THE EFFECTS OF CEMENT-STEM DEBONDING IN THA ON THE LONG-TERM FAILURE PROBABILITY OF CEMENT

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Abstract—The damage accumulation failure scenario is one of the most prominent ones of cemented THA reconstruction, and involves the accumulation of mechanical damage in materials and interfaces due to repetitive dynamic loading, eventually resulting in gross loosening. This study addresses this scenario by combining finite element techniques with the theory of continuum damage mechanics, to analyze the damage accumulation process in the cement mantle. It was investigated how damage accumulation was affected by stem-cement debonding, and what the effects of a layer with poor bone quality around the cement mantle were. For the unbonded stem, it was determined if clinical migration rates can be explained by failure of the cement mantle, and whether cement failure promotes the formation of a pathway for debris at the stem-cement interface.

It was found that stem-cement debonding not only elevated the initial stress levels with a factor of about two to three as demonstrated in earlier studies, but remained to have an impact on the failure process of the cement mantle. Stem-cement debonding accelerated the failure process by a factor of four, and promoted the formation of a pathway for debris at the stem-cement interface, particularly when the bone support to the cement mantle was reduced. The amount of subsidence was only substantial when the damaged cement mantle was surrounded by a layer of bone with reduced stiffness.

This study supports the hypothesis that the survival of cemented THA is enhanced by a firm and lasting bond between the stem and the cement mantle, although this may be difficult to realize clinically. © 1997 Elsevier Science Ltd

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INTRODUCTION

Damage accumulation and particulate reaction failure scenarios are currently the most prominent in total hip arthroplasty (THA) (Huiskes, 1993). The former involves the accumulation of mechanical damage in materials and interfaces due to repetitive, dynamic loading. This damaging process can lead to cement failure, or interface disruption and eventually to gross loosening. This scenario has been identified clinically by Jasty et al. (1991), who analyzed retrieved specimens and found that cement damage was almost always associated with stem-cement debonding, and most cracks had been generated at the corners of the stem. The same crack patterns were found in our own material, consisting of five autopsy retrieved specimens. In all specimens, cement cracks were initiated at the corners of the stem, which had debonded from the cement mantle (Fig. 1). Stress analyses indicate that stem-cement debonding causes higher cement stresses (Mann et al., 1995; Verdonschot and Huiskes, 1996a), which could explain the higher crack densities around the debonded sites found in the retrieved specimens.

When the cement material has failed, the particulate reaction failure scenario may become more prominent. Through the defects in the cement mantle, wear particles can be transported from the stem-cement to the cement-bone interface. This process is enhanced when the stem debonds from the cement mantle and a pathway is formed to transport the particles from the joint space to the fractured sites (Anthony et al., 1990). These wear particles activate macrophages, resulting in local osteolysis. This process gradually degrades the bone, promotes interface motions, and propagates to gross loosening. In its turn, the particulate-reaction failure scenario affects the damage accumulation scenario, as the bone support to the cement mantle is gradually reduced.

This study addresses the damage accumulation failure scenario with respect to the cement mantle, and the particulate reaction failure scenario relative to the formation of a pathway at the stem-cement interface. It was investigated how the failure process of the cement mantle is affected by the characteristics of the stem-cement interface and a reduction of bone support, and whether clinical migration rates can be explained by failure of the cement mantle, and whether cement failure promotes the formation of a pathway for debris at the stem-cement interface.

METHODS

A CT-based, three-dimensional FE model of a proximal femur was created (Huiskes et al., 1992) and a cemented femoral stem was introduced in the model. The thickness of the cement mantle was variable (2 mm minimal thickness, 4 mm typical thickness) depending on the endosteal shape of the femur. Directly distal to the tip no cement was present, simulating a space created by a special designed centralizer which is sometimes used in conjunction with polished implants. The model contained 2420 8-node isoparametric elements, 281...
gap-elements (to simulate the debonded stem–cement interface) and 3660 nodal points (Fig. 2). The validity of the model was tested by comparing the results produced by the present model to models with other (higher and lower) mesh densities. It was found that the results in terms of number of cracks and total accumulated damage were not very sensitive to mesh density with deviations within ten percent. For each bone element the average apparent density \( \rho \) (g cm\(^{-3}\)) was determined using the CT data and Young's moduli (MPa) could be calculated using the equation established by Carter and Hayes (1977):

\[
E = c \rho^3
\]  

with \( c = 3790 \) (MPa(g cm\(^{-3}\))^\(^{-3}\)), and a maximal Young's modulus of 20 GPa.

