THE INITIATION OF FAILURE IN CEMENTED FEMORAL COMPONENTS OF HIP ARTHROPLASTIES

MURALI JASTY, WILLIAM J. MALONEY, CHARLES R. BRAGDON, DANIEL O. O’CONNOR, TERESA HAIRE, WILLIAM H. HARRIS

From Massachusetts General Hospital and Harvard Medical School, Boston

We studied 16 femora retrieved at post-mortem from symptomless patients who had a satisfactory cemented total hip arthroplasty from two weeks to 17 years earlier, with the aim of delineating the initial mechanisms involved in loosening. Only one specimen showed radiographic evidence of loosening; the other 15 were stable to mechanical testing at 17.0 Nm of torque.

In all 16 specimens, the cement–bone interface was intact with little fibrous tissue formation. By contrast, separation at the cement–prosthesis interface and fractures in the cement mantle were frequent. The most common early feature was debonding of the cement from the metal, seen at the proximal and distal ends of the prosthesis. Specimens which had been in place for longer also showed circumferential fractures in the cement, near the cement–metal interface, and radial fractures extending from this interface into the cement and sometimes to the bony interface. The most extensive cement fractures appeared to have started at or near sharp corners in the metal, or where the cement mantle was thin or incomplete. Fractures were also related to voids in the cement.

The time relationship in this series suggested that long-term failure of the fixation of cemented femoral components was primarily mechanical, starting with debonding at the interface between the cement and the prosthesis, and continuing as slowly developing fractures in the cement mantle.

An understanding of the mechanisms which initiate the failure of fixation is essential if the longevity of cemented femoral components is to be improved. Controversy continues over attempts to identify the critically weak member or members of the structural composite of bone, cement and metal. There are four vulnerable regions: the cement–prosthesis interface, the cement itself, the cement–bone interface, and the bone. All four have been variously implicated as the site at which failure starts, based on the evidence of clinical follow-up and histological studies of material obtained at revision operations (Fornasier and Cameron 1976; Charnley 1979; Gruen, McNeice and Amstutz 1979; Stauffer 1982; Sutherland et al 1982; Jasty et al 1984, 1986). Such studies, however, cannot define the initial causes of loosening, since clinical radiographs are inadequate to identify the really early changes, and, by the time revision is performed, most of the critical information has been obscured.

Charnley (1979) believed that loosening of cemented femoral components was primarily due to mechanical factors, and that cement was well tolerated by the skeleton, even in the long term. Others have maintained that fibrous tissue invariably forms at the cement–bone interface as a result of biological reactions to the implanted foreign materials, and that these biological events lead to loosening (Fornasier and Cameron 1976; Sutherland et al 1982; Jasty et al 1986). There are, in addition, many variations and controversies within these two major points of view.

Even assuming that mechanical events are most important in initiating loosening, the specific details are far from clear. Many factors have been implicated. These include the strength of cement, the strength of the cement–prosthesis interface, the strength of the cement–bone interface, the alignment of the metal in the cement mantle, the thickness of the cement mantle, the design of the component, and the modulus of the implanted device (Charnley 1979; Crowninshield et al 1980; Stauffer 1982; Sutherland et al 1982; Tarr et al 1982; Crowninshield and Tolbert 1983; Ahmed, Raab and Miller 1984; Burke,
The present study of 16 femora, we carried out additional morphological and fractographic studies of the cement and its interfaces.

MATERIALS AND METHODS

Sixteen whole cadaveric femora were harvested at postmortem from 12 patients (Table I). Four intact contralateral femurs were also collected. Clinical information was obtained for each patient from their physicians, medical records and family members. There were seven women and five men, aged from 43 to 98 years at the time of death. The initial diagnosis had been osteoarthritis in eight patients (12 femora), hip fracture in two, avascular necrosis in one, and was unknown in one. The implants had been in place from 0.5 to 210 months (Table I).

The soft tissue was removed from each specimen and it was radiographed. Radiolucencies were recorded for the zones described by Gruen et al (1979) and radiographic loosening was assessed by the criteria of Harris and McGann (1986). The stability of the implant was assessed by a torque wrench micrometer (Maloney et al 1989). This applied a torsional load of 17.0 Nm at 2.8 Nm increments to the femoral component, this force being based on those shown for stair climbing by Davy et al (1988), and measured the resultant micromotion.

