Theories of wear and loosening in hip prostheses

Wear-induced loosening vs loosening-induced wear—a review

Bengt Mjöberg

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.

Department of Orthopedics, Uppsala University Hospital, S-751 85 Uppsala, Sweden
Tel +46-18 66 30 00. Fax -18 50 94 27
Submitted 94-04-14. Accepted 94-05-09

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, Jones and Hungerford 1987, Pazzaglia et al. 1987, Goodman et al. 1988, Howie and Vernon-Roberts 1988, Howie et al. 1988, Santavirta et al. 1990a, Willert et al. 1990a, 1990b, Fornasier et al. 1991, Cooper et al. 1992, Schmalzried et al. 1992, Maloney et al. 1993).

An alternative explanation is that loosening results from early prosthetic instability (due to insufficient initial fixation or early loss of fixation), and that "late loosening" is the result of late detection rather than of late occurrence of loosening (Mjöberg 1991).

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 (Judet 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

1954, Mittelmeier and Singer 1956). A Judet hemiprosthesis 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. Polytetrafluorethylene (Teflon®) was the first polymer material used for the acetabular component, which initially was uncemented (Charnley 1961). Polytetrafluorethylene has a low coefficient of friction but poor wearing properties. The material was soon abandoned because of early prosthetic failures; abraded particles of polytetrafluorethylene (in contrast to particles of polyethylene) 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 (polyoxymethylene 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 component. 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-pressure lavage, adequate cement filling, and more experienced surgeons) (Andersson et al. 1972, Volz and Wilson 1977, Beckenbaugh and Ilstrup 1978, Halawa et al. 1978, Charnley 1979, Carlsson and Gentz 1980, Eftekhar and Pawluk 1980, Harris et al. 1982, Krause et al. 1982, Carter et al. 1983, Kristiansen and Jensen 1985, Paterson et al. 1986, Mulroy and Harris 1990) and the cemented metal-on-polymer 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 transportation 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 quantity of wear particles and this foreign-body reaction. In cases of abundant foreign material extensive cellular 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, Willert et al. 1990b): if the quantity of wear products exceeds the transportation capacity, wear products should accumulate and fill the joint cavity with caseous debris. (For example, the large size of the wear particles of polytetrafluorethylene 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 granulomas, periprosthetic bone resorption and eventually prosthetic loosening. Accordingly, the failures of the early Charnley and of the Christiansen prostheses were related to the poor wearing properties of polytetrafluorethylene and polyacetal, respectively, and to cellular reaction to the abraded particles.

Cement disease, metallosis, and plasticosis

Loosening of the cemented hip arthroplasty may be associated with a considerable osteolysis (Harris et al. 1976, Carlsson et al. 1983, Scott et al. 1985, Jones and Hungerford 1987, Huddleston 1988, Tallroth et al. 1989, Willert et al. 1990a). The biomechanical properties of the cement were thought to be responsible, and the phenomenon was called cement disease (Jones and Hungerford 1987). Similar osteolysis has, however, also been seen in failed uncemented hip prostheses; in prostheses made of cobalt-chromium alloy (Brown and Ring 1985, Buchert et al. 1986, Mahoney and Dimon 1990, Maloney et al. 1990, Santavirta et al. 1990a, Borssén et al. 1991, Cooper et al. 1992, Grigoris et al. 1993) as well as in those made of titanium alloy (Agins et al. 1988, Lombardi et al. 1989, Maloney et al. 1990, Tanzer et al. 1992).

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 hemiprostheses (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 polytetrafluorethylene (Teflon®) particles and one specimen of polyethylene particles into his own thigh. He discovered that the polytetrafluorethylene (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 metallic hemiprostheses 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 phagocytosed the smallest wear particles, i.e. metal particles. Cellular damage did not occur in those cells which phagocytosed 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 (Brånemark 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 periprosthetic 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 periprosthetic 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 periarticular 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

In clinical research, correlations have been demonstrated between hip prosthetic loosening and demographic and physiological variables (age, gender, body weight, physical activity, index diagnosis, hip dysplasia, protrusio acetabuli, width of femoral canal, and previous hip surgery), operative technique (see the paragraph on operative technique in "Historical background" above), prosthetic design (size of prosthetic components, degree of eccentricity of the acetabular component, and neck length of the femoral component), positioning (containment of the acetabular component and varus/valgus position of the stem), friction and wear (Tönnis and Asai 1976, Beckenbaugh and Ilstrup 1978, Carlsson and Gentz 1980, Ramadier et al. 1980, Sutherland et al. 1982, Buchhorn et al. 1984, Kristiansen and Jensen 1985, Mathiesen et al. 1986, Paterson et al. 1986, Wroblewski 1986, Johnsson et al. 1988, Morrey and Ilstrup 1989, Ahnfelt et al. 1990, Garía-Cimbrelo and Manuera 1992).

