Letters to The Editor

Professionalism in Publishing

To The Editor:

I applaud JBJS for emphasizing the American Academy of Orthopaedic Surgeons' mandatory Standards of Professionalism (SOPs) for publishing and reporting in the editorial "Professionalism in Publishing" (2006;88:2323-5) by Brand et al. Thank you for raising awareness of the AAOS Professional Compliance Program (PCP), the SOPs on Research and Academic Responsibilities, and the AAOS grievance procedure.

I wish to point out that this editorial contains an error related to the procedure for grievances and their appeals as well as a couple of statements that may lead to some confusion. Near its end, the editorial states, "(w)hen the AAOS Board of Directors makes a recommendation for action, either the grievant or the respondent may file an appeal." Actually, the Board of Directors receives recommendations on professional compliance actions from the AAOS Committee on Professionalism and, if there is an appeal, from the AAOS Judiciary Committee. Grievants and/or respondents may appear before the board as well. However, Board decisions are final regarding any professional compliance actions taken against AAOS Fellows or members. Board decisions take effect immediately and are not subject to further AAOS review, consideration, or appeal. Appeals are heard by the Judiciary Committee before consideration by the Board of Directors.

The editorial notes, "While the AAOS guidelines are intended for AAOS Fellows…." The term "guidelines" may imply to some individuals recommendations that readers can consider, and perhaps modify, to fit their needs. The SOPs for Research and Academic Responsibilities set out baseline mandatory minimum standards for acceptable conduct, not guidelines. Also, the SOPs apply not only to active, inactive, and emeritus AAOS Fellows, but also to AAOS members, including more than 4000 residents and candidates. Fellows and members (except international and honorary) make up the sole population that can become grievants and respondents.

I sincerely hope that contributors to JBJS will not be parties to grievances filed with AAOS for alleged violations of the SOPs. However, AAOS is working hard to ensure that all grievances will be heard and adjudicated under a system that provides fairness and due process to both the grievant and the respondent.

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Disclosure: The author did not receive any outside funding or grants in support of his research for or preparation of this work. Neither he nor a member of his immediate family received payments or other benefits or a commitment or agreement to provide such benefits from a commercial entity. No commercial entity paid or directed, or agreed to pay or direct, any benefits to any research fund, foundation, division, center, clinical practice, or other charitable or nonprofit organization with which the author, or a member of his immediate family, is affiliated or associated.

R.A. Brand, J.J. Jacobs, and J.D. Heckman reply:

The Editors appreciate Dr. Mandell's clarification of these important points.

Richard A. Brand, MD
Editor-in-Chief,
Clinical Orthopaedics and Related Research

Joshua J. Jacobs, MD
Consulting Editor for Research,
The Journal of Bone and Joint Surgery,
American Volume

James D. Heckman, MD
Editor-in-Chief,
The Journal of Bone and Joint Surgery,
American Volume

These letters originally appeared, in slightly different form, on jbjs.org. They are still available on the web site in conjunction with the article to which they refer.

This letter and response will also be published in *Clinical Orthopaedics and Related Research*.

Influence of Acetabular Coverage on Hip Survival After Free Vascularized Fibular Grafting for Femoral Head Osteonecrosis

To The Editor:

I read with great interest the article entitled "Influence of Acetabular Coverage on Hip Survival After Free Vascularized Fibular Grafting for Femoral Head Osteonecrosis," (2006;88:2152-8), by Roush et al., and the commentary by Steinberg and Steinberg[1]. Indeed, this article and the commentary by Marvin Steinberg[1] represent the views and thoughts of two senior authors with opposing opinions on how to treat osteonecrosis. Urbaniak[2] supports vascularized bone-grafting, whereas Steinberg[3] supports avascular cancellous bone-grafting. While the intent of the article by Roush et al. was not to document the clinical efficacy of either type of bone-grafting, the conclusions made therein invariably influence one's ability to recognize a potential limitation of free vascularized fibular grafting, and it is from this observation that I would like to offer a few comments.