The specimens were then prepared by dehydration in calcium chloride pellets, embedded in a rectangular block of surgical bone cement and then sectioned transversely at 5 mm intervals, using a high-speed, water-cooled, circular saw (DoAll, Newton, Massachusetts) with an aluminium ceramic blade. We avoided the use of solvents, other and alcohol; this and the use of a high-speed water-cooled saw allowed the preparation of sections with minimal artefacts in the bone cement.

Contact radiographs were taken of each section, and its surfaces were gently polished on a surface grinder with 600 grit silicon carbide paper. After further dehydration in calcium chloride pellets, thin layers of gold were applied to the surfaces of the specimens using a Sputter coater. The specimens were then examined in a scanning electron microscope (SEM) equipped with a back-scatter detector (Cambridge Instruments, Cambridge, Massachusetts). These techniques made it possible to study the cement, prosthesis and bone with minimal artefacts.

Fractographic studies were then carried out to assess the presence of fatigue fractures in the cement and on its interfaces with metal and with bone. In some specimens, the metal was removed from the cement mantle to allow scanning electron microscopy of the surface. In other specimens, where partial fractures through the cement mantle were already present, these were completed acutely so that the fracture surfaces could be evaluated for the presence of fatigue markings. Representative sections were prepared for histology after the prosthesis had been removed from the sections.

Table I. Details of 16 hip specimens removed from symptomless patients who had died after cemented hip arthroplasty

<table>
<thead>
<tr>
<th>Hip</th>
<th>Prosthesis</th>
<th>Sex:age</th>
<th>Motion (µm)</th>
<th>Debonding cement-prosthesis Site†</th>
<th>Cement fracture site‡</th>
<th>Cement:</th>
<th>Motion (µm)</th>
<th>Debonding cement-prosthesis Site†</th>
<th>Cement fracture site‡</th>
</tr>
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<tr>
<td>0.5</td>
<td>HD-2</td>
<td>M:67</td>
<td>50</td>
<td>D</td>
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<td>None</td>
<td>0.8</td>
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<tr>
<td>3</td>
<td>Omnifit</td>
<td>M:71</td>
<td>13</td>
<td>D</td>
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<td>None</td>
<td>1.0</td>
<td>P</td>
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<tr>
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<td>300</td>
<td>P</td>
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<td>P</td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>D</td>
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<td></td>
<td></td>
<td></td>
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</tr>
<tr>
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<td>58</td>
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<td>F:85</td>
<td>100</td>
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<td>P</td>
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<td>P</td>
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<tr>
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<tr>
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<td>P</td>
<td>M</td>
<td></td>
<td></td>
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<tr>
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<td>NA†</td>
<td>P</td>
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<td>P</td>
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<td>4.0</td>
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<tr>
<td>137</td>
<td>CAD</td>
<td>F:71</td>
<td>13</td>
<td>P</td>
<td>1.0</td>
<td>P</td>
<td>1.0</td>
<td>D</td>
<td>1.0</td>
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<td>144</td>
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<td>M:NA†</td>
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<td>P</td>
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<td>P</td>
<td>2.0</td>
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</tr>
<tr>
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<td>M:71</td>
<td>25</td>
<td>P</td>
<td>2.0</td>
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<td>M:58</td>
<td>NA†</td>
<td>Total</td>
<td>P</td>
<td>M</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td>Müller</td>
<td>F:67</td>
<td>25</td>
<td>P</td>
<td>4.0</td>
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<td>4.0</td>
<td>P</td>
<td>4.0</td>
</tr>
<tr>
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<td>Müller</td>
<td>F:76</td>
<td>15</td>
<td>P</td>
<td>3.0</td>
<td>P</td>
<td>4.0</td>
<td>P</td>
<td>4.0</td>
</tr>
<tr>
<td>210</td>
<td>Müller</td>
<td>F:76</td>
<td>3</td>
<td>P</td>
<td>4.0</td>
<td>P</td>
<td>2.0</td>
<td>P</td>
<td>2.0</td>
</tr>
</tbody>
</table>

* period implanted in months/side
† NA, not available
‡ P, proximal; M, middle; D, distal
To confirm that artefacts were not produced by the mechanical testing, embedding or sectioning processes, femoral components of identical design were implanted into the five intact femoral specimens using identical surgical techniques. These femurs were then subjected to the mechanical and fractographic studies.

