Theories of wear and loosening in hip prostheses
Wear-induced loosening vs loosening-induced wear—a review

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The observation of periprosthetic granulomas containing wear debris around apparently well-fixed as well as around loose-fitting prosthetic components has led to the development of the hypothesis of wear-induced loosening. However, the hypothesis of wear-induced loosening can neither explain the rapid early prosthetic migration detected by roentgen stereophotogrammetry nor the epidemiology of clinical failure without supplementary ad hoc assumptions. By contrast, apart from explaining the rapid early prosthetic migration detected by roentgen stereophotogrammetry, the theory of early loosening can explain the development of wear granulomas as well as to a great extent the epidemiology of clinical failure.

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The development of hip arthroplasty can be divided into two: before and after the introduction of bone cement. The Smith-Petersen mold arthroplasty, the Thompson, the Moore and the Judet hemiarthroplasties and the McKee total arthroplasty were all uncemented. Although early results were often good, late results were not so good due to pain from movements between prosthesis and bone and subsequent bone destruction.

With prosthetic fixation with bone cement, clinical results improved, the cemented hip arthroplasty became a success and it was thought that bone erosion would not occur unless the prosthesis became loose (McKee and Watson-Farrar 1966). However, initially apparently well-fixed prosthetic components were sometimes observed, after several years, to cause local bone resorption and subsequent clinical loosening. Wear particles, cellular reaction and granuloma formation were observed in the periprosthetic tissue and many authors claimed that late prosthetic loosening was due to a foreign-body reaction to wear particles from cement, metal, or polyethylene (Willert and Semlitsch 1977, Vernon-Roberts and Freeman 1977, D'Aubigné and Postel 1950). The early results were encouraging, but after a few years prosthetic failure and severe periprosthetic tissue reaction with concomitant loss of bone stock were recorded (d'Aubigné and Postel 1962).

As the cause of loosening is controversial, new prosthetic designs, materials and procedures have been introduced, often on uncertain theoretical grounds. In this review, the two current hypotheses of prosthetic loosening are presented and discussed: loosening as a result of foreign-body reaction to wear particles and loosening as a result of early prosthetic instability.

Wear and loosening of hip prostheses

Historical background
The Smith-Petersen Vitallium mold arthroplasty was designed to be loose-fitting (Smith-Petersen 1939). The clinical results were graded good or excellent in just over one half of the patients (Law 1962), but acetabular protrusion and gradual resorption of the femoral head both occurred frequently.

The Judet hemiprosthesis was made of polymethylmethacrylate and had a short stem that was designed to traverse the remainder of the neck and to penetrate the lateral cortex of the femur (Judit and Judet 1950). The early results were encouraging, but after a few years prosthetic failure and severe periprosthetic tissue reaction with concomitant loss of bone stock were recorded (d'Aubigné and Postel 1962).
et al. 1954, Mittelmeier and Singer 1956). A Judet hemi-
prosthesis made of nylon gave similar results (Levy et al. 1954).

In 1958 Charnley introduced the use of bone cement when he anchored a Thompson prosthesis within the femoral shaft. Polytetrafluoroethylene (Teflon®) was the first polymer material used for the acetabular component, which initially was unce-
mented (Charnley 1961). Polytetrafluoroethylene has a low coefficient of friction but poor wearing prop-
erties. The material was soon abandoned because of early prosthetic failures: abraded particles of polytet-
rafluoroethylene (in contrast to particles of polyethy-
lene) were found to produce giant-cell granulomas and voluminous masses of caseous debris (Charnley 1963).

The cementing of the acetabular component was introduced in 1960 by McKee (McKee and Watson-Farrar 1966), and the polyethylene acetabular component was introduced by Charnley in 1962. The high-wear resistance of the ultra-high molecular weight polyethylene made it possible to attain a low frictional torque by using a small (22 mm) diameter femoral head. However, in contrast to the cemented femoral component, a radiolucency commonly developed early around the cemented acetabular component. For a short period of time, therefore, Charnley used an uncemented press-fit acetabular component, but because of a high incidence of early clinical failure cementing was reintroduced in 1965 (Charnley 1979).

