RUSH UNIVERSITY MEDICAL CENTER

2013 RUSH ORTHOPEDICS JOURNAL

ALL IN THE FAMILY: During an illustrious career in which he helped transform care for spinal deformity patients, Ronald L. DeWald, MD (left), inspired many other orthopedic surgeons to follow in his footsteps, including his son Christopher DeWald, MD (right). Read the *Rush Orthopedic Journal's* exclusive interview (page 61) in which Ronald and Christopher DeWald discuss the evolution of spinal deformity treatment—and training.

To view the 2013 *Rush Orthopedics Journal* online or to view past issues of the journal, please visit the Rush website at www.rush.edu/orthopedicsjournal.

> "I'VE HAD THE PRIVILEGE OF REPRESENTING AAOS, ITS MEMBERS, AND OUR PROFESSION, BOTH IN THE UNITED STATES AND ABROAD. I'VE WORKED WITH BRILLIANT, PASSIONATE, AND DEDICATED PEOPLE."

Chairman's Letter

In my chairman's letter in the 2011 *Rush Orthopedics Journal*, I shared the news that I had joined the presidential line of the American Academy of Orthopaedic Surgeons (AAOS). Since then, I've had the privilege of representing AAOS, its members, and our profession both in the United States and abroad. I've worked with brilliant, passionate, and dedicated people. And I've had many remarkable experiences, including meeting with lawmakers in the White House and on Capitol Hill, and attending President Obama's second inauguration.

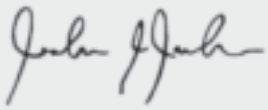
But the most memorable moment was on March 21, 2013, when I became the 81st president of AAOS during the academy's annual meeting in Chicago. Accepting the leadership mantle in my hometown, just a few miles from the medical center where I have spent most of my career, I couldn't help but feel humbled and honored. Of the 81 AAOS presidents, I am the only one from Rush and the first from Chicago in 50 years.

I first became involved with AAOS in the early 1990s, when I was a junior attending at Rush. I was invited by the then-vice president of the academy, Bernard Morrey, MD, to serve on the biomedical engineering committee. He had learned of my interest in engineering and thought I would be a valuable addition to the committee. With that invitation, I embarked on what has been a long and rewarding relationship with the premier professional organization in orthopedic surgery.

It's interesting that during my fledgling years with AAOS, the most significant benefit of membership was continuing medical education. The educational opportunities afforded by AAOS are still top-notch. What has changed is the increasing importance of the academy in communication, health care quality, and advocacy.

One of AAOS's key aspirational goals is to be *the* source of musculoskeletal information, the authority to which people will turn for clarifications, reactions, and opinions. Now more than ever, the procedures orthopedic surgeons perform—most notably total hip and total knee replacement—are under tremendous scrutiny. An increasing number of stories suggest that these surgeries are being over-utilized and inappropriately used. These are the sorts of misconceptions that orthopedic surgeons and our societies must attempt to debunk. As president of AAOS, it's my job to be aware of important developments affecting our profession, including the latest research findings and clinical advances, so that, if needed, the academy can respond quickly in the best interests of our patients and our profession. I am honored to be leading AAOS as we address these and other challenges head-on.

I am equally proud to be chairman of the Department of Orthopedic Surgery at Rush, which has been my professional home since I was a joint replacement surgery fellow here in 1987. My talented and accomplished colleagues in the department have once again authored a superb collection of articles for the *Rush Orthopedics Journal*. I encourage you to take a look. And this year's interview with spinal deformity surgery pioneer Ronald L. DeWald, MD, conducted by his son, esteemed spinal deformity surgeon Christopher DeWald, MD, is a must-read (see page 61). ■



Joshua J. Jacobs, MD

The William A. Hark, MD-Susanne G. Swift Professor of Orthopedic Surgery

Chairman, Department of Orthopedic Surgery

Rush University Medical Center

AT THE ANNUAL MEETING of the American Academy of Orthopaedic Surgeons (AAOS) in March, new AAOS President Joshua J. Jacobs, MD (left), presented his predecessor, John Tongue, MD, with a medallion honoring Tongue's year of presidential service. Photo courtesy of the American Academy of Orthopaedic Surgeons.

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Chairman and professor, Department of Orthopedic Surgery

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Assistant professor, Department of Orthopedic Surgery

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Director, Adult Reconstructive Orthopedic Surgery Fellowship Program

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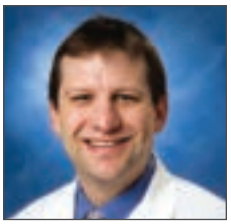
Professor, Department of Orthopedic Surgery

Jorge O. Galante, MD, DMSc

The Grainger Director, Rush Arthritis and Orthopedics Institute

Professor, Department of Orthopedic Surgery



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Associate professor, Department of Orthopedic Surgery

FELLOWS

Christopher Anderson, MD
Medical school - University of Kansas School of Medicine
Residency - University of Kansas School of Medicine-Wichita

Michael Cross, MD
Medical school - Vanderbilt University School of Medicine
Residency - Hospital for Special Surgery

Demetri Economedes, DO
Medical school - Philadelphia College of Osteopathic Medicine
Residency - Philadelphia College of Osteopathic Medicine

Chris Estes, DO
Medical school - Arizona College of Osteopathic Medicine
Residency - Banner Good Samaritan Medical Center

Julius Oni, MD
Medical school - Howard University College of Medicine
Residency - NYU Langone Medical Center/Hospital for Joint Diseases

Brian Swinteck, MD
Medical school - Wake Forest University School of Medicine
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Elliott Vann, MD
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Residency - Brooke Army Medical Center

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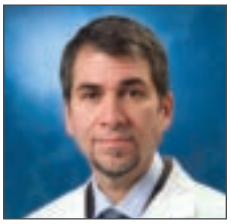
Director, Section of Hand and Elbow Surgery

Professor, Department of Orthopedic Surgery

Robert W. Wysocki, MD

Assistant professor, Department of Orthopedic Surgery



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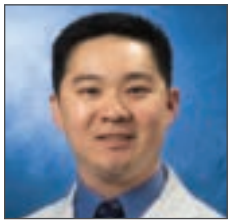
Director, Section of Foot and Ankle Surgery

Assistant professor, Department of Orthopedic Surgery

Johnny L. Lin, MD

Assistant professor, Department of Orthopedic Surgery



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Assistant professor, Department of Orthopedic Surgery

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Rush University Professor of Orthopedic Oncology

Vice chairman and professor, Department of Orthopedic Surgery

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Director, Section of Pediatric Surgery

Assistant professor, Department of Orthopedic Surgery

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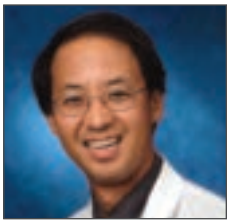


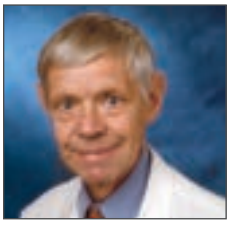
Howard S. An, MD

Director, Division of Spine Surgery

The Morton International Professor of Orthopedic Surgery

Professor, Department of Orthopedic Surgery

Director, Spine Surgery Fellowship Program



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Assistant professor, Department of Orthopedic Surgery

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Assistant professor, Department of Orthopedic Surgery

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Residency - Thomas Jefferson University Hospitals

Joseph Lee, MD

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Residency - Columbia University Medical Center

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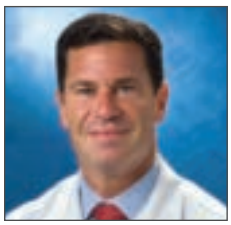
Professor, Department of Orthopedic Surgery
Director, Sports Medicine Fellowship Program

Charles A. Bush-Joseph, MD

Professor, Department of Orthopedic Surgery

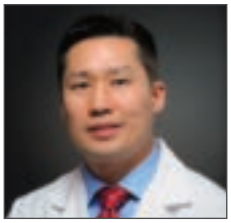


Brian J. Cole, MD, MBA

Director, Rush Cartilage Restoration Center
Professor, Department of Orthopedic Surgery

Brian Forsythe, MD

Assistant professor, Department of Orthopedic Surgery



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Residency - The Ohio State University Medical Center

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Deborah J. Hall

Instructor, Department of Orthopedic Surgery

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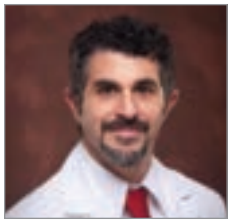

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Assistant professor, Department of Orthopedic Surgery

Markus A. Wimmer, PhD

Co-director, the Joan and Paul Rubschlager Motion Analysis Laboratory

Director, the Joan and Paul Rubschlager Tribology Laboratory

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Professor, Department of Orthopedic Surgery

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Not pictured:
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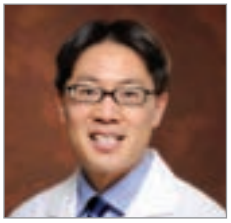
Assistant professor, Department of Orthopedic Surgery

SPORTS MEDICINE RESEARCH LABORATORY



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Alfons Fischer, PhD

Visiting professor, Department of Orthopedic Surgery

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Not pictured:

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Medical school - Case Western Reserve University School of Medicine

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Andrew J. Riff, MD
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Medical school - Tufts University School of Medicine

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Robert A. Sershon, MD
Medical school - Rush Medical College

Matthew W. Tetreault, MD
Medical school - University of Pittsburgh School of Medicine

“FURTHER STUDY COULD VALIDATE A CLASSIFICATION SYSTEM THAT WOULD CLARIFY WHICH PATIENTS ARE BEST TREATED BY NONOPERATIVE CARE, DECOMPRESSION, OR ARTHRODESIS.”