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).

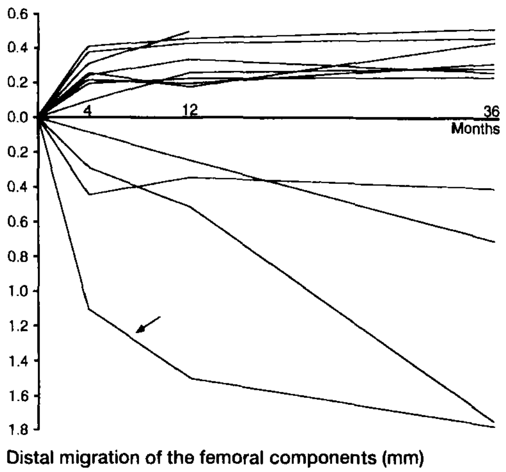

Figure 1. Migration along the longitudinal axis of the migrating 8/16 acetabular (above) and 4/14 femoral (below) components followed by roentgen stereophotogrammetry for 3 years (Two femoral components excluded because of inadequate bone marking with tantalum balls). Arrow indicates the only femoral component associated with slight pain initially at weight-bearing. Note that 8/16 acetabular and 10/14 femoral components did not migrate (significance limit = 0.2 mm) during the observation period; and that in all the cases but two (one acetabular and one femoral), the migration was seen within 4 months after surgery. (Redrawn with permission from *Acta Orthop Scand* 1990; 61: 273–4).

However, this intimate contact does not exclude the possibility of prosthetic wobbling micromovements around an axis near the dome of the acetabular component (Perren 1984). These micromovements will cause joint fluid to be pumped in and out of the prosthetic interfaces, and the resulting pressure waves may interfere with perfusion and oxygenation of bone (Franzén et al. 1993). The layer of devitalized bone thus formed is resorbed by osteoclasts (Chambers 1980, Vaes 1988) and the cement is gradually undermined. The micromovements transport joint fluid and wear particles to the leading edge of bone resorption, where the smallest particles penetrate furthest. This bone resorption may expand locally (Pierson and Harris 1993) and eventually joint fluid may force it way through the acetabulum into the pelvis (Hattrup et al. 1988, Reigstad and Røkkum 1992). The expelled joint fluid (containing wear debris and bone detritus) will be partially resorbed and may be invaded by granulation tissue (Landells 1953).

In the cemented hip arthroplasty for arthrosis most femoral components are well-fixed, in contrast to the acetabular components (Charnley 1979, Utz et al. 1986, Mjöberg et al. 1990, Mulroy and Harris 1990). 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 (Huo 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 osseointegration 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 (Brånemark et al. 1977, Perren 1984, Pilliar et al. 1986). At roentgen stereophotogrammetry all fully-threaded acetabular components (Snorrason and Kärrholm 1990), half of the porous-coated acetabular components fixed with screws (Kärrholm and Snorrason 1992) and almost all uncemented femoral components (Wykman et al. 1988, Nistor et al. 1991, Kärrholm and Snorrason 1993) exhibited migration within 1–2 years. Thus, the focal osteolysis reported in some uncemented acetabular (Santavirta 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, Santavirta et al. 1990a, Borssén et al. 1991, Cooper 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). Similarly, differences in prosthetic neck length, varus/valgus position, frictional torque, and acetabular component eccentricity (due to design or wear) have a secondary influence on the process of loosening.

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, Garía-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 polytetrafluorethylene acetabular component (Charnley 1963) may be due to prosthetic loosening, at least in part. Elusion of fluorides from polytetrafluorethylene 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

Agins H J, Alcock N W, Bansal M, Salvati E A, Wilson P D, Pellicci P M, Bullough P G. Metallic wear in failed titanium-alloy total hip replacements. J Bone Joint Surg (Am) 1988; 70 (3): 347–56.

Ahnfelt L, Herberts P, Malchau H, Andersson G B J. Prognosis of total hip replacement. A Swedish multicenter study of 4664 revisions. Acta Orthop Scand 1990; 61 (Suppl 238): 1–26.