Roush et al. retrospectively reviewed a consecutive series of 200 hips in 160 patients with osteonecrosis of the femoral head who had undergone free vascularized fibular grafting. They found that, of the hips with a center-edge angle of ≤30°, 55% demonstrated progression of collapse and 45% were converted to a total hip arthroplasty. In contrast, of the hips with a center-edge angle of >30°, only 10% demonstrated progression of collapse and only 6% were converted to a total hip arthroplasty. Roush et al. encouraged the reader to consider acetabular dysplasia as an independent risk factor with a negative influence on prognosis and cited a study of children with congenital dislocation of the hip, by Gregosiewicz and Wosko[4] , to support their position. However, one must carefully consider this conclusion. Gregosiewicz and Wosko[4] reported that the highest risk for os-

teonecrosis in children is associated with a number of factors: (1) an age less of than six months, (2) severe acetabular dysplasia, (3) the use of an abduction apparatus such as the Frejka pillow by outpatients, and (4) a "frog-leg" position afte reduction. The observation by Gregosiewicz and Wosko[4] implies increased contact forces on a soft, predominantly cartilaginous femoral head after reduction. An age of less than six months correlates well with the congenital nature of acetabular dysplasia. In contrast, the mean age at the time of surgery in the article by Roush et al. was 33.6 years, with the worst degree of collapse being only 3 mm (in eleven patients). If the intent of Roush et al. was to imply a causative role for acetabular dysplasia, then one would think that the adult hips in their series would have been more arthritic, particularly after thirty-three years. Clearly, one must contemplate how dysplastic hips with a center-edge angle of ≤30° functioned for an average of thirty-three years and then had development of a primary bone disease (i.e., osteonecrosis), with the etiologic associations being known in 75% of the 200 hips, only to collapse after free vascularized fibular grafting. Roush et al. seemed to suggest that the failed femoral heads would have survived had it not been for the acetabular dysplasia. Yet the acetabular dysplasia was present prior to free vascularized fibular grafting. Could these dysplastic hips have benefited from a different joint preservation procedure? Although Steinberg and Steinberg[1] suggest that perhaps the femoral heads with the lower center-edge angles were deformed, implying a propensity to collapse, one must recognize how free vascularized fibular grafting may potentiate the demise of a femoral head with a dysplastic acetabulum. Thus, is it the dysplastic acetabulum that portends a poor outcome, as suggested by Roush et al., or is it the treatment chosen, i.e., free vascularized fibular grafting?

The surgical technique of free vascularized fibular grafting as described by Urbaniak et al.[2] comprises thorough débridement of the femoral head. The core tract, ranging in diameter from 16 to 19 mm, is designed to avoid occlusion of the peroneal vessels and to prevent tension on the anastomosis. This large core tract likely destabilizes the femoral head and neck and potentiates collapse where contact forces are greatest, i.e., a dysplastic acetabulum. Although Urbaniak et al.[2] described passing a guidewire into the necrotic lesion within the femoral head, it is far more important that the starting point of the guidewire along the lateral cortex be situated to prevent tension on the anastomosis once the large core tract is created. This requirement likely determines the position of the fibula and may prevent optimal placement in view of the acetabular dysplasia. Roush et al. fell short of identifying this potential limitation of free vascularized fibular grafting and concluded by asking the reader to preoperatively quantify the extent of dysplasia for prognostic and *possibly* surgical planning purposes. One wonders what other surgical plans exist when Roush et al. comment that "the surgical procedure has remained essentially unchanged since the publication of our original reports."

Mont et al.[5] and Rosenwasser et al.[6] demonstrated that avascular bone-grafting combined with thorough débridement can be successfully applied to select patients with osteonecrosis of the femoral head and that good outcomes can be achieved. Continued emphasis on the role of the *vascularized* fibula in the treatment of osteonecrosis might invariably prevent one from recognizing the features that vascular techniques (i.e., free vascularized fibular grafting) and avascular techniques (i.e., the trapdoor procedure as described by Mont et al.[5] and the lightbulb procedure as described by Rosenwasser et al.[6]) have in common, namely, thorough débridement. Importantly, the thorough débridement of the trapdoor/lightbulb procedure leaves the femoral neck substantially intact. Thus, when the acetabulum is dysplastic, could more femoral heads be saved with use of avascular techniques that provide better subchondral support after thorough débridement?

I commend Roush et al. for critically reviewing the failures of free vascularized fibular grafting in a series of 200 hips, but I strongly believe that the article would have been more helpful had the authors discussed how the surgical technique of free vascularized fibular grafting, having not changed in nearly twenty years, may have contributed to destabilizing a femoral head with increased contact forces due to acetabular dysplasia. The work of the senior authors, Urbaniak[2] and Steinberg[7], is well recognized in the literature. However, as a new generation of orthopaedists develops interest in this devastating disease, we must recognize that perhaps free vascularized fibular grafting cannot be uniformly applied to all hips as implied by Roush et al. More importantly, treatment protocols should focus on the features that vascular and avascular bone-grafting techniques have in common when such features are associated with good clinical outcomes.

