Wear Debris in Total Joint Replacements

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Abstract

In vivo degradation of prosthetic implant materials is increasingly recognized as a major factor limiting the durability of total joint arthroplasty. In vivo degradation occurs primarily by means of wear processes that can generate large quantities of particulate debris. This debris can stimulate an adverse local host response leading to periprosthetic bone loss, which can compromise implant fixation and bone stock. The authors review the basic mechanisms of implant degradation and the host response to particulate degradation products, particularly in the context of the pathogenesis of osteolysis. Submicron polyethylene particles (mean size, 0.5 μm) are the dominant type of wear particle present in periprosthetic tissues associated with uncemented hip replacements. Polyethylene wear can be minimized by improving the quality of the polyethylene, avoiding use of large-diameter (greater than 28 mm) femoral heads in total hip arthroplasty, and improving the design and fabrication of modular connections, which can be important sources of three-body wear particles. Advances in the understanding of the basic mechanisms of osteolysis are critical to the development of preventive measures that will minimize the clinical impact of this phenomenon.

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Wear debris—its generation and the subsequent tissue reaction to it—has emerged as a central problem limiting the long-term longevity of total joint replacements. Since the inception of the low-friction arthroplasty concept in the late 1960s, it has been recognized that wear is a significant issue. Certainly, Sir John Charnley's early experience with polytetrafluorethylene acetabular components points to the disastrous consequences of accelerated articular wear. Nearly two decades ago, Willert and Semlitsch[1] published a seminal paper that serves as the basis for much of the current understanding of the relationship of articular wear debris to periprosthetic bone loss and aseptic loosening. These authors were among the first to propose that the generation of wear debris may eventually overload local afferent transport mechanisms, leading to accumulation within and around the joint and subsequently to periprosthetic bone resorption and aseptic loosening.

The study of wear and the biologic response to wear debris is truly a multidisciplinary effort involving concepts from a variety of fields, among them tribology (the study of friction, lubrication, and wear), materials science, mechanical engineering, histopathology, biochemistry, and molecular biology. Tools from each of these disciplines must be brought to bear in order to understand the mechanisms of particle generation, as well as the mechanisms of tissue response to such particles. This review traces the progress in understanding the tissue reaction to wear debris with regard to physical and biologic mechanisms, clinical ramifications of wear debris–tissue interactions, and current strategies to minimize the clinical impact of wear.

Mechanisms of Debris Generation

In broad terms, the generation of debris from implanted materials can be conceptualized as occurring from two independent, though not mutually exclusive, processes: wear and corrosion. Wear involves the loss of

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material in particulate form as a consequence of relative motion between two surfaces. Real surfaces are not atomically smooth, but possess undulations (peaks and valleys). Two materials placed together under load will be in contact over only a small area of the higher peaks, or asperities. Atomic interactions occur at the individual points of contact; when two surfaces slide relative to each other, these interactions are disrupted, with a finite probability that localized failure will occur in one or the other sliding surface. This results in the release of material in the form of particles, or wear debris. The particles may be lost from the system, may be transferred to the counterface, or may remain between the sliding surfaces. There are primarily three processes that can cause wear: (1) abrasion, by which a harder surface plows grooves in a softer material; (2) adhesion, by which a softer material is smeared onto a harder counter surface, forming a transfer film; and (3) fatigue, by which alternating episodes of loading and unloading result in the formation of subsurface cracks, which propagate to form particles that are shed from the surface.[2]

The second mechanism by which debris can be generated is corrosion. Unlike wear, corrosion is governed by electrochemical phenomena and generally applies only to metallic implant materials. Some authors consider in vivo oxidation of polyethylene a form of corrosion; however, unlike metallic corrosion, polyethylene oxidation is a chemical (as opposed to an electrochemical) process. Metallic corrosion involves metal release on an ionic level; however, particulate matter can be formed by precipitation of metal salts in the aqueous media, or particles may be released by selective (grain boundary) corrosion.

Corrosion and wear processes can often be synergistic. For example, the generation of metallic wear debris due to adhesion, abrasion, or fatigue can lead to the generation of very fine particulate matter within the tissues. This, in turn, presents an enormous surface area available for electrochemical processes. Some of the local cellular events that occur in response to wear debris may, in fact, be mediated in part by the effect of metal salts or organometallic complexes. Furthermore, certain wear processes, such as fretting (wear produced by small cyclic interpart motions), may accelerate corrosion by disrupting passivating oxide films. This is probably the dominant mechanism of generation of particulate corrosion products from joint replacement implants, given the fact that the two metallic implants currently in use (titanium-base alloy and cobalt-base alloy) are self-passivating and have an oxide layer that serves as an effective barrier to generalized and localized (pitting and crevice) corrosion.

