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The theory of early prosthetic loosening — a concise overview

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Abstract

The theory of early prosthetic loosening states that loosening is initiated during or shortly after surgery, and that the subsequent progression of loosening is affected by biomechanical factors and periprosthetic fluid pressure fluctuations. Later and secondary to loosening, wear particles may affect the progression of loosening. The loosening may increase subclinically for a long period of time and may, when detected, be misinterpreted as a late occurrence of loosening. This concise overview presents the essential features of this theory as applied to hip replacements. Aspects discussed are insufficient initial fixation, early loss of fixation, biomechanical factors, periprosthetic fluid pressure fluctuations, periprosthetic osteolysis, and wear particles.

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Introduction

The theory of early prosthetic loosening is based on a few postulates (the hypothetico-deductive method) that concur with convincing evidence from both clinical and experimental research and states that prosthetic loosening (defined as migration) is initiated during or shortly after surgery and that the subsequent progression of loosening is affected by biomechanical factors and periprosthetic fluid pressure fluctuations.[1][2] This overview presents the essential features of this theory as applied to hip replacements.

Initiation of loosening

The initial fixation may be insufficient due to *poor interlock* (inadequate cement filling, the interposition of tissue debris, etc.)[3][4][5] or because of *poor bone quality* (osteoporosis, rheumatoid arthritis, etc.).[6][7][8][9] Adequate initial fixation does, however, not eliminate the risk of loosening; *resorption of a layer of a necrotic bone bed*(which begins within a few weeks

of surgery) may result in early loss of otherwise optimal fixation.[10][11] However, if loosening is not initiated, a prosthetic component will be well-fixed.

Biomechanical factors

The progression of hip prosthetic loosening, *if initiated*, is affected by the magnitude of the mechanical stress to which the prosthetic components are exposed, which varies according to the patient's body weight and level of physical activity, as well as on the offset of the femoral component (Figure 1), the joint friction (providing friction torque), and the eccentricity of the acetabular component (Figure 2).