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Disclosure: The author did not receive any outside funding or grants in support of his research for or preparation of this work. The author, or a member of his immediate family, received, in any one year, payments or other benefits in excess of $10,000 or a commitment or agreement to provide such benefits from a commercial entity (Orthopedic Sciences, Inc. [OSI]). No commercial entity paid or directed, or agreed to pay or direct, any benefits to any research fund, foundation, division, center, clinical practice, or other charitable or nonprofit organization with which the author, or a member of his immediate family, is affiliated or associated.

T.F. Roush, S.A. Olson, R. Pietrobon, L. Braga, and J.R. Urbaniak reply:

We appreciate Dr. Brannon's comments and questions. He raises several necessary points of clarification.

First, he correctly emphasizes the limitations of citing the article by Gregosiewicz and Wosko[4] pertaining to the causation of osteonecrosis in children with known congenital hip dislocation. His mention of the discrepancy regarding the age of the patients in that series and the age of the patients in our report is indeed accurate. We cited that article to demonstrate the paucity of literature regarding the relationship between osteonecrosis and developmental dysplasia of the hip. The primary utility that we found from that article was related to its postulation that osteonecrosis tended to be more severe in hips with developmental dysplasia than in those without dysplasia. Clearly, any additional extrapolation from the article by Gregosiewicz and Wosko[4] would be inaccurate because of the age and disease differences between their series of patients and ours. The article by Hadley et al.[8], which was also cited in our manuscript, was instead a more appropriate validation of our hypothesis as it em-

phasized the increased contact stresses on the femoral head in patients with developmental dysplasia of the hip.

The second point raised by Dr. Brannon pertained to the causative agent of the poor outcomes for dysplastic hips as cited in our report. We certainly now believe that the dysplastic acetabulum in itself portends a worse outcome for hips with osteonecrosis that are treated with free vascularized fibular grafting. We are not in a position to attribute the poor results to the free vascularized fibular grafting treatment chosen as every patient in our study had the same procedure and patients with an increased center-edge angle tended to do quite well after the procedure. Furthermore, as Drs. Steinberg and Steinberg[1] point out, the 39% overall rate of progressive collapse of the femoral head and conversion to total hip arthroplasty that we found in our series compares favorably with reports in the existing literature regarding non-arthroplasty treatments of osteonecrosis of the femoral head. This is particularly striking because 30% (sixty) of the 200 hips in our series demonstrated some degree of developmental dysplasia (a center-edge angle of ≤25°). To investigate this issue further, we would need to compare the rates of developmental dysplasia of the hip in other series, which are not accessible because these values have not been routinely recorded. It is our hope that some degree of assessment of developmental dysplasia of the hip will be employed and recorded in the future to further elucidate the role of this condition in the outcome of osteonecrosis treatment and perhaps to devise a more refined treatment strategy for these patients.

Dr. Brannon also questioned whether the core tract made during the free vascularized fibular grafting procedure may potentiate collapse of dysplastic hips. This is a distinct possibility, although we did not address this issue in our study. Perhaps a lower-diameter threshold of core tract exists when the presumed increase in contact force from a dysplastic acetabulum is at work? By this rationale, smaller core tracts (such as those made during nonvascularized fibular grafting), the use of porous tantalum implants[9], or core decompression may play a larger role in the treatment of those patients. Before those techniques can be recommended in this setting, however, basic science studies evaluating the diameter threshold of core tracts in the setting of developmental dysplasia of the hip would need to be performed.

A further point of desired clarification regarded our statement that concluded the abstract: "An estimation of the degree of hip dysplasia should be included in the preoperative assessment of patients with osteonecrosis of the femoral head for prognostic and possibly surgical planning purposes." Rather than planning a variation of the free vascularized fibular grafting procedure, our intent with this statement is to challenge future researchers and investigators to consider alternative procedures in the setting of developmental dysplasia of the hip and osteonecrosis. In particular, this pertains to procedures addressing the deficient acetabulum.

Finally, Dr. Brannon questioned whether femoral heads may be better saved with the use of avascular techniques that provide better subchondral support after thorough débridement. This concern is quite valid and, although theoretically accurate, will require further elucidation by obtaining a more accurate assessment of critical thresholds of core tract and graft strength in the setting of developmental dysplasia of the hip.

In conclusion, we appreciate the insightful questions and comments expressed by Dr. Brannon regarding our study. After proposing that a relationship exists between (a lack of) acetabular coverage and the outcomes of free vascularized fibular grafting treatment for osteonecrosis of the femoral head, our results pose many more questions regarding the optimal treatment for these patients. We look forward to further research that addresses this important problem.

