Letters to the Editor

Dear Editor:

It was with great interest that we read the article by Chappell et al., "Effect of Fatigue on Knee Kinetics and Kinematics in Stop-Jump Tasks" (July 2005, pages 1022-1029). The article addresses an important problem, given the anecdotal evidence from athletes, clinicians, and coaches, suggesting that ACL injuries and many other injuries occur most often when athletes are fatigued. However, although the experiments presented in this article appear to be well designed, there are inconsistencies in the terminology used to define the loads calculated from the inverse dynamics approach. The inconsistent definitions of loading in this article appear to have led to a conclusion regarding fatigue and ACL injury that is not supported by the results of this study.

The inconsistencies in terminology used in this article lie in the reporting of internal versus external loading. Studies that report loads calculated from inverse dynamics maintain a convention of reporting either net externally applied loads from ground reaction, gravity, or inertial forces, or net internal forces from the muscles, passive soft tissues, and bone that must balance these external loads. However, in this article, the authors appear to have mixed these 2 conventions. For example, the knee flexion/extension moment during landing is reported by the authors as an extension moment (Figure 8). Because the ground reaction force and inertia of the body center of mass must act to flex the knee, this extension moment must be the internal moment that the knee extensor mechanism must generate to counteract the external flexion moment. In contrast, the authors report a knee valgus moment in female subjects during landing. It appears they are reporting an externally applied valgus moment in this case, apparently to be consistent with the literature the authors cite and the results of other studies. However, changing the convention from internal to external load without a clear definition can lead to a misinterpretation of the results. In fact, the inconsistencies in defining the load convention appear to contribute to a misinterpretation of the results in this study.

The meaning of the anterior-posterior shear forces existing at the knee during landing appears to have been misinterpreted, as a result of not consistently defining internal versus external loads. The force shown in Figure 5 in this article does not reflect the force in the ACL, as suggested in this article, but rather the opposite. The posterior ground reaction force due to braking results in a loading situation at the knee that protects the ACL rather than endangering it. The externally applied force at the ground is transmitted up through the ankle, resulting in an external force that pushes the tibia backward. Intuitively, this posterior force from the ground is analogous to a posterior drawer test, which causes the ACL to become slack and the posterior cruciate ligament to become taut. However, the authors interpret an increase in this force as a worse condition for ACL injury. Thus, the conclusion that fatigue-induced increases in this shear force may increase strain on the ACL and risk of injury in both female and male subjects is not supported based on the results of this article. In fact, the opposite conclusion fits these results, that the increase in shear force due to fatigue, as shown in Figure 7, protects the ACL from injury. Future studies should focus on the other components of loading that change owing to fatigue to determine which ones would lead to a greater risk of ACL injury in fatigued athletes.

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REFERENCES


Dear Editor:

We read with great interest the recent article by Chappell et al titled “Effect of Fatigue on Knee Kinetics and Kinematics in Stop-Jump Tasks” (July 2005, pages 1022-1029). The authors concluded that “increased anterior shear force indicates a possible increased strain on the ACL and thus an increased risk for ACL injury.” This interpretation of knee kinetics is, however, incorrect. During the stop jump, the body undergoes a large deceleration, and there is an external ground reaction force pushing the tibia posteriorly. The authors’ free body diagram of the lower leg (Figure 1) correctly shows that the femur exerts an anterior shear force on the tibia, as required to balance a posterior force from the ground. However, the only anatomical structures that can pull the tibia anteriorly are the posterior cruciate ligament and, at low flexion angles, the patellar tendon. The ACL can therefore only be strained if the shear force of the patellar tendon becomes larger than the required total anterior shear force, but no attempt was made to estimate this effect. Even if such analysis were added, the fact remains that an increased posterior ground reaction force will decrease (not increase) the strain in the ACL. This is consistent with the well-known drawer test, in which an anterior, not posterior, external force is applied to strain the ACL.

Therefore, the correct interpretation is, in our opinion, that the increased anterior tibial shear force, as reported by Chappell et al, makes the sagittal plane force balance safer for the ACL, and not
Figure 1. Free body diagram of forces acting on the lower leg during a stop-jump task. From Chappell JD, Herman DC, Knight BS, Kirkendall DT, Garrett WE, Yu B. Effect of fatigue on knee kinetics and kinematics in stop-jump tasks. Am J Sports Med. 2005;33:1022-1029.

less safe. This may well be a protective adaptation to compensate for other effects of fatigue that would put the ACL at increased risk, such as decreased flexion angles and increased valgus loading. If the interpretations of Chappell et al were incorporated into injury prevention programs, one might erroneously advise athletes not to have such adaptations.