Wear Rates

During the initial relative motion of surfaces, a large number of asperities break, resulting in a high wear rate. This is termed the "wearing-in period." The real contact area increases, and the two surfaces can be said to have adapted to each other. With the passage of time, the wear rates decrease and eventually become linearly dependent on the contact force and sliding distance.[2] This is termed "steady-state wear."

Many efforts have been made to measure the steady-state wear rates of various articulating couples in vitro. The results of such studies have been difficult to interpret and apply due to the many variables playing a role (e.g., test geometry, material pair selection, load transfer setup, and selection of lubricant). In general terms, the harder of the two bearing materials will wear less rapidly. In a metal-polymer pair, the polymer wears almost exclusively; in a metal-ceramic pair, the metal will wear to a greater extent. The estimated in vitro wear rates for the socket (in hip-joint simulation studies) range from 0 to 3,000 mm^3/year, depending on such factors as the type of couple employed, the test condition, and the lubricant used.[3] Extraneous debris can significantly influence in vitro wear rates.

There is also a great deal of variability in in vivo wear rates, generally measured in radiographic follow-up studies of total joint replacements. Radiographic wear measurements are usually expressed as linear wear rates, whereas in vitro studies generally report volumetric wear. Volumetric wear is actually the more critical of the two measurements because it can be directly related to the number of wear particles presented to the periprosthetic fluids, which typically is on the order of billions of particles per year.[4]

For the hip, linear wear rates of 25×10^{-6} (ceramic on ceramic) to 2.26 mm/year (Teflon on stainless steel) have been reported.[3] For the most common wear couple currently in use in the United States, cobalt-base alloy and ultrahigh-molecular-weight polyethylene (UHMWPE), wear rates are typically on the order of 0.1 mm/year.[3] Linear wear rates of this magnitude generally do not directly affect the function of the joint; however, significantly higher rates could lead to joint dysfunction due to impingement of the femoral neck on the acetabular component.

Clinical wear rates would be expected to increase with increasing physical activity, weight of the patient, size of the femoral head, roughness of the metallic counterface, and oxidation of the polyethylene. In contrast, clinical wear rates would be expected to decrease with

increasing polyethylene thickness[5] and molecular weight.

Osteolysis due to Wear

Clinical Features

Periprosthetic bone loss, or osteolysis, presents either as diffuse cortical thinning or as a focal cyst-like lesion. The latter may involve the metaphyseal trabecular bone, the diaphyseal cortical bone, or both. Charnley was among the first to recognize the phenomenon of endosteal osteolysis in cemented total hip arthroplasty (THA), initially describing it as an "alteration in the texture of the cortex." Subsequently, several authors have described the phenomenon of osteolysis in association with loose cemented femoral components[6] (Fig. 1).