During the pioneering years much attention was directed towards wear resistance (Galante and Rostoker 1973, Weightman 1977). The Christiansen trunnion-bearing total hip prosthesis was introduced in 1970. The design included a polyacetal (polyoxy-
ethylene or Delrin®) coating positioned between the trunnion and the prosthetic head, a large (37 mm) diameter femoral head, and an acetabular component made of polyacetal. The major articulation was intended to occur in the trunnion joint and not between the prosthetic head and the acetabular com-
ponent. The objective was to reduce friction and wear (Christiansen 1974). However, the clinical results were poor and about one third of these prostheses implanted in Sweden during the 1970’s have been revised because of loosening (Ahnfelt et al. 1990). Common findings at revision were jamming of the trunnion joint (Alho et al. 1984), severe wear of the acetabular component (Sudmann et al. 1983), and periprosthetic giant-cell granulomas (Ohlin and Kindblom 1988). The prosthesis has been taken off the market.

The operative technique improved (removal of
articular cartilage from the acetabulum, preservation of subchondral bone in the acetabulum, drill-holes in the acetabulum, cortical support to the stem, i.e., removal of the cancellous bone in the femoral canal, plugging of the femoral canal, brushing, high-pres-
sure lavage, adequate cement filling, and more ex-
sen and Jensen 1985, Paterson et al. 1986, Mulroy and Harris 1990) and the cemented metal-on-poly-
mer hip arthroplasty thus became one of the most successful of all orthopedic operations. However, the results still deteriorated with time because of loosening.

The hypothesis of a wear product transporta-
tion equilibrium
Initially apparently well-fixed prosthetic components were sometimes observed after several years to cause local bone resorption, subsequently leading to clinical loosening. Wear particles (of cement, metal, polyethylene, etc.) and numerous macrophages and giant-cells were observed in the periprosthetic tissue obtained at revision surgery. Histological examinations demonstrated a correlation between the quan-
tity of wear particles and this foreign-body reaction. In cases of abundant foreign material extensive cell-
ular reaction with granuloma formation was seen. Wear particles were found to be transported to the lymph nodes draining the hip. The hypothesis of a wear product transportation equilibrium was (and still is) put forward (Willert and Semlitsch 1977, Vernon-Roberts and Freeman 1977, Willett et al. 1990b): if the quantity of wear products exceeds the transportation capacity, wear products should accumu-
late and fill the joint cavity with caseous debris. (For example, the large size of the wear particles of polytetrafluoroethylene was thought to interfere with transportation of these particles away from the joint (Charnley 1979).) The wear particles were claimed to cause cellular reaction resulting in infiltrating gran-
ulomas, periprosthetic bone resorption and eventi-
ally prosthetic loosening. Accordingly, the failures of the early Charnley and of the Christiansen prostheses were related to the poor wearing properties of poly-
tetrafluoroethylene and polyacetal, respectively, and to cellular reaction to the abraded particles.

Cement disease, metallosis, and plasticosis
Loosening of the cemented hip arthroplasty may be

Metallic ions and particles can be released by electrochemical corrosion, fretting corrosion and by wear. Increased levels of metallic ions in the urine or in the serum have been observed in patients with a metal-on-metal prosthesis (Coleman et al. 1973, Jones et al. 1975) and in patients with a loose prosthetic component (Dorr et al. 1990, Jacobs et al. 1991), and metal sensitivity as a cause of loosening has been suspected (Evans et al. 1974, Jones et al. 1975, Vernon-Roberts and Freeman 1977, Svensson et al. 1988, Lalor et al. 1991). Also, metallic particles have been suggested to cause local osteolysis and loosening (Vernon-Roberts and Freeman 1977, Pazzaglia et al. 1985, 1987). However, osteolysis has also been described around Judet hemiprostheses made of acrylic or nylon (d'Aubigné and Postel 1954, Levy et al. 1954, Mittelmeier and Singer 1956) and around uncemented all-polyethylene acetabular components (Wilson-MacDonald et al. 1990, Grigoris et al. 1993).

Thus, the role of polyethylene wear debris has recently been emphasized (Santavirta et al. 1990a, Willert et al. 1990b, Wilson-MacDonald et al. 1990, Fornasier et al. 1991, Cooper et al. 1992, Schmalzried et al. 1992, Grigoris et al. 1993). Indeed, it has been suggested that the polyethylene wear particles migrate along the intact bone-cement interface (Howie et al. 1988, Fornasier et al. 1991, Schmalzried et al. 1992). However, doubts about the role of polyethylene arise from observations of similar periprosthetic osteolysis seen in uncemented ceramic-on-ceramic prostheses (Mahoney and Dimon 1990, Borssén et al. 1991) and in uncemented metallic hemiprosthesis (Kozinn et al. 1986, Tallroth et al. 1989).