Predictors of Dynamic Instability in Degenerative Spondylolisthesis

WILLIAM SLIKKER III, MD / JOE Y. B. LEE, MD | KRZYSZTOF SIEMIONOW, MD / ALEJANDRO A. ESPINOZA ORÍAS, PHD / HOWARD S. AN, MD

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According to Kirkaldy-Willis and Farfan, the pathomechanics of lumbar spine degeneration occurs in three progressive phases.[1] In the early dysfunction stage, biochemical changes occur, and microscopic damage occurs over time in the intervertebral disc, as does synovitis of the facet cartilage. As degeneration progresses, there is decreased intervertebral disc height and subluxation of the facet joints, leading to increased instability between adjacent vertebral bodies. In the final stage, spondylosis occurs from formation of apophyseal disc osteophytes, and the facet joints undergo hypertrophic arthropathy. This model of degeneration and restabilization has been supported by various cadaveric studies, but very few clinical studies have been undertaken to confirm this hypothesis of lumbar spine degeneration.[2-8]

Despite the progressive ideas of Kirkaldy-Willis and Farfan,[1] the natural history of degenerative spondylolisthesis (DS) is still poorly understood. Matsunaga and colleagues examined the natural history of DS by following 40 patients for a mean of 8.25 years.[9] Patients with intervertebral disc narrowing, spur formation, subcartilaginous sclerosis, or ligamentous ossification did not have slip progression, suggesting that these morphologic changes represented a mechanism for restabilization. We need to clarify the correlation between slip progression and clinical deterioration as well as the need for surgical stability.

We do not understand well the relationship of dynamic instability and DS. We do not fully know if dynamic instability contributes to DS over time, or if increased dynamic instability correlates to the degree of spondylolisthesis. Attempts to define dynamic instability in the literature have reported it as greater than 3 mm of translation of one vertebral body on another or greater than 10° of motion between adjacent endplates when comparing flexion and extension radiographs.[10-11]

The objective of this study is to determine predictors of dynamic instability in relation to DS, thereby increasing our understanding of DS natural history and the need for surgical stabilization. We hypothesize that increased spondylotic changes and decreased disc height correlate with decreased intervertebral angular and translational motion in patients with DS.

MATERIALS AND METHODS

An institutional review board (IRB) approved a retrospective review, which we performed on data from 125 patients with diagnosis of L4-L5 DS who had undergone decompression and fusion from 2005 to 2011. We analyzed radiographs of the lumbar spine in neutral, flexion, and extension views to determine degree of slip, disc height, translational motion, angular motion, and lumbar lordosis. We measured these variables using electronic templates and a standard medical image visualization program (MedView; MedImage, Ann Arbor, Michigan), as shown in Figure 1.

We calculated intraclass correlation coefficients (Cronbach α) on SPSS, v. 15, (IBM; Armonk, New York) to compare the inter- and intraobserver correlation for the measurements collected by two independent observers for degree of slip, disc height, translational motion, angular motion, and lumbar lordosis.

We evaluated radiographs of our patients for spondylotic changes, including osteophyte spur formation, subcartilaginous sclerosis, and facet hypertrophy at L4-L5.[12,13] In addition, patients who had undergone magnetic resonance imaging (MRI) as part of their preoperative evaluation, were analyzed using the Pfirrmann classification to determine their grade of intervertebral disc degeneration at L4-L5[14] (Figure 2). We defined patients as having dynamic instability if they had greater than 3 mm of translation of one vertebral body on another or greater than

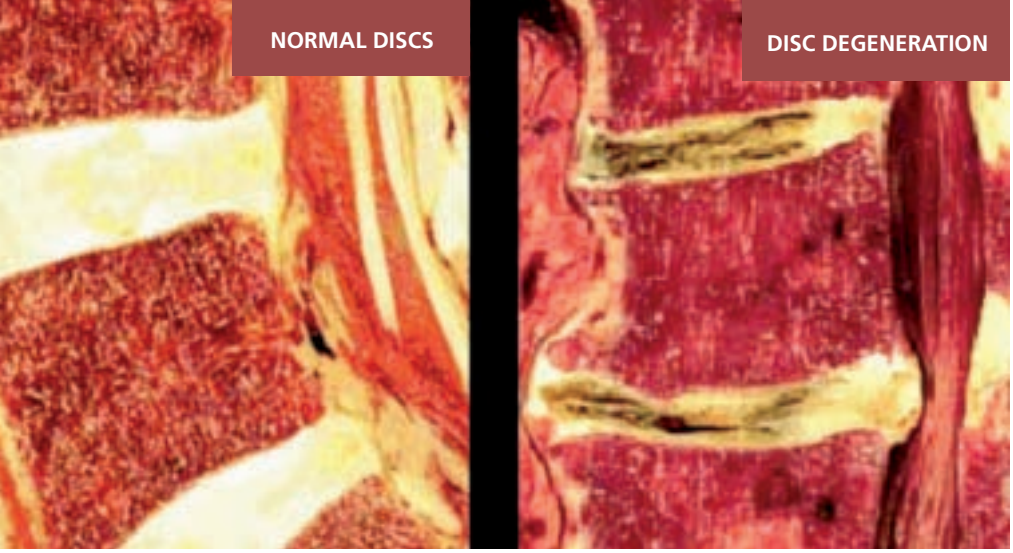

FIGURE 1.

Histological examination of the lumbar spine demonstrating normal intervertebral discs and degenerative changes including decreased disc height.

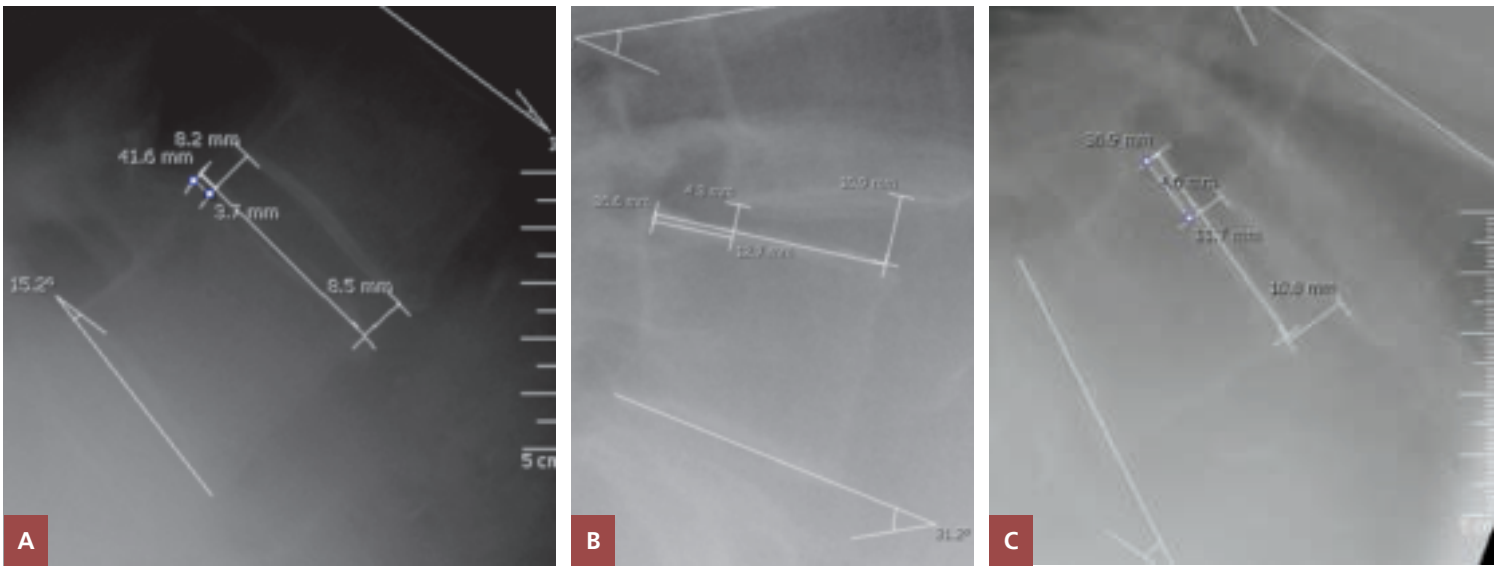

FIGURE 2.