RESULTS

In 15 of 16 cases, the radiographs showed no evidence of loosening of the femoral components (Fig. 1). Only one of the 16 specimens showed definite radiographic loosening, with radiolucency at the cement–metal interface, but two others had areas of focal endosteal osteolysis without component loosening. On mechanical testing, 11 prostheses had micromotion less than 100 μm, two had between 100 and 200 μm and one had 300 μm (Table I). Measurements were not available for the remaining two specimens; one contained a grossly loose prosthesis, and the other was not tested because it was very osteoporotic.

In none of the 16 cases was there morphological evidence of loosening at the cement–bone interface, the trabecular bone being intimately associated with and interdigitated with the cement (Fig. 2). Careful histological examination of all these interfaces showed extensive fibrous tissue formation in only one specimen. In all the others, fibrous tissue was rare at the cement–bone interface. Occasional patches were observed in five specimens at a few areas, but not exceeding four to five cells thick; the fibrous tissue did not surround the mantle, or significantly interfere with cement–bone interdigitation.

Even though 15 of the 16 hips showed no clinical, radiographic or physical evidence of loosening and all of the cement–bone interfaces appeared well fixed in all femora, some debonding of the cement–metal interface was seen in every specimen. In four specimens, there was a layer of collagenous and fibrinous tissue about 50 to 100 μm thick between the femoral component and the surrounding cement. This established that these gaps had existed in vivo and were not artefacts of the biomechanical testing or cutting the femur into sections.

Debonding was complete in two hips retrieved at 118 and 156 months respectively. The 118-month specimen showed no radiographic separation at the cement–metal interface, but the other (156 months) showed radiographic separation in the proximal lateral region on the anteroposterior radiograph and was loose to manual palpation.

In the other 14 femora the debonding visible on cut sections was focal and confined to either the proximal anterior surface or, in nine femora, to both locations (Table I). It seemed that debonding usually occurred early after surgery, since it was seen in the distal 1 to 2 cm in all but one specimen. Proximal debonding involved the anterior and medial aspects only in two femora; in eight it had progressed to involve the entire

![Fig. 1](https://example.com/fig1.png)

Radiographs of both femora retrieved from a patient who had bilateral total hip arthroplasty ten years previously. There is no radiographic evidence of loosening.

![Fig. 2](https://example.com/fig2.png)

Serial sections through a pair of femora. On the left a Müller prosthesis had been implanted 17 years earlier; on the right implantation had been performed in the laboratory after death. The left side shows good cement apposition, extensive bone remodelling, the formation of secondary circumferential trabeculae, and ingrowth of bone into the cement.
Scanning electron micrograph showing an incomplete fracture through the cement mantle (small solid arrows) originating at the cement-metal interface. This shows separation (hollow arrows). $P = \text{prosthesis}, C = \text{cement}$.

SEM of another incomplete fracture through the cement mantle. The prosthesis had loosened from the cement mantle and had been removed from the section.

circumference of the prosthesis. Except for two prostheses with total debonding, the middle region of the metal remained well fixed to the cement mantle. Debonding tended to be most extensive in the specimens retrieved late (Table I).

Fractures in the cement mantle itself were found on cut sections around all prostheses which had been in use for over three years (Figs 3 to 9). In some specimens, we identified fatigue striations of the fracture surfaces (Fig. 10). The striations established that the fractures in the cement mantles were due to fatigue loading over many

Fractures through the cement mantle at 13 years. The prosthesis had migrated into retroversion, but the cement-bone interface was intact. The arrows indicate cement fractures.

There is a complete fracture through the cement mantle (solid arrow) on the medial side near a corner of the prosthesis. Hollow arrows indicate the separation at the cement-prosthesis interface.

There is a circumferential fracture in the cement mantle near the cement-prosthesis interface.
Fig. 8
Section through the region of radiological osteolysis in a femur with a well fixed femoral component, showing fractures in the mantle, with some fragmentation of the cement in this region.

Fig. 9
Fractures in varying stages of development in the cement (C = cement, P = prosthesis, b = bone, FV = fracture through void, Fb = fracture originating at the surface of the cement).

Fig. 10a
Fig. 10b
Fig. 10c
Fatigue striations in a cement mantle fracture. These show that the cement had undergone fatigue failure over many cycles of stress in normal use. Figure 10a – Low magnification scanning electron micrograph of a fracture surface shows a glassy fatigue-fracture zone surrounded by an irregular acute catastrophic fracture zone. Curved, regularly spaced fatigue markings are seen around a large void in the cement. Adjacent fractures pass through other voids. Figure 10b – An intermediate-magnification micrograph of the fatigue zone. Figure 10c – A high-magnification view of the centre of Figure 10b showing fine parallel fatigue striations and plastic deformation, due to repetitive high loads during use in vivo.