To investigate the effect of reduced bone support to the cement due to bone degradation at the cement–bone interface, the stiffnesses of the bone elements directly adjacent to the cement mantle were reduced by a factor of 100. Hence, bone degradation was assumed to be present from the beginning of the failure process. For all bone elements, Poisson's ratio was 0.35. Young's modulus for the cement material was set at 2200 MPa, and Poisson's ratio at 0.3 (Saha and Pal, 1984). Young's modulus and Poisson ratio for the stainless steel prosthetic material were 200 GPa and 0.28, respectively.

The loading case simulated the one generated during the stance phase of walking with a force of 2450 N acting on the prosthetic head, with angles of 23° in the frontal plane and 6° in the sagittal plane (Bergmann et al., 1993). Muscle forces were assumed acting on the greater trochanter with a total force of 1650 N, with angles of 24° in the frontal plane and 15° in the sagittal plane (directed towards anterior) estimated from Crowninshield and Brand (1981) and Dostal and Andrews (1981). This load was assumed to be applied to the structure repeatedly, in cycles from zero to the maximal value.

To investigate the effects of stem cement interface characteristics on the damage process, the interface was assumed to be either completely bonded or unbonded. In the latter case, the friction coefficient was 0.25 (Hampton, 1981; Mann et al., 1991; Verdonschot and Huiskes, 1996a)

The theory of Continuum Damage Mechanics (CDM) was implemented to simulate the accumulation of microcracks during loading (Chaboche, 1988b; Lemaitre, 1984; Paas et al., 1990; Verdonschot and Huiskes, 1992). The details of the method are described in an earlier paper (Verdonschot and Huiskes, 1995) and involves the introduction of a damage variable \( D \), with \( 0 \leq D \leq 1 \). If \( D = 0 \), the material is fully intact, and if \( D = 1 \), it has 'cracked'. The stress situation in the cement mantle is typically three-dimensional, which requires a tensorial
variable \( (D) \) to describe the damage produced in the material. In that case, damage can accumulate in multiple directions, and the material can eventually 'crack' in three perpendicular directions.

How this theory of CDM was implemented in the FE code is schematically shown in Fig. 3. The FE code provided the stress tensors \( (\sigma_{ip})_{\text{global}} \) in the global coordinate system in every integration point within the cement elements. Subsequently, the principal stresses \( (\sigma_{ip})_{\text{principal}} \) and their rotations \( (R_{ip}) \) were calculated. In the case that a principal stress component was tensile, damage was assumed to occur perpendicular to that plane. The rate of micro-crack development was determined from fatigue experiments on uncentrifuged bone cement specimens (Davies et al., 1987), which provide a relation between the stress amplitude \( \sigma \) and the number of cycles to failure \( N \) at that stress level, according to

\[
\log N = -4.68 \log \sigma + 8.77. \tag{2}
\]

Using the Palmgren–Miner rule (Miner, 1945), the damage per cycle in each principal stress (or damage) direction was determined, and stored in the local damage tensor \( (\sigma_{ip})_{\text{local}} \) of the global coordinate system. Subsequently, this cyclic damage tensor was added cycle times to the damage tensor which was already present \( (D_{ip})_{\text{global}} \) until a temporary damage tensor \( (D_{ip})_{\text{local}} \) was obtained that had a new principal value of one. In this case, cycles, indicates how many times the cyclic damage tensor should be added to the already existing damage to obtain complete damage in a new direction. After this procedure had been performed for all integration points, the timestep used in the current increment was set to the minimal value of cycles. Subsequently, the old damage tensors \( (D_{ip})_{\text{global}} \) were updated by adding timestep times the cyclic damage tensors \( (D_{ip})_{\text{local}} \) for all integration points. This approach ensures that damage becomes complete in a direction in at least one integration point in every calculation increment. A macro crack was assumed to have occurred when the damage in a direction \( (d_i) \) was more than 0.95. In that case, a cracking option available in the MARC finite element code was activated (MARC Analysis Corporation, Palo Alto, CA). This option allowed initiation of cement cracks perpendicular to the damage direction. The cracking option ensures no stiffness in tensile and shear direction at the integration point, whereas full stiffness is restored in the case that the crack would be forced to close, due to compressive stresses. This results in nonlinear, anisotropic material behavior of the cement material after the material was locally cracked.

