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Direct evidence of "damage accumulation" in cement mantles surrounding femoral hip stems retrieved at autopsy: Cement damage correlates with duration of use and BMI

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ABSTRACT

The "damage accumulation" phenomenon has not been *quantitatively* demonstrated in clinical cement mantles surrounding femoral hip stems. We stained transverse sections of 11 postmortem retrieved femoral hip components fixed with cement using fluorescent dye-penetrant and quantified cement damage, voids, and cement–bone interface gaps in epifluorescence and white light micrographs. Crack density (Cr.Dn), crack length-density (Cr.Ln.Dn), porosity, and cement–bone interface gap fraction (c/b-gap%) were calculated, normalized by mantle area. Multiple regression tests showed that cement damage (Cr.Ln.Dn. & Cr.Dn.) was significantly positively correlated ($r^2=0.98$, $p<0.001$) with "duration of use" and body mass index ("BMI") but not cement mantle "porosity". There were significant interactions: "duration of use"*"BMI" was strongly predictive ($p<0.005$) of Cr.Dn.; and "duration of use"*"porosity" was predictive ($p=0.04$) of Cr.Ln.Dn. Stem related cracks accounted for approximately one fifth of Cr.Dn and one third of Cr.Ln.Dn. The mean c/b-gap% was 13.8% but it did not correlate ($r^2=0.01$, $p=0.8$) with duration of use. We concluded that duration-dependent fatigue damage accumulation occurred during *in vivo* use. BMI strongly influenced cement crack length and the rate of new crack formation over time. Voids did not increase the rate of crack initiation but appeared to have promoted crack growth over time. Although not progressive, substantial bone resorption at the cement–bone interface appeared to be common.

1. Introduction

Several series of *in vitro* and *in silico* studies of cemented femoral stem function in total hip arthroplasty (THA) (Cristofolini et al., 2003; McCormack and Prendergast, 1999; Race et al., 2003; Stolk et al., 2004) have been predicated upon the assumption that fatigue damage accumulation occurs in clinical cement mantles. This assumption seemed reasonable since previous workers had documented the existence of fatigue cracks in revision-retrieved cement (Culleton et al., 1993; Topoleski et al., 1990) and in postmortem retrieved stem/cement/bone constructs (Jasty et al., 1991). However, the commonly described "damage accumulation" phenomenon has yet to be *quantitatively* demonstrated in clinical cement mantles. On the contrary, Bishop et al. (2009), who examined an impressively large number of cemented total hip arthroplasty (THA) postmortem retrievals, reported that there was no sign of damage accumulation related to *in vivo* duration of use.

Along with the assumption of damage accumulation, it has long been thought that cement voids cause stress concentrations and initiate cracks in clinical mantles, leading to fatigue failure. A further factor that naturally fits within the damage accumulation scenario is patient obesity. It would seem likely that patients with a higher body mass index (BMI) would generate higher stresses in their cement mantles leading to greater fatigue damage rates.

The unproven status of the damage accumulation scenario, combined with the suspected roles of cement porosity and patient obesity, led us to our primary research question: **(1)** Does cement mantle damage correlate with *in vivo* duration of use and is it influenced by cement mantle porosity or patient body-mass-index (BMI)?

The function of the cement mantle is to support the stem within the bone. Therefore, in considering damage accumulation within the cement mantle, it is appropriate to consider also the mechanical competence of the stem/cement and cement/bone interfaces. Previous workers have suggested that cement damage originates at the stem–cement interface (Jasty et al., 1991), which prompted our second research question: **(2)** Does cement damage preferentially develop at the stem–cement interface?

Several previous studies of retrieved cemented components have suggested that the cement bone interface remains "intact"

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