Alho A, Søreide O, Bjersand A J. Mechanical factors in loosening of Christiansen and Charnley arthroplasties. Acta Orthop Scand 1984; 55: 261–6.

Andersson G B J, Freeman M A R, Swanson S A V. Loosening of the cemented acetabular cup in total hip replacement. J Bone Joint Surg (Br) 1972; 54 (4): 590–9.

Anthony P P, Gie G A, Howie C R, Ling R S M. Localised endosteal bone lysis in relation to the femoral components of cemented total hip arthroplasties. J Bone Joint Surg (Br) 1990; 72 (6): 971–9.

Beckenbaugh R D, Ilstrup D M. Total hip arthroplasty. A review of three hundred and thirty-three cases with long follow-up. J Bone Joint Surg (Am) 1978; 60 (3): 306–13.

Betts F, Wright T, Salvati E A, Boskey A, Bansal M. Cobalt-alloy metal debris in periarticular tissues from total hip revision arthroplasties. Metal contents and associated histologic findings. Clin Orthop 1992; 276: 75–82.

Boniface R J, Cain P R, Evans C H. Articular responses to purified cartilage proteoglycans. Arthritis Reuma 1988; 31 (2): 258–66.

Borssén B, Kärrholm J, Snorrason F. Osteolysis after ceramic-on-ceramic hip arthroplasty. A case report. Acta Orthop Scand 1991; 62 (1): 73–5.

Brien W W, Salvati E A, Betts F, Bullough P, Wright T, Rimnac C, Buly R, Garvin K. Metal levels in cemented total hip arthroplasty. A comparison of well-fixed and loose implants. Clin Orthop 1992; 276: 66–74.

Brooker A F Jr., Collier J P. Evidence of bone ingrowth into a porous-coated prosthesis. A case report. J Bone Joint Surg (Am) 1984; 66 (4): 619–21.

Brown G C, Lockshin M D, Salvati E A, Bullough P G. Sensitivity to metal as a possible cause of sterile loosening after cobalt-chromium total hip-replacement arthroplasty. J Bone Joint Surg (Am) 1977; 59 (2): 164–8.

Brown I W, Ring P A. Osteolytic changes in the upper femoral shaft following porous-coated hip replacement. J Bone Joint Surg (Br) 1985; 67 (2): 218–21.

Brånemark P-I, Hansson B O, Adell R, Breine U, Lindström J, Hallén O, Öhman A. Osseointegrated implants in the treatment of the edentulous jaw. Experience from a 10-year period. Scand J Plast Reconstr Surg 1977; 11 (Suppl 16): 1–132.

Buchert P K, Vaughn B K, Mallory T H, Engh C A, Bobyn J D. Excessive metal release due to loosening and fretting of sintered particles on porous-coated hip prostheses. Report of two cases. J Bone Joint Surg (Am) 1986; 68 (4): 606–9.

Buchhorn U, Willert H-G, Semlitsch M, Weber H. Dimensionsänderungen der Polyäthylen-Hüftpfannen bei Müller-Hüftendoprothesen. Ein Bericht über die Meßmethoden und ihre klinische Bedeutung. Z Orthop 1984; 122 (2): 127–35.

Carlsson Å, Möller H. Implantation of orthopaedic devices in patients with metal allergy. Acta Derm Venerol 1989; 69: 62–66.

Carlsson Å S, Gentz C-F. Mechanical loosening of the femoral head prosthesis in the Charnley total hip arthroplasty. Clin Orthop 1980; 147: 262–70.

Carlsson Å S, Gentz C-F, Linder L. Localized bone resorption in the femur in mechanical failure of cemented total hip arthroplasties. Acta Orthop Scand 1983; 54 (3): 396–402.

Carlsson Å S, Magnusson B, Möller H. Metal sensitivity in patients with metal-to-plastic total hip arthroplasties. Acta Orthop Scand 1980; 51 (1): 57–62.

Carter D R, Vasu R, Harris W H. Periacetabular stress distributions after joint replacement with subchondral bone retention. Acta Orthop Scand 1983; 54: 29–35.

Chambers T J. The cellular basis of bone resorption. Clin Orthop 1980; 151: 283–93.

Charnley J. Arthroplasty of the hip. A new operation. Lancet 1961; i: 1129–32.

Charnley J. Tissue reactions to polytetrafluorethylene. Lancet 1963; ii: 1379.