The development of the damage process in the cement mantle was evaluated by considering the principal values \( (d) \) of the damage tensor in the integration points

\[
(D_{ip})_{\text{principal}} = \begin{bmatrix}
d_1 & 0 & 0 \\
0 & d_2 & 0 \\
0 & 0 & d_3
\end{bmatrix}_{ip}. \tag{3}
\]

It was considered how the maximal value \( (d_{\text{max}}) \) of the principal values \( (d) \) was distributed in the cement mantles, and how the total damage \( (D_{\text{tot}}) \) and the total number of macroscopic cracks \( (N_{\text{cracks}}) \) in the cement mantle developed with the number of loading cycles. The total damage \( (D_{\text{tot}}) \) accumulated in the cement mantle was defined as the total of the three principal values \( (d) \) of the damage tensor, summed over all integration points \( (N_{ip}) \)

\[
D_{\text{tot}} = \sum_{ip=1}^{N_{ip}} \sum_{i=1}^{3} (d_{ip}). \tag{4}
\]

The total number of cracks \( (N_{\text{cracks}}) \) generated in the cement mantle was determined by the number of principal values of the damage tensors in the integration points which were larger than 0.95,

\[
N_{\text{cracks}} = \sum_{ip=1}^{N_{ip}} \sum_{i=1}^{3} (cr_{ip})_{ip}
\]

where

\[
(cr_{ip})_{ip} = \begin{cases}
0 & \text{if } (d_{ip}) < 0.95, \\
1 & \text{if } (d_{ip}) \geq 0.95.
\end{cases} \tag{5}
\]
RESULTS

Debonding of the stem–cement interface affected the damage process in the cement mantle considerably. Assuming bonded stem–cement interface conditions, the tensile bending stresses at the lateral exterior of the cement mantle produced damage at these particular sites. The unbonded stem produced high tensile stresses at the corners of the stem. Hence, damage was initiated in these regions. The damage accumulated with the number of loading cycles, until the material failed. (Figs 4 and 5). Due to the difference in load-transfer mechanism between the bonded and unbonded stems, the damage directions were different as well. The bonded stem produced damage directed predominantly in longitudinal and radial directions, whereas the unbonded one generated damage oriented mainly in the circumferential, hoop direction. Hence, the cement mantles gradually lost their integrities in these directions, respectively. In the cracked areas, zero-stiffness in the damaged directions occurred, leading to stress release in those directions (Fig. 5).

The rate of the damage process was higher in the case of a debonded stem, and was further increased by bone degradation. Assuming unbonded stem–cement interface conditions, the damage rate was approximately four times higher as compared to the case with a bonded stem–cement interface (Fig. 6). The total damage ($D_{tot}$) accumulated in the cement mantle is represented by integration points that are cracked ($d_{max} > 0.95$), and those that are partly damaged ($0.0 < d_{max} < 0.95$). After 25 million loading cycles, the total amount of damage accumulated in the whole cement mantle ($D_{tot}$) was 197, and the number of cracks ($N_{cracks}$) was 44 in the case of bonded stem–cement interface conditions, whereas these values were considerably higher ($D_{tot}$ was 792, and $N_{cracks}$ was 520) when this interface was assumed to be unbonded. The effect of bone degradation on the damage rate was relatively small in the case of the bonded stem. In the case of an unbonded stem, however, it had considerable effects. The total amount of damage ($D_{tot}$)
Cement-stem debonding

Fig. 6. The development of the total amount of damage built up in the cement mantle as a function of the number of loading cycles. Debonding accelerated the damage process. Reducing the stiffness of the bone layer adjacent to the cement mantle had only an effect in case of an unbonded stem-cement interface. In that case the damage rate was elevated.