**Experimental and histological investigations**

The cellular reaction to foreign particles has been examined experimentally and studied histologically in periprosthetic tissue specimens obtained from hip arthroplasties. The results of several of these investigations are contradictory; significant reactions to particles of polymethylmethacrylate, metal, and polyethylene are reported, as well as no or almost no reaction. Thus, different authors have arrived at different conclusions about the importance of the material and size of the wear particles in loosening.

Cohen (1959) observed that coarse powders of cobalt-chrome alloy and stainless-steel both caused a minimal reaction when injected subcutaneously into rats, whereas a fine powder of cobalt-chrome alloy caused a mild reaction and a fine powder of stainless steel caused a moderate reaction. Disregarding the reactions to the individual materials, the variation in degree of reaction could be explained by increased total surface area and by increased corrosion of small particles.

Charnley (1963) injected subcutaneously two specimens of polytetrafluoroethylene (Teflon®) particles and one specimen of polyethylene particles into his own thigh. He discovered that the polytetrafluoroethylene (in contrast to the polyethylene) particles produced a transient systemic reaction and a chronic local inflammation, and suggested that this was probably due to the elusion of fluorides. (Analogously, polyacetal particles do release formaldehyde (Dumbleton 1979), which is locally irritating.)

Stinson (1964) found that polymethylmethacrylate, polyethylene and nylon particles inserted into muscle and into the knee-joint of guinea-pigs induced an extremely mild chronic inflammatory reaction, similar around each polymer, and suggested that factors other than the chemical composition of these implants determined the tissue response. Escalas et al. (1976), however, found that polyethylene particles implanted in muscle of rabbits caused a more pronounced inflammatory reaction.

Willert and Semlitsch (1977) observed wear particles and a foreign-body reaction in periprosthetic tissue specimens taken at revision surgery for failed cemented hip arthroplasty, and proposed the hypothesis of the wear product transportation equilibrium described above. If the quantity of wear products exceeded the transportation capacity, it was suggested that the wear products accumulated and caused cellular reaction resulting in infiltrating granulomas, periprosthetic bone resorption and eventually prosthetic loosening.

Linder et al. (1983) noticed concomitant bone resorption and bone formation at the bone-cement interface in failed total hip arthroplasties and suggested that the bone formation was an attempt by the
body to stabilize the prosthesis—which, however, failed in the presence of implant movement. Metallic or polyethylene wear particles were not always present in granulomatous tissue and the neighboring soft tissue membrane. The polymethylmethacrylate wear particles in the soft tissue membrane caused an extremely mild reaction and they concluded that the tissue reaction was caused by prosthetic instability.

Kozinn et al. (1986) examined the interface membrane from failed uncemented hip metallo-hemi-prostheses and found that the inflammation was mild in the absence of polymethylmethacrylate and polyethylene debris, even in the presence of moderate metallic debris. These findings were corroborated by Lennox et al. (1987) who examined the interface membrane from failed cemented and uncemented hip prostheses. Specimens from the cemented implant membrane were abundant in macrophages and giant-cells, mainly in areas adjacent to the bone surface as opposed to the cement surface. However, Lennox et al. suggested that the membrane developed as a response to polymethylmethacrylate particles (produced by micromovements).

Pazzaglia et al. (1987) examined periprosthetic tissue specimens obtained from failed cemented hip arthroplasties and observed cellular damage to occur only in those cells which phagocyted the smallest wear particles, i.e. metal particles. Cellular damage did not occur in those cells which phagocyted polymethylmethacrylate and polyethylene wear particles. They concluded that metal particles were the most harmful because of their small size.