Example of measurement method for Cobb angle, anterolisthesis, and anterior and posterior disc height. We measured Cobb angle from the superior endplate of L4 to the inferior endplate of L5. **A,** We calculated anterolisthesis by measuring the amount of anterior translation of L5 compared to L4 in millimeters. **B,** We calculated anterior and posterior disc heights by measuring the intervetebral space between L4 and L5 endplates at the anterior and posterior edge of the L4 vertebral body. **C,** These measurements are done in neutral, flexion, and extension.

10° of motion between adjacent endplates when comparing flexion and extension radiographs. We excluded patients with isthmic spondylolisthesis, degenerative spondylolisthesis at L5-S1, retrolisthesis, laterolisthesis, or insufficient radiographs. We used analysis of variance (ANOVA) to find significant associations between dynamic instability and radiographic, MRI, and demographic factors.

RESULTS

A total of 125 patients met the inclusion criteria for the study (Table 1). The average age of our population was 70 years old, with 65% being female. The most common occupation was a desk job (36%), and most patients reported that they were retired at the time of surgery. Only 10 patients in the study had jobs that involved active manual labor at the time of surgery.

In all patients, the average amount of anterolisthesis was 6.7 mm on neutral radiographs, 6.4 mm on extension radiographs, and 7.9 mm on flexion radiographs. The mean L4-L5 Cobb angle was 18.9° on neutral radiographs, 20.3° on extension radiographs, and 15.9° on flexion radiographs. There were 19 patients (15%) with grade 0 spondylotic changes, 33 (26%) with grade 1 spondylosis, 26 (21%) with grade 2 spondylosis, and 47 (38%) with grade 3 spondylosis. Eighty-three of the patients had preoperative MRIs that were graded using the Pfirrmann grading system. None of the patients had grade I disc degeneration, 7% had grade II, 35% had grade III, 29% had grade IV, and 29% had grade V. The intraclass correlation coefficients (Cronbach α) for degree of slip, disc height, translational motion, angular motion, and lumbar lordosis demonstrated an intraobserver reliability of > 0.91 and an interobserver reliability of > 0.93.

	AGE	GENDER	OCCUPATION	CIGARETTE SMOKERS	L4-L5 COBB ANGLE: NEUTRAL	L4-L5 COBB ANGLE: EXTENSION	L4-L5 COBB ANGLE: FLEXION	ANTEROLISTHESIS IN NEUTRAL	ANTEROLISTHESIS IN EXTENSION	ANTEROLISTHESIS IN FLEXION
Mean for all patients	70	65% female	Mostly desk work and/or retired	16%	18.9°	20.3°	15.9°	6.7 mm	6.4 mm	7.9 mm

TABLE 1.

Patient Demographics

MEAN VALUES*	ALL PATIENTS (N = 125)	NO DYNAMIC INSTABILITY (N = 86)	DYNAMIC INSTABILITY (> 3 MM OF TRANSLATION OR > 10°; N = 39)	NO DYNAMIC INSTABILITY VS INSTABILITY (*P* VALUE)
Cobb (change in Cobb angle in flexion/extension)	5.36° ± 0.42°	3.38° ± 0.26°	9.68° ± 0.88°	< .0001
Translation (change in olisthesis in flexion/extension)	1.92 mm ± 0.12	1.39 mm ± 0.09	3.07 mm ± 0.22	< .0001
Anterior Disc Height	10.2 mm ± 0.40	9.80 mm ± 0.49	11.26 mm ± 0.63	= .0862
Posterior Disc Height	5.80 mm ± 0.24	5.44 mm ± 0.29	6.59 mm ± 0.40	= .0245

**Data are reported as mean ± standard error of mean (SEM).*

TABLE 2.

Patients with Dynamic Instability Compared to Patients Without Dynamic Instability

Thirty-nine (31%) patients met criteria for dynamic instability, which was significantly correlated with preserved disc height ($P < .05$) (Table 2). The presence of increased spondylotic changes was significantly correlated with decreased translational motion ($P < .05$). Furthermore, there was a significant correlation between advanced disc degeneration on MRI (as defined by the Pfirrmann grading) with decreased angular motion ($P < .05$).

There was no correlation between slip percentage on neutral radiographs and dynamic instability on flexion/extension radiographs. Additionally, lumbar lordosis was not correlated with slip percentage or with translational motion.

DISCUSSION

In this study we attempted to determine predictors for dynamic instability that could be easily and reliably measured by the clinician on neutral plain radiographs. Identifying dynamic instability can aid in clinical decision making and further elucidate the natural history of DS. The Spine Patient Outcomes Research Trial (SPORT) demonstrated superior outcomes when patients with DS were treated with posterior decompression and arthrodesis as compared to nonsurgical management.[15,16] Dynamic instability may determine progression of slippage, even though the current degree of slippage has not been shown to correlate with instability. Along with such judgments, clinical factors are of paramount importance in deciding whether to advise nonoperative care, decompression only, or arthrodesis.[14]

Dynamic instability was found to be inversely related to the amount of disc degeneration and spondylotic changes. In addition, this study provides a reliable way for clinicians to measure dynamic instability on neutral, plain radiographs in the office. Using the system demonstrated in Figure 1, we found a high intra- and interreliability between 2 observers, which supports the method's reproducibility and generalizability for other clinicians.

While lumbar lordosis was not correlated with slip percentage or with translational motion, prior studies have proposed that

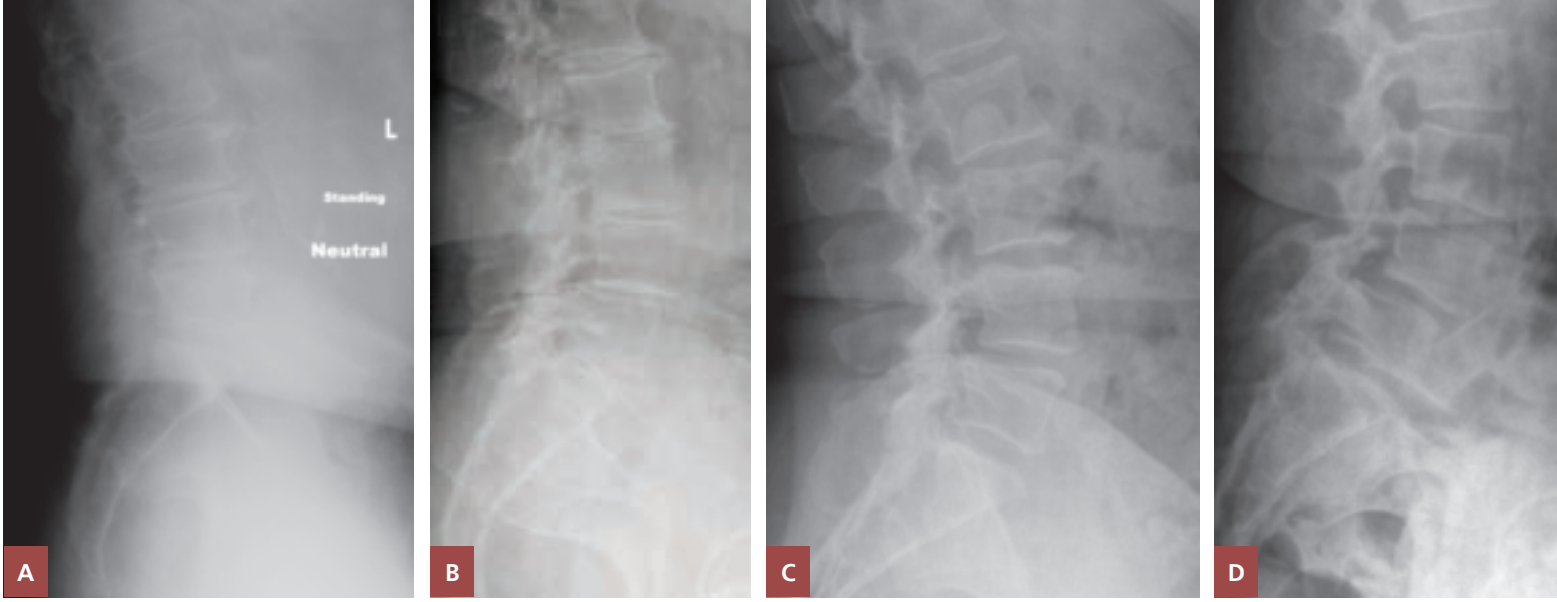

FIGURE 3.

Lateral standing radiographs of 4 different patients with degenerative spondylolisthesis, showing varying disc height, anterior translation, and spondylosis. **A,** Grade 1 instability: Disc height narrowed or collapsed and less than grade 1 anterior translation. **B,** Grade 2 instability: Disc height narrowed and anterior translation equal or greater than grade 1. **C,** Grade 3 instability: Disc height preserved and less than grade 1 anterior translation. **D,** Grade 4 instability: Disc height preserved and anterior translation equal or greater than grade 1.

patients with increased lumbar lordosis may be at a higher risk for progression of DS. Also, the Cobb angle of L4-L5 on neutral radiographs is not correlated with angular motion on flexion and extension views. It seems logical that patients with increased Cobb angle between L4-L5 may be at a higher risk for dynamic instability; however, this was not the case in our study.