Charnley J. Low friction arthroplasty of the hip. Theory and practice. Springer Verlag, Berlin, Heidelberg, New York 1979: 25–40.

Christiansen T. A combined endo- and total hip prosthesis with trunnion-bearing. The Christiansen prosthesis. Acta Chir Scand 1974; 140: 185–8.

Cohen J. Assay of foreign-body reaction. J Bone Joint Surg (Am) 1959; 41 (1): 152–66.

Coleman R F, Herrington J, Scales J T. Concentration of wear products in hair, blood, and urine after total hip replacement. Br Med J 1973; 1: 527–9.

Cook S D, Barrack R L, Thomas K A, Haddad R J. Quantitative analysis of tissue growth into human porous total hip components. J Arthroplasty 1988; 3 (3): 249–62.

Cooper R A, McAllister C M, Borden L S, Bauer T W. Polyethylene debris-induced osteolysis and loosening in uncemented total hip arthroplasty. A cause of late failure. J Arthroplasty 1992; 7 (3): 285–90.

d'Aubigné R M, Postel M. Functional results of hip arthroplasty with acrylic prostheses. J Bone Joint Surg (Am) 1954; 36 (3): 451–75.

Dorr L D, Bloebaum R, Emmanual J, Meldrum R. Histologic, biochemical, and ion analysis of tissue and fluids retrieved during total hip arthroplasty. Clin Orthop 1990; 261: 82–95.

Dumbleton J H. Delrin as a material for joint prostheses—a review. In: Corrosion and degradation of implant materials. ASTM STP 684 (Ed. Syrett B C, Acharya A). American Society for Testing and Materials, Philadelphia 1979: 41–60.

Eftekhar N S, Pawluk R J. Role of surgical preparation in acetabular cup fixation. Hip 1980: 308–28.

Elves M W, Wilson J N, Scales J T, Kemp H B S. Incidence of metal sensitivity in patients with total joint replacements. Br Med J 1975; 4: 376–8.

Engh C A, Bobyn J D, Glassman A H. Porous-coated hip replacement. The factors governing bone ingrowth, stress shielding, and clinical results. J Bone Joint Surg (Br) 1987; 69 (1): 45–55.

Escalas F, Galante J, Rostoker W, Coogan P. Biocompatibility of materials for total joint replacement. J Biomed Mater Res 1976; 10: 175–95.

Evans E M, Freeman M A R, Miller A J, Vernon-Roberts B. Metal sensitivity as a cause of bone necrosis and loosening of the prosthesis in total joint replacement. J Bone Joint Surg (Br) 1974; 56 (4): 626–42.

Fornasier V, Wright J, Seligman J. The histomorphologic and morphometric study of asymptomatic hip arthroplasty. A postmortem study. Clin Orthop 1991; 271: 272–82.

Franzén H, Mjöberg B. Wear and loosening of the hip prosthesis. A roentgen stereophotogrammetric 3-year study of 14 cases. Acta Orthop Scand 1990; 61 (6): 499–501.

Franzén H, Mjöberg B, Rydholm U. Metal backing improves the survival of surface replacement of the hip. Arch Orthop Trauma Surg 1993; 112 (6): 257–9.

Galante J O, Rostoker W. Wear in total hip prostheses. An experimental evaluation of candidate materials. Acta Orthop Scand 1973; 44 (Suppl. 145): 3–46.

García-Cimbrelo E, Manuera L. Early and late loosening of the acetabular cup after low-friction arthroplasty. J Bone Joint Surg (Am) 1992; 74 (8): 1119–29.

George R C, Chrisman O D. The role of cartilage polysaccharides in osteoarthritis. Clin Orthop 1968; 57: 259–65.

Gil-Albarova J, Lacleriga A, Barrios C, Cañadell J. Lymphocyte response to polymethylmethacrylate in loose total hip prostheses. J Bone Joint Surg (Br) 1992; 74 (6): 825–30.

Goodman S B, Fornasier V L, Kei J. The effects of bulk versus particulate polymethylmethacrylate on bone. Clin Orthop 1988; 232: 255–62.

Goodman S B, Fornasier V L, Lee J, Kei J. The effects of bulk versus particulate titanium and cobalt chrome alloy implanted into the rabbit tibia. J Biomed Mat Res 1990a; 24 (11): 1539–49.

Goodman S B, Fornasier V L, Lee J, Kei J. The histological effects of the implantation of different sizes of polyethylene particles in the rabbit tibia. J Biomed Mat Res 1990b; 24 (4): 517–24.