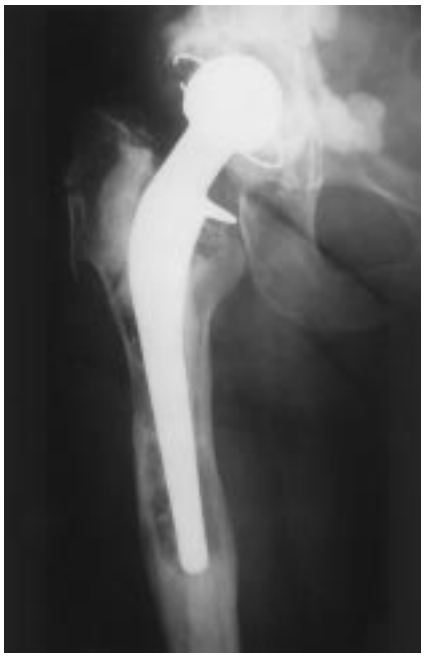


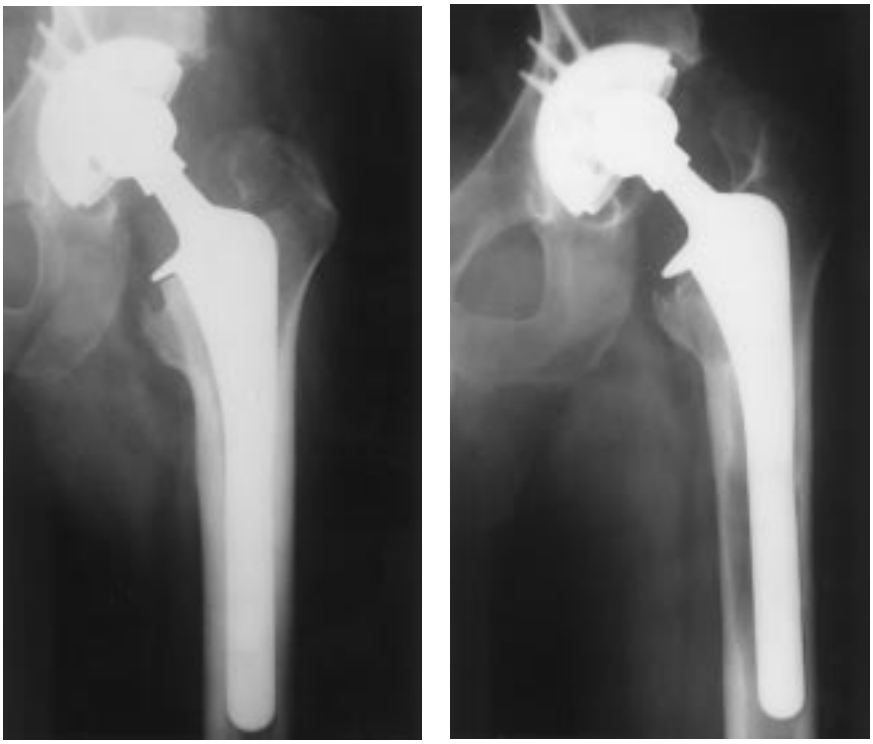
Fig. 1 Anteroposterior radiograph of the hip of a patient with an aseptically loose cemented titanium-base-alloy–UHMWPE total hip replacement. There is evidence of debonding in the proximal lateral cement-metal interface, with large areas of focal endosteal bone loss adjacent to the femoral stem.

Focal osteolysis in association with stable cemented femoral components has also been described by several authors. Maloney et al[7] reported 25 cases of focal femoral osteolysis in radiographically stable cemented femoral implants. The time interval between implantation and the appearance of the femoral lytic lesion ranged from 40 to 168 months. The rate of radiographic progression was variable; in one case, the lesion progressed to gross loosening of the femoral component. In 60% of the patients, the osteolytic area corresponded to either a cement-mantle defect or a focus of very thin cement. A direct communication between the joint and the focal lesion through the stem-cement interface and a cement-mantle defect has been postulated as an important element in the pathogenesis of focal osteolysis in cemented implants, as demonstrated by Anthony et al.[8]

The occurrence of osteolysis in both well-fixed and loosely cemented total hip replacements gave rise to the misnomer "cement disease." On the basis of histologic studies demonstrating cement debris associated with macrophages, giant cells, and vascular granulation tissue, it was initially thought that the reaction to particulate polymethylmethacrylate produced these lesions. Recently, however, osteolysis has been recognized in association with both loose and well-fixed uncemented implants, demonstrating that the absence of acrylic cement does not preclude the occurrence of osteolysis. Analyzing data from three centers with a minimum follow-up of 2 years, Maloney et al[9] reported focal femoral osteolysis in 3% of 474 consecutive radiographically stable uncemented cobalt-base and titanium-base-alloy total hip replacements. In our recent review of THA with uncemented titanium-base alloy,[10] 8% of 110 radiographically stable hips showed focal femoral osteolysis at an average 5.5-year follow-up. The average interval to the appearance of a radiographic lesion was 50 months (range, 36 to 63 months). These patients with femoral osteolysis and radiographically stable hips were asymptomatic (mean Harris Hip Score, 94; range, 77 to 100), except for one patient who experienced mild thigh pain.

There was no difference in any demographic or radiologic variable between patients with femoral osteolysis and those without, with the exception that osteonecrosis of the femoral head was the preoperative diagnosis more frequently in patients with osteolysis (55%) than in those without osteolysis (29%). This apparent relationship between osteolysis and prior osteonecrosis is probably attributable to the fact that patients with osteonecrosis tend to be younger and more active on average than the THA population at large. Therefore, this subset of patients place greater demands on the articulation, which may result in more wear, more debris generation, and more osteolysis. Radiographically (Fig. 2), these lesions were most common in the vicinity of the distal aspect of the femoral stem (Gruen zones 3 to 5) and were typically associated with endosteal scalloping of the proximal medial femoral cortex (Gruen zone 7). Generally, these lesions tended to be progressive. It appeared from our review that osteolysis was observed earlier and at a higher incidence with stable uncemented femoral components than with cemented components, at least for the type of uncemented design used in this patient population (Harris-Galante prosthesis [HGP], Zimmer, Warsaw, Ind).

