Implant stability, histology, RSA and wear—more critical questions are needed

A view point

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I discuss the radiostereometry (RSA) of prosthetic micromovement from clinical and histological viewpoints. Even in non-migrators there is a wide variety of interfaces with various resistances to trigger factors, such as mechanical forces and sequels of wear. I suggest that the various forms of bone resorption seen with conventional radiography are caused by such trigger factors and that their anatomical appearance is determined by the structure of the interface obtained at surgery.

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Does wear really cause prosthetic loosening? This question has recently been asked in a review in this Journal (Mjöberg 1994). The author contends that loosening is caused by an early loss of fixation, that wear products arrive at the interface secondary to implant instability and that stability can be defined on the basis of RSA (radiostereometry), whereas histology is a doubtful tool in evaluating prosthetic stability.

My purpose with this review is to put the evaluation of RSA into perspective against the background of clinical and histological observations.

Clinical implications of migration

In the extensive literature on prosthetic migration after hip and knee replacement (for review, see Ryd 1992), 3 general patterns are seen: 1) continuous migration, 2) slight migration for 3–12 months followed by stability, and 3) no migration. The proportions vary with implantation site.

Although the clinical result may be successful initially, it appears that continuous migration implies a poor long-term prognosis for prosthetic survival. However, this also has a positive consequence. If a new prosthetic design and/or anchorage principle is shown to have a disproportionate number of early continuous migrators it should be regarded with a degree of scepticism and withheld from widespread use until more data are gathered, all for the benefit of our patients.

If one accepts that early and continuous migration may herald later loosening, the question then arises whether one can be correspondingly optimistic about the other 2 migratory patterns, which make up the majority of cases. Is the migratory pattern, as defined by RSA, specific enough to distinguish between future failure and permanent fixation?

General observations about micromotion

Migration is a form of micromotion which implies an irreversible shift in implant position that takes place over time and requires serial measurements with intervals of several months. The term micromotion is sometimes also used to describe a small reversible movement on provocation, also called inducible displacement (Ryd 1992). These 2 modes of motion are not necessarily correlated (Ryd 1986).

This lack of a relationship may well be due to the fact that, once a mature interface has developed, these movements take place at different sites: The interface can be regarded as a connection between an implant and surrounding bone, with or without an intervening soft tissue membrane (Figure 1). Inducible displacement can theoretically take place within the implant itself (loose-fitting parts), as a slip between implant and membrane, as a deformation within the membrane or as an elastic deformation within the bone. It does not take place at the junction of the membrane and the bone, since they are joined together.

Migration (if deformation of the implant itself can be excluded, which also includes subsidence within the cement, and if a continuous slip between the implant and membrane can be ruled out) can only be the result of bone resorption or bone impaction due to mechanical overload, i.e., the result of osteoclastic

Figure 1. Showing fibrocartilage below a non-migrating unicondylar tibial component, 7 years postoperatively. (Reproduced with permission from Ryd L and Linder L. On the correlation between micromotion and histology. J. Arthroplasty 4; 303, 1989, Churchill Livingstone, New York).

activity at the membrane-bone junction or the result of the healing of an impaction fracture. In summary, migration generally is the result of events that do not apply to inducible displacement, which should be kept in mind when terms like *stability* and *loosening* are used.

These considerations are of profound importance for discussions on implant stability in general and RSA and histology in particular. Histology, by its very nature, is a method that depicts one point in time, and therefore cannot be used to study migration, which is a dynamic process. At best, histology can demonstrate a high rate of bone turnover at the bone-membrane junction, but it cannot determine whether there is a net loss of bone. Histology can, however, give valuable information about the basis for inducible displacement.

