# Correspondence

# Fixation and loosening of hip prostheses

*To the Editor:*

It was with great interest that I read the recent review article by Dr. Bengt Mjöberg entitled "Fixation and loosening of hip prostheses" (*Acta Orthop Scand* 1991; 62 (5): 500–508).

Although heat injury to the underlying bone may play a role in the early migration of prosthetic components, to imply that heat injury is the major cause of late loosening (given that the cement technique and bone stock are adequate) is rather short-sighted.

Heat injury occurs in all cemented prostheses to a greater or lesser degree (1). However, not all prostheses with conventional cement technique have been shown to migrate or become loose. Indeed, in the study by Dr. Mjöberg (2) in which cements with high and low exotherms were used, only five of 12 acetabular components and one of 12 femoral components were shown by RSA to migrate in the high exotherm group (one of the components migrated in the low exotherm group). If heat injury is a major contributing factor, why did not all the acetabular components in the high exotherm group migrate? Dr. Mjöberg suggests that perhaps body weight, physical activity, etc. may have lead to these differences; however, no further analysis is given. Furthermore, other characteristics of the low exotherm cement ("better particle size gradation, resulting in closer packing and thus reduced mean interspace while still permitting an acceptable dough viscosity") may contribute to the improved results (2).

Our group previously reported the histological results of the implantation of doughy versus preformed, cooled cement plugs of the same volume in the rabbit tibia (3). At 4 months, the interface was similar histologically in the two groups. A fibrous tissue membrane, with the occasional histiocyte, encapsulated the implants. The surrounding bone was also similar in both groups. We concluded that heat injury and monomer leakage are not important factors, or their effects have been completely reversed by 4 months.

The definition of prosthetic loosening is a hotly debated topic. There has been no consensus on the subject. Furthermore, many prostheses that have migrated or are "radiographically loose" by one rating system or another are not clinically loose, i.e., they are not painful or disabling enough to require surgical revision. RSA is a useful tool in accurately delineating the degree of prosthetic *migration*. However, to my knowledge, it has never been shown that components that undergo migration of only a few tenths of a millimeter in the first few months postoperatively *always* go on to continued migration and progressive loosening.

What is the etiology of prosthetic loosening? Clearly, the phenomenon is multifactorial. Aseptic loosening is both mechanical and biological. The mechanical aspects include prosthetic design and material properties, technique of implantation, quality of bone stock, etc. The biological aspects include the myriad of host responses to the implantation of a foreign body and its byproducts. The heat injury to the surrounding bone is just one aspect of this biological response. If this etiology were as important as is implicated by Dr. Mjöberg, then component loosening should be more pervasive.

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## References

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*To the Editor:*

The comments of Dr. Goodman contain some fundamental misunderstandings.

Heat injury does *not* occur in all cemented hip prostheses (1), which is a fact that is important to know to understand the mechanism of loosening. The temperature at the bone-cement interface is dependent on several factors, such as the initial bone temperature, the specific heat generation, the heat conduction, the local cement thickness, etc. (2); and the threshold temperature for heat injury to the acetabular bone is probably not exceeded until the cement thickness goes beyond about 4–5 mm (3). Thus, the fibrous tissue membrane surrounding the 0.2-g cement plugs in the experiment of Dr. Goodman and coworkers can certainly not be explained by heat injury during the polymerization: the surgical trauma to the bone marrow is a more probable explanation (4, 5).

Roentgen stereophotogrammetry can reveal migration of one or both prosthetic components in many asymptomatic hips during the first postoperative year; *some* of these components will probably fail in the future. As pointed out in my review (6), some readers may argue against that a slightly migrating prosthetic component is to be regarded as loose, as such a component usually remains asymptomatic for many years. However, if the definition of loosening is broadened, the definition of nonloosening becomes correspondingly narrower. The advantage of having a narrow definition of prosthetic fixation (rather than of prosthetic loosening) is that components that fulfill this criterion will probably not fail. Such a definition is, of course, unsuitable for clinical decisions on surgical revision, but it is very suitable in research when comparing different prosthetic designs and concepts.

Although pathologic reactions may be complex, an etiology can be simple. Loosening of hip prostheses can be explained as a result of an early prosthetic instability due to *insufficient initial fixation* because of an inadequate interlock or a weak cancellous bone bed, or due to *loss of fixation* because of resorption of a layer of heat-injured bone; other explanations are apparently not necessary. However, if some additional factors (not only having a secondary influence on the process of loosening) can be proved to be necessary, these should, naturally, be included in the etiology of prosthetic loosening, but not until then.

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