While the incidence of osteolysis in stable implants was 8% at minimum 4.5-year follow-up, it was 14.9% at minimum 8-year follow-up, demonstrating that the incidence of



A B

Fig. 2 Anteroposterior radiographs of the hip of a patient who underwent THA with an uncemented titanium-base-alloy Harris-Galante prosthesis with a cobalt-base-alloy–UHMWPE articulating couple. **A,** Image obtained in the early postoperative period. **B,** Image obtained 103 months postoperatively demonstrates significant endosteal bone loss in both a cystic pattern (Gruen zones 5 to 7) and a linear pattern (Gruen zones 2 to 4).

femoral osteolysis in this patient population increased with time. Since osteolysis is usually asymptomatic, long-term radiologic follow-up of all patients after THA, especially those with uncemented implants, is strongly recommended to identify this process prior to the occurrence of major complications secondary to progressive bone loss. Other authors have reported a 10% to 20% incidence of focal femoral osteolysis at 2- to 9-year follow-up with other uncemented implant systems fabricated from both cobalt- and titanium-base alloy.[6]

Acetabular osteolysis has received less attention, but it does occur in association with both cemented and uncemented acetabular components. For uncemented components, the incidence depends on the type of acetabular component and the length of follow-up. Incidences of 46% at 5- to 7-year follow-up with a cobalt-base-alloy porous-coated implant (PCA, Howmedica, Rutherford, NJ), 28% at 6-year follow-up with a cobalt-base-alloy acetabular component (AML, DePuy, Warsaw, Ind), and 1.2% at minimum 5-year follow-up with the titanium-base-alloy HGP acetabular component have been reported.[6] This difference in incidences is thought to be due to differences in the thickness of the UHMWPE insert, the relative stability of the UHMWPE insert within the metal backing, the congruence of the insert with respect to the concave surface of the metal backing, the femoral head diameter, the quality of the polyethylene, or a combination of these factors.

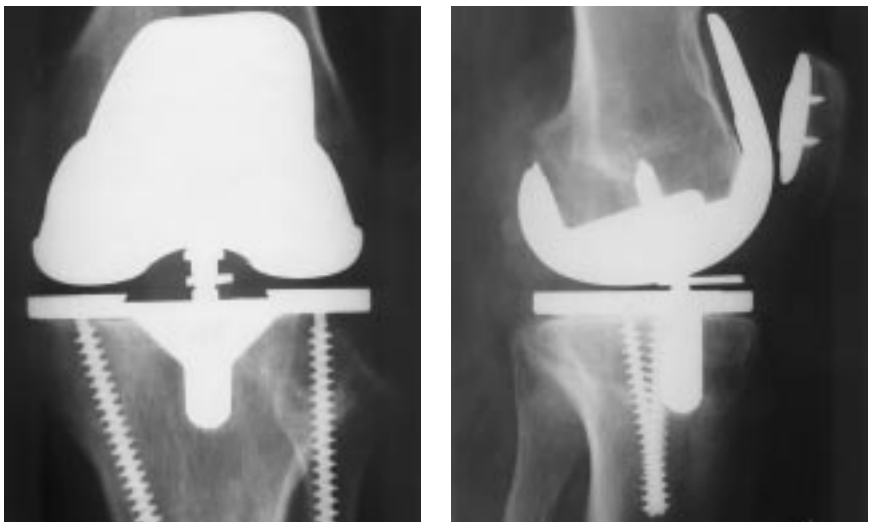
Radiologically, it is possible to recognize two types of acetabular lesions. Periacetabular lesions are seen primarily in the periphery of the acetabulum. Retroacetabular lesions are seen centrally and infiltrate the body of the ilium and/or (occasionally) the body of the ischium.

Osteolysis associated with total knee arthroplasty (TKA) has been reported infrequently. Peters et al[11] reported an incidence of 16% in a cementless cobalt-alloy device at an average of 35 months after surgery. The medial aspect of the proximal tibia was the most common site for bone resorption, and the screw-bone interface seemed to be a preferential pathway for progression of this process (Fig. 3). The histologic findings in this series were similar to those reported for lesions about the hip; however, there were particular design features of the prosthesis used in that study that may have led to accelerated polyethylene and metal wear.

It is unclear why osteolysis is reported more frequently about the hip than about the knee. Factors such as differential mechanisms of hip and knee wear resulting in different polyethylene particle geometry and size, differences in joint volume, and differences in interfacial barriers to migration of debris have all been postulated to account for this apparent disparity.