In this study we found that as the Pfirrmann MRI score increases, representing increasing disc degeneration, the amount of translation decreases. These findings, along with decreased dynamic instability in the setting of spondylotic changes, suggest dynamic instability is not present in severe spondylolisthesis due to disc degeneration and restabilizing spondylotic changes. Increased dynamic instability might actually represent the potential for future slippage and progression of spondylolisthesis before restabilization mechanisms have taken effect.

We demonstrated that retained disc height is correlated with increased dynamic instability. These findings are in agreement with the natural history of DS proposed by Kirkaldy-Willis,[1] and future prospective longitudinal studies are needed to determine the exact contribution of dynamic instability to the progression of DS. However, this study's findings help elucidate some of the predictors of dynamic instability in patients with DS.

We suggest a classification of degenerative spondylolisthesis based on lateral standing radiographs to analyze 4 different patterns of degenerative spondylolisthesis (Figure 3). Grade 1 instability is represented by the motion segment with disc height narrowing or collapse and less than grade 1 anterior translation. These patients typically have minimal motion on flexion and extension radiographs, and decompression without fusion might be appropriate if surgery is considered. Those with Grade 2 instability have decreased disc height and anterior translation equal or greater than grade 1. These patients might or might not have significant motion on dynamic radiographs, and spondylosis might be minimal or significant. Whether to perform fusion with instrumentation in addition to decompressive laminectomy might depend on patient specific factors; however, fusion is recommended to prevent further instability. Grade 3 instability may be in the motion segment with preserved disc height and less than grade 1 anterior translation and minimal spondylosis changes. Even though the anterior translation is not significant, these patients typically show significant motion on dynamic radiographs, and fusion is typically recommended. Grade 4 instability is represented by preserved disc height and anterior translation equal to or greater than grade 1. In these patients, there is significant instability following decompression, and fusion with stable instrumentation or interbody fusion should be considered. In order for this classification to be valid, further studies are needed to determine the ability to prognosticate instability or assess responses to varying treatments, including decompression alone, versus different types of stabilization and fusion.

CONCLUSION

In patients with DS, preserved disc height was significantly associated with dynamic instability. This finding may represent a greater potential for slip progression over time in these patients. In contrast, disc height loss, disc degeneration on MRI, and spondylotic changes such as subchondral sclerosis, facet hypertrophy, and spur formation, were related to decreased dynamic instability. This finding is consistent with the idea that restabilization mechanisms may be related to a decreased chance of slip progression. Further study could validate a classification system that would clarify which patients are best treated by nonoperative care, decompression, or arthrodesis. ■

References and financial disclosures are available online at www.rush.edu/orthopedicsjournal.

"WE HAVE DEVELOPED A NOVEL METHOD FOR PRECISELY MEASURING THE JOINT DISTANCE, SPECIFICALLY TAILORED TO THE COMPLEX 3D FACET GEOMETRY ... USING 3D SUBJECT-BASED CT MODELS."

3D Topographic Analysis of Lumbar Facet Joint Degeneration In Vivo

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Facet joints play an important role in controlling kinematics of the motion segment and load transmission in the spine.[1-3] The facet joints are synovial joints that undergo degenerative osteoarthritic changes similar to those of other weight-bearing joints.[4] Extensive motion and loading conditions can contribute to facet joint osteoarthritis (OA), which has been considered as a potential source of low back pain and disability. Some authors have estimated that 15% to 45% of chronic low back pain comes from the facet joints.[5]

Osteoarthritis is, in general, characterized radiographically by joint-space narrowing, subchondral bone sclerosis, and osteophyte formation.[6,7] Among these parameters, joint-space width has been considered a close correlate to cartilage degeneration.[6,8] For the facet joint, narrowing of the joint space, thinning of articular cartilage, and subarticular cortical bone hypertrophy are frequently observed changes due to the aging process.[8,9]

In clinical practice, plain radiography remains the main screening technique to assess OA changes. This is accomplished by measuring the distance between articulating surfaces (also known as joint distance or joint width) on planar x-ray images. However, facet joint OA evaluation by plain radiogram requires special techniques due to 3-dimensional (3D) orientation of the facet joint.[8] Since its appearance as a clinical diagnostic tool, computed tomography (CT) has been commonly used to provide a more accurate and anatomically correct evaluation of the facet joint geometry. Various parameters, such as facet joint orientation and joint area, have been previously reported based on CT methods.[3,10-13] The results of these studies indicate that variation of orientation of the facet joint is associated with age-related changes in load bearing and development of lumbar facet joint OA.[11-13] Pathria et al, Weishaupt et al, and Kalichman et al used transverse CT as the diagnostic method in the assessment of lumbar facet joint OA.[8,14,15] Using this approach, they were able to identify the abnormalities associated with facet joint OA progression. However, these studies are solely qualitative in nature, and facet joint space narrowing was evaluated within a limited number of transverse slices.

Macroscopic evaluation of the whole facet joint surface using mapping systems allowed detailed description of the extent and location of the cartilage degeneration.[16,17] Analysis of the topographical patterns within anatomically defined zones on the surface of the facet joint enables investigation of the effects of segmental lumbar motion on facet joint degeneration process.

We have developed a novel method for precisely measuring the joint distance, specifically tailored to the complex 3D facet geometry in the form of a continuous distribution of all the distances between the joint surfaces throughout the entire facet surface, using 3D subject-based CT models.[18,19] This method is essentially a detailed mapping system that evaluates the extent and location of the facet degeneration by depicting the narrowing of the facet joints. In this paper, we use the terms *facet joint space width* or *facet joint distance* interchangeably to refer to the same parameter. With the present study we aimed to determine lumbar facet joint space width within clinically relevant topographical zones in vivo and its correlations with age, level, and presence of low back pain.

MATERIALS AND METHODS

By signing an informed consent form, 96 volunteers (45 females, 51 males) agreed to participate in this study, which was approved by the institutional review board (IRB). The average age was 37.6 years

	MALE (N = 51)		FEMALE (N = 45)	
	Asymptomatic (n = 33)	Symptomatic (n = 18)	Asymptomatic (n = 29)	Symptomatic (n = 16)
20s	10	3	9	2
30s	11	9	11	5
40s	9	3	7	4
50s	3	3	2	5

TABLE 1

Study Population Categorized by Number of Individuals (N = 96) in the Subcategories of Gender, Symptoms, and Age

(range, 22-59 years). We categorized those subjects with low back pain as symptomatic subjects and healthy subjects as asymptomatic subjects (Table 1).

We performed a lumbar CT (1.0-mm axial slices) scan (Volume Zoom; Siemens, Malvern, PA) on each subject, in supine position. We traced the facet joint surfaces from axial Digital Imaging and Communications in Medicine (DICOM) images in a custom-written program (Microsoft Visual C++) using a tablet digitizer (Wacom Intuos3; Wacom, Saitama, Japan). Our team took particular care to identify and exclude osteophyte formations from the joint surface. Otsuka et al.[13] previously described the tracing methodology. In total, we created 1,920 individual facet joint surface models and exported them as point-cloud data sets. Subsequently, we created triangular surface meshes from point-cloud data in the custom-written program.

We set a local coordinate system in order to establish a mapping system on the facet joint and calculated a normal vector for each mesh element and a mean normal vector of all normal vectors through the entire surface (Figure 1). Then we calculated the center of the facet joint surface and set this location as the origin of the local coordinate system. We defined the mean normal vector as one of the coordinate system axes. This axis and the CT coordinate system pointing towards the cranial direction formed a plane. We determined the second coordinate in this plane to be perpendicular to its mean normal vector and directed towards the cranial direction. We determined the third coordinate by the cross product between the first and the second vectors (Figure 2).

Next, we defined five topographic zones, namely central, superior, inferior, medial and lateral, in 3D space, according to the following procedure. We converted Cartesian coordinates for each point-cloud data point to a spherical coordinate system with the origin set at each facet joint surface model's area center (Figure 2). We defined the outer margin of the facet joint surface by the points that had the longest distance from the origin within a virtual cone with a vertex angle of 30°.[20] We defined the margin of the central zone so that its shape is analogue (concentric) to the outer margin of the facet joint surface and the area of the central zone is one-fifth of the whole facet joint surface area. We divided the peripheral area, outside of the central zone, into four zones defined by an angular parameter in the spherical coordinate of each point (superior zone: ± 45°; medial zone: 45°-135°; inferior zone: 135°-225°; lateral zone: 225°-315°) from the cranial axis (Figure 3A). The angles represent projected angles on a plane

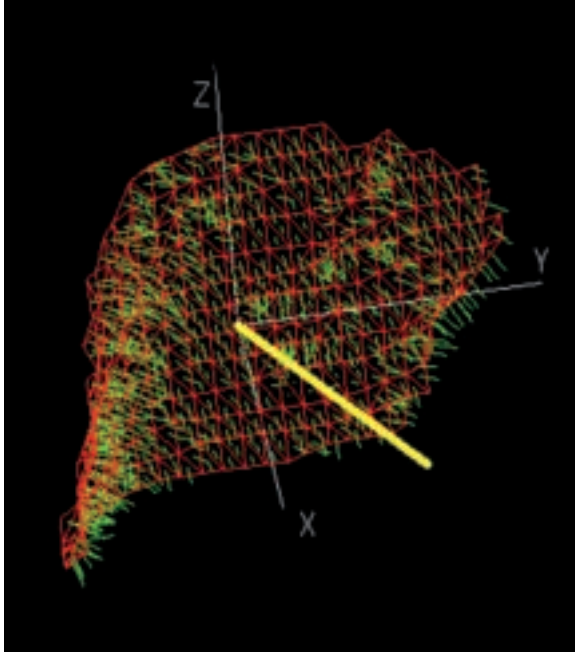

FIGURE 1.

