

Review Article

The theory of early prosthetic loosening — a concise overview

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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 after surgery) may result in early loss of otherwise optimal fixation.^{[10][11]} However, if loosening is not initiated, a prosthetic component will remain 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).

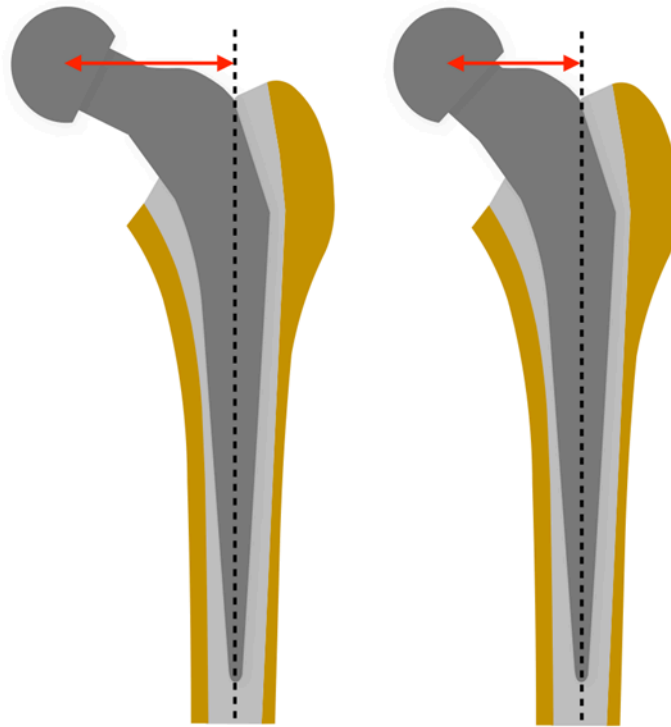


Figure 1. Femoral component offset. Femoral components with a high offset (compared with a low offset) are exposed to greater torque around the longitudinal axis during walking and, especially, when rising from a chair or climbing stairs (which should be avoided during the healing period). Thus, loosened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

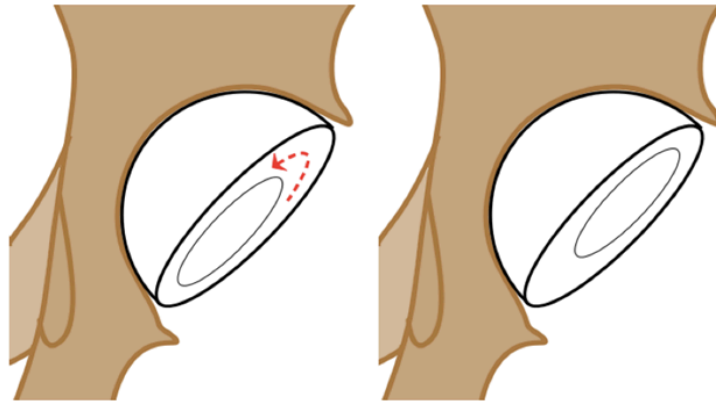


Figure 2. Acetabular component eccentricity. In 15 hip arthroplasties, where the polyethylene cup *by design* was thicker at its upper part to cope with possible wear in the plastic (to the left), the cups rotated through 180 degrees so that, within 3 years, the thicker part was situated at the lower 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.^[21]

Wear particles

Wear particles cannot (contrary to 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 after 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.