Histologic Features

We have reviewed the histologic appearance of periprosthetic tissues from patients with femoral osteolysis associated with uncemented implants who underwent revision surgery at our institution.[6] The findings were qualitatively similar for patients with loose



A B

Fig. 3 Radiographs of a patient with a painful uncemented cobalt-base-alloy TKA. **A,** Anteroposterior radiograph shows a large area of tibial bone loss associated with the proximal aspect of the lateral tibial screw. **B,** Lateral radiograph demonstrates a large area of femoral bone loss adjacent to the anterior flange of the femoral component.

implants and those with well-fixed implants, but more particulate wear debris was observed in association with loose implants. The joint pseudocapsule revealed hypertrophic synovitis with areas of necrosis, intense histiocytic infiltration, and occasional foreign-body giant cells and lymphocytes. There was no evidence of acute inflammation. Many of the histiocytes contained fine, opaque black granules. Strongly birefringent particles from the submicron range up to approximately 50 μm in size were seen under polarized light. The larger particles were associated with foreign-body giant cells.

Specimens obtained from the femoral membrane in the vicinity of the osteolytic lesions demonstrated dense fibrous tissue with foci of intense histiocytic infiltration and with foreign-body giant cells. Lymphocytes were scarce, and there was no evidence of an acute inflammatory process. Isolated areas demonstrated fine, opaque black granules within the histiocytes, similar to those seen in the capsule but less numerous. Under polarized light, minute, strongly birefringent particles, characteristic of polyethylene, were observed within the cytoplasm of the histiocytes (Fig. 4). Nearly all of the femoral components demonstrated bone ingrowth under backscattered scanning electron microscopy. In one case, a histiocytic infiltrate associated with resorption lacunae in the ingrown bone was present within the porous coating in a loose component, suggesting trabecular bone failure either as a result of or aided by the resorption process.

The histologic appearance of osteolysis seen in association with cementless implants was similar to that seen in association with cemented implants, except that the latter demonstrated large numbers of polymethylmethacrylate particles (or voids representing the location of particles dissolved during tissue processing).[8]

Particle Analysis

We evaluated the joint pseudocapsule and interfacial membranes from patients with osteolysis associated with uncemented titanium-alloy–UHMWPE implants, utilizing electron microprobe analysis, analytic electron microscopy, and Fourier transform infrared spectroscopy for the determination of the identity and amount of particulate wear debris. Both tissues contained particles of titanium alloy (size range, less than 1 μm to 20 μm), but many fewer metallic particles were found if the components were well fixed at revision surgery. Fourier transform infrared spectroscopy positively identified UHMWPE particles as small as 5 μm (smaller particles are beyond its resolution). The superior resolution of the analytic electron microscope facilitated identification of silicate and stainless-steel particles in the submicron size range.

We recently conducted a parallel study to characterize the composition and morphology of wear debris from periprosthetic tissues.[12] The tissues were recovered from osteolytic areas in patients undergoing revision of uncemented titanium-alloy total hip replacements (mean implantation

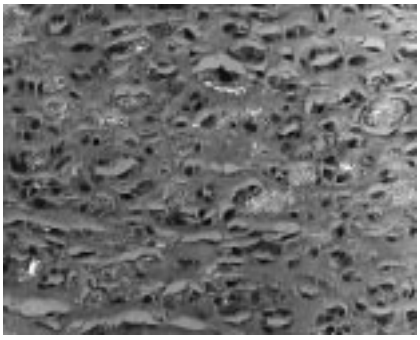


Fig. 4 Polarized light photomicrograph of tissue obtained from an osteolytic lesion in a patient with a loose titanium-base-alloy HGP after 64 months in situ. Note plump histiocytes with numerous intracellular birefringent polyethylene particles (hematoxylin-eosin; original magnification X200).

time, 62 months; range, 8 to 114 months). The composition of the particulate debris was characterized, and particle-size analysis was performed with the use of scanning electron micrographs of the recovered debris. This study revealed that 70% to 90% of the recovered particles were submicron UHMWPE (mean size, approximately 0.5 μm). Similar findings have been reported by other laboratories.[13,14] In our study, smaller quantities of titanium alloy, commercially pure titanium, and bone particles were also identified. Stainless-steel and silicate particles were relatively rare. Thus, volumetrically, submicron UHMWPE particles seem to be the dominant wear product present in the periprosthetic tissues of patients with osteolysis associated with uncemented implants.