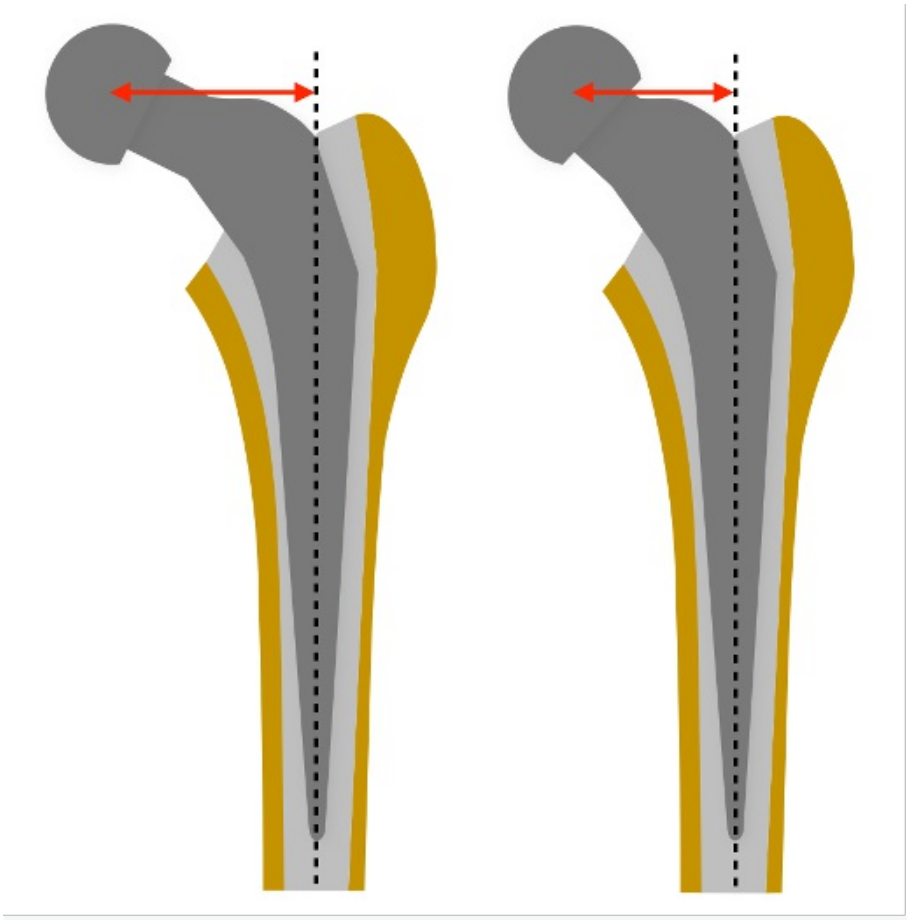

Figure 1. Femoral component offset. Femoral components with a high offset (compared with a low offset) are exposed to greater torque around the longitudinal axis during walking and, especially, when rising from a chair or climbing stairs (which should be avoided during the healing period). Thus, loosened femoral components with a high offset can be expected to develop large micromovements faster and result in earlier clinical failure.

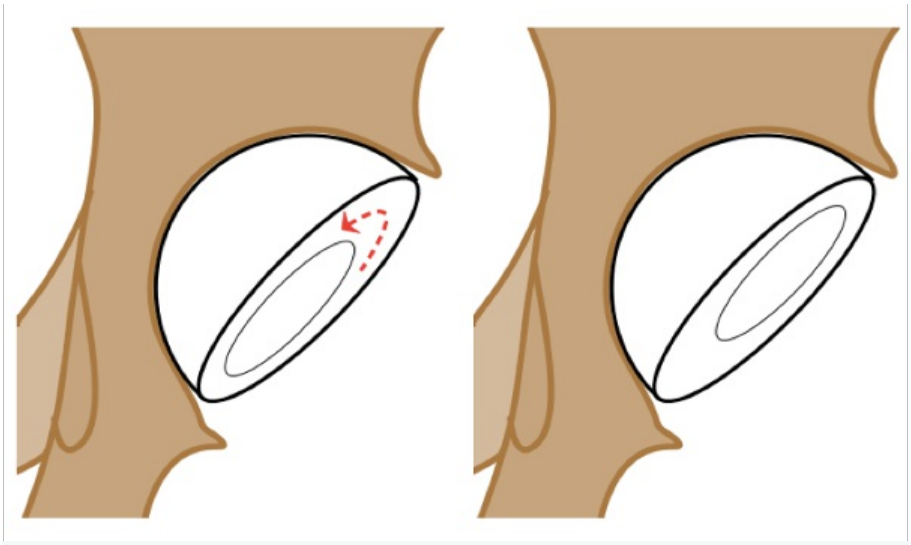

Figure 2. Acetabular component eccentricity. In 15 hip arthroplasties, where the polyethylene cup *by design* was thicker at its upper part to cope with possible wear in the plastic (to the left), the cups rotated through 180 degrees so that, within 3 years, the thicker part was situated at the lower pole of the acetabulum (to the right).[12] After theoretical calculations and mechanical experiments, the authors concluded that the cause of this abnormal rotation was a torque occurring at each step due to the eccentric design of the cup. — Acetabular eccentricity *due to wear* will also cause a torque. Thus, loosened acetabular components with increased wear can, for purely biomechanical reasons, be expected to develop large micromovements faster and result in earlier clinical failure.

Periprosthetic fluid pressure fluctuations and osteolysis

The micromovements of a loosened prosthesis[13][14][15] (or the pumping action of a loose polyethylene liner in an acetabular shell with screw holes[16][17]) may cause devitalizing periprosthetic fluid pressure fluctuations and fluid jets leading to focal periprosthetic osteolysis. The mechanism appears to be that the pressure spikes cause osteocyte death and that these necrotic osteocytes release DAMPs (damage-associated molecular patterns, danger signals, or alarmins),[18][19] which, via a recently clarified unique pattern recognition receptor, reinforce osteoclastogenesis.[20] The prosthetic micromovements and the periprosthetic osteolysis may then reinforce each other and increase subclinically during a long period of time. Eventually, the loosening may be detected on standard radiographs and give the impression of a late occurrence of loosening. Although the existence of genuine late onset of loosening can never be completely ruled out (because it is impossible to prove a negative), late loosening is probably a misinterpretation of late-detected loosening.[2]