Resolution of RSA and histology

RSA is our most sensitive tool for non-invasive study of prosthetic stability. It has a resolution of about 100 microns. Histology, on the other hand, has a resolution that is about 2 magnitudes greater. This difference would perhaps not be very important if the same phenomenon were studied. However, with histology, cellular reactions are studied, with RSA only the effects of cellular activity, in the form of a measurable distance. This, in turn, has the important implication that RSA is a late detector of early cellular activity, which can be quite varied, just as loosening may be a late manifestation of early migration. Today few studies attempt to span the gap between radiology and cellular anatomy.

Therefore, unfortunately we are unable to correlate the massive literature on interface histology (in which the migratory pattern is unknown) with the literature on migration (in which the histology is unknown). Above all, we must not extrapolate RSA results into specific histologic categories. This will certainly lead to the question: At what level of resolution do we find clinical relevance? Is it permissible to draw conclusions about the long-term behavior of a prosthesis that does not migrate during the first few years or can there still be dangers ahead?

Non-migrators, histology

Non-migrators represent a mechanically adequate implant fixation. Does this also mean that non-migrators represent a biologically sound situation?

In a study of the histology of three non-migrating unicondylar tibial components, Ryd and Linder (1989) showed that the components were supported by a 2 mm layer of soft tissue, mainly fibrocartilage (Figure 1).

In Charnley's sockets there was a soft tissue lining in 100 percent of cases (Malcolm 1990). In most RSA studies of socket migration, about 50 percent exhibit migration, while the remainder are stable. This should indicate that even implants bordered by a fibrous membrane do not necessarily migrate (or migrate so little at so slow a rate that this goes undetected by RSA, so that they are defined as stable).

It would seem reasonable to assume that successful osseointegrated implants do not migrate, although this has never been shown by RSA. Interestingly, in an RSA study of femoral knee components, the only one revised because of migration showed bony ingrowth (Nilsson et al. in press). Nevertheless, from the above it is clear that non-migrators, far from representing a uniform interface anatomy, encompass at least two subsets: osseointegration and soft tissue, including fibrocartilage. The question then is whether these interfaces of clinically completely successful and non-migrating implants are also qualitatively identical.

Osseointegration represents a mechanical barrier to the flux of fluid along the interface. However, it is interesting to speculate on how the avascular fibrocartilage in Figure 1 was nourished during the 7 years when the implant was in service. It is generally held that normal articular cartilage is nourished by diffusion both from the subchondral bone and from the synovial fluid. If this fibrocartilage is nourished in the same way, then it would be an indirect proof that fluid is actually being pumped in and out of the interface region. It is likely that, for example, wear particles take the same route.

The corollary of this reasoning would be that, even in non-migrators, there may be qualitative biological differences between interfaces which, in the long term, may prove important if trigger factors of

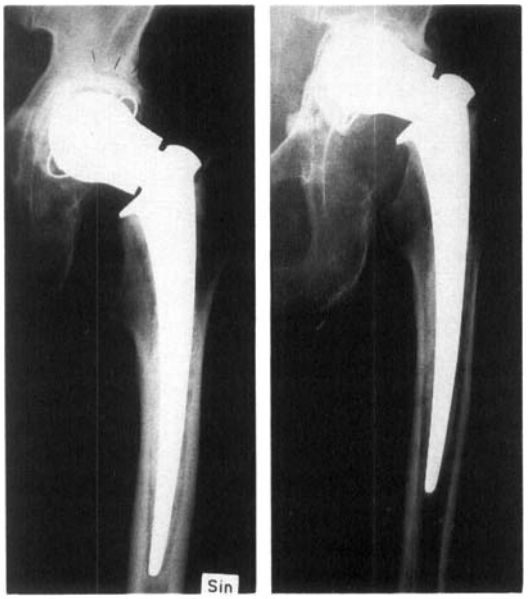

Figure 2. Radiographs of the same hip, 1 and 12 years after a Christiansen THR. Note the socket migration and the intact femoral component.

various kinds are added. How and when are these interfaces formed and what are the possible trigger factors?