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Scott G. McLean, PhD
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DOI: 10.1177/0363546505283276

Authors’ Response: We thank Drs Ajit M. Chaudhari and Thomas P. Andriacchi, and Drs Antonie J. van den Bogert and Scott G. McLean for their comments on our recent publication entitled “Effect of Fatigue on Knee Kinetics and Kinematics in Stop-Jump Tasks” (July 2005).

Drs Chaudhari and Andriacchi expressed their concern for our definition of knee moments. We used knee-joint resultants through the entire text in this publication, which Drs Chaudhari and Andriacchi referred to as internal forces and moments. We did not change the convention from internal to external load in this publication. We did, however, find an error in our original Figure 9 after reading Drs Chaudhari and Andriacchi’s comments, which we think is the cause of the confusion in terminology. The signs for valgus and varus moments in Figure 9 should be reversed. Accordingly, the valgus and varus moments mentioned in the presentation and discussion of the results should all be reversed. We sincerely apologize for this error and the confusion due to this error, and we thank Drs Chaudhari and Andriacchi for drawing our attention to the error. The corrected Figure 9 in our original publication is shown here in Figure 1.

Drs Chaudhari and Andriacchi and Drs van den Bogert and McLean expressed similar concerns on our use of the proximal tibial anterior shear force as an indicator of ACL loading, stating that the anterior-posterior component of the ground-reaction force vector has the same effect on the ACL or PCL as a drawer test and that the posterior ground-reaction force protects the ACL.

The anterior-posterior component of the ground-reaction force vector acting on the foot during the stop-jump task does not have the same effects as a posterior drawer test. During the drawer test, the tester applies an anterior or posterior draw force at the proximal end of the tibia in an open kinetic chain of the lower extremity (the distal end of the kinetic chain is free). During the landing of the stop-jump task, the ground applies a ground-reaction force with a posterior component on the foot in a closed kinetic chain of the lower extremity (the distal end of the kinetic chain is fixed on the ground). The mechanical effects of the external anterior or posterior force on the ACL or PCL are not the same in these 2 situations. A posterior drawer test bears little resemblance to the situation described by Drs Chaudhari and Andriacchi and Drs van den Bogert and McLean. Clinicians can be assured that if they have a patient who is seated and whose leg is hanging from the examining table, pushing the foot posteriorly will not stretch the PCL. The posterior force on the foot makes the knee flex, not translate.

A posteriorly directed force is applied to the foot during the landing of the stop-jump task. This force would cause the knee to flex. If motor control demands required the patient or athlete to remain standing, then a quadriceps force must balance the tendency of flexion because of the posterior ground-reaction force; this would require quadriceps contraction to resist the external knee flexion moment with an internal extension moment. The quadriceps applies force on the tibia through the extensor mechanism and the patellar tendon. The quadriceps contraction can provide an anterior shear force on the tibia with the knee at a small flexion angle.

Our use of proximal tibial anterior shear force as an indicator of ACL loading in the stop-jump task is based on careful analysis of the lower biomechanics and experimental data. The ACL loading
Figure 2. Major knee forces in the sagittal plane. $F_{\text{Ham}}$, hamstring tendon force; $F_{\text{ACL}}$, ACL loading; $F_{\text{pr}}$, patellar tendon force; $\phi$, ACL elevation angle; $\beta$, hamstring tendon-tibial shaft angle; $\alpha$, patellar tendon-tibial shaft angle.

TABLE 1
Sagittal Plane Kinematic and Kinetic Data at Peak Ground-Reaction Force Collected From a Stop-Jump Trial of a Female Recreational Athlete

<table>
<thead>
<tr>
<th>Data</th>
<th>Horizontal</th>
<th>Vertical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee joint center coordinates, m</td>
<td>0.1885</td>
<td>0.4835</td>
</tr>
<tr>
<td>Ankle joint center coordinates, m</td>
<td>0.1974</td>
<td>0.1384</td>
</tr>
<tr>
<td>Peak ground-reaction forces, N</td>
<td>-356</td>
<td>637</td>
</tr>
<tr>
<td>Center-of-pressure coordinates, m</td>
<td>0.3350</td>
<td>0</td>
</tr>
</tbody>
</table>

*The positive horizontal axis is pointing in the anterior direction; the positive vertical axis is pointing upward. The knee flexion angle is 20°.