Increased by a factor of three when bone support to the cement mantle was reduced. Secondary (and tertiary) cracks perpendicular to the primary ones were absent around the bonded stem, and only two were created in the unbonded case. Bone degradation around the unbonded stem, led to about 180 secondary cracks after 25 million loading cycles.

In all cases, the rate of the damage process reduced over time, due to tensile stress relief in the directions perpendicular to the cracks. Initially, the highest tensile stress (6.9 MPa) was generated in the case of the unbonded stem-cement interface with degraded bone quality (Fig. 7). Hence, this case produced the highest initial damage rate. Around the bonded stem, peak tensile stresses were relatively low, and were hardly affected by bone degradation.

Subsidence of the debonded stem in the cement mantle was considerably increased by degradation of the bone around the cement mantle. Similar to radiographic measurements, the amount of subsidence was defined as the size of the gap created between the proximal (coronal) prosthetic surface and the proximal cement (Fowler et al., 1988). When the loads were applied for the first time to the structure with the unbonded prosthesis, the stem subsided 25 microns in the cement shaft (Fig. 8). The size of this gap increased to about 50 microns after 25 million loading cycles. In the case with bone degradation the stem subsidence on 30 microns after the first load application, 5 microns more than when the bone was intact. However, as the damage process continued, the effect of bone degradation on the amount of subsidence became more distinct. After 25 million loading cycles, the stem had subsided about 400 microns within the cement mantle.

The debonded stem produced a pathway for debris when loaded. This pathway was increased during the damaging process when the cement–bone interface was deficient. The formation of a pathway for debris at the stem–cement interface was judged by considering the interface area that was in contact with cement. In the unloaded situation, 100% of the interface area was in contact. After the first load application, the stem loaded the cement mantle primarily at its corners. Hence, a pathway from proximal to distal was created at the stem–cement interface at the flat surfaces of the stem (Fig. 9). During the damage process, this pattern
Fig. 8. The amount of subsidence of the stem within the cement mantle defined by the size of the gap created between the prosthetic surface and the proximal cement as a function of the number of loading cycles. When the loads were applied to the structure with unbonded prosthesis, the stem subsided about 25 microns in the cement shaft. The size of this gap increased to about 50 microns after 25 million loading cycles. Reducing the bone quality led to a considerable increase of subsidence.

Fig. 9. The contact areas at the stem-cement interface. Due to small rotations of the stem within the cement mantle, a pathway is formed from proximal to distal. When the cement-bone interface is deficient, the pathway is more substantial.

remained relatively stable in the case that the cement mantle was supported by normal bone. However, in the degraded bone case, the rotations of the stem within the cement mantle increased leading to an enlargement of the space between the stem and the cement mantle, enhancing the pathway mechanism.

DISCUSSION

The FE model used in the analyses and the application of the theory of CDM used in this study are based on a number of assumptions and limitations, which should be kept in mind when interpreting the results. In the FE model, an idealized implantation was assumed and the stem had a straight, double tapered collarless shape, and tip-bearing was prevented by assuming a space under the tip, created by a centralizer. Other prosthetic shapes may lead to less subsidence, particularly when they are provided with collars or flanges. However, this was not investigated in this study. Only one loading case, applied repeatedly, was considered, simulating the stance phase of the gait cycle. The mechanical properties of the bone were assumed to remain constant in time, and to be linear elastic and isotropic. These assumptions are not entirely realistic, particularly for the degraded bone layer around the cement mantle. In the model this layer had an average thickness of about four millimeters and an average Young's modulus of 90 MPa. Retrieval studies indicate that the layer around the cement mantle often consists of fibrous tissue with a thickness of two to five millimeters (Anthony et al., 1990; Justy et al., 1986), and a tangent modulus of about 1 MPa (Hori and Lewis, 1982). Hence, the effects of poor bone quality rather than those of soft tissue interposition were investigated.

In the application of the theory of CDM, uncertainties exist about the assumptions that damage did not have any effect on the mechanical properties of the acrylic cement until the cement had cracked and the actual damage rate was based on uni-directional tensile stress fatigue experiments. In an earlier study we applied the same method, and found that it led to damage rates that are higher than occur in reality, particular when frictional interfaces are present in the structure (Verdonschot and Huiskes, 1995).

