

Correspondence

Is the proximal bone resorption around the femoral stem after hip arthroplasty really caused by reduced stress?

Sir,—The proximal bone resorption (often associated with mid-thigh pain) commonly seen around apparently distally well-fixed cementless stems has been supposed to be caused by removal of stress (according to Wolff's law) and therefore called *stress-shielding*. In order to bring about a more physiological loading of the bone, the isoelastic prosthesis and a large number of amazingly different calcar-loading cementless prostheses were introduced in the 1970s and '80s (Morscher 1984), but most of them failed because of loosening. Since then, when most poorly performing cementless prostheses have been taken off the market and the operative technique has improved, both proximal bone resorption and thigh pain have decreased (Engh et al. 2009, Khanuja et al. 2011). Although the etiology of the thigh pain has not been clarified, the most accepted hypothesis is that it in some way derives from stress concentration at the tip of the stem (Lavernia et al. 2004).

The bone resorption has consistently been described to increase progressively proximally to the distal load area—and has also been reported (Bodén et al. 2006, Capello et al. 2009, Yamada et al. 2009) to increase for several years. These observations are difficult to explain on the basis of stress-shielding because, from a logical point of view, the proximal bone must (from the very beginning) either be in contact or not in contact with the implant (i.e. either stress-shielded or not stress-shielded). This means that the pathophysiological mechanism of the proximal bone resorption remains enigmatic.

In addition, bone resorption due to reduced stress has been prevented experimentally by just 4 two-second physiological strain cycles a day (Rubin and Lanyon 1984). Hence: Is the proximal bone resorption around the femoral stem after hip arthroplasty really caused by reduced stress? Probably not.

Proposed mechanism for both proximal bone resorption and thigh pain: Although a prosthetic stem would be well-fixed distally, the difference in flexural stiffness between

the prosthetic stem and the surrounding proximal bone may cause micromovements between the two during normal daily activities: the more proximal, the larger the micromovements (Figures 1–2). These micromovements (e.g. at heel strike) may cause short bursts of high fluid pressure in the interstice between the stem and the proximal bone, which is enough to induce osteolysis (Fahlgren et al. 2010) through a complex series of sometimes significantly painful inflammatory responses to the necrotic bone (Rock and Kono 2008). A similar mechanism for bone resorption and thigh pain, of course, is also applicable to clearly loose femoral components.

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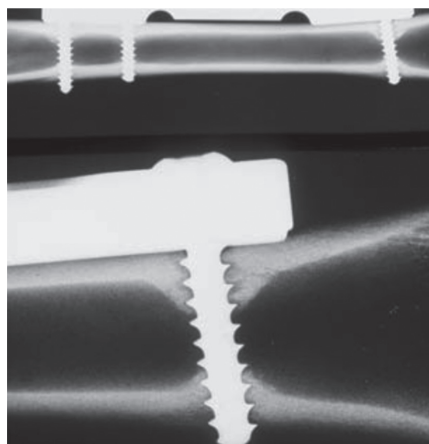


Figure 1. "An instrumented plate (above) allows monitoring of the force transmission to the single screw on the right bridging a segment of a sheep tibia. The two screws on the left lock that end of the plate tightly to the bone. When the sheep stands on this leg, the tibia shortens by a few micrometres. Therefore the single screw undergoes a changing load perpendicular to the long axis of the screw, and loosening at the implant-to-bone surface is seen. This results in 'unstable obstruction'. Loosening of the implant is produced without the load exceeding the strength at the interface." From Perren (2002) with permission.

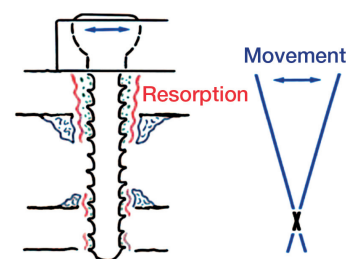


Figure 2. Diagram to show the mechanics of Figure 1: "For a given initial condition the plate pulls and pushes the screw head backwards and forwards. The non-locked screw tilts around an axis within the remote cortex. There is marked resorption where the screw moves while the screw thread near the axis of rotation does not show bone resorption. Similarly, the amount of callus formed corresponds to the amount of displacement or instability." From Perren (2002) with permission.

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