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These letters originally appeared, in slightly different form, on jbjs.org. They are still available on the web site in conjunction with the article to which they refer.

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Knowing What Our Primary Care Providers Need To Know

To The Editor:

I applaud the efforts of Lynch and colleagues to further enlighten us on the crisis in musculoskeletal education in the United States. Their article "Important Demographic Variables Impact the Musculoskeletal Knowledge and Confidence of Academic Primary Care Physicians" (2006;88:1589-95) indicates that our medical providers lack a consistent basic fund of musculoskeletal knowledge, which our medical schools should be providing to every graduate. I do have several comments.

As a rheumatologist who has spent his entire career educating internal medicine residents in the area of musculoskeletal medicine, I do not think the Freedman and Bernstein twenty-five-item assessment test[1] accurately reflects the issues most primary care internists face in practice. It is a one-size-fits-all test and is really an orthopaedic assessment that is heavily weighted toward orthopaedic trauma (nine of the twenty-five items). While this may be more relevant to certain primary care providers (family practice physicians or rural internists), it does not reflect the musculoskeletal problems seen on a daily or weekly basis by most primary care internists. I know that chairs of medical departments have endorsed the test, but I am not sure that in most cases they would be the best group to evaluate ortho-

paedic issues. I understand a more directed assessment is under study currently.

My second comment is more general. A previous paper on this topic by Lynch et al.[2] delineated what a rural internist was actually seeing in clinic with regard to musculoskeletal problems. I think this type of information should be our starting point for teaching future generations of primary care providers. Teaching should address their future needs rather than be based on a hypothetical curriculum generated by expert opinion and panels. While a good place to start, this cannot substitute for evidence-based curriculum development. Knowing what they need to know will help us know what to teach and allow us to develop more useful assessment tools.

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Disclosure: The author did not receive any outside funding or grants in support of his research for or preparation of this work. Neither he nor a member of his immediate family received payments or other benefits or a commitment or agreement to provide such benefits from a commercial entity. No commercial entity paid or directed, or agreed to pay or direct, any benefits to any research fund, foundation, division, center, clinical practice, or other charitable or nonprofit organization with which the author, or a member of his immediate family, is affiliated or associated.

J.R. Lynch, G.A. Schmale, D.C. Schaad, and S.S. Leopold reply:

We thank Dr. Gardner for his thoughtful comments concerning our paper, and we agree that this issue warrants further study.

Dr. Gardner's first concern is that the twenty-five-item test originally created by Drs. Freedman and Bernstein does not accurately reflect the musculoskeletal knowledge required of practicing primary care physicians. The specific issue is that some questions concern topics in orthopaedic trauma, which may be beyond the scope of practice of some primary care providers. Indeed, a minority of the questions do refer to musculoskeletal trauma (36%, nine of twenty-five questions). However, these particular questions do not ask the primary care physician for management decisions; rather, they test the ability of the physician to recognize orthopaedic emergencies and identify the anatomic structures that might be in jeopardy when these emergencies occur. While this might not be relevant to the practice of every provider, certainly these issues are likely to come up in the practices of primary care physicians whose scope of practice includes covering high school sports teams, urgent care facilities, and walk-in clinics. Perhaps more importantly, it is worth recognizing that the remainder of the examination—approximately two-thirds of the overall test—covers what would be considered "general practice" by any definition, including diagnoses such as arthritis, compressive neuropathies, back pain, health maintenance screening as it relates to the musculoskeletal system, and common infectious and oncological concerns. The initial evaluation, treatment, and appropriate referral of patients with these conditions are routinely performed by primary care physicians. In fact, the physicians tested in this study performed worse on questions dealing with office-based musculoskeletal care than they did on those dealing with orthopaedic emergencies. For instance, 89% of the participating physicians were able to recognize compartment syndrome as a surgical emergency needing appropriate referral; however, only 58% of the providers understood the difference between osteoporosis and osteomalacia. This must be considered within the purview of a primary care physician, given that in one study osteoporosis was found to be the third most common musculoskeletal problem addressed by primary care physicians[2]. Lastly, to our knowledge, the test instrument created by Drs. Freedman and Bernstein is the only previously published, field-tested examination of musculoskeletal knowledge that has been endorsed by program directors of internal medicine programs from across the country, who, incidentally, weighted the importance of this test more heavily than it was originally weighted by the test's creators[3].