Polygon mesh (red lines) of the facet surface, normal vectors of each polygon (green lines), and average surface normal vector (yellow lines). Coordinates represent CT coordinate system.

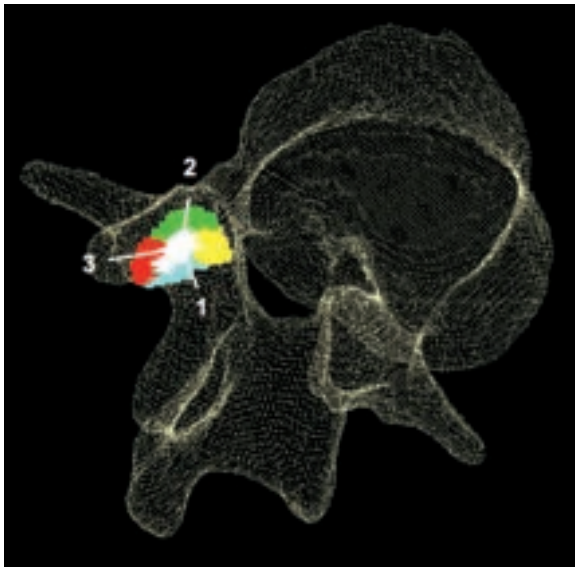

FIGURE 2.

Local coordinate system and 5 zones on facet joint surface showing direction of mean surface normal vector (1), cranial direction (2), direction orthogonal to axis 1 and axis 2 (3); superior zone (green), lateral zone (red), inferior zone (cyan), medial zone (yellow), central zone (white).

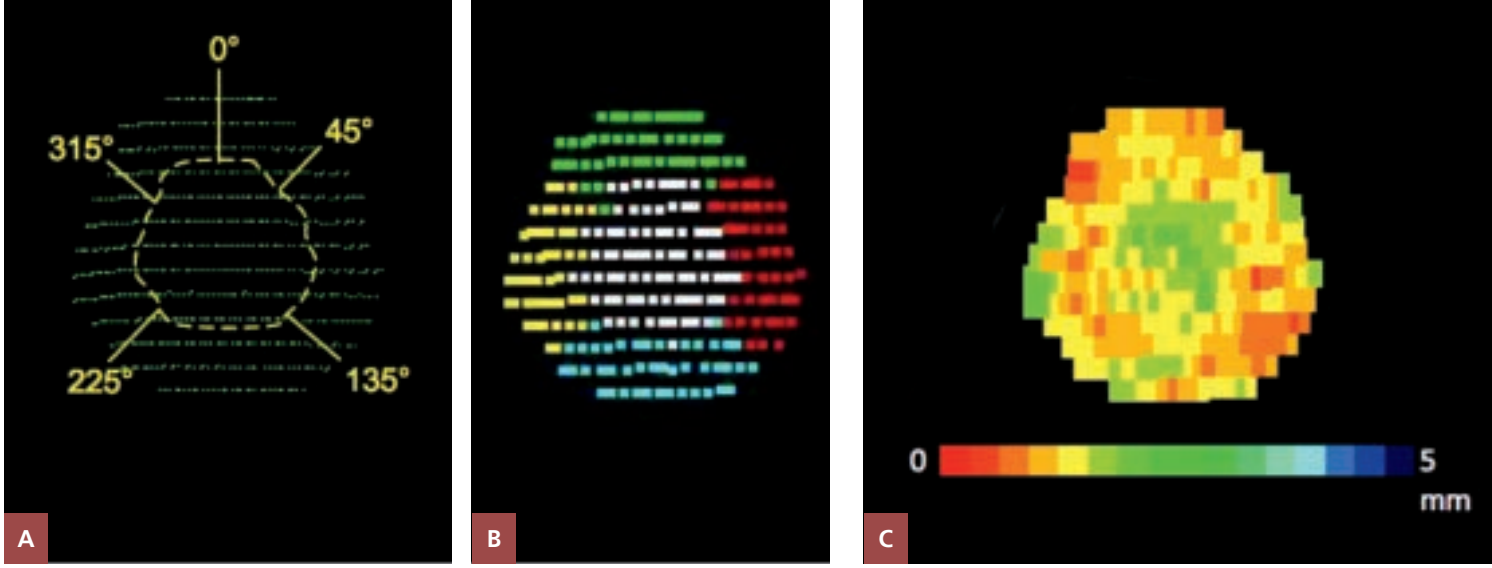

FIGURE 3.

Facet joint surface zoning. **A,** Definition of peripheral zones based on angular parameters. The 0° direction corresponds to the cranial direction of axis 2 in Figure 2. **B,** The five zones based on the facet-based coordinate system. **C,** Facet joint space width distribution map.

perpendicular to the mean normal vector of the facet joint surface.

We calculated facet joint space width as the least distance between each pair of opposing surfaces (inferior facet, superior facet), and we calculated distances between points from opposing surfaces as described elsewhere.[21,22] By averaging the least facet joint distances within each zone, we calculated the mean facet joint space width for each zone.

To evaluate differences between zones, levels, ages, and symptoms, we used analysis of variance (ANOVA) and Fisher post hoc tests. We evaluated differences between right and left sides and gender comparisons with an unpaired *t* test and reported results as mean and standard deviation. Significance was set to $\alpha = 0.05$.

RESULTS

The average facet joint space width (± SD) was 1.93 ± 0.51 mm for the central zone, 1.75 ± 0.48 mm for the superior zone, 1.63 ± 0.49 mm for the inferior zone, 1.48 ± 0.44 mm for the medial zone, and 1.65 ± 0.48 mm for the lateral zone.

Statistical correlation between facet joint space width distribution in right and left facet joints showed no side preference; therefore, we used all facet joints individually in the analysis of zones, gender, age, and symptoms. Gender comparison showed greater width distribution in males in all zones ($P < .0001$). Overall, space width was significantly lower in symptomatic subjects ($P < .0001$).

Level comparison showed significant differences. The average facet joint space width for the asymptomatic group (Figure 4) was 1.58 ± 0.36 mm at L1/L2, 1.84 ± 0.42 mm at L2/L3, 1.88 ± 0.41 mm at L3/L4, 1.95 ± 0.43 mm at L4/L5, and 1.63 ± 0.41 mm at L5/S1. The facet joint space width at L4/L5 was significantly greater than all levels ($P < .0001$) except L3/L4 ($P < .003$). The narrowest facet joint space width was measured at L1/L2 and L5/S1 levels with mutual significant difference ($P < .02$). When compared with other levels, L1/L2 and L5/S1 were significantly smaller ($P < .0001$). The average facet joint space width for the symptomatic group was 1.24 ± 0.36 mm at L1/L2, 1.51 ± 0.53 mm at L2/L3, 1.7 ± 0.59 mm at L3/L4, 1.76 ± 0.59 mm at L4/L5, and 1.38 ± 0.56 mm at L5/S1. Similar to the asymptomatic group (Figure 5), facet joint space width at L4/L5 was significantly larger than that at the other levels

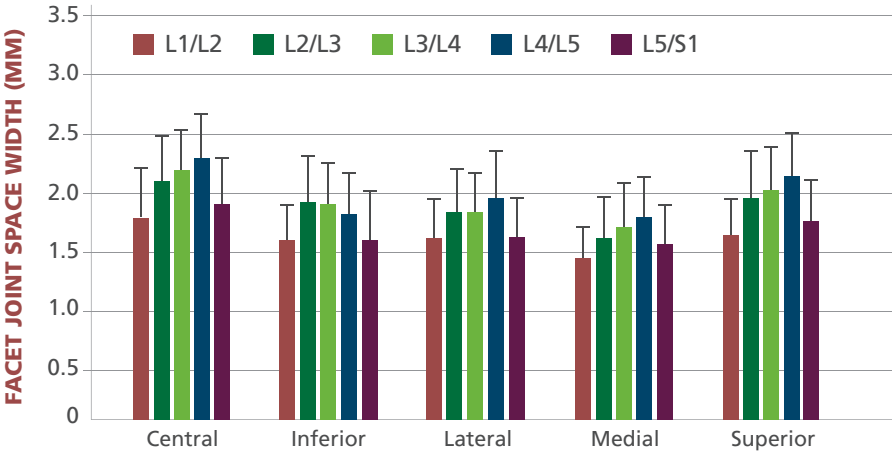

FIGURE 4.