Howie and Vernon-Roberts (1988) demonstrated that a single relatively high dose of cobalt-chrome particles injected into rat knee joints caused a synovitis similar to that seen in a failed arthroplasty, but they pointed out that the observed cellular reaction would not be expected in human periprosthetic tissue because of gradual release of wear particles in the latter. In another experiment, Howie et al. (1988) inserted a non-weight-bearing cement plug through the knee joint into the distal femur of the rat. They repeatedly injected polyethylene particles into the joint, beginning as early as 2 weeks after the cement plug implantation, and found that this caused osteolysis around the cement plug. They concluded that wear particles caused bone resorption by provoking macrophages and giant-cells to release osteoclast-stimulating factors. However, besides macrophages and giant-cells, particles of polyethylene also reached the bone-cement interface indicating an insufficient biological barrier (Bränemark et al. 1977). In Man this biological barrier develops within 5 months (Linder et al. 1988) and prevents the spreading of wear particles from the joint cavity into the bone-cement interface (Linder and Carlsson 1986). Thus, a probable explanation is that synovial fluid (and debris) was pressed into the bone-cement interface as a consequence of excessive cartilage detrition and abrasion, which must have occurred when the large quantity of polyethylene particles became trapped between the articular surfaces, damaged the cartilage and produced cartilaginous debris which in turn caused a severe secondary synovitis (George and Chrisman 1968, Boniface et al. 1988).

Goodman et al. (1988, 1990b) found that a single dose of polymethylmethacrylate or polyethylene particles injected into the bone marrow of the rabbit tibia caused a foreign-body reaction similar to that seen in a failed arthroplasty (whereas injected particles of titanium or cobalt-chrome alloy seemed to be inert (Goodman et al. 1990a)). However, as pointed out by Howie and Vernon-Roberts (1988), such a reaction would probably not occur in human peri-prosthetic tissue because of the gradual release of the wear particles.

Herman et al. (1989) concluded from an experimental study that both a large cement surface and cement powder caused macrophages to release osteoclast-stimulating factors. Murray and Rushton (1990), however, from another experimental study suggested that phagocytosis of any foreign particles (in contrast to the bulk material) will cause macrophage activation, provided there are enough particles.

Santavirta et al. (1990b, 1991b) examined peri-prosthetic tissue specimens taken from failed cemented and uncemented hip arthroplasties with and without granulomas. They observed active fibroblasts in "simple" loosening, but little fibroblast reaction in the granulomatous tissue. Metallic and polyethylene debris was not restricted to cases of granuloma formation. They suggested that the "granulomatosis" was a peculiar immunopathological entity different from "simple" loosening. In an experimental study, Santavirta et al. (1991a) found that polymethylmethacrylate powder is essentially an immunologically inert material. In contrast, Gil-Albarova et al. (1992) reported an activated immune response to polymethylmethacrylate in patients with loosening of cemented hip prosthesis, regardless of the presence of granulomas.

Fornasier et al. (1991) examined specimens retrieved at autopsy from patients with successful hip arthroplasty and observed areas with macrophages in the bone-cement interface in these apparently stable prosthetic components. They suggested a mechanism called directional exocytosis, i.e., a transcellular
transportation of wear particles to the leading edge of bone resorption, where the particles stimulated bone resorption.

Betts et al. (1992) found similar relative amounts of metallic elements in the periprosthetic tissue of failed cemented hip prostheses (made of cobalt-chrome alloy) as in the prostheses themselves. They found some prostheses with very little metallic debris. These findings argue against electrochemical corrosion, and the authors concluded that much of the metallic debris seen at revision was generated after the prosthesis became significantly loose. They detected polymethylmethacrylate and polyethylene particles in only half of the periprosthetic tissue sections.

Lee et al. (1992) discovered no difference in the size of metallic particles in perarticulare tissue specimens obtained from failed cemented hip prostheses made of titanium-alloy, cobalt-chrome alloy and stainless steel. They concluded that an increased rate of early failure of titanium alloys cannot be explained by the size of the metallic particles. They noticed larger mean size of polyethylene particles from titanium-alloy prostheses than from either cobalt-chrome alloy or stainless-steel alloy prostheses.

Schmalzried et al. (1992) examined specimens retrieved at autopsy from cemented acetabular components. They found a soft-tissue layer present only at the periphery of apparently stable acetabular components, an intimate contact between bone and cement in central regions at the acetabular dome, and a transition zone containing macrophages and polyethylene particles (but no polymethylmethacrylate particles). They assumed that late loosening of the acetabular component is biological in nature (as opposed to mechanical loosening of the femoral component); bone resorption developing at the transition zone and progressing along the bone-cement interface towards the dome was suggested to occur as a result of the macrophage reaction induced by small polyethylene particles spontaneously migrating toward the leading edge of bone resorption.