Trigger factors

Micromovements (inducible displacement) have long been regarded as a reason for the formation and maintenance of a fibrous membrane between bone and implant. Unlike a fracture fixation device, a joint prosthesis never becomes redundant mechanically: it is critically dependent on an equilibrium between mechanical loads and the tissue resistance to them. Episodes of overload and fatigue phenomena in the implant bed may cause tissue reactions that change the mechanical environment, leading in time to a vicious spiral. There seems to be no spontaneous healing mechanism once this spiral has been set in motion.

A joint prosthesis also differs from other static implants in bone, since it has a number of interfaces that produce wear particles. Such particles are released gradually and the biological environment around the prosthesis therefore is not the same on, e.g., day 300 and day 3000. The wear particles are ingested by cells in the synovium and cause synovitis by activating macrophages to release inflammatory mediators, bone resorbing factors and other cytokines. In all probability there is also an increased amount of joint fluid. It is for future research to determine whether it is pressure waves in the joint fluid, cytokines produced in the joint capsule (Ohlin et al. 1990), or the wear particles themselves that represent the important change.

Can these new conditions trigger an adverse reaction in the interface tissue, leading to implant loosening, even if there are initially no signs of prosthetic migration?

Observations concerning wear

In Scandinavia the Christiansen hip prosthesis represents a large-scale clinical failure. It is interesting to note that the failures were mainly confined to the socket side (Ohlin 1990). Very often we encounter patients with few symptoms but with radiographic appearances such as that in Figure 2. The radiograms show the same hip after 1 year and after 12 years. The femoral component, having been inserted with a 1st generation cementing technique, is completely unaffected although it has suffered much wear, the socket having been worn through. Why do the femur and the acetabulum behave so differently when both must have been exposed to a massive load of plastic particles?

Part of the answer may lie in the way bone reacts to bone cement: bone cement is not a glue and, if it is placed on a sclerotic or dense trabecular surface, microinterlock will not occur. There is increasing clinical evidence that intrusion of cement into cancellous bone is beneficial as regards radiolucencies, but there are hardly any histological studies of the effects of 3rd or 4th generation cementing techniques. Figure 3, representing 3rd generation cementing in the femur, may serve as a reference. Cement was injected well into the cancellous bone present in one aspect of the femur, whereas in the opposite part

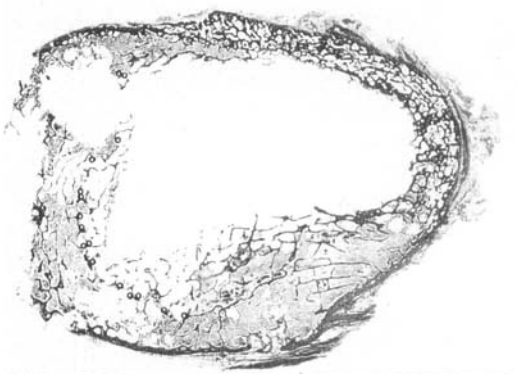

Figure 3. Histological cross-section of the proximal femur in a case of 3rd generation cementing. The cement has been dissolved out. Red circles denote direct contact between osteoid and cement (Goldner stain).

there was no cancellous bone. Only in the cancellous bone were direct contacts between osteoid and cement seen; in the rest of the interface a 1–2 cell layer of fibrous tissue and macrophages bordered the cement. Bone cement seems to require absolute stability as well as time to develop a direct bone-cement contact, possibly through microinterlock.