Pathogenesis

The pathogenesis of focal osteolysis is currently under intense scrutiny. In 1983, Goldring et al[15] opened up a new avenue of orthopaedic research when they described a synovium-like membrane at the bone-cement interface in patients with loose total hip replacements. This membrane had the capacity to produce large amounts of prostaglandin E_2 (PGE_2) and collagenase—substances that possess bone-resorbing activity. Many investigators have subsequently studied the relationship of macrophage and fibroblast secretory products to aseptic loosening and osteolysis.

We have conducted a series of investigations in an effort to delineate the pathogenesis of osteolysis. In these studies we have shown the following: (1) Levels of interleukin-1 (IL-1), a potent proinflammatory cytokine with bone-resorbing activity, were significantly elevated in explants of interfacial membranes from failed uncemented total hip replacements.[16] (2) Phagocytosable particles (those measuring 10 μm or less) of unalloyed titanium and polymethylmethacrylate could stimulate the secretion of IL-1 and PGE_2 from mouse peritoneal macrophages in a dose- and time-dependent manner, whereas nonphagocytosable particles (those measuring more than 10 μm) had little effect.[17] (3) Unalloyed titanium particles measuring 1 to 3 μm had the capacity to enhance the bone-resorbing activity of these macrophages in a dose-dependent manner in a bone-organ culture system.[17] (4) Prostaglandin E_2 and IL-1 inhibition could only partially block the latter effect, indicating that macrophage-mediated bone resorption involves a complex cascade of cytokine-mediator interactions.[17] Further research is needed to clarify the role of the various bone-resorbing agents and the role of the various particulate species in periprosthetic bone loss.

Recently, more sophisticated methods have been applied to study the problem of periprosthetic bone loss.[18] These include immunohistochemistry and in situ hybridization, both of which are powerful tools that can help unravel the basic cellular mechanisms leading to the observed clinical entities of focal osteolysis and aseptic loosening. Work is under way at several centers utilizing these techniques.

Jiranek et al[18] have demonstrated that IL-1β messenger RNA (mRNA) is present predominantly in macrophages, whereas IL-1β protein is present on both macrophages and fibroblasts. This suggests that macrophages actively secrete this cytokine, which is subsequently bound to both macrophages and fibroblasts. Our laboratory has demonstrated that IL-1β is a dominant cytokine present in periprosthetic granulomatous tissue, measured either as protein (by immunochemical techniques) or as mRNA (by using the polymerase chain reaction, a powerful technique that amplifies extremely small quantities of mRNA).[19] Furthermore, cells of the interfacial membrane have a high latent capacity for IL-1α and IL-6 secretion in response to a change in the microenvironment, suggesting potential roles for these two cytokines in particle-stimulated, macrophage-mediated bone resorption.

In summary, it is hypothesized that wear-particle generation and migration into the joint cavity and periprosthetic space may stimulate macrophage recruitment and phagocytosis, as proposed by Willert and Semlitsch.[1] This, in turn, stimulates secretion of various cellular mediators that interact and modify the activities of one another, resulting in either histiocytic or osteoclastic bone resorption.

Material and Design Considerations

Wear particles can originate from a number of different sites. In acetabular and tibial components, they can originate from the articular or nonarticular surface of the polyethylene, the metal backing, or the fixation screws. In other components, stems, coatings, metallic articular surfaces, and modular connections can all potentially generate particulate debris. Surgical tools, bone, remnants of surface processing of the prosthetic device, and the catalyst used in the synthesis of polyethylene can also be sources of particulate debris.

In most studies, UHMWPE is the predominant particle. Most likely, the bulk of this debris originates from the articular surface and has easy access to the proximal medial femoral cortex and the trochanteric region in the hip. Localized osteolytic lesions in these areas are common, but their clinical significance is limited unless large granulomatous lesions develop.

Osteolysis remote from the articulation presents a more complex

problem. For example, the finding of UHMWPE debris in the vicinity of the distal aspect of a well-fixed THA femoral stem suggests a communication between the joint space and the most remote regions of the femoral periprosthetic space.[20]

In noncircumferentially coated devices and press-fit devices without a coating, a space can often be recognized between the cortical shell that forms around the implant and the metallic surface of the implant. The space can be an actual cavity or can be occupied by loose connective tissue. In both instances, direct access of particulate material to the distal femoral canal is possible. Autopsy specimens of noncircumferentially coated devices from our implant retrieval pool have shown the presence of histiocytes in cavities surrounding the uncoated regions of the THA femoral component. These histiocytes demonstrated particulate intracellular birefringent material with the same characteristics identified in histiocytes in tissues from the joint capsule. Similar findings have been observed in our canine uncemented THA model.