Grigoris P, Roberts P, McMinn D J W. Failure of uncemented polyethylene acetabular components. J Arthroplasty 1993; 8 (4): 433–7.

Gruen T A, McNeice G M, Amstutz H C. "Modes of failure" of cemented stem-type femoral components. A radiographic analysis of loosening. Clin Orthop 1979; 141: 17–27.

Halawa M, Lee A J C, Ling R S M, Vangala S S. The shear strength of trabecular bone from the femur, and some factors affecting the shear strength of the cement-bone interface. Arch Orthop Trauma Surg 1978; 92 (1): 19–30.

Harris W H, McCarty J C Jr., O'Neill D A. Femoral component loosening using contemporary techniques of femoral cement fixation. J Bone Joint Surg (Am) 1982; 64 (7): 1063–7.

Harris W H, Schiller A L, Scholler J M, Freiberg R A, Scott R. Extensive localized bone resorption in the femur following total hip replacement. J Bone Joint Surg (Am) 1976; 58 (5): 612–8.

Hattrup S J, Bryan R S, Gaffey T A, Stanhope C R. Pelvic mass causing vesical compression after total hip arthroplasty. Case report. Clin Orthop 1988; 227: 184–9.

Herman J H, Sowder W G, Anderson D, Appel A M, Hopson C N. Polymethylmethacylate-induced release of bone-resorbing factors. J Bone Joint Surg (Am) 1989; 71 (10): 1530–41.

Hodgkinson J P, Maskell A P, Paul A, Wroblewski B M. Flanged acetabular components in cemented Charnley hip arthroplasty. Ten-year follow-up of 350 patients. J Bone Joint Surg (Br) 1993; 75 (3): 464–7.

Howie D W, Vernon-Roberts B. The synovial response to intraarticular cobalt-chrome wear particles. Clin Orthop 1988; 232: 244–54.

Howie D W, Vernon-Roberts B, Oakeshott R, Manthey B. A rat model of resorption of bone at the cement-bone interface in the presence of polyethylene wear particles. J Bone Joint Surg (Am) 1988; 70 (2): 257–63.

Huddleston H D. Femoral lysis after cemented hip arthroplasty. J Arthroplasty 1988; 3 (4): 285–97.

Huo M H, Salvati E A, Lieberman J R, Betts F, Bansal M. Metallic debris in femoral endosteolysis in failed cemented hip arthroplasties. Clin Orthop 1992; 276: 157–68.

Jacobs J J, Skipor A K, Black J, Urban R M, Galante J O. Release and excretion of metal in patients who have a total hip-replacement component made of titanium-base alloy. J Bone Joint Surg (Am) 1991; 73 (10): 1475–86.

Jasty M, Bragdon C R, Maloney W J, Haire T, Harris W H. Ingrowth of bone in failed fixation of porous-coated femoral components. J Bone Joint Surg (Am) 1991a; 73 (9): 1331–7.

Jasty M, Maloney W J, Bragdon C R, O'Connor D O, Haire T, Harris W H. The initiation of failure in cemented femoral components of hip arthroplasty. J Bone Joint Surg (Br) 1991b; 73 (4): 551–8.

Johnsson R, Thorngren K-G, Persson B M. Revision of total hip replacement for primary osteoarthritis. J Bone Joint Surg (Br) 1988; 70 (1): 56–62.

Jones D A, Lucas K, O'Driscoll M, Price C H G, Wibberley B. Cobalt toxicity after McKee hip arthroplasty. J Bone Joint Surg (Br) 1975; 57 (3): 289–96.

Jones L C, Hungerford D S. Cement disease. Clin Orthop 1987; 225: 192–206.

Judet J, Judet R. The use of an artificial femoral head for arthroplasty of the hip joint. J Bone Joint Surg (Br) 1950; 32 (2): 166–73.

Kozinn S C, Johanson N A, Bullough P G. The biologic interface between bone and cementless femoral endoprostheses. J Arthroplasty 1986; 1 (4): 249–59.

Krause W R, Krug W, Miller J. Strength of the cement-bone interface. Clin Orthop 1982; 163: 290–9.

Kristiansen B, Jensen J S. Biomechanical factors in loosening of the Stanmore hip. Acta Orthop Scand 1985; 56 (1): 21–4.

Kärrholm J, Snorrason F. Migration of porous coated acetabular prostheses fixed with screws. Roentgen stereophotogrammetric analysis. J Orthop Res 1992; 10 (6): 826–35.