Of course, Dr. Gardner is right that no examination can cover all topics, and none can be completely relevant to all providers. We also agree that there might be other examinations that could be developed to test particular groups of primary care providers or to emphasize different kinds of musculoskeletal content. We encourage Dr. Gardner and others interested in these topics to write and, importantly, to validate other test instruments and to perhaps examine other populations to see whether the findings that we observed at a top academic primary care program generalize well to other physician populations. If such validation can be made, perhaps it will prompt the changes not only to medical school education but also to graduate medical education and continuing medical education that our work suggests are necessary.

Again, we thank Dr. Gardner for his interest, and encourage his work toward the creation of evidence-based assessment tools that will help improve the musculoskeletal knowledge and confidence of primary care providers.

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These letters originally appeared, in slightly different form, on jbjs.org. They are still available on the web site in conjunction with the article to which they refer.

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Custom Patellofemoral Replacement in the Presence of Trochlear Dysplasia

To The Editor:

I read with interest the article "Custom Patellofemoral Arthroplasty of the Knee" (2006;88:1475-80), by Sisto and Sarin. It is not uncommon for young patients with isolated patellofemoral arthritis to have a dysplastic trochlea. Instead of being concave, the trochlea is flat or even convex. In such a setting, I have always worried that a custom implant that duplicates the articulating anatomy of the patient's patella (convex) will lead to an unstable construct. The authors do not appear to have encountered this problem. Would they comment on this?

As an aside, I think that their patellofemoral replacement (or any other) would be equally indicated in frail and elderly pa-

tients who are unlikely to have deterioration of the other compartments in their lifetime.

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Disclosure: The author did not receive any outside funding or grants in support of his research for or preparation of this work. Neither he nor a member of his immediate family received payments or other benefits or a commitment or agreement to provide such benefits from a commercial entity. No commercial entity paid or directed, or agreed to pay or direct, any benefits to any research fund, foundation, division, center, clinical practice, or other charitable or nonprofit organization with which the author, or a member of his immediate family, is affiliated or associated.

D.J. Sisto and V.K. Sarin reply:

We thank Dr. Grelsamer for his interest in our study and for the opportunity to discuss the custom approach to patellofemoral arthroplasty in more detail. Dr. Grelsamer correctly observes that our published series does not include patients with a flat or convex femoral trochlea. While we agree that the treatment of patients with isolated patellofemoral arthritis and concomitant severe trochlear dysplasia can be a challenge, we believe that a custom approach to patellofemoral arthroplasty is a reasonable treatment option for this indication.

The posterior (bone-contact) surface of the custom patellofemoral implant is designed to replicate the native surface and is defined by a preoperative computed tomography scan. In contrast, the prosthetic femoral trochlea is designed to conform to the articular radius of the mating patellar implant and is thickened laterally and medially along its borders to compensate for any lack of native medial-lateral stability. The thickness of the custom implant along the patellar tracking arc is designed to reestablish the anterior position of the femur.

Stability of the implant construct and extensor mechanism is fundamental to the successful outcome of any patellofemoral arthroplasty, including the custom approach. The presence of trochlear dysplasia, as Dr. Grelsamer points out, further underscores the importance of achieving a stable and balanced extensor mechanism intraoperatively. A convex trochlea may increase the tendency to overstuff the patellofemoral joint, and this possibility must be addressed during the design of the custom implant and during the implantation procedure.

Finally, we agree with Dr. Grelsamer that patellofemoral arthroplasty is indicated in elderly patients with isolated patellofemoral arthritis whose medial-lateral compartments would not be expected to become symptomatic during their lifetime. The purpose of our study, though, was to report on the use of custom patellofemoral arthroplasty in a younger population.

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These letters originally appeared, in slightly different form, on jbjs.org. They are still available on the web site in conjunction with the article to which they refer.

Is Arthroscopic Release Indicated?

To The Editor:

We read the article entitled "Arthroscopic Release and Latissimus Dorsi Transfer for Shoulder Internal Rotation Contractures and Glenohumeral Deformity Secondary to Brachial Plexus Birth Palsy" (2006;88:564-74), by Pearl et al., with interest. We congratulate the authors for demonstrating the potential for glenohumeral remodeling in children with brachial plexus birth injuries. We appreciate their attempts to clarify the surgical indications for tendon transfer as opposed to release of the internal rotation contracture. However, we take issue with their belief that arthroscopic release adds anything, and we believe that it may, in fact, be somewhat inadequate. The authors state that "releasing the subscapularis from its origin failed in one of five children." The authors do not clarify whether those failures were in patients with posterior dislocation or subluxation or in patients in whom the humeral head was centered. When the glenohumeral joint is centered, we have never encountered such a failure. For children with long-standing subluxation or dislocation, we have taken an individualized approach. After performing our subscapular slide, we release tight structures anteriorly by means of intramuscular lengthening of the pectoralis, partial release of the coracobrachialis tendon, partial coracoidectomy, and/or release of the coracohumeral ligament. Using this approach, we have never failed to achieve equivalent full external rotation of the affected shoulder. We do not immobilize the shoulder in full external rotation postoperatively for fear of overstretching these structures and causing too much weakness and loss of internal rotation power. We wonder whether the authors are immobilizing shoulders in too much external rotation postoperatively.