Another factor which affects the damage process, but was neglected here, is the visco-elasticity of acrylic cement. In a long-term creep simulation, Verdonschot and Huiskes (1997) have shown that creep of acrylic
cement may reduce the average tensile cement stress by approximately 50%, which will reduce the damage rate in the cement mantle. As a consequence of the assumptions in the FE model, and in the application of the theory of CDM, the results are merely indicative, and conclusions should be limited to general trends.

The stress levels in the cement mantle were relatively low as compared to those reported in the literature. The peak tensile stress generated around the bonded stem in this study was 2.7 MPa, whereas Mann et al. (1992) reported a value of 4.3 MPa and Harrigan and Harris (1992) a value of 5.9 MPa. In the present study debonding increased the peak stress by a factor of 2.6, whereas this factor was 1.7–3 in the studies reported by Mann et al. (1992) and Crowninshield and Tolbert (1983). Regarding the fact that these values are highly sensitive to the FE mesh, prosthetic shape, material properties and loading conditions, the model used in the present study can be considered as adequate for the purpose as posed in the introduction.

The crack patterns around the unbonded stem formed realistic patterns, relative to results of Jasty et al. (1991) as also found in our own retrieved material. To our knowledge, crack patterns similar to the ones generated around the bonded stems modeled in this study have not been reported in the literature. However, this may be due to the fact that in virtually all retrieved specimens the stem–cement interface had (partly) debonded.

Although one might expect that stresses would increase with the number of loading cycles, it was found that they decreased over time. The fractured cement mantle acted as a spacing material between the stem and the bone, thereby reducing the tensile stresses and increasing the compressive stresses in the cement mantle. Therefore the damage rate declined with the number of loading cycles, and the theoretical mechanical survival time of the structure became infinite. Of course, the biological survival time of the reconstruction is then confined by the effects of debris, particulate reactions and other factors.

The effect of bone degradation on the damage rate was higher in the case of an unbonded stem–cement interface as compared to a bonded one. This can be explained by the fact that for bonded stems the load-transfer is governed by the bending stiffnesses of stem and bone (Huiskes, 1980). A change of stiffness of the endosteal bone hardly changes the bending stiffness and therefore has little effect on the stress levels in the cement mantle. Assuming unbonded stem–cement interface conditions, however, subsidence of the stem may occur as the cement mantle fractures. The amount of subsidence is limited by the constraining capacity of the cement and the bone. Reducing the stiffness of the bone leads to an increase of subsidence and a subsequent elevation of cement stresses. In this way, a reduced bone support accelerates the damage process of the cement mantle.

The pathway for debris at the stem–cement interface may already be formed before cracks have developed in the cement. During the damage process, a proximal to distal pathway was formed for wear particles to be transported to distal areas, as proposed by Anthony et al. (1990). Cement failure had little effect on the extent of the pathway, as long as the bone support to the cement mantle was maintained. In the case of degraded bone around the cement mantle, however, it became much larger.

Cement failure had only minor effects on the amount of subsidence of the stem within the cement mantle, provided that the bone bed was normal. Based on the present study, it seems that cement failure alone cannot be responsible for prosthetic subsidence values sometimes reported in the literature (Fowler et al., 1988). However, when the bone layer surrounding the cement mantle is osteolyzed or a soft-tissue layer is formed around the cement mantle (Anthony et al., 1990; Jasty et al., 1986), the present results indicate that the subsidence of the stem may increase considerably. In that way, prosthetic subsidence becomes an indicator of the progress of the failure process of the THA reconstruction, as has been reported by Kährholm et al. (1994).

This study demonstrates that stem–cement debonding not only elevates the initial stress levels in the cement mantle, as demonstrated in earlier studies, but remains to have an impact during the whole failure process of the cement mantle. Stem–cement debonding accelerates the failure process, and promotes the formation of a pathway for debris at the stem–cement interface, particularly when the bone support to the cement mantle is reduced. Hence, this study supports the hypothesis that the survival of cemented THA is enhanced by a firm and lasting bond between the stem and the cement mantle (Harris, 1992), although this may be difficult to realize clinically.

REFERENCES