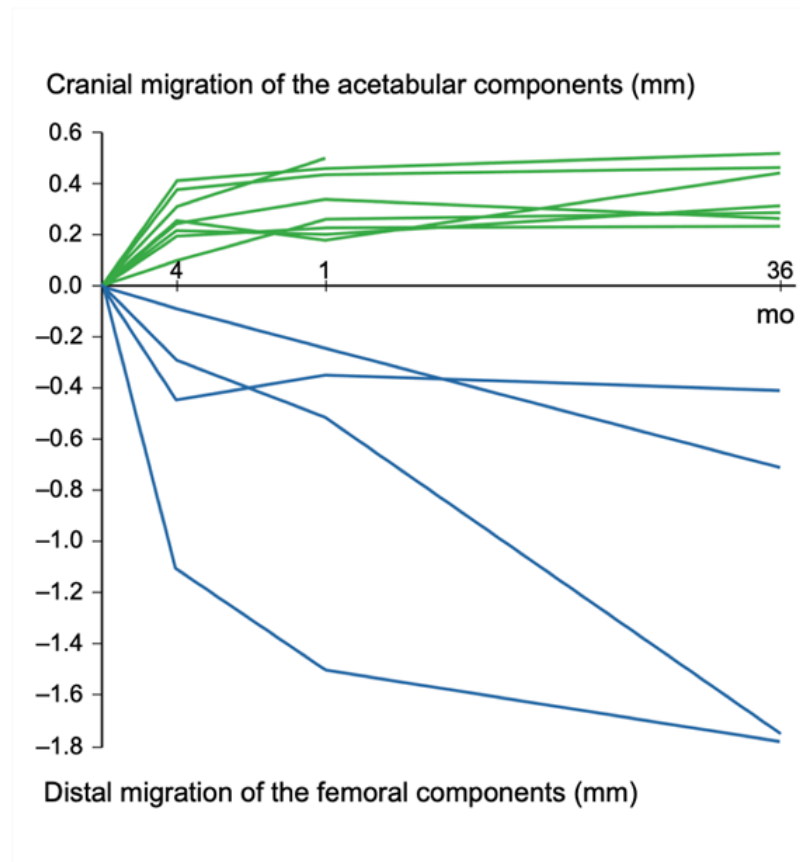


Figure 3. Prosthetic migration along the longitudinal axis. Migration of the migrating eight acetabular (green) and four femoral components (blue) in the series followed by radiostereometric analysis during a period of 3 years [eight acetabular and ten femoral components did not pass the limit (0.2 mm)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

References

1. [△]Mjöberg B. *The theory of early loosening of hip prostheses. Orthopedics* 1997; 20: 1169-1175 [PMID: 9415912]
2. ^{a, b, c}Mjöberg B. *Is early migration enough to explain late clinical loosening of hip prostheses? EFORT Open Rev* 2020; 5: 113-117 [DOI: 10.1302/2058-5241.5.190014]
3. [△]Krause WR, Krug W, Miller J. *Strength of the cement-bone interface. Clin Orthop Relat Res* 1982; (163): 290-299 [DOI: 10.1097/00003086-198203000-00043]
4. [△]Kristiansen B, Jensen JS. *Biomechanical factors in loosening of the Stanmore hip. Acta Orthop Scand* 1985; 56: 21-24 [DOI: 10.3109/17453678508992972]
5. [△]Dohmae Y, Bechtold JE, Sherman RE, Puno RM, Gustilo RB. *Reduction in cement-bone interface shear strength between primary and revision arthroplasty. Clin Orthop Relat Res* 1988; (236): 214-220 [DOI: 10.1097/00003086-198811000-00029]
6. [△]Franzén H, Mjöberg B, Önnarfält R. *Early loosening of femoral components after cemented revision. A roentgen stereophotogrammetric study. J Bone Joint Surg Br* 1992; 74: 721-724 [DOI: 10.1302/0301-620X.74B5.1527]

