. Vital biomechanics. Proposed general concepts for skeletal adaptations to mechanical usage. *Calc Tissue Int* 1987;42:145–156.
19. Pauwels F. *Gesammelte Abhandlungen zur Funktionellen Anatomie des Bewegungsapparates*. Translated *Biomechanics of the locomotor apparatus*. Berlin: Springer-Verlag; 1980. P. Maquet and R. Furlong, translators.
20. Huiskes R, Weinans H, Grootenboer HJ, Dalstra M, Fudala B, Slooff TJ. Adaptive bone-remodeling theory applied to prosthetic-design analysis. *J Biomech* 1987;20:1135–1150.

21. Cowin SC, Hegedus DH. Bone remodeling I: a theory of adaptive elasticity. *J Elasticity* 1976;6: 313–326.
22. Carter DR. Mechanical loading history and skeletal biology. *J Biomech* 1987;20:1095–1109.
23. Turner TM, Sumner DR, Urban RM, Rivero DP, Galante JO. A comparative study of porous coatings in a weight-bearing total hip arthroplasty model. *J Bone Joint Surg* 1986;68A:1396–1490.
24. Sumner DR, Turner TM, Urban RM, Galante JO. Remodeling and ingrowth of bone at two years in a canine cementless total hip-arthroplasty model. *J Bone Joint Surg* 1992;74A:239–250.
25. Bergmann G, Siraky J, Rohlmann A, Koelbel R. A comparison of hip joint forces in sheep, dog and man. *J Biomech* 1984;12:907–921.
26. Van Rietbergen B, Huiskes R, Weinans H, Sumner DR, Turner TM, Galante JO. The mechanism of bone remodeling and resorption around press-fitted THA stems. *J Biomech* 1993;26:369–382.
27. Weinans H, Huiskes R, van Rietbergen B, Sumner DR, Turner TM, Galante JO. Adaptive bone remodeling around bonded noncemented THA: a comparison between animal experiments and computer simulations. *J Orthop Res* 1993;11:500–513.
28. Turner TM, Sumner DR, Urban RM, Galante JO. Femoral remodeling associated with uncoated and porous-coated cementless total hip arthroplasty. *Transactions of the 33rd Meeting of the Orthopedic Research Society* 1987;12:408.
29. West JD, Mayor MB, Collier JP. Potential errors inherent in quantitative densitometric analysis of orthopaedic radiographs. *J Bone Joint Surg* 1987;69A:58–64.
30. Engh CA, McGovern TF, Bobyn JD, Harris WH. A quantitative evaluation of periprosthetic bone-remodeling after cementless total hip arthroplasty. *J Bone Joint Surg* 1992;74A:1009–1020.
31. Huiskes R. Stress shielding and bone resorption in THA: clinical versus computer-simulation studies. *Acta Orthop Belg* 1993;59(Suppl. I):118–129.
32. Weinans H, Huiskes R, Grootenboer HJ. Trends of mechanical consequences and modeling of a fibrous membrane around femoral hip prostheses. *J Biomech* 1990;23:991–1000.
33. Rubin PJ, Rakotomana RL, Leyvraz PF, Zysset PK, Curnier A, Heegaard JH. Frictional interface micromotions and anisotropic stress distribution in a femoral total hip component. *J Biomech* 1993; 26:725–740.
34. Weinans H, Huiskes R, Grootenboer HJ. Quantitative analysis of bone reactions to relative motions at implant–bone interfaces. *J Biomech* 1993;26:1271–1281.
35. Huiskes R. Failed innovation in total hip replacement: diagnosis and proposals for a cure. *Acta Orthop Scand* 1993;64(6):699–716.