Finally, we see no logic in performing releases through the arthroscope. One would not release a heel cord contracture with ankle arthroscopy. In addition to the risk to the axillary nerve (with one patient in the study losing 40° of elevation), four patients "had severe functional loss of internal rotation, and this prompted consideration of additional intervention, such as an internal rotation osteotomy." Alain Gilbert has abandoned anterior release at the insertion of the subscapularis for this very reason[1]. We believe that anterior tenotomy of the subscapularis renders too much functional loss of internal rotation.

Because five (15%) of thirty-three patients had a serious complication, we believe that this approach must be reconsidered.

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Disclosure: The authors did not receive any outside funding or grants in support of their research for or preparation of this work. Neither they nor a member of their immediate families received payments or other benefits or a commitment or agreement to provide such benefits from a commercial entity. No commercial entity paid or directed, or agreed to pay or direct, any benefits to any research fund, foundation, division, center, clinical practice, or other charitable or nonprofit organization with which the authors, or a member of their immediate families, are affiliated or associated.

M.L. Pearl, B.W. Edgerton, P.A. Kazimiroff, and K. Wong reply:

We thank Dr. Price and colleagues for their interest and the opportunity to further clarify our approach to the internal rotation contractures that develop in so many of these children. It was our frustration with methods similar to the ones that they de-

scribe that led us to explore and develop the arthroscopic approach. Approximately 70% of children who present with internal rotation contractures will have glenohumeral deformity[2,3]. The children with centered glenohumeral joints to whom the correspondents allude represent a minority. For the majority of patients with advanced contractures and deformity, there is increasing evidence that the described surgical method does not consistently relocate the glenohumeral joint and may impede optimal skeletal development.

Van der Sluijs and colleagues[4] and Birch and colleagues[5] independently reported on yet another method of releasing the subscapularis, one that releases it from its insertion, aiming for a step lengthening of the tendon while preserving the anterior capsule (when possible). Both groups of authors reported that in patients with severe contractures it is not possible to achieve reduction of the glenohumeral joint without releasing the anterior capsule. In fifteen of the nineteen patients in the series of van der Sluijs et al., release of the contracture could not be achieved without release of the anterior capsule. In addition, Birch and colleagues postulated that in many instances, excessive retroversion results in an external rotation contracture once the glenohumeral joint is reduced. For retroversion of >40°, they recommended an internal rotational osteotomy as part of the same procedure[5]. In that study, seventy of 183 patients were managed with combined soft-tissue release and rotational osteotomy. Another recent study, by Waters and Bae, demonstrated that procedures that avoid the anterior capsule fail to result in glenohumeral remodeling[6], further establishing that extra-articular procedures are incompletely effective in such cases. So the question becomes, for children with severe contractures and/or those with advanced glenohumeral deformity, do surgeons who do not address the subscapularis tendon and the underlying joint capsule consistently achieve a complete release that will allow glenohumeral remodeling? The foregoing studies and our own suggest not.

It is not clear why the correspondents compare attempted arthroscopic release of an extra-articular structure such as the Achilles tendon with our procedure, but, contrary to their intention, this comparison does highlight the appeal of minimizing surgical trauma with percutaneous and arthroscopic approaches. The comparison also brings to light the reality that most caretakers of these children have considerable expertise in areas other than shoulder surgery (neurosurgery, plastic surgery, hand surgery, and pediatric orthopaedics). Only surgeons who are experienced in shoulder arthroscopy should consider this form of management. Our program, and others that have adopted the arthroscopic approach, combine the efforts of a hand/peripheral nerve surgeon and a shoulder specialist to address the complexity of many of these problems.