$F_{\text{ACL}}$ due to sagittal plane biomechanics in the stop-jump task can be expressed as

$$F_{\text{ACL}} = \frac{F_{\text{AP}}}{\cos \phi},$$

(1)

where $F_{\text{AP}}$ is the knee anterior draw force and $\phi$ is the ACL elevation angle (Figure 2). According to McLean et al.,² the knee anterior draw force can be expressed as

$$F_{\text{AP}} = F_{\text{pr}} \sin \alpha - F_{\text{Ham}} \sin \beta - F_{\text{ks}},$$

(2)

where $F_{\text{pr}}, F_{\text{Ham}},$ and $F_{\text{ks}}$ are the patellar tendon force, hamstring tendon force, and proximal tibial anterior shear force (knee-joint resultant force in the anterior direction), respectively; while $\alpha$ and $\beta$ are the patellar tendon-tibial shaft angle and hamstring tendon-tibial shaft angle, respectively (Figure 2). It is quite clear in the inverse dynamics of the lower extremity that $F_{\text{ks}}$ is correlated to the posterior ground-reaction force. We believe that the concern shown in our use of $F_{\text{ks}}$ as an indicator of ACL loading and the claim that the posterior ground-reaction force protects the ACL are mainly based on the role of $F_{\text{ks}}$ in Equation 2 and the obvious correlation between $F_{\text{ks}}$ and the posterior ground-reaction force.

What Drs Chaudhari and Andricchi and Drs van den Bogert and McLean did not realize is that $F_{\text{pr}}$ is also correlated to the posterior ground-reaction force. The posterior ground-reaction force on the foot creates an external flexion moment relative to the knee, which needs to be balanced by an internal knee extension moment. The greater the posterior ground-reaction force is, the greater the internal knee extension moment and the greater the $F_{\text{pr}}$ are because the knee tension moment is created by the $F_{\text{pr}}$. The effect of the posterior ground-reaction force on $F_{\text{pr}}$ is much greater than that on $F_{\text{ks}}$, because the moment arm of the posterior ground-reaction force relative to the knee is much greater than that of $F_{\text{pr}}$. The moment arm of $F_{\text{pr}}$ is less than 0.05 m (according to Smidt), whereas the vertical distance from the ground to the center of the knee joint in the stop-jump task is more than 0.4 m. This condition means that an increase of 1 N in the posterior ground-reaction force will result in an increase of more than 8 N in $F_{\text{pr}}$. The effect of the external flexion moment on ACL loading has been acknowledged when describing the mechanism of ACL injuries while skiing in a recent letter to the editor by Drs McLean, van den Bogert, and others.³ The decreased knee flexion angle will not increase ACL loading without a large internal knee extension moment because of quadriceps muscle contraction. These considerations combined together indicate that the increasing posterior ground-reaction force will increase ACL loading and that there should be a positive correlation between $F_{\text{ks}}$ and $F_{\text{ACL}}$ when the knee flexion angle is small.

The effect of the posterior ground-reaction force on ACL loading can be clearly demonstrated using a simplified simulation based on the real kinematic and kinetic data for a randomly selected stop-jump trial of a randomly selected female subject in our database (Table 1). To simplify the simulation, we neglected the mass and moment of inertia of the lower leg and foot. Hamstring co-contraction was not considered in this simulation. The patellar tendon moment arm, patellar tendon-tibial shaft angle, and ACL elevation angle were estimated from the knee flexion angle as 0.481 m, 19°, and 55°, respectively.¹ The actually observed peak posterior and vertical ground-reaction forces during landing were 356 N and 637 N, respectively. The peak posterior ground-reaction force was increased to 656 N with a 100-N increment. The ACL loading was calculated using Equations 1 and 2.

TABLE 2
Simulation Results

<table>
<thead>
<tr>
<th>Posterior Ground-Reaction Force, N</th>
<th>Knee Extension Moment, N·m</th>
<th>Proximal Tibial Anterior Shear Force, N</th>
<th>Patellar Tendon Force, N</th>
<th>ACL Loading, N</th>
</tr>
</thead>
<tbody>
<tr>
<td>356</td>
<td>82</td>
<td>340</td>
<td>1712</td>
<td>368</td>
</tr>
<tr>
<td>456</td>
<td>132</td>
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<tr>
<td>656</td>
<td>230</td>
<td>640</td>
<td>4789</td>
<td>1573</td>
</tr>
</tbody>
</table>

The American Journal of Sports Medicine
The results of this simple simulation show that increasing the posterior ground-reaction force results in increased ACL loading as well as proximal tibial anterior shear force (Table 2). Neglecting linear acceleration forces, angular acceleration moment, and gravitational forces should not have a significant effect on the results of this simulation. Adding hamstring co-contraction would only increase the ACL loading when the knee angle is small.