Cycles and not due to a single loading. In general, specimens less than ten years in situ showed small incomplete fractures while the specimens in place more than ten years all showed large complete cement mantle fractures.

The fractures were oriented either circumferentially at the cement–metal interface (see Fig. 7), or radially, appearing to propagate away from this interface (see Figs 3, 4, 5 and 6). Both types of fracture were usually associated with debonding of cement from metal: the circumferential fractures appeared to lead to further separation at the cement–metal interface (Fig. 7). The radially oriented fractures, sometimes extending completely through the cement and sometimes partially, were usually associated with debonding and were usually located at or near the corners of the prosthesis (Figs 4 and 6) on the medial, anterior and posterior surfaces. The fractures were wider at the prosthetic side than toward the bone. In spite of these separations and fractures, the cement–bone interface was intact in all cases.

Defects in the cement mantle were also frequently associated with fractures in the cement and debonding. This was most common in defects in the proximal-medial and distal-lateral regions. In the one implant that appeared loose on contact radiographs, a defect in the
proximal-medial cement mantle led to several radial fractures and migration of the prosthetic component into retroversion (Fig. 5). In two other cases, the defects in the distal-lateral mantle were surrounded by numerous fractures of the adjacent thin cement. In the two cases which showed areas of focal endosteal osteolysis, there were numerous fractures through the adjacent thin cement with some fragmentation (Fig. 8).

Many fractures in the cement seemed to originate at the prosthesis–cement interface, but under scanning electron microscopic examination there were also numerous fractures in varying stages of development which appeared to originate from voids in the cement (Figs 9 and 11). For example, in the 40-month specimen in which the prosthesis tip was debonded, the cement surface adjacent to the tip was examined. Small fractures were minimal sporadic fibrous tissue, but the metal prosthesis was completely debonded and surrounded by a layer of fibrous tissue. Numerous radial fractures in the cement had allowed the prosthesis to subside into retroversion. Even though the prosthesis was radiographically loose and was mobile on manual palpation, the patient had had no pain, presumably because the cement–bone interface had not yet loosened.

There were no cement fractures or debonding at the cement–prosthesis interface in any of the sections made from the five specimens of contralateral femora. The fibrous tissue in some areas of debonding in the specimens retrieved at post-mortem, and the fatigue striations at the fractured surfaces established that the findings in the retrieved specimens were not artefacts from either the post-mortem mechanical testing or the sectioning of the specimens.

**DISCUSSION**

Our study of cemented femoral components retrieved from patients after as long as 17 years use provided many insights into possible mechanisms of failure. The different periods of physiological loading in vivo permitted assessment of biological phenomena such as changes at the bone–cement interface as well as an assessment of the fatigue damage to the methacrylate bone cement. We tried to elucidate the sequence of events at the initiation of failure.

In all cases non-fragmented cement was tolerated biologically over a very long term. All the specimens showed excellent apposition with trabecular bone and intimate interdigitation with the surface of the cement with no significant intervening fibrous tissue. Similar findings have been reported in specimens from total hip replacements performed by Charnley (Charnley 1979; Malcolm, personal communication). Observations at revision surgery on patients with loose femoral components led to the suggestion that the fibrous tissue at the cement–bone interface plays a major role in initiating prosthetic loosening (Willert et al 1974; Goldring et al 1986), but our study supports the alternative view that the fibrous tissue at this interface in well-cemented femoral components is secondary to prosthetic loosening rather than the primary cause.

We detected localised areas of separation of the cement–metal interface in all femora after three years, and some had fibrous tissue formation at this interface. Despite this, none of them had any loosening or significant fibrous tissue formation at the cement–bone interface. Even in the one femur with radiographic evidence of femoral loosening, excellent interdigitation had been maintained everywhere between the cement and bone and the patient had had no symptoms. In this specimen, the cement–metal separation was visible on the radiographs, and the prosthesis was confirmed to be loose on manual palpation. In the other specimens, the cement–metal separations were probably too small to be...
detected on the clinical radiographs, but with time they might have become larger. It is accepted that some patients with loose prostheses may not develop clinical symptoms until much later (Dorr, Takei and Conaty 1983; Hodgkinson, Shelley and Wroblewski 1988), and it is likely that our patient would have eventually developed pain.

