venous pressure is perhaps a more important pain mechanism over time.

7. As long as cartilage is intermittently loaded, which it always is, we doubt that the size of the contact area is significant in terms of cartilage nutrition.

8. The advancement of the tidemark associated with thickening of the subchondral plate may be the most significant change that occurs in osteoarthrotic joints. Deterioration of articular cartilage could well be the result as much of increased remodeling of the metaphysis as wear of cartilage. Thinned cartilage is especially at risk for horizontal fibrillation, which unlike vertical splits will be fatal to cartilage integrity. Catastrophic cartilage "wear" is most likely from fragmentation, but slow thinning, in the absence of fragmentation, probably comes from advancement of the tidemark rather than detrition. We believe OA is not a final common pathway but rather a final common result. There is no pathophysiological cascade of changes that occurs in lockstep in all cases. One can achieve a final common result by a variety of pathways.

9. OA preferentially appears in individuals of high bone mass. We suspect they are individuals with minor incoordination resulting in repetitive impulsive loading of their joints. Certain joints, we believe, are relatively immune from OA because they are spared the impulsive loads, due to particular anatomic or other factors. The ankle is the best example.

10. Secondary OA does occur in joints with earlier injuries, diseases, or deformities but interestingly has a variable penetrance. Minor incoordination may be an important variable determining this penetrance.

11. Osteoporosis and osteoarthrosis (primary or secondary) are divergent disorders.

12. OA is a physiological imbalance of joints from mechanical factors. Inflammatory changes are secondary. Thus, the term osteoarthrosis is appropriate and the term osteoarthritis is misleading.

We are all grateful for Professor Hulth's many significant contributions to our understanding of osteoarthrotic process and to his thoughtful and insightful letter. My cowriters and I continue to be greatly encouraged by his observations and support.

ERIC L. RADIN, M.D.
Henry Ford Hospital
Detroit, Michigan

Dear Sir:

We congratulate Dr. Incavo and his coauthors[1] on their excellent study of an extremely common problem with cementless femoral components. Laboratory investigations of clinical problems are important and help each of us as orthopedic surgeons make intelligent treatment decisions. However, we would invite some reinterpretation of their conclusions in relation to the clinical situation.

They suggest (on page 179) that the surgeon remove the prosthetic stem (or broach) once a crack is created and then cerclage the femur before reinserting the component. Theoretically, this would be the ideal way. Practically, it is nearly impossible since the crack is almost always produced when the surgeon attempts to obtain an extremely tight fit at final seating during the procedure. Once the prosthesis is inserted, the fit obtained is usually good, and it would be unreasonable to expect the surgeon to remove the prosthesis, cerclage it, and then impact it again. This approach incurs all the risks of removal of a fairly stable prosthesis with the risk of possible further crack propagation on second insertion of the prosthesis. It is easier to immediately cerclage the prosthesis in place with perhaps more than one wire, a solution that is not commented on in the present study. In our own experience with hoop-stress calcar fractures,[3] no difference in clinical outcome

was found whether zero, one, or two cerclage wires were used to gain stability. These clinical findings were based on intraoperative surgical assessment in the clinical and roentgenographic outcome of patients who suffer intraoperative *stable* fractures.

In addition, the authors suggest that postoperative weight bearing should be delayed for all fractures regardless of stability. They state that regardless of the patterns or stability of the fracture, longitudinal cracks may be a risk for catastrophic failure during normal weight bearing, and that postoperative weight bearing should be delayed. In our experience, patients were treated with a standard weight-bearing protocol and demonstrated no significant difference in their outcome. In addition to our study, Sharkey *et al.*[4] and Mallory *et al.*,[2] who are quoted in the present work, have both demonstrated no difference in the clinical outcome of patients with stable femoral fractures in cementless arthroplasty. We believe the present author's conclusion that patients with these fractures not be allowed to weight bear is unfounded.

CARLOS LAVERNIA, M.D.
MICHAEL A. MONT, M.D.
DAVID S. HUNGERFORD, M.D.
KENNETH A. KRACKOW, M.D.
The Johns Hopkins University
Department of Orthopaedic Surgery
Baltimore, Maryland

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Reply to Lavernia *et al.*

Dear Sir:

We thank Drs. Lavernia, Mont, Hungerford, and Krackow for their comments and observations concerning longitudinal crack propagation of the femur around uncemented femoral prostheses.

When we began this study, we hoped to demonstrate sufficient stability so that weight bearing could be allowed postoperatively for patients sustaining a proximal fracture during surgery. Admittedly, this was a difficult proposition since we were using embalmed anatomic specimen bone. Additionally, there are many variables in surgical practice that we could not address. The prosthesis we examined has design characteristics that should increase the risk of femoral crack propagation (*i.e.*, very large anteroposterior and mediolateral dimensions, an anatomic bow, and no collar). Nonetheless, in only two of eight specimens tested did the force to propagate the fracture exceed three times body weight (70 kg). We are pleased to hear that in Dr. Lavernia's experience standard postoperative weight bearing demonstrated no significant difference in outcome.

In preliminary studies performed in our laboratory, we also examined the use of more than one cerclage wire to prevent crack propagation. We found that using two wires provided no additional benefit. The next step