Revision Total Hip Arthroplasty

**FAILURE MECHANISMS OF HIP ARTHROPLASTY**

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Although periprosthetic fracture, dislocation, component fracture, and sepsis are some mechanisms by which hip arthroplasty components fail, aseptic loosening and the contribution of particulate debris to the loosening process are the major contributions to failure. These last two contributions will be examined.

**GENERAL MECHANISM OF FAILURE FOR CEMENTED AND CEMENTLESS IMPLANTS**

Loosening and osteolysis were originally reported around cemented devices. Charnley, with his initial Teflon experience, noted the phenomenon relatively early. This phenomenon was coined “cement disease” in the late 1970s and early 1980s. However, as the experience with cementless prostheses developed, it was recognized that loosening and destructive bone loss (osteolysis) also occurred around cementless devices. The observation of loosening and osteolysis around both cemented and cementless devices helped investigators recognize that any debris generated in the total hip arthroplasty (THA) construct could contribute to the loosening and osteolytic process. Once debris is generated, it induces the destruction of the prosthesis composite bone interface, causing further destruction of bone. This process is thought to involve the macrophage cell line and the release of cytokines and other agents that propagate the process of bone destruction. The material (metal, polyethylene, and cement) as well as the size and morphology of the debris particles may be important in determining the biological response.

**CEMENTED COMPONENT FAILURE**

Mechanical and biological processes are responsible for cemented component failure in the THA construct. Autopsy retrievals of well-functioning THAs have demonstrated cracks in the cement surrounding femoral components, with debonding of the cement from the prosthesis, and preservation of the bone cement interface. In addition, cement cracks in areas of thin cement mantles have been associated with areas of osteolysis. These osteolytic areas have been noted to contain macrophage cell lines laden with polyethylene and cement debris. The cracks in the thin cement mantles provide access channels for particulate debris. On the acetabular side of the construct, in autopsy retrievals, a progressive macrophage-laden membrane has been demonstrated to develop from the periphery toward the dome of the component. The membrane is rich in polyethylene particulate debris. Hence, on the acetabular side of the construct, loosening may be more related to biological processes than to mechanical processes, with the biological response to the small (nanometer-size) polyethylene debris generated at the bearing surface being the main culprit.

**CEMENTLESS COMPONENT FAILURE**

The mechanisms of failure for cementless components have not been as well elucidated as have those for cement components. In addition, the introduction of modularity in the THA construct occurred during the same period that cementless devices began to be implanted. We now appreciate, from the early cementless hip experience, that femoral components with patch or non-circumferential coatings provided access channels for debris from the bearing surface to migrate to the tip of the prosthesis, as well as...
debris from the non-porous portion of the femoral component to migrate to the bearing surface. This mechanism has been responsible for the massive osteolysis demonstrated around some patch-coated femoral devices. Components with sparse areas for ingrowth have been noted to fail through the bone adjacent to the ingrowth pads (by a fatigue failure mode), presumably because of inadequate ingrowth area. Extensively porous-coated femoral components, although small in numbers, have been demonstrated to fail by fatigue fracture through the component when massive stress shielding of bone has occurred. Bone loss of up to 50% has been noted on dual energy radiograph absorptiometry imaging of some extensively coated femoral devices.

On the acetabular side of the cementless construct, failure has been associated with osteolysis and polyethylene wear. Use of large 32 mm femoral heads and, hence, thin polyethylene liners was largely responsible for many early cementless acetabular component failures. However, the contributions of non-conforming acetabular liner-shell interfaces, poor acetabular liner capturing mechanisms, and inadequate modular taper connections have now been recognized.

DISCUSSION

Mechanisms of failure may differ between cementless and cemented THA devices, but mechanical and biological mechanisms probably contribute to the failure of both forms of fixation. Eliminating, or at least minimizing, the generation of particulate debris is paramount in avoiding loosening of and osteolysis around hip arthroplasty components. This will become a more challenging proposition with the use of modular components.

REFERENCES