In the case of circumferentially coated devices, access to the remote aspects of the implant-bone interface appears to be restricted. While the overall incidence of femoral osteolysis associated with THA may not be less with circumferentially coated implants, the lesions tend to be proximal to the porous coating, at least in the initial stages. As the process evolves, however, it may progress distally.

With regard to the two types of acetabular lesions, the peripheral lesion is probably related to wear debris originating from the joint cavity, and is similar to lesions seen in the area of the proximal medial femoral cortex or at the greater trochanter. The polyethylene debris responsible for the retroacetabular lesions may originate from the convex side of the acetabular insert, gaining access to the bone by means of holes in the shell created during the manufacturing process. However, retroacetabular lesions have been observed in cementless implants even in the absence of holes in the shell. The volume of the debris generated from the polyethylene is no doubt related to a number of variables, including the smoothness of the concave metallic surface of the acetabular component, the tolerance between the polyethylene and the metal shell, and the relative stability of the insert. For example, failure of the locking mechanism, which allows free motion of the polyethylene liner within the shell, could generate a significant volume of polyethylene debris from the convex surface in the absence of eccentricity of the head and significant wear at the articular (concave) surface of the insert. This mode of failure may be more frequently observed in the future as the time of implantation of earlier modular designs increases.

Metallic debris may originate from stems as a result of stem-bone fretting. This would be expected for loose implants in which gross interfacial motion is present. This may also be the case in proximally fixed stems, as significant motion can occur between the distal portion of the stem and the surrounding bone.

Fretting and corrosion at modular junctions have been recently recognized as important potential sources of particulate debris (Fig. 5). This phenomenon has been described in femoral THA components with tapers and heads made of similar metals (cobalt-base alloy) as well as in tapers and heads made of the mixed-metal combination of a cobalt-base-alloy head on a titanium-base-alloy neck.[21] This process involves a number of variables, including the metallurgic state of the implants, the dimensions of the coupling, manufacturing tolerances, and taper geometry. It is believed that fretting initiates the process by removing passivating films. This in turn allows corrosion of the underlying metal surface.[21] Furthermore, we have shown that corrosion products formed at the head-neck junction can migrate to the joint pseudocapsule, the articular surface of the polyethylene insert, and the femoral interfacial membrane. In addition, these corrosion products can be found in osteolytic lesions within the femoral canal.

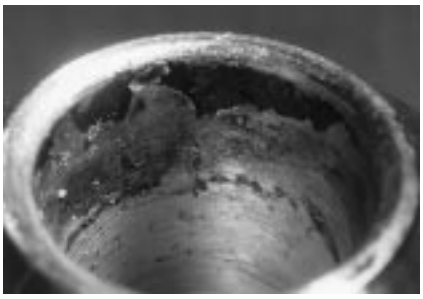


Fig. 5 Interior of the taper in a modular cobalt-base-alloy femoral head retrieved after 71 months in situ from a patient with femoral osteolysis. There is evidence of severe corrosive attack near the rim (original magnification X10).

In our investigations, a number of other particulate species have been recovered. These include silicates (remnants from the surface processing used to finish the metallic stems), the presence of which has been linked to excessive wear at the metallic counterface,[22] and stainless-steel particles (contaminants from the surgical instruments or debris from cerclage wires used to stabilize an intraoperative femoral fracture or a trochanteric osteotomy). While the significance of the corrosion products and stainless-steel and silicate particles has not been fully elucidated, they could potentially stimulate macrophages. In addition, these particles can migrate to the joint space and act as third bodies, thereby increasing polyethylene wear.

A major question regarding the pathogenesis of periprosthetic bone loss is related to the relative contribution of each of the particulate species to the overall process. In vitro cell-culture studies in our laboratories have demonstrated that the macrophage and fibroblast response to particulate debris is a function of particle size, composition, and dose. However, particles of different compositions may exhibit differential cytotoxicities[23] when introduced in a large bolus in cell-culture studies, precluding a direct comparison of their in vivo stimulatory effects. These issues are being studied by researchers at several centers, utilizing fabricated and/or retrieved particulate materials in cell cultures. In spite of incomplete knowledge, there is a growing consensus that polyethylene particles are the most biologically active, if for no other reason than that, by virtue of their sheer numbers and small size, they give rise to an enormous surface area for interaction with the surrounding tissues. A great deal of further research is required to resolve these issues.