**Epidemiologic observations**


These correlations should be explained by the theory of prosthetic loosening. Many of these correlations cannot be accounted for by the hypothesis of wear particles causing prosthetic loosening by foreign-body reaction without it being supplemented by ad hoc-assumptions, such as the assumption of a peculiar immunological reaction (Santavirta et al. 1990b, 1991b), the assumption of transcellular transportation of wear particles to the leading edge of bone resorption in stable prosthetic components (Fornasier et al. 1991), and the assumption of biological loosening of the acetabular component as opposed to mechanical loosening of the femoral component (Schmalzried et al. 1992).

**The theory of early loosening**

**Early loosening due to insufficient initial fixation or due to early loss of fixation**

Using roentgen stereophotogrammetry prosthetic migration can be detected in many asymptomatic hips as early as 4 months postoperatively (Figure 1). However, many of these migrating prosthetic components may appear well-fixed for a long period of time at radiographic examination. If loosening does occur, it is probably initiated at an early stage: insufficient initial fixation because of inadequate interlock or because of weak cancellous bone bed and early loss of fixation due to resorption of a layer of heat-injured bone both cause prosthetic instability and progressive bone resorption (Mjöberg 1991). Indeed, early signs of loosening can often be detected at conventional radiography (Paterson et al. 1986, Hodgkinson et al. 1993).

**Loosening may cause granulomas**

Although intimate contact between bone and cement has recently been demonstrated in the acetabulum, bone resorption commonly develops circumferentially at the intra-articular margin and progresses along the bone-cement interface towards the dome of the acetabular component (Schmalzried et al. 1992).
In cases of poor initial interlock at the cement-bone interface (i.e., inefficient filling of cement), micromovements will cause damage or necrosis of a layer of bone tissue, as will a heat injury of the bone. The injured bone tissue will act as a stimulus for osteoclast recruitment and activation (Chambers 1980, Vaes 1988) and subsequent bone resorption will undermine the cement. However, the most common cause of loosening of the cemented femoral component in primary hip arthroplasties, besides inadequate filling of cement, is residual weak cancellous bone in the femoral canal. Whichever of these is the cause, the cement will fracture (“debonding” of the stem) if it is not adequately supported by cortical bone (Markolf and Amstutz 1976, Weinans et al. 1990, Jasty et al. 1991b). Then, joint fluid (and wear debris) may be pumped under high pressure, by prosthetic micromovements, from the gap between the stem and the cement through the defect in the cement mantle into the interface, where it will interfere with the perfusion and oxygenation of bone (Anthony et al. 1990). The ejected joint fluid will be partially resorbed and the inspissated wear debris (and bone detritus) may be invaded by granulation tissue (Landells 1953). This explains the high concentration of wear debris found in these granulomatous lesions (Hu et al. 1992). Eventually a growing granuloma may jeopardize the strength of the femur.

Bone ingrowth into some porous-coated hip-prosthetic components has been found at histological examination (Brooker and Collier 1984). Engh et al. 1987, Cook et al. 1988). This suggests ossointegration of the uncemented implant. However, bone ingrowth has been reported in prostheses despite progressive migration and even clinical failure (Cook et al. 1988, Jasty et al. 1991a). Thus, the reliability of evaluating prosthetic fixation histologically is doubtful. A prerequisite for bone ingrowth into an implant is initial stability, only extremely small movements being tolerated (Bränemark et al. 1977, Perren 1984, Pilliar et al. 1986). At roentgen stereophotogrammetry all fully-threaded acetabular components (Snorrason and Kährholm 1990), half of the porous-coated acetabular components fixed with screws (Kährholm and Snorrason 1992) and almost all uncemented femoral components (Wykman et al. 1988, Nistor et al. 1991, Kährholm and Snorrason 1993) exhibited migration within 1–2 years. Thus, the focal osteolysis reported in some uncemented acetabular (Sanavirta et al. 1990a, Wilson-MacDonald et al. 1990, Cooper et al. 1992, Grigoris et al. 1993) and in some uncemented femoral (Kozinn et al. 1986, Lombardi et al. 1989, Tallroth et al. 1989, Maloney et al. 1990, Sanavirta et al. 1990a, Borssén et al. 1991, Cooper 1990).
et al. 1992, Tanzer et al. 1992, Grigoris et al. 1993) components is probably a consequence of insufficient initial fixation of the prosthesis to the bone (or of the polyethylene liner to the metal shell in some uncemented metal-backed acetabular components (Maloney et al. 1993)). The similarity of the osteolytic lesions in cemented and uncemented arthroplasties suggests a similar pumping mechanism.