In conclusion, our analyses and calculations demonstrated that

1. The proximal tibial anterior shear force is an indicator of ACL loading when the knee flexion angle is small;
2. Increasing the posterior ground-reaction force increases ACL loading when the knee flexion angle is small; and
3. The posterior ground-reaction force does not protect the ACL when the knee flexion angle is small.

Finally, we would like to thank The American Journal of Sports Medicine for this opportunity to clarify an important issue in studies related to noncontact ACL injuries.

Bing Yu, PhD
Jonathon J. Chappell, MD
William E. Garrett, Jr, MD, PhD

Dear Editor:

As I was perusing the July edition of AJSM, I read with great interest the article of Caffey et al, in which the investigators study the effect of radiofrequency (RF) energy on human articular cartilage (“Effects of Radiofrequency Energy on Human Articular Cartilage: An Analysis of 5 Systems,” July 2005, pages 1035-1039). The study describes, in simulated operating room conditions, the testing of 5 commercially available RF probes attached to a customized jig to standardize a minimum contact pressure of each probe tip to 2.0 g on human articular cartilage.

As I have tested and published in Arthroscopy on the chondrocyte viability and metabolic activity after treatment of bovine articular cartilage with bipolar RF using the ACD-50 probe, I would like to rebut the experimental design and express the numerous weaknesses of the Caffey et al article. The data obtained by these investigators on articular cartilage testing are quite different than the data we have published.

The first issue I would like to address is that in our clinical experimental setting, the maximum cell death observed with the ACD-50 was 109.4 ± 22.1 % at a power setting of 4. I see that the Caffey et al investigators chose a different power setting of 3 for the probe testing, which may affect cell death.

Another issue that seems misleading is the title, which expresses the effects of RF on human articular cartilage. The tissue tested by the investigators is listed as aged articular cartilage–osteoarthritic (OA) tissue. These testing samples were taken from patients undergoing total knee procedures who were aged 57 to 72 years. The cores obtained and tested from aged/OA patients have different structures compared to mature articular cartilage. In aged/OA patients, the calcified layer is quite different; the superficial layer lacks cell viability. It is well known that aged articular cartilage is hypacellular, with apoptotic cells present in the articular cartilage matrix.

The article of Caffey et al does not show histology or confocal microscopy of control specimens. No ablation of the tissue is described in their assessment of the application of the RF probe. A zone of ablation is generally seen with the majority of bipolar RF devices (see Figure 1).1

The list of references seems incomplete. The authors list only 1 reference in 2004, Liao et al. Why could they not continue the search and refer, within the same year, to the work of Amiel et al, which was also published in Arthroscopy? They could refer in the article to the numerous publications related to the positive effect(s) of RF treatment for chondral lesions.

In an attempt to simulate an arthroscopic environment with controls, the authors have chosen to use a nonphysiologic application, applying direct pressure without probe movement, which in no way is replicated during a normal surgical procedure. The specimens were placed in a metal container, and the only circulation that took place was with a motorized propeller. This does not replicate the normal physiologic flow of saline in juxtaposition to probe application and would obviously have significant implications in terms of the generation of heat. The results could only be applied to their experimental design and not to the clinical environment, where the probes used in arthroscopy are used in a free-hand technique. Control over duration of application and amount of pressure could only lie in the hands of the surgeon, and only the surgeon could have the determination and skill to apply these instruments.

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REFERENCE


Author's Response: We welcome the feedback from Dr Amiel at his Connective Tissue Biochemistry Lab with his letter to the editor of The American Journal of Sports Medicine. We do not feel that
there are “numerous weaknesses” or “fundamental flaws” in our study as much as there is a difference of opinion about study design. We defined a well-controlled model, under a room-temperature fluid medium similar to everyday arthroscopy, to precisely control energy delivery to human articular cartilage. Five common commercially available RF probes were investigated using the manufacturers’ recommended energy levels. Every model can have drawbacks; yet, our experimental setup was one of the most precise methods used to date for the evaluation of RF heat transfer to human articular cartilage.