Kärrholm J, Snorrason F. Subsidence, tip and hump micromovements of noncoated ribbed femoral prostheses. Clin Orthop 1993; 287: 50–60.

Lalor P A, Revell P A, Gray A B, Wright S, Railton G T, Freeman M A R. Sensitivity to titanium. A cause of implant failure? J Bone Joint Surg (Br) 1991; 73 (1): 25–8.

Landells J W. The bone cysts of osteoarthritis. J Bone Joint Surg (Br) 1953; 35 (4): 643–9.

Law W A. Late results in vitallium-mold arthroplasty of the hip. J Bone Joint Surg (Am) 1962; 44 (8): 1497–1517.

Lee J-M, Salvati E A, Betts F, DiCarlo E F, Doty S B, Bullough P G. Size of metallic and polyethylene debris particles in failed cemented total hip replacements. J Bone Joint Surg (Br) 1992; 74 (3): 380–4.

Lennox D W, Schofield B H, McDonald D F, Riley L H Jr. A histologic comparison of aseptic loosening of cemented, press-fit, and biologic ingrowth prostheses. Clin Orthop 1987; 225: 171–91.

Levy L J, Lipscomb C P, McDonald H C. Complications of Judet arthroplasty due to foreign-body reaction to nylon prostheses. J Bone Joint Surg (Am) 1954; 36 (6): 1175–80.

Linder L, Carlsson Å, Marsal L, Bjursten L M, Brånemark P-I. Clinical aspects of osseointegration in joint replacement. A histological study of titanium implants. J Bone Joint Surg (Br) 1988; 70 (4): 550–5.

Linder L, Carlsson Å S. The bone-cement interface in hip arthroplasty. A histologic and enzyme study of stable components. Acta Orthop Scand 1986; 57 (6): 495–500.

Linder L, Lindberg L, Carlsson Å. Aseptic loosening of hip prostheses. A histologic and enzyme histochemical study. Clin Orthop 1983; 175: 93–104.

Lombardi A V Jr., Mallory T H, Vaughn B K, Drouillard P. Aseptic loosening in total hip arthroplasty secondary to osteolysis induced by wear debris from titanium-alloy modular femoral heads. J Bone Joint Surg (Am) 1989; 71 (9): 1337–42.

Mahoney O M, Dimon J H III. Unsatisfactory results with a ceramic total hip prosthesis. J Bone Joint Surg (Am) 1990; 72 (5): 663–71.

Maloney W J, Jasty M, Harris W H, Galante J O, Callaghan J J. Endosteal erosion in association with stable uncemented femoral components. J Bone Joint Surg (Am) 1990; 72 (7): 1025–34.

Maloney W J, Peters P, Engh C A, Chandler H. Severe osteolysis of the pelvis in association with acetabular replacement without cement. J Bone Joint Surg (Am) 1993; 75 (11): 1627–35.

Markolf K L, Amstutz H C. A comparative experimental study of stresses in femoral total hip replacement components. The effects of prostheses orientation and acrylic fixation. J Biomech 1976; 9 (2): 73–9.

Mathiesen E B, Lindgren U, Reinholt F P, Sudmann E. Wear of the acetabular socket. Comparison of polyacetal and polyethylene. Acta Orthop Scand 1986; 57 (3): 193–6.

McKee G K, Watson-Farrar J. Replacement of arthritic hips by the McKee-Farrar prosthesis. J Bone Joint Surg (Br) 1966; 48 (2): 245–59.

McKellop H A, Sarmiento A, Schwinn C P, Ebramzadeh E. In vivo wear of titanium-alloy hip prostheses. J Bone Joint Surg (Am) 1990; 72 (4): 512–7.

Mittelmeier H, Singer L. Anatomische und histologische Untersuchungen von Arthroplastgelenken mit Plexiglas-Endoprothesen. Möglichkeiten und Grenzen der Gelenkneubildung. Arch Orthop Unfallchir 1956; 48: 519–560.

Mjöberg B. Fixation and loosening of hip prostheses. A review. Acta Orthop Scand 1991; 62 (5): 500–8.

Mjöberg B, Franzén H, Selvik G. Early detection of prosthetic-hip loosening. Comparison of low- and high-viscosity bone cement. Acta Orthop Scand 1990; 61 (3): 273–4.

Morrey B F, Ilstrup D. Size of the femoral head and acetabular revision in total hip-replacement arthroplasty. J Bone Joint Surg (Am) 1989; 71 (1): 50–5.