Secondary factors influencing the process of loosening

According to the theory of early loosening the time lag to clinical failure of different migrating prosthetic components is (apart from by varying degrees of early prosthetic instability) dependent on the degree of stress during normal human activity. This is variable in different patients because of differing body weight and level of physical activity. It is also variable for different components because of differing prosthetic design, positioning, friction and wear. For example, the femoral component is exposed to greater torsional stress than the acetabular component during walking and, especially, when climbing stairs and rising from a chair. Thus, slightly loose femoral components can be expected to develop larger micromovements and result in earlier clinical failure than slightly loose acetabular components (Figure 1).

Loosening may cause excessive wear

Acetabular component migration has been found to be related to the depth of acetabular wear (Buchhorn et al. 1984, Wroblewski 1986, Garfa-Cimbrelo and Manuera 1992) indicating a causal relationship. This correlation can be explained as a consequence of prosthetic instability (as opposed to representing a reaction to wear debris). An unstable prosthetic component pumps joint fluid (and wear debris) in and out of the interfaces during walking (Charnley 1961, Gruen et al. 1979, Wroblewski et al. 1987). When the prosthetic micromovements have caused cement fragmentation, cement particles are pumped into the joint cavity and cause excessive wear (Revell et al. 1978, Franzén and Mjöberg 1990, McKellop et al. 1990).

The frequent failure of the Christiansen prosthesis (Ahnfelt et al. 1990), the often severe wear of its polyacetal acetabular component (Sudmann et al. 1983) and the common periprosthetic giant-cell granulomas (Ohlin and Kindblom 1988) may suggest that wear debris is a factor leading to osteolysis and clinical failure. However, polyacetal has a higher frictional coefficient than polyethylene (Mathiesen et al. 1986); the higher torque of a polyacetal acetabular component compared with a polyethylene acetabular component can be expected to cause larger micromovements of a slightly loose acetabular component. Once prosthetic micromovements have caused cement fragmentation and the cement particles have been pumped into the joint cavity, the particles may cause excessive wear. In the initially slightly loose Christiansen acetabular component both increased friction and eccentricity contribute to the high torque and early failure.

Similarly, the early failure and severe periprosthetic tissue reaction of the uncemented polytetrafluoroethylene acetabular component (Charnley 1963) may be due to prosthetic loosening, at least in part. Elusion of fluorides from polytetrafluoroethylene particles may aggravate local inflammation (as may the release of formaldehyde from polyacetal particles (Dumbleton 1979)) but the production of particles is probably mainly secondary to loosening.

Metal sensitivity

The risk of metal sensitivity after metal-on-polyethylene hip arthroplasty seems to be very small (Carlsson et al. 1980, Pazzaglia et al. 1986) and even a manifest metal sensitivity probably does not cause prosthetic loosening (Carlsson and Möller 1989). Well fixed metal-on-polyethylene prostheses made of stainless steel, cobalt-chromium alloy, and titanium alloy exhibit low metal levels in the synovial fluid, whereas loosening may cause release of large amounts of metal into synovial fluid and surrounding tissues (Dorr et al. 1990, Brien et al. 1992). Thus, the metal sensitivity associated with prosthetic loosening is probably a result of loosening (Elves et al. 1975, Brown et al. 1977).

Conclusion

Loosening of hip prostheses can be explained as a result of an early prosthetic instability (due to insufficient initial fixation or early loss of fixation), where certain risk factors (such as body weight, physical activity, varus/valgus position, prosthetic neck length, etc.) have a secondary influence on the process of loosening. The theory of early loosening can explain both the rapid early prosthetic migration detected by roentgen stereophotogrammetry and to a great extent the epidemiology of clinical failure. This
is not true of the hypothesis of wear particles causing prosthetic loosening by foreign-body reaction, without it being supplemented by ad hoc assumptions.

References