Wear particles

Wear particles cannot (contrary what is assumed in the widely accepted hypothesis of particle disease[21][22][23][24])

initiate prosthetic loosening for several reasons: Firstly, histological studies indicate that a stable implant has a biological barrier that prevents wear particles from entering into the bone-cement[25] or into the bone-prosthesis interface.[26] Secondly, even if the biological barrier were defective, experiments have shown that uncontaminated particles do not induce osteolysis.[27][28] Thirdly, radiostereometric analysis indicates that loosening is initiated within a few weeks of surgery and thus long before any significant amounts of wear particles are produced (Figure 3). However, later and *secondary to loosening*, wear particles may affect the progression of loosening as described below – if they appear in the interface.

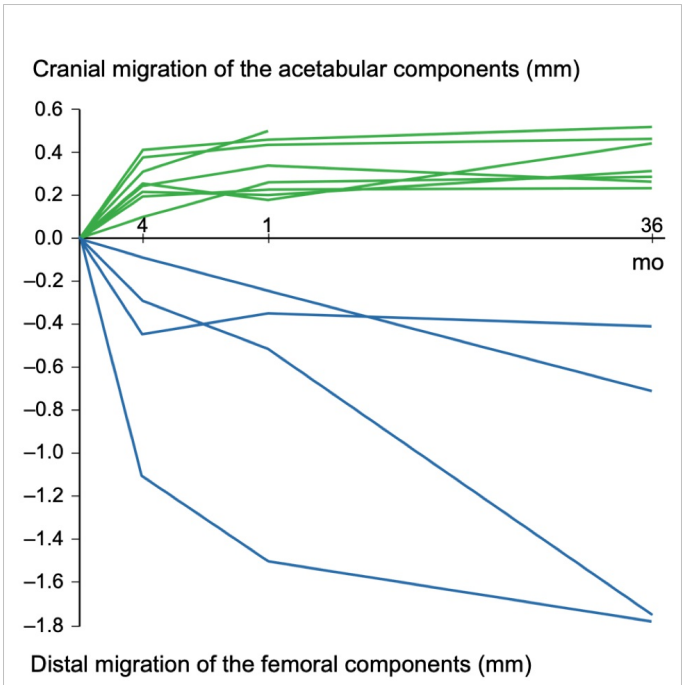

Figure 3. Prosthetic migration along the longitudinal axis. Migration of the migrating eight acetabular (green) and four femoral components (blue) in the series followed by radiostereometric analysis during a period of 3 years [eight acetabular and ten femoral components did not pass the limit (0.2 mm) for significant migration]. Note that in almost all cases, the migration was detected within 4 months of surgery. From Mjöberg *et al.*[29] with permission.

Already during what should have been the healing period (which lasts up to 6–9 months after surgery), some loose *cemented* components probably produce significant amounts of cement particles due to abrasive micromovements at bone-cement interface. These cement particles may become DAMPs-coated and thereby inhibit bone ingrowth and prevent osseointegration.[30] The larger the early migration and the larger the abrasive micromovements, the more DAMPs-coated bone-formation-inhibiting cement particles in the bone-cement interface. For cemented prostheses, therefore, atraumatic surgery and initial prosthetic stability are crucial in ensuring a low risk of loosening.[2]

Polyethylene wear is slow unless promoted by three-body wear due to cement particles that have become trapped between the joint surfaces,[31][32][33] i.e. *uncemented* prostheses produce very small amounts of polyethylene particles during the healing period. If prosthetic stability is achieved during the healing period, the bone-prosthesis interface will (as mentioned) be sealed by a biological barrier against wear particles entering from the joint cavity.[25][26] This may explain why certain uncemented femoral components (unlike cemented femoral components) may withstand an early migration and still achieve bone ingrowth and even osseointegration.[34][35][36]

Conclusions

Prosthetic loosening is primarily due to inadequate initial fixation or an early loss of fixation, and secondarily due to biomechanical factors and periprosthetic fluid pressure fluctuations. Later, DAMPs-coated wear particles, if they appear in the interface, may inhibit bone ingrowth and thereby affect the progression of loosening. The loosening may increase subclinically during a long period of time and may, when detected, be misinterpreted as a late occurrence of loosening.

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