Mulroy R D Jr., Harris W H. The effect of improved cementing techniques on component loosening in total hip replacement. An 11-year radiographic review. J Bone Joint Surg (Br) 1990; 72 (5): 757–60.

Murray D W, Rushton N. Macrophages stimulate bone resorption when they phagocytose particles. J Bone Joint Surg (Br) 1990; 72 (6): 988–92.

Nistor L, Blaha J D, Källström U, Selvik G. In vivo measurements of relative motion between an uncemented femoral total hip component and the femur by roentgen stereophotogrammetric analysis. Clin Orthop 1991; 269: 220–7.

Ohlin A, Kindblom L-G. The ultrastructure of the tissue surrounding the Christiansen total hip. Acta Orthop Scand 1988; 59 (6): 629–34.

Paterson M, Fulford P, Denham R. Loosening of the femoral component after total hip replacement. The thin black line and the sinking hip. J Bone Joint Surg (Br) 1986; 68 (3): 392–7.

Pazzaglia U E, Ceciliani L, Wilkinson M J, Dell'Orbo C. Involvement of metal particles in loosening of metal-plastic total hip prostheses. Arch Orthop Trauma Surg 1985; 104: 164–74.

Pazzaglia U E, Dell'Orbo C, Wilkinson M J. The foreign body reaction in total hip arthroplasties. A correlated light-microscopy, SEM, and TEM study. Arch Orthop Trauma Surg 1987; 106: 209–19.

Pazzaglia U E, Minoia C, Gualtieri G, Gualtieri I, Riccardi C, Ceciliani L. Metal ions in body fluids after arthroplasty. Acta Orthop Scand 1986; 57: 415–8.

Perren S M. The induction of bone resorption by prosthetic loosening. In: The cementless fixation of hip endoprostheses (Ed. Morscher E). Springer Verlag, Berlin, Heidelberg, New York, Tokyo 1984: 39–41.

Pierson J L, Harris W H. Extensive osteolysis behind an acetabular component that was well fixed with cement. A case report. J Bone Joint Surg (Am) 1993; 75 (2): 268–71.

Pilliar R M, Lee J M, Maniatopoulos C. Observations on the effect of movement on bone ingrowth into porous-surfaced implants. Clin Orthop 1986; 208: 108–13.

Ramadier J O, Lelong P, Dupont J Y. Rotation anormale de certaines cupules cotyloïdiennes excentrées scellées. Revue Chir Orthop 1980; 66: 507–14.

Reigstad A, Røkkum M. An intrapelvic granuloma induced by acetabular cup loosening. Acta Orthop Scand 1992; 63 (4): 465–6.

Revell P A, Weightman B, Freeman M A R, Vernon-Roberts B. The production and biology of polyethylene wear debris. Arch Orthop Trauma Surg 1978; 91: 167–81.

Santavirta S, Hoikka V, Eskola A, Konttinen Y T, Paavilainen T, Tallroth K. Aggressive granulomatous lesions in cementless total hip arthroplasty. J Bone Joint Surg (Br) 1990a; 72 (6): 980–4.

Santavirta S, Konttinen Y T, Bergroth V, Eskola A, Tallroth K, Lindholm T S. Aggressive granulomatous lesions associated with hip arthroplasty. Immunopathological studies. J Bone Joint Surg (Am) 1990b; 72 (2): 252–8.

Santavirta S, Konttinen Y T, Bergroth V, Grönblad M. Lack of immune response to methyl methacrylate in lymphocyte cultures. Acta Orthop Scand 1991a; 62 (1): 29–32.

Santavirta S, Konttinen Y T, Hoikka V, Eskola A. Immunopathological response to loose cementless acetabular components. J Bone Joint Surg (Br) 1991b; 73 (1): 38–42.

Schmalzried T P, Kwong L M, Jasty M, Sedlacek R C, Haire T C, O'Connor D O, Bragdon C R, Kabo J M, Malcolm A J, Harris W H. The mechanism of loosening of cemented acetabular components in total hip arthroplasty. Analysis of specimens retrieved at autopsy. Clin Orthop 1992; 274: 60–78.

Scott W W Jr., Riley L H Jr., Dorfman H D. Focal lytic lesions associated with femoral stem loosening in total hip prosthesis. Am J Roentgenol 1985; 144 (5): 977–82.