Facet joint space width for asymptomatic subjects.

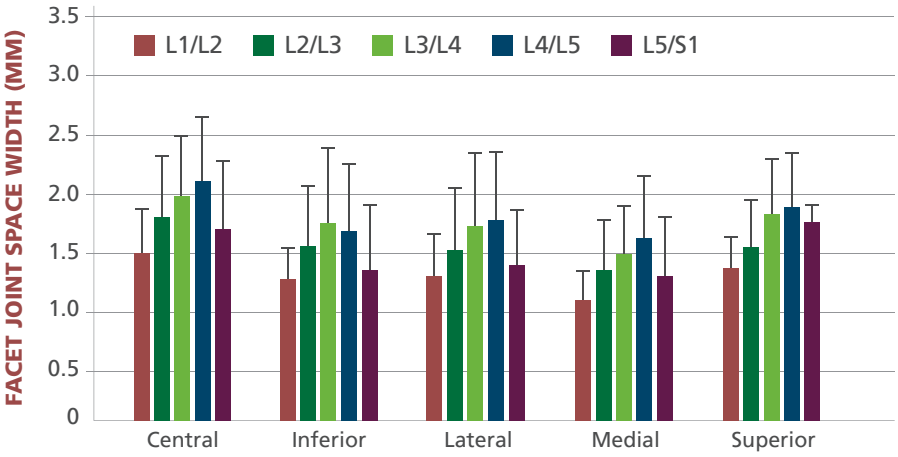

FIGURE 5.

Facet joint space width for symptomatic subjects.

($P < .0001$) except L3/L4 ($P = .1$). The smallest facet joint space widths were again measured at L1/L2 and L5/S1; however, there were significant differences between them ($P < .001$). L1/L2 was different than every other level ($P < .0001$), while L5/S1 was different than L3/L4, L4/L5 level ($P < .0001$), and L2/L3 ($P < .003$).

The facet joint space width was always larger in the central zone than in the peripheral zones ($P < .0001$) for both the symptomatic group and the asymptomatic groups. When compared to the superior zone, inferior zone space width was significantly narrower in the asymptomatic group ($P < .0001$) and in the symptomatic group ($P < .04$). Space width within the medial zone was the smallest for both groups ($P < .0001$).

The data presented in Figure 6 show changes in facet joint space width distribution at different levels with respect to age. Facet joint space width changes started in the fourth decade in the peripheral zones for L1/L2. For L2/L3, narrowing occurred only at the lateral zone in the fifth decade. At L3/L4, narrowing occurred during the fourth decade in lateral and superior zones, and facet joint space width increased in the fifth decade. Changes in the L4/L5 level involved all lateral zones in the fourth and fifth decade and central zone in the fifth decade. For L5/S1, narrowing started as early as in the third decade in the inferior zones and implicates all remaining zones after the fourth decade (see Figure 6).

DISCUSSION

The present study measured facet joint space width distribution in vivo to estimate the extent and location of the facet joint degeneration, using subject-based facet joint 3D CT models. The results of the present study showed that overall facet joint space width at L5/S1 was narrower than that in L3/4 and L4/5. This finding is consistent with a previous study that evaluated facet degeneration using CT grading.[23] The zonal analysis in the present study demonstrated that facet joint space width was narrower in the inferior and medial regions of the facet joint. Furthermore, our data show narrowing of the facet joint space width in the inferior region evident as early as in the third decade. Although previous cadaver-based studies demonstrated facet joint cartilage degeneration in younger cohorts, the present study is the first to demonstrate early degenerative changes in the facet joint in vivo, using clinically available CT-data in a quantitative manner.

Macroscopic studies of the whole cartilage surface have been conducted using human cadaveric lumbar spines.[16,17] The analysis of the whole facet joint surface is beneficial to compare cartilage degeneration against the structural 3D characteristics and biomechanical functions of the facet joint.

In order to describe the extent and location of the cartilage lesions in the facet joint, it is crucial to create a mapping system specific to the facet joint. Swanepoel et al defined in a quantitative manner the central and peripheral regions in the facet joint surface, based on the distance of the center of the damaged area from the center of the whole facet joint surface.[16] Tischer et al further divided the facet joint surface into 5 topographic zones including the central, superior, medial, inferior, and lateral regions; in spite of this, they did not describe a detailed mapping method.[17] In a cadaveric study, the exposed facet joint surface can be rotated manually so that the joint surface becomes parallel to a graphical image plane, enabling analysis. However, the facet joint surface orientation must be defined in 3D space for in vivo analyses. In the present study, we defined joint surface 3D orientation by its mean normal vector,[13] which was also part of a facet-centered local coordinate system (Figure 2). The geometry of the facet joint is often assumed as an ellipse, and a local facet joint coordinate system can be set to coincide with the axes of such ellipse.[3,24] Under the elliptical shape assumption, eigenvectors can be used to determine the local coordinates in 3D[20]; however, the shape of the facet joint is not always elliptical, and the directions of the axes other than the normal direction of the facet surface cannot be determined if the shape of the facet is close to a circle. Furthermore, the orientation of the axis of the ellipse declines from the anatomical axis,[24] which may obscure the interpretation of the topographic differences in relation to the lumbar spine segmental motion.

The results of the present study showed smaller widths in the facet joint peripheral zones, consisting of superior, lateral, inferior, and medial zones, when compared to the central zone. This finding is

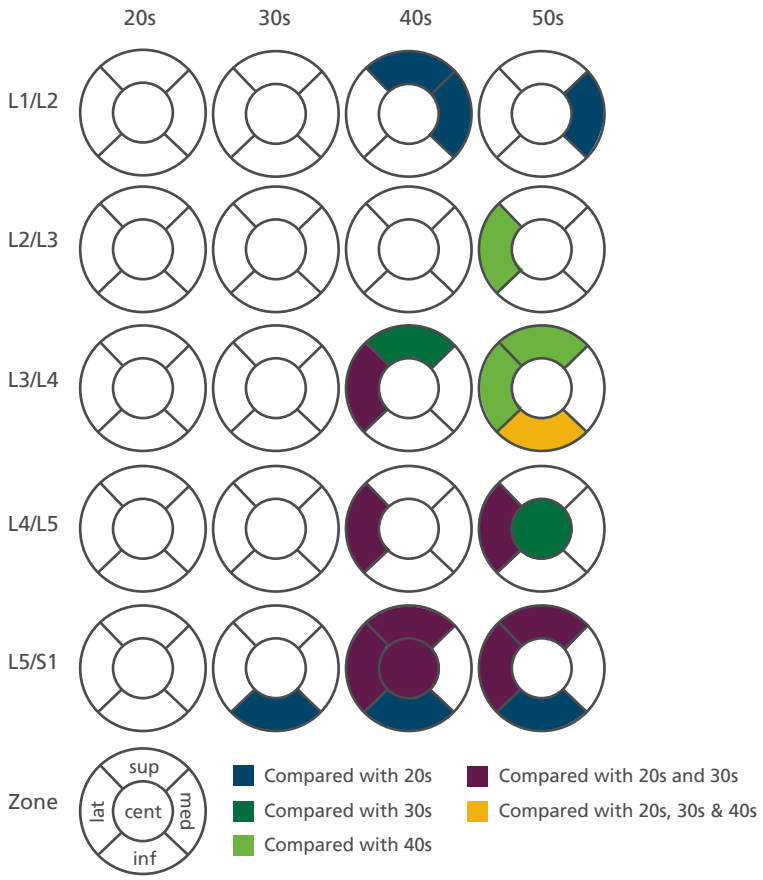

FIGURE 6.

Changes in facet space width with age. Color code indicates significant differences ($P < .05$) among different age groups within each spinal level and each zone. The subdivisions in the circle correspond to the anatomical zones of the facet joint that were defined in Figure 3 and are shown on the facet model in Figure 2.

consistent with previous cadaveric studies focused on the distribution of cartilage degeneration in vitro.[16,17] Tischer et al observed that most cartilage defects are located in the superior region of the superior facet and in the inferior region of the inferior facet.[17] They postulated that cartilage degeneration in the superior portion of the superior facet was caused by more cartilage contact with the opposing inferior facet in this region during full flexion, whereas the cartilage degeneration in the inferior portion of the inferior facet with pressure accumulation originated from bone-to-bone contact of the inferior portion of the inferior facet with superior facet during full extension.[1,17] Increased load transmission through the inferior portion of the inferior facet is also postulated in lordotic posture and erect posture with heavy loads.[1] Since segmental lordosis is reported to be higher at L5/S[1,25] early onset of the facet joint space narrowing in the inferior region at L5/S1 measured in the present study may be explained by the higher lordosis in this level, in addition to the higher load transmission in the lower lumbar levels. This study has shown lateral region facet joint narrowing at L3/L4, L4/L5, and L5/S1 in the fourth decade. Segmental lumbar axial rotation movement is mainly restricted by the facet joint,[1] and high contact pressures in the lateral region of the facet joint have been estimated during axial rotation.[26] This loading pattern during axial rotation may cause relatively early onset of facet joint space narrowing in the lateral region of the facet joint.