In hip surgery, the acetabulum often presents to the cement a hard, subchondral bony surface, whereas the femur by its nature presents a cancellous surface. Just as it is easy to achieve good bone-cement interdigitation in the proximal femur, it is correspondingly difficult to do so in the peripheral parts of the acetabulum, except in the anchoring holes. Subjected to early loading, the cement in these areas will most likely be bordered by soft tissue from a very early stage. The development of this thin membrane is the result of tissue-healing in the face of a poor interlock, and not necessarily the result of heat trauma (which seems to be subliminal in the majority of cases, Toksvig-Larsen et al. 1991), nor the result of wear. This is borne out by observations from serial radiographs that zones in the acetabulum can often be traced back to the 3-month radiograms, but the soft tissue may extend further since a membrane a few cell layers thick is not detected on clinical radiograms (Linder 1994). This does not mean that the socket is loose (a jetty is not unstable simply because water moves underneath it), but it means that fluid may have access to at least the peripheral parts of the interface almost from the time of surgery, and that the total area of bone/cement interdigitation may be rather small.

Granulomata

The patterns of wear granulomata, seen in the femur as endosteal scallopings and recently in relation to uncemented acetabular cups, are intriguing. A common feature is that they seem to be contained, i.e., they appear at first as localized lesions. A risk factor with cemented femoral components has been shown to be metal-bone contact, usually at the tip. With uncemented cups, screw-holes seem to be a common feature, which is also true at the knee (Peters et al. 1992, Maloney et al. 1993), but recently cystic lesions have been shown to originate adjacent to the edge of uncemented sockets as well (Schmalzried et al. 1994).

The observation of polyethylene wear particles in these lesions suggests that a pathway has been opened up between the joint cavity and a small part of the interface. This would require a mechanical component at the site of the future lesion and/or a trigger factor in the joint. It is not within the scope of this review to speculate on the sequence of events leading to localized bone loss, but doubts about wear particles as the primary event are not unfounded. For example, in the femur, high pressures have been measured in these lesions even after slight movement of the hip (Anthony et al. 1990), and little is known about the local effects of such pressure waves occurring with every step. Because of their dimensions, water molecules, as well as soluble, biologically active molecules, must reasonably precede even submicron particles. However, as Willert et al. pointed out (1990), it remains to be shown whether fluid transport is the only mechanism by which wear particles are transported.

The anatomy and function of the interface

A loaded implant must work in concert with the surrounding tissue. Thus, for example, osseointegration of an implant is not sufficient in itself, if the supporting bone is too weak to cope with dynamic loads. It has also become increasingly evident that the interface must in some way be able to withstand the added impact of trigger factors.

Concerning function, it may be relevant to talk about tight interfaces and open ones, that is, whether or not fluid can move along the interface. As is seen from the above, this distinction bears no relation to migration, nor does it exclude the possibility that a tight interface can become an open interface through mechanical or biological factors.

Osseointegration would naturally represent a tight interface but, under certain circumstances, even a soft tissue interface may act as a tight interface. For instance, with bone cement in cases of good interlock, and with porous-surfaced implants, where collagenous fibers run into the depth of the coating, much like Sharpey's fibers, rather than create a bursa around the implant.

In an open interface, the following sequence cannot be exluced: the membrane formed between the cement and the bone is relatively stable at first, so that bone remodeling goes on at a pace in which bone formation and resorption are reasonably balanced. Gradually, since inducible displacements are present, cytokines (Ohlin et al. 1990) and wear products accumulate at the interface, affect macrophages, which stimulate bone resorption and reduce the rate of new bone formation (Goodman 1994). The net result will be bone loss and eventually a detectable migration.

There need not be an antagonism between the seemingly disparate theories of implant fixation, wear, loosening and bone resorption. Rather, there may be an interaction of mechanical and biological

factors such that what we observe today are different expressions of a common phenomenon, bone resorption, in a variety of settings. For example, cystic granuloma formation may be a localized form of bone resorption seen in cases of tight interfaces, whereas a linear radiolucency and migration may be the typical expression of bone resorption in an open interface situation. These interfaces have various initial appearances, related to the design of the implants and the anchorage principle, but also very much to the performance of the surgeon who implanted the devices.

The thoughtful surgeon will understand that various systems have different side-effects and safety margins, and should thereby be better able to use the implants to their full advantage.

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