7. [△]Önsten I, Bengnér U, Besjakov J. Socket migration after Charnley arthroplasty in rheumatoid arthritis and osteoarthritis. A roentgen stereophotogrammetric study. *J Bone Joint Surg Br* 1993; 75: 677-680 [DOI: 10.1302/0301-620X.75B5.8376420]
8. [△]Snorrason F, Kärrholm J, Holmgren C. Fixation of cemented acetabular prostheses. The influence of preoperative diagnosis. *J Arthroplasty* 1993; 8: 83-90 [DOI: 10.1016/s0883-5403(06)80112-9]
9. [△]Aro HT, Alm JJ, Moritz N, Mäkinen TJ, Lankinen P. Low BMD affects initial stability and delays stem osseointegration in cementless total hip arthroplasty in women: a 2-year RSA study of 39 patients. *Acta Orthop* 2012; 83: 107-114 [DOI: 10.3109/17453674.2012.678798]
10. [△]Sih GC, Connelly GM, Berman AT. The effect of thickness and pressure on the curing of PMMA bone cement for the total hip joint replacement. *J Biomech* 1980; 13: 347-352 [DOI: 10.1016/0021-9290(80)90014-7]
11. [△]Toksvig-Larsen S, Franzén H, Ryd L. Cement interface temperature in hip arthroplasty. *Acta Orthop Scand* 1991; 62: 102-105 [DOI: 10.3109/17453679108999232]
12. [△]Ramadier JO, Lelong P, Dupont JY. Rotation anormale de certaines cupules cotyloïdiennes excentrées scellées [Rotational displacement of eccentric cups cemented in the acetabulum (author's transl)]. *Rev Chir Orthop Reparatrice Appar Mot* 1980; 66: 507-514 [PMID: 6451002]
13. [△]Aspenberg P, van der Vis H. Fluid pressure may cause periprosthetic osteolysis. Particles are not the only thing. *Acta Orthop Scand* 1998; 69: 1-4 [DOI: 10.3109/17453679809002344]
14. [△]Skoglund B, Aspenberg P. PMMA particles and pressure--a study of the osteolytic properties of two agents proposed to cause prosthetic loosening. *J Orthop Res* 2003; 21: 196-201 [DOI: 10.1016/S0736-0266(02)00150-X]
15. [△]Fahlgren A, Bostrom MP, Yang X, Johansson L, Edlund U, Agholme F, Aspenberg P. Fluid pressure and flow as a cause of bone resorption. *Acta Orthop* 2010; 81: 508-516 [DOI: 10.3109/17453674.2010.504610]
16. [△]Manley MT, D'Antonio JA, Capello WN, Edidin AA. Osteolysis: a disease of access to fixation interfaces. *Clin Orthop Relat Res* 2002; (405): 129-37 [DOI: 10.1097/00003086-200212000-00015]
17. [△]Walter WL, Walter WK, O'Sullivan M. The pumping of fluid in cementless cups with holes. *J Arthroplasty* 2004; 19: 230-234. [DOI: 10.1016/j.arth.2003.10.005]
18. [△]Rock KL, Kono H. The inflammatory response to cell death. *Annu Rev Pathol* 2008; 3: 99-126 [DOI: 10.1146/annurev.pathmechdis.3.121806.151456]
19. [△]Murao A, Aziz M, Wang H, Brenner M, Wang P. Release mechanisms of major DAMPs. *Apoptosis* 2021; 26: 152-162 [DOI: 10.1007/s10495-021-01663-3]