Smith-Petersen M N. Arthroplasty of the hip. A new method. J Bone Joint Surg 1939; 21 (2): 269–88.

Snorrason F, Kärrholm J. Primary migration of fully-threaded acetabular prostheses. A roentgen stereophotogrammetric analysis. J Bone Joint Surg (Br) 1990; 72 (4): 647–52.

Stinson N E. Tissue reaction induced in guinea-pigs by particulate polymethylmethacrylate, polythene and nylon of the same size range. Br J Exp Pathol 1964; 46: 135–46.

Sudmann E, Havelin L I, Lunde O D, Ratt M. The Charnley versus the Christiansen total hip arthroplasty. A comparative clinical study. Acta Orthop Scand 1983; 54: 545–52.

Sutherland C J, Wilde A H, Borden L S, Marks K E. A ten-year follow-up of one hundred consecutive Müller curved-stem total hip-replacement arthroplasties. J Bone Joint Surg (Am) 1982; 64 (7): 970–82.

Svensson O, Mathiesen E B, Reinholt F P, Blomgren G. Formation of a fulminant soft-tissue pseudotumor after uncemented hip arthroplasty. A case report. J Bone Joint Surg (Am) 1988; 70 (8): 1238–42.

Tallroth K, Eskola A, Santavirta S, Konttinen Y T, Lindholm T S. Aggressive granulomatous lesions after hip arthroplasty. J Bone Joint Surg (Br) 1989; 71 (4): 571–5.

Tanzer M, Maloney W J, Jasty M, Harris W H. The progression of femoral cortical osteolysis in association with total hip arthroplasty without cement. J Bone Joint Surg (Am) 1992; 74 (3): 404–10.

Tönnis D, Asai H. Untersuchungen über die Lockerungsraten verschiedener Hüftgelenksprothesen und unterschiedlicher Halslängen. Arch Orthop Unfallchir 1976; 86: 317–32.

Utz J A, Lull R J, Galvin E G. Asymptomatic total hip prosthesis. Natural history determined using Tc-99m MDP bone scans. Radiology 1986; 161 (2): 509–12.

Vaes G. Cellular biology and biochemical mechanism of bone resorption. A review of recent developments on the formation, activation, and mode of action of osteoclasts. Clin Orthop 1988; 231: 239–71.

Vernon-Roberts B, Freeman M A R. The tissue response to total joint replacement prostheses. In: The scientific basis of joint replacement (Ed. Swanson S A V, Freeman M A R). Pitman Medical, Tunbridge Wells 1977: 86–129.

Volz R G, Wilson R J. Factors affecting the mechanical stability of the cemented acetabular component in total hip replacement. J Bone Joint Surg (Am) 1977; 59 (4): 501–4.

Weightman B. Friction, lubrication and wear. In: The scientific basis of joint replacement (Ed. Swanson S A V, Freeman M A R). Pitman Medical, Tunbridge Wells 1977: 46–85.

Weinans H, Huiskes R, Grootenboer H J. Trends of mechanical consequences and modeling of a fibrous membrane around femoral hip prostheses. J Biomech 1990; 23 (10): 991–1000.

Willert H-G, Bertram H, Buchhorn G H. Osteolysis in alloarthroplasty of the hip. The role of bone cement fragmentation. Clin Orthop 1990a; 258: 108–21.

Willert H-G, Bertram H, Buchhorn G H. Osteolysis in alloarthroplasty of the hip. The role of ultra-high molecular weight polyethylene wear particles. Clin Orthop 1990b; 258: 95–107.

Willert H-G, Semlitsch M. Reactions of the articular capsule to wear products of artificial joint prostheses. J Biomed Mater Res 1977; 11 (2): 157–64.

Wilson-MacDonald J, Morscher E, Masar Z. Cementless uncoated polyethylene acetabular components in total hip replacement. Review of five- to 10-year results. J Bone Joint Surg (Br) 1990; 72 (3): 423–30.

Wroblewski B M. 15-21-year results of the Charnley low-friction arthroplasty. Clin Orthop 1986; 211: 30–5.

Wroblewski B M, Lynch M, Atkinson J R, Dowson D, Isaac G H. External wear of the polyethylene socket in cemented total hip arthroplasty. J Bone Joint Surg (Br) 1987; 69 (1): 61–3.

Wykman A, Selvik G, Goldie I. Subsidence of the femoral component in the noncemented total hip. A roentgen stereophotogrammetric analysis. Acta Orthop Scand 1988; 59 (6): 635–7.