As discussed in our report, the existing literature is woefully inadequate in describing the loss of internal rotation that comes from any method of treatment, and better methods are needed to quantify and document this concern. This is certainly true of the clinical results reported by the correspondents and in all other clinical series that employ similar methodology. It is somewhat ironic that our attempt to deal with this issue candidly has become a point of vulnerability in a field that has, until recently, skirted the issue. It is a mistake to think that children who undergo a release of the subscapularis from its origin have normal subscapularis function. The truncated and atrophied subscapularis muscle can be seen on magnetic resonance imaging and corresponds to a limitation of active internal rotation on clinical examination. In fact, loss of internal rotation in some of our earlier open cases exceeded that seen in many of our arthroscopic cases. Yes, it is true that we would opt for improved internal rotation in nearly all of our patients. However, at the present time, the state of the art does not offer these children a perfect solution that provides for a complete range of motion in all directions with normal glenohumeral development. We concur with Dr. Birch et al. that a contemporary surgical approach must achieve glenohumeral reduction for children with skeletal remodeling potential and then must restore the functional orientation of the arm if needed. This can be done with open or arthroscopic means, in one or more operations.

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These letters originally appeared, in slightly different form, on jbjs.org. They are still available on the web site in conjunction with the article to which they refer.

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Patellar Complications Following Distal Femoral Replacement After Bone Tumor Resection

To The Editor:

In "Patellar Complications Following Distal Femoral Replacement After Bone Tumor Resection" (2006;88:2225-30), Schwab et al. draw our attention to an aspect of prosthetic reconstruction after a distal femoral resection that is important and often overlooked. They, correctly in my view, state, "the position of the joint line deserves special attention . . ." Unfortunately, I believe they do not appreciate the cause of their most common complication, which is elevation of the joint line. This causes so-called patella baja and patellar impingement. The authors reference an article by Thorpe et al. and suggest that some of their patients may have had fibrous bands as described in that study[1]. The patients in the study by Thorpe et al. did not have patella baja caused by fibrous bands, and they were cured with an arthroscopic release of the bands. Schwab et al. mention scarring of the patellar tendon that was possibly related to relative devascularization. The reference they cite describes only the vascularity of the patella, not of the patellar tendon, and actually notes that the vascular supply is abundant[2]. To support a theory that the patellar tendon shortens in the postoperative period, the au-

thors should show serial lateral radiographs demonstrating this shortening.

The surgeon controls the position of the joint line. The amount of bone removed from the tibia must be equal to the thickness of the tibial component, including the polyethylene, if the joint line is to be maintained. If insufficient bone is removed, or if a thicker polyethylene component is added to lengthen the reconstruction, the joint line is raised. The authors recognize this but then indicate that they measure the femoral resection with the tibial resection and combine these to decide the length of the reconstruction. The femur should be measured separately from the tibia, and the femoral component should equal the length of the femur that was resected. The tibial component should be equal to the thickness of the tibia that was resected. If the reconstruction is short, the femur should be lengthened. The thickness of the tibial polyethylene should not be increased. Implanting a thicker tibial polyethylene tray raises the joint line, producing patella baja and risking patellar impingement. Their mean LT/HI (patellar tendon length/height of patellar tendon insertion) ratio of 1.3 is clearly abnormal and indicates that, on the average, the joint lines are abnormally high.

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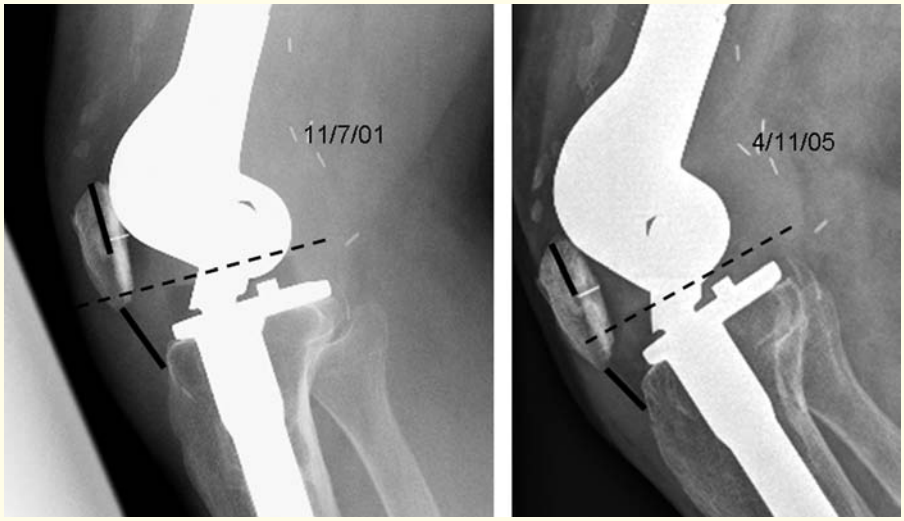

Fig. 1

Lateral radiographs of the knee show the changing relationship of the patellar tendon length over three years and five months. Heavy dark lines define the tendon length and its relationship to the patellar length. It was well beyond the patellar button marker initially, and it was well short of it at the time of the last follow-up. The dashed lines highlight the top of the polyethylene tibial bearing.