20. [△]Andreev D, Liu M, Weidner D, Kachler K, Faas M, Grüneboom A, Schlötzer-Schrehardt U, Muñoz LE, Steffen U, Grötsch B, Killy B, Krönke G, Luebke AM, Niemeier A, Wehrhan F, Lang R, Schett G, Bozec A. Osteocyte necrosis triggers osteoclast-mediated bone loss through macrophage-inducible C-type lectin. *J Clin Invest* 2020; 130: 4811–4830 [DOI: 10.1172/JCI134214]
21. [△]Goodman SB, Gibon E, Pajarinen J, Lin TH, Keeney M, Ren PG, Nich C, Yao Z, Egashira K, Yang F, Konttinen YT. Novel biological strategies for treatment of wear particle-induced periprosthetic osteolysis of orthopaedic implants for joint replacement. *J R Soc Interface* 2014; 11: 20130962 [DOI: 10.1098/rsif.2013.0962]
22. [△]Sukur E, Akman YE, Ozturkmen Y, Kucukdurmaz F. Particle disease: A current review of the biological mechanisms in periprosthetic osteolysis after hip arthroplasty. *Open Orthop J* 2016; 10: 241–251 [DOI: 10.2174/1874325001610010241]
23. [△]Kandahari AM, Yang X, Laroche KA, Dighe AS, Pan D, Cui Q. A review of UHMWPE wear-induced osteolysis: the role for early detection of the immune response. *Bone Res* 2016; 4: 16014 [DOI: 10.1038/boneres.2016.14]
24. [△]Goodman SB, Gallo J. Periprosthetic osteolysis: mechanisms, prevention and treatment. *J Clin Med* 2019; 8: 2091 [DOI: 10.3390/jcm8122091]
25. [△]Linder L, Carlsson ÅS. The bone–cement interface in hip arthroplasty. A histologic and enzyme study of stable components. *Acta Orthop Scand* 1986; 57: 495–500 [DOI: 10.3109/17453678609014777]
26. [△]Sundfeldt M, Widmark M, Johansson CB, Campbell P, Carlsson LV. Effect of submicron polyethylene particles on an osseointegrated implant: an experimental study with a rabbit patello–femoral prosthesis. *Acta Orthop Scand* 2002; 73: 416–424 [DOI: 10.1080/00016470216314]
27. [△]Aspenberg P, Herbertsson P. Periprosthetic bone resorption. Particles vs movement. *J Bone Joint Surg Br* 1996; 78: 641–646 [PMID: 8682835]
28. [△]Skoglund B, Larsson L, Aspenberg PA. Bone-resorptive effects of endotoxin-contaminated high-density polyethylene particles spontaneously eliminated in vivo. *J Bone Joint Surg Br* 2002; 84: 767–773 [DOI: 10.1302/0301-620X.84B5.11775]
29. [△]Mjöberg B, Franzén H, Selvik G. Early detection of prosthetic-hip loosening. Comparison of low- and high-viscosity bone cement. *Acta Orthop Scand* 1990; 61: 273–274 [DOI: 10.3109/17453679008993518]
30. [△]Mjöberg B. Hip prosthetic loosening and periprosthetic osteolysis: A commentary. *World J Orthop* 2022; 13: 574–577 [DOI: 10.5312/wjo.v13.i6.574]
31. [△]Willert HG, Bertram H, Buchhorn GH. Osteolysis in alloarthroplasty of the hip. The role of bone cement fragmentation. *Clin Orthop Relat Res* 1990; (258): 108–121. [PMID: 2203567]

32. [△]McKellop HA, Sarmiento A, Schwinn CP, Ebrahmdadeh E. In vivo wear of titanium-alloy hip prostheses. *J Bone Joint Surg Am* 1990; 72: 512-517 [PMID: 2324137]
33. [△]Wang A, Essner A. Three-body wear of UHMWPE acetabular cups by PMMA particles against CoCr, alumina and zirconia heads in a hip joint simulator. *Wear* 2001; 250: 212-216. [DOI: 10.1016/S0043-1648(01)00643-3]
34. [△]Weber E, Sundberg M, Flivik G. Design modifications of the uncemented Furlong hip stem result in minor early subsidence but do not affect further stability: a randomized controlled RSA study with 5-year follow-up. *Acta Orthop* 2014; 85: 556-561 [DOI: 10.3109/17453674.2014.958810]
35. [△]Aro E, Alm JJ, Moritz N, Mattila K, Aro HT. Good stability of a cementless, anatomically designed femoral stem in aging women: a 9-year RSA study of 32 patients. *Acta Orthop* 2018; 89: 490-495 [DOI: 10.1080/17453674.2018.1490985]
36. [△]Floerkemeier T, Budde S, Lewinski GV, Windhagen H, Hirschler C, Schwarze M. Greater early migration of a short-stem total hip arthroplasty is not associated with an increased risk of osseointegration failure: 5th-year results from a prospective RSA study with 39 patients, a follow-up study. *Acta Orthop* 2020; 91: 266-271 [DOI: 10.1080/17453674.2020.1732749]

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