Dr Amiel did not like our cartilage source. As human tissue is difficult to come by, we were able to obtain human knee tissue with minimal degenerative changes (less than grade 3 or 4 by Outerbridge classification) that were macroscopically intact. We understand that these tissues can have age-related changes, yet we preferred this tissue over healthy animal articular cartilage and feel that this more closely represents the clinical human situation and provides valuable scientific information. Clearly, grade 3 cartilage demonstrated easy destruction and a much greater response to RF application. More important, we precisely measured and controlled the pressure of the RF wand application, an important variable for any RF experiment. In general, lack of control of potential sources of error is what constitutes a fundamentally flawed study. As stated, we observed a great variability in tissue changes when attempting “hand control” and determined that this error must be controlled by study design. Unfortunately, few studies have controlled for this variable. Dr Amiel found less cell death (around 109 µm) with his study. With “light touch” RF application, his uncontrolled energy application may have minimized thermal energy contact to his bovine tissue, resulting in the smaller penetration numbers in his published sample size of 12 specimens.

Dr Amiel did not like our confocal microscopy photograph. Our images were consistent, and this was a representative image from more than 120 specimens tested. Our controls show no effect with normal confocal surfaces. Histologic examination was not the focus of this study.

We disagree that any “numerous publications” in the literature demonstrate the positive effects of RF treatment in chondral lesions. Actually, the literature consistently demonstrates poor study designs with poor control of thermal applications on animal cartilage with few human articular cartilage effects studied to date.

Our circulating bath was not meant to simulate the clinical environment. This was our chosen model, and although we recognize that the fluid environment has a documented effect, the fluid influences were not the focus of this study, and again, the fluid environment was well controlled from one experimental condition to the next. The thermal effects of 1 and 3 seconds of applications in this large basin of saline have minimal effects to the overall temperature of the bath setup. Probe movement, as controlled and investigated in other studies with longer times of thermal exposure, was not the purpose or design of our study of minimal energy application. We discussed this in our published article.

Overall, the purpose of our study was to raise the level of understanding to the orthopaedic surgeon who sees articular cartilage lesions on a daily basis. We attempted to control thermal application, which has not been adequately done to date. Treatment of articular cartilage lesions has become increasingly common, and a rigid scientific approach to evaluate the arthroscopic treatment of them is critical to our patients.

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DOI: 10.1177/0363546505284190

**Dr Hughston’s Legacy**

**A Dedication to the Fundamentals**

Dear Editor:

As of September 6, 2005, it has been 1 year since the passing of Dr Jack Hughston, the first editor emeritus of this journal. Over these past 12 months, I have often found myself thinking about his life and what his legacy may be for us as orthopaedic surgeons with an interest in sports medicine. Although I am sure that there are many aspects of his work and character that have been and likely will be detailed by others, I would like to offer a perspective on an important aspect of his legacy I believe pertinent to all disciplines within orthopaedic surgery. Dr Hughston is certainly to be remembered as one of the founders of sports medicine worldwide and for his love of the knee, in particular. In recent years, however, his work has often been perceived by younger surgeons as rather antiquated and perhaps even passé. However, his absolute dedication to the fundamentals of medicine and clinical diagnosis is still vitally important and likely to indefinitely remain so.

Dr Hughston stressed a thorough knowledge of knee anatomy—especially the extra-articular anatomy—that, despite the availability of modern computer programs and newer representation techniques, remains generally poorly taught and poorly understood. Perhaps of most importance, he emphasized the necessity of a thorough history and a complete, documented knee examination before proceeding with ancillary diagnostic procedures including musculoskeletal imaging. He realized that the orthopaedic surgeon must be the one person responsible for properly assessing and integrating all of the data available to arrive at an accurate clinical diagnosis before choosing which treatment recommendations (especially operative) to suggest to the patient. This basic principle is just as important, if not more so, in today’s era of the wide availability of advanced musculoskeletal imaging technology. He would be disappointed but hardly surprised at the current trend for many orthopaedic surgeons to essentially dispense with an adequate history and physical examination in favor of arriving at a clinical diagnosis solely by means of reading the radiologist’s impressions of the MR images. It is ironic and disconcerting in the extreme that the easy access to advanced musculoskeletal imaging technology, especially the MRI, has led to a situation that Dr Hughston greatly feared—the atrophying of basic diagnostic skills of orthopaedic surgeons by the increasing overreliance on the radiologist’s interpretation of the joint of a patient with whom he or she has never spoken, much less examined. I describe this unfortunate and all-too-common situation (allowing the radiologist to make the diagnosis) to our residents as “pushing in the mental clutch.” Dr Hughston knew that there are no easy paths to the acquisition