This report shows that narrowing of the facet joint width was more prominent for symptomatic subjects. Our previous work has shown relationships between mean facet joint space width and disc height.[19] Several studies have supported the theory that facet joint degeneration is secondary to intervertebral disc degeneration.[4,27] However, macroscopic studies on facet joint degeneration using cadaveric specimens demonstrated early initiation of facet degeneration and did not support the correlation between the facet degeneration and intervertebral degeneration.[16,28] Future studies are required to clarify relationships between the extent and location of facet degeneration and facet joint kinematics in vivo and to identify possible causes for low back pain associated with facet joint degeneration.

This study is not without limitations. Particularly, the small sample sizes for some age groups affect the study's statistical power. Second, in the present study, we determined joint space width using the least-distances method to measure the space between the superior and inferior facet joint surfaces; therefore, estimation of cartilage thinning at individual facet joints is rendered impractical.

Although we calculated normal vectors for all mesh elements throughout each facet joint surface, only the mean normal vector was used to determine the facet-based local coordinate system in the present study. Regional variation of the normal vector orientation could be used for analyses of the curvature of the facet surface, which may be an important factor causing regional differences in cartilage degeneration via different loading conditions within a facet joint surface. Future studies will address the contribution of regional curvature variations to regional differences in facet joint degeneration.

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“THE MINIMALLY INVASIVE SURGERY (MIS) APPROACH OFFERS THE BENEFITS OF DIRECT ACCESS TO THE VENTRAL SPINE WHILE MINIMIZING INCISIONAL TRAUMA AND EXTENSIVE TISSUE DISSECTION.”

Cutting-Edge Techniques for Minimally Invasive Thoracic Spine Surgery

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Over the past century, spine surgeons have developed surgical approaches to the anterior elements of the thoracic spine. Minimally invasive surgery (MIS) of the spine is a rapidly developing system at the cutting edge of this field. Traditional open approaches to the thoracic spine require thoracotomies that breach the pleural space, double-lumen endotracheal tubes for single-lung ventilation, postoperative chest tubes, and prolonged hospitalization. Thoracoscopic approaches to the spine reduce some of the exposure-related morbidity; however, these techniques also require single-lung ventilation and postoperative chest-tube drainage. By using the MIS tubular retractions system, surgeons obviate or reduce those morbidities.

INDICATIONS

We use a MIS lateral retropleural thoracic discectomy and corpectomy to treat unstable fracture patterns; primary or metastatic tumors; infection (eg, osteomyelitis or spondylodiscitis); and large, symptomatic disc herniations.[1,2] The MIS lateral retropleural approach may be utilized in tumor surgery for decompression and stabilization of compressive systems caused by metastatic cancer. Surgery is potentially curative for primary vertebral tumors, and therefore it is essential to remove the entire pathologic lesion.

Posterior thoracic disc herniations (TDH) are often asymptomatic, with symptomatic herniations being relatively rare compared to those of the cervical and lumbar spine.[3-6] Large TDHs may be heavily calcified and occupy significant canal space, resulting in thoracic spinal cord compression and myelopathy.[7,8] An MIS lateral retropleural approach for discectomy with or without a partial corpectomy and fusion may be considered in these cases.[9]

PATIENT EVALUATION

Evaluation of a patient for MIS anterior thoracic surgery should focus on disease location and comorbidities. A history of previous pulmonary conditions is a relative indication for the MIS lateral retropleural approach over other approaches, because single-lung ventilation is not routinely used.[1] However, severe pulmonary disease should preclude any surgery. Patients with preexisting lung disease should undergo pulmonary function testing, because thoracic surgery can lead to significant intraoperative and postoperative complications. A history of prior thoracotomy, chest trauma, or thoracic spine surgery that causes pleural scarring may complicate the retropleural approach.[10] We check for anatomic abnormalities such as aberrant blood vessels or anomalous vasculature locations that may preclude exposure.

TECHNIQUE

Positioning and Exposure

The patient is positioned in the lateral decubitus position (on the side) on a radiolucent table (Figure 1A). The surgeons stand behind the patient, and the fluoroscopic C-arm and mounted retractor arm are in front of the patient. In general, we place the tubular retractor such that the open end faces the spinal canal/cord allowing an unobstructed view of the neural compression.

The operative level is identified under biplanar fluoroscopy (x-ray guidance), and we mark the anterior and posterior vertebral lines over the lateral chest wall. We make a 3-4 cm incision directly lateral to the index vertebra along the course of the superior margin of the rib.[11] The rib is exposed subperiosteally, reflecting the neurovascular bundle inferiorly. The intercostal space is retracted, or if needed, we will resect approximately 2 cm of lateral rib overlying the vertebra (Figure 1B).[10,12] This rib may be utilized as autologous bone graft for fusion. We place a series of tubular dilators through the intercostal space under fluoroscopic guidance, retracting the pleura and lung anteriorly (Figure 1C).[12] An expandable split-blade retractor is placed over the dilators and secured to the flexible table-mounted retractor arm. The vertebral

bodies and intervertebral discs above and below are exposed subperiosteally. We then cauterize the segmental artery and resect it as proximally as possible.[10,12] At this point, the remaining procedure differs for discectomy alone vs corpectomy with reconstruction depending on the pathology.

Case 1: Discectomy

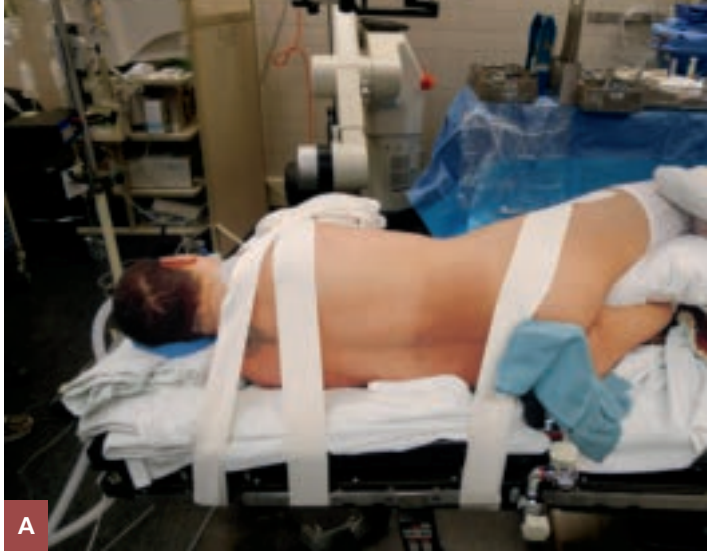
The patient is a 28-year-old female with a large thoracic herniated disc, which is causing progressive myelopathy. Decompressive laminectomy was previously performed by another surgeon without improvement of myelopathic symptoms (Figures 2A and 2B).

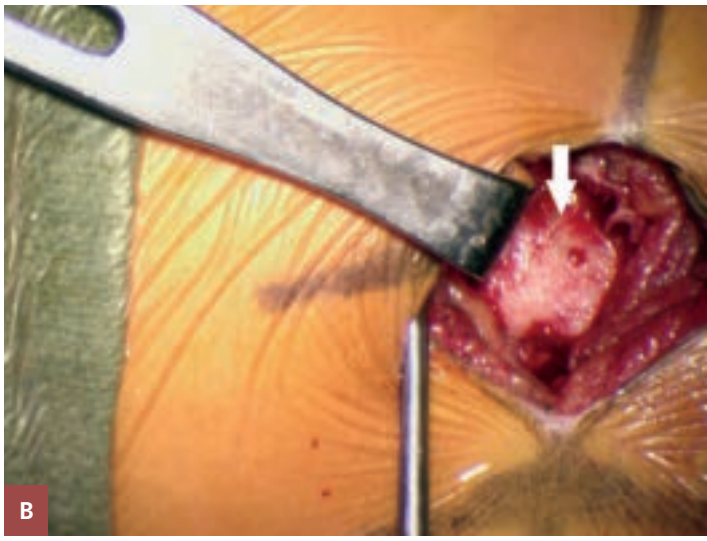
We perform a discectomy by creating a trough anterior to the canal, drilling the posterior third of the vertebral bodies straddling the disc space and the superior half of the inferior pedicle (Figures 2C and 2D).[13] A thin shell of the posterior cortex with the herniated disc is left in place, protecting the dura. We then remove the remaining posterior cortex and herniated disc by gently pulling anteriorly into the trough, thereby dissecting away from the dura. Large, calcified herniated discs may be adhered to the posterior longitudinal ligament (PLL) and dura, which may lead to inadvertent durotomy.[11,14] Fusion is not necessary in most cases; however, if large portions of the vertebral bodies are removed, then we will perform a fusion using the resected portion of rib as an autograft.[13]

Case 2: Corpectomy and Reconstruction

The patient is a 28-year-old female with an expansile giant cell tumor with vertebral body destruction and cord compression.