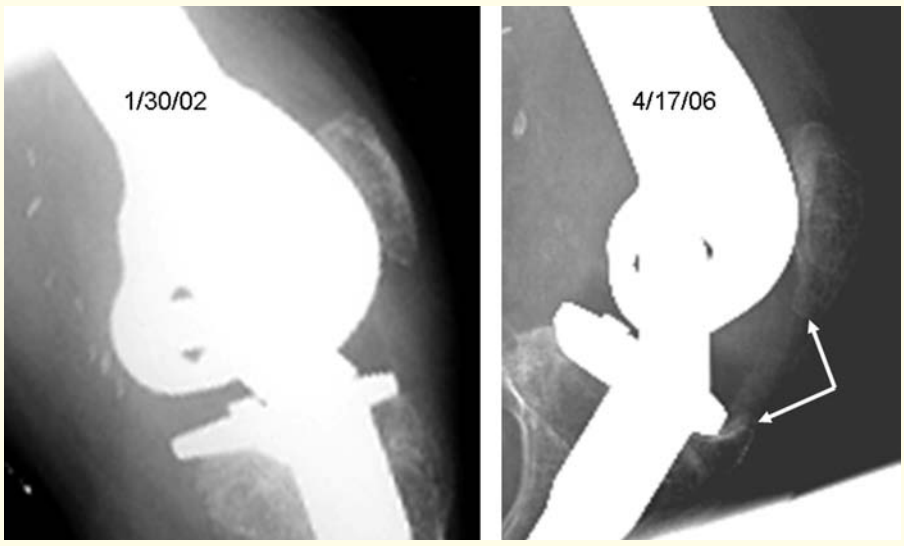

Fig. 2

During four years and three months of follow-up, bone grew from the tibia and from the inferior pole of the patella (arrows), shortening the functional patellar tendon.

Disclosure: The author did not receive any outside funding or grants in support of his research for or preparation of this work. Neither he nor a member of his immediate family received payments or other benefits or a commitment or agreement to provide such benefits from a commercial entity. No commercial entity paid or directed, or agreed to pay or direct, any benefits to any research fund, foundation, division, center, clinical practice, or other charitable or nonprofit organization with which the author, or a member of his immediate family, is affiliated or associated.

J.H. Healey and J.H. Schwab reply:

We are delighted that our article sparked discussion of the importance of reestablishing the joint line in knee replacements after tumor resection. Dr. Springfield presents a clear tutorial on how to accomplish this: match the femoral resection and replacement lengths, and then match the tibial resection and replacement lengths. This tautology is everyone's goal. For various reasons, we were not always able to achieve it.

Nevertheless, discussion of the average technical adequacy of the procedure misses the point. Citing the high joint line in our patients as the cause ("their mean LT/HI . . . ratio of 1.3 is clearly abnormal and indicates that, on the average, the joint lines are abnormally high"), Dr. Springfield overlooks the paradox that the LT/HI ratio was low in patients with impingement (0.9). This vitiates his argument. Curiously, the high ratios that he understandably criticizes (1.4) were found in patients without any impingement. When the reader looks at the data, it is clear that the ratios are not so important and another cause for patellar impingement should be sought. Even when the joint line is reproduced accurately (in our case, with a minimum 15 to 17-mm tibial cut, 3-mm metal-backed tray, and either a 12 or 14-mm polyethylene tibial bearing), the problem still occurs. Why?

The vascular hypothesis is just that—a hypothesis. These cases are much different from regular joint resurfacing procedures. All of the synovium, capsule, and fat pad are removed, and the geniculate vessels are interrupted. The "abundant blood supply" described by Kayler and Lyttle does not exist in these reconstructed joints. The open question is whether this devascularization is sufficient to contribute to patellar contracture and impingement.

Finally, the two appended figures (Figs. 1 and 2) document the changes that occur to the patellar tendon length, the inferior pole of the patella, and, in children, the anterior tibial plateau. There is little doubt that this phenomenon is real.

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These letters originally appeared, in slightly different form, on jbjs.org. They are still available on the web site in conjunction with the article to which they refer.

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