Strategies for Prevention of Osteolysis

The basic strategy designed to address the problem of osteolysis should incorporate methods to decrease the periprosthetic particulate burden. Polyethylene wear remains the most serious and elusive problem. A number of factors govern the polyethylene wear rate, including femoral-head diameter and polyethylene thickness. Femoral heads with diameters of 32 mm have been associated with increased volumetric polyethylene wear; therefore, it is our current practice to use 28-mm heads. With smaller, metal-backed acetabular components (50 mm or less), the use of a 22-mm head becomes advisable to maintain a greater thickness of polyethylene.

Manufacturing flaws, such as fusion defects and foreign-body inclusions, have also been suggested as contributory to adverse polyethylene wear properties. These problems are currently being addressed with attention to polyethylene quality control and development of improved fabrication modalities.

Ceramic heads have been introduced as another method of decreasing polyethylene wear. While their performance clinically and in laboratory environments indicates that polyethylene wear can be decreased, the introduction of a ceramic head may pose additional problems—most significantly, fracture of the ceramic component. Furthermore, their benefit in the clinical setting has not been demonstrated conclusively.

The elimination of polyethylene is another approach being investigated clinically in various centers. With the realization that early problems may have been related to the design and not the articulation, there has been a renewed interest in the application of metal-metal bearings. In addition, ceramic-ceramic bearings have been used in Europe for over 10 years. However, approved clinical application of either wear couple in the United States is still several years away.

Metallic wear is also being addressed. Nitriding and nitrogen ion implantation have been introduced to decrease the potential for abrasive wear and fretting in titanium-alloy stems. This approach may also be of value in cobalt-alloy stems. Fabrication of metallic bearing surfaces with extremely low roughness can be expected to decrease articular wear rates. A polished metal head can be made as smooth as a ceramic head. Polishing of the stem will remove surface asperities and decrease particle generation from stem-bone fretting. In addition, polishing will minimize silicate contamination. For surgeons and manufacturers to continue to benefit from the advantages of modularity, a great deal of attention needs to be directed toward optimizing modular designs. Forthcoming design improvements in modular connections will address manufacturing tolerances, taper geometry, and metallurgic processing to minimize the incidence and severity of the mechanically assisted crevice corrosion process that has been demonstrated.[21] In general terms, increased modularity should be applied with caution.

Design improvements should also be taking place in acetabular prostheses. These should include improved tolerances between the polyethylene insert and the metal backing, improved surface finish on the metallic concave surfaces, secure locking mechanisms, and the avoidance of holes on the convex portion of the acetabular prosthesis.

Implant fixation is also an important variable. It is believed that more extensive circumferential porous coatings will improve fixation as well as reduce the likelihood of polyethylene transport to the distal portions of the femoral canal. Surgical technique has an important role in that initial rigid fixation will facilitate bone ingrowth (in uncemented applications) and thereby minimize motion between the bone and the implant. Meticulous cement technique to ensure an adequate cement mantle of uniform thickness is important to diminish the likelihood of cement-mantle defects, which can predispose to focal osteolysis. The surgeon also needs to pay careful attention to the intraoperative assembly of modular connections. This includes careful cleaning and drying of the head-neck or stem-sleeve couplings of femoral THA components, ensuring that polyethylene inserts used in THA and TKA are fully seated with locking mechanisms correctly engaged, and avoiding mismatch of modular components due to inappropriate

coupling of components from different manufacturers or improper sizing of components from a single manufacturer.

Summary

Wear in total joint replacement is a complex phenomenon with important clinical ramifications. Submicron particulate wear debris, especially polyethylene debris, appears to be central to the pathogenesis of osteolysis. Efforts are under way to limit the generation of polyethylene wear debris by improving the quality of the polyethylene, improving the bearing characteristics of the femoral head and/or condyle counterface, improving the stability of modular connections, avoiding large-diameter (more than 28 mm) femoral heads in THA, and avoiding excessively thin (less than 5 to 6 mm) polyethylene components in THA and TKA. Access of articular wear debris to remote locations may be limited by avoiding noncircumferential porous coatings in uncemented implants, holes in the metal backing of uncemented acetabular components, and cement-mantle defects.

Issues related to wear of prosthetic implant materials will continue to dominate efforts to improve the performance and longevity of total joint replacements. Both engineering and biologic advances are crucial in order to understand the mechanisms of particle generation from orthopaedic implant materials and to understand the host response to such particles. The clinician must be cognizant of these issues to be able to critically evaluate prosthetic design innovations before their widespread clinical application. Relatively long follow-up periods (5 to 10 years) may be required to determine the efficacy of some currently proposed improvements.

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