Our results suggest that loosening at the cement–bone interface is a late event: it seems to follow the mechanical changes which initiate the loosening process. With time, cement fracture may lead to fragmentation of methylmethacrylate or ingress of polymeric debris. This may then be responsible for membrane formation at the bone–cement interface and for the bone lysis that may accompany loosening, as seen locally in two of our specimens.

In these two specimens the prostheses did not show radiographic loosening, and both patients were asymptomatic. However, sections showed that localised cement mantle fractures had already taken place, and that fragmentation in these regions had led to local destruction of the cement–bone interface. It is likely that if this process extends to a larger area, the prosthesis would lose its bony support, become grossly loose and produce clinical symptoms.

Numerous large and small fractures in the cement were found in several cases in which there was no radiographic or mechanical evidence of loosening at the cement–bone interface. This occurred in the presence of good cement–bone apposition with no fibrous tissue formation at this interface. Small fractures within the cement mantle were seen around voids in the cement as early as five years after the arthroplasty. Larger fractures through the entire thickness of the cement mantle were found only in the ten-year to 17-year specimens. These findings suggest that cement undergoes accumulated fatigue damage in vivo well before gross loosening is manifest.

It could be argued that the cement fractures were incidental findings with no relationship to loosening, since these patients had good function with no significant pain. However, our study suggests that the cement fractures are not benign. In one case they were associated with loosening and migration of the femoral component within the cement mantle and in two other cases, they were associated with focal osteolysis secondary to the liberation of particulate methylmethacrylate. In addition, we found evidence that the cement fractures were progressive, increasing in number and extent with time, so that eventually more of the specimens might have shown loosening or osteolysis. We have already reported on the role of fragmented methylmethacrylate from localised cement fractures in causing bone lysis around well fixed cemented femoral components (Maloney et al 1990).

Lucencies at the cement–prosthesis interface, and cement fractures visible on clinical radiographs, have been used as the criteria to diagnose prosthetic loosening even when the patients may not have significant pain (Stauffer 1982; Harris and McGann 1986). Our findings confirm that separation at the cement–prosthesis interface and fractures of the cement mantle become visible on the clinical radiographs only when they are large. It seems that many smaller fractures and separations occur in patients with total hip replacements, and are not, for some time, visible on clinical radiographs. It is probable that these small fractures can propagate, become larger, and eventually be visible on the clinical radiographs.

Patients may not develop significant pain for months or years after the appearance of radiographic loosening (Dorr et al 1983; Hodgkinson et al 1988) as shown in one of our cases. However, it is likely that, with time, the other patients, especially the two with focal lysis, might also have developed radiographic loosening, and, with more time, significant clinical symptoms. We chose only asymptomatic patients since we were looking for the initial changes which might lead to failure of fixation. We interpret our findings as showing that cement undergoes accumulated fatigue damage before clinical or radiographic loosening is apparent.

Local debonding at the cement–metal interface, seen in some areas in almost all of our cases, is very important. This debonding started to occur very soon after the arthroplasty: it was noted at the distal end of the prosthesis in one case after only 15 days. This localised debonding may be one of the earliest features in the initiation of prosthetic loosening. With time, it may progress and involve the entire cement–prosthesis interface, as seen in two of our specimens, and eventually become visible on the clinical radiographs. Debonding at the cement–metal interface may also cause marked increases in the stresses which occur within the cement, leading to additional mechanical failures of the cement mantle (Crowninshield and Tolbert 1983). The poor strength of the cement–metal interface has been shown in previous experimental studies: the cement itself is not an adhesive (Ahmed et al 1984). Clinical studies have also shown that failure at the cement–metal interface may be a cause for loosening (Fornasier and Cameron 1976; Stauffer 1982).

Long-term success may well be improved by such measures as eliminating the sharp corners of the metal prosthesis (Crowninshield et al 1980), strengthening of the cement–metal interface by precoating or creating a roughened surface (Ahmed et al 1984), reducing the porosity of bone cement by centrifugation (Burke et al 1984) or vacuum-mixing, curetting the fine trabecular bone in the proximal metaphysis, and maintaining an adequate thickness of the cement mantle (Crowninshield et al 1980; Tarr et al 1982). However, more long-term studies are needed to evaluate the effect of such improvements on the longevity of cemented total hip replacements. There is already some clinical evidence.
that long-term success is extended by the use of newer components without sharp corners and by modern cementing techniques (Harris and McGann 1986; Mulroy and Harris 1990).

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REFERENCES