This case requires a corpectomy to remove the tumor as the compressive pathology. We expose the dura by removing the pedicle with a high-speed burr. The thecal sac serves as a guide to the proximity of the spinal canal.[2,10,15] The intervertebral discs above and below are resected with angled curettes and rongeurs. The borders of the corpectomy are now clearly visible. We resect the vertebral body with a high-speed burr, rongeurs, and curettes until a thin portion of the anterior vertebra is preserved with the anterior longitudinal ligament (ALL) (Figure 3A).[12] If possible, the PLL is left intact unless it is part of the compressive pathology.[2] In order to reconstruct the anterior vertebra, we insert an





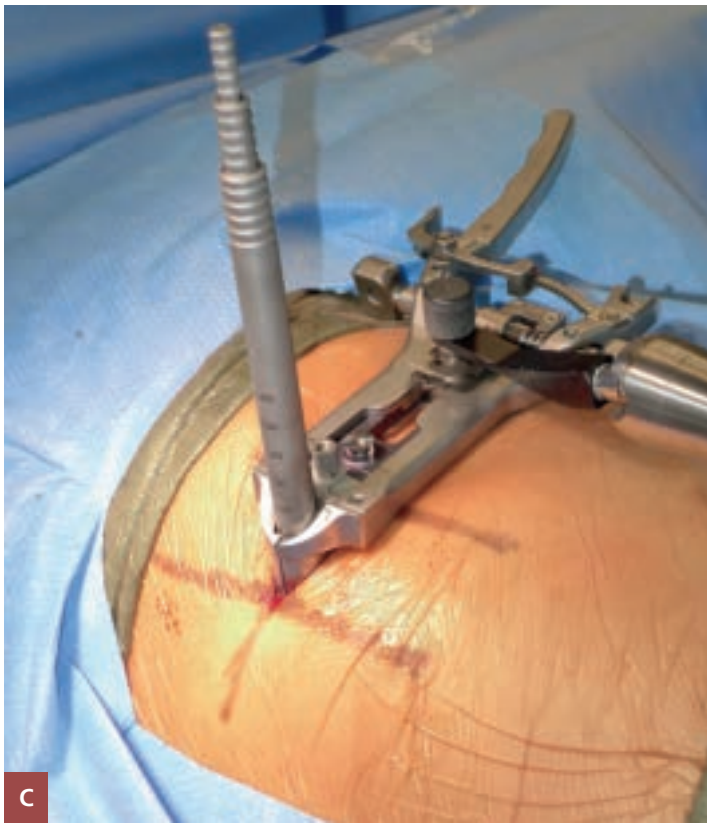


FIGURE 1.

A, The patient is secured in the lateral decubitus position. **B,** Intraoperative photograph in which approximately 2 cm of the rib has been resected and the parietal pleura (arrow) overlying the lung is now visible. **C,** After the plane between the parietal pleura and endothoracic fascia is defined, a series of tubular dilators are placed, retracting the lung anteriorly and exposing the lateral vertebral body. An expandable retractor is then placed over the tubular dilators and secured to the table.

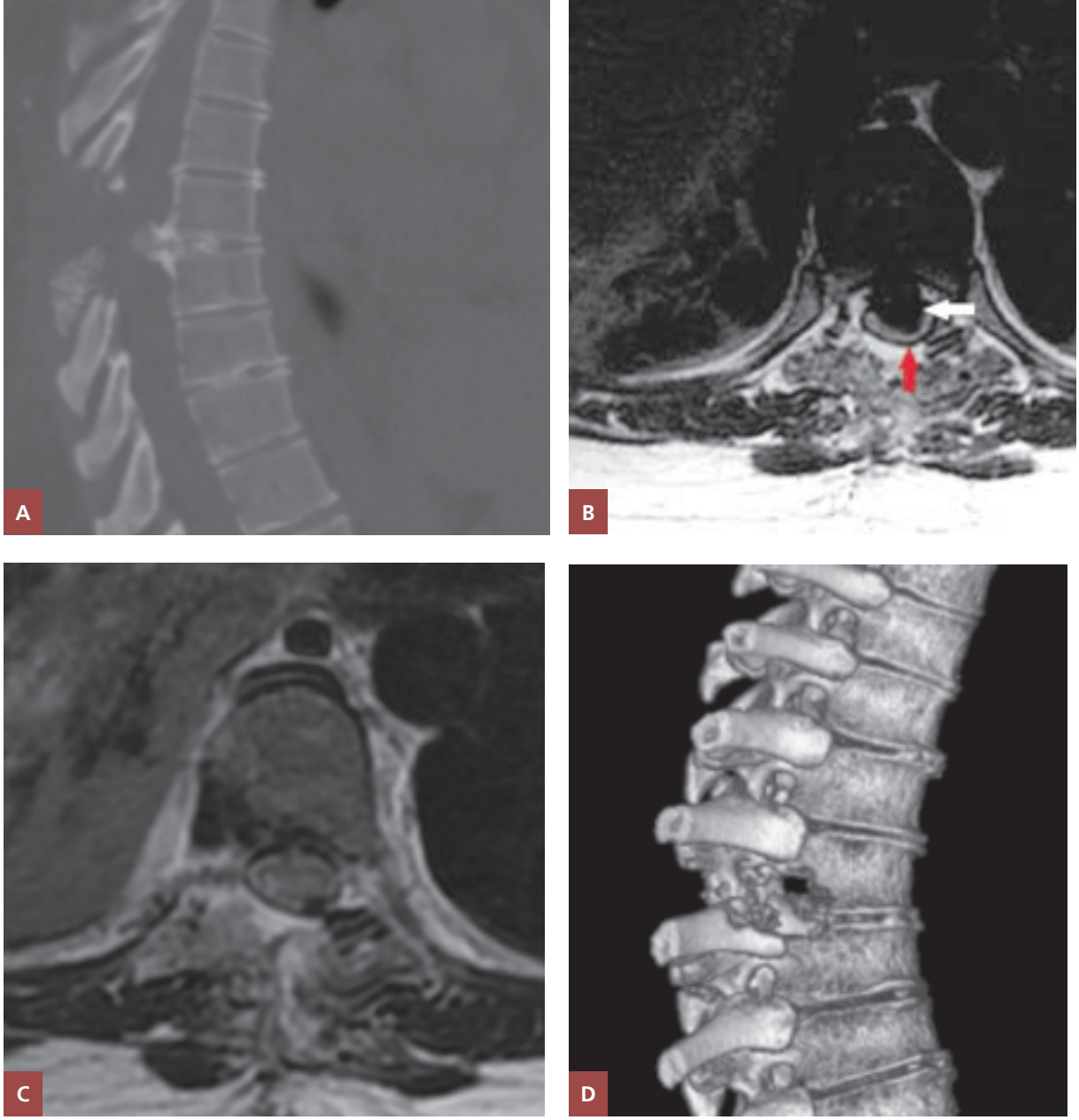


FIGURE 2.

Case 1: A patient undergoing minimally invasive thoracic discectomy for a large herniated disc causing spinal cord compression. **A,** Preoperative sagittal computed-tomography (CT) image of a patient with a massive calcified herniated nucleus pulposus (white arrow) at T7/8. Posterior laminectomy and discectomy were previously attempted by an outside surgeon. **B,** Axial MRI image of the same patient demonstrating the herniated disc (white arrow) that occupies most of the canal space as well as the spinal cord (red arrow) being compressed and displaced posteriorly. Postoperative axial **C,** and 3D reconstruction **D,** demonstrating partial vertebrectomy and discectomy (arrowheads) of the same patient after a minimally invasive lateral retropleural approach. The spinal cord (red arrow) is no longer compressed.

expandable titanium cage into the defect and expand it under lateral fluoroscopy until the desired sagittal alignment is attained (Figure 3B).[11,12] Autologous bone graft or a substitute surrounds the cage to promote fusion. Fixation is then completed with ventrolateral plating of the adjacent vertebrae using a dual-rod construct.[12] Posterior pedicle screws and rods may also be used as supplemental fixation and are placed in either the lateral decubitus position or when the patient is repositioned prone on a Jackson table (Figures 3C and 3D).[16,17]

OUTCOMES

Numerous articles report the early postoperative outcomes of MIS techniques for the treatment of tumors, trauma, infection, and disc herniations.[10,11,14,17-19]

Several studies report on the outcomes of an MIS lateral retropleural discectomy for symptomatic TDHs.[9,14,18] Uribe et al compared their results to a review of the literature and found the MIS group, as compared to the open group, demonstrated a trend towards shorter operative times (182.0 vs 229.3 minutes), reduced blood loss (290.0 vs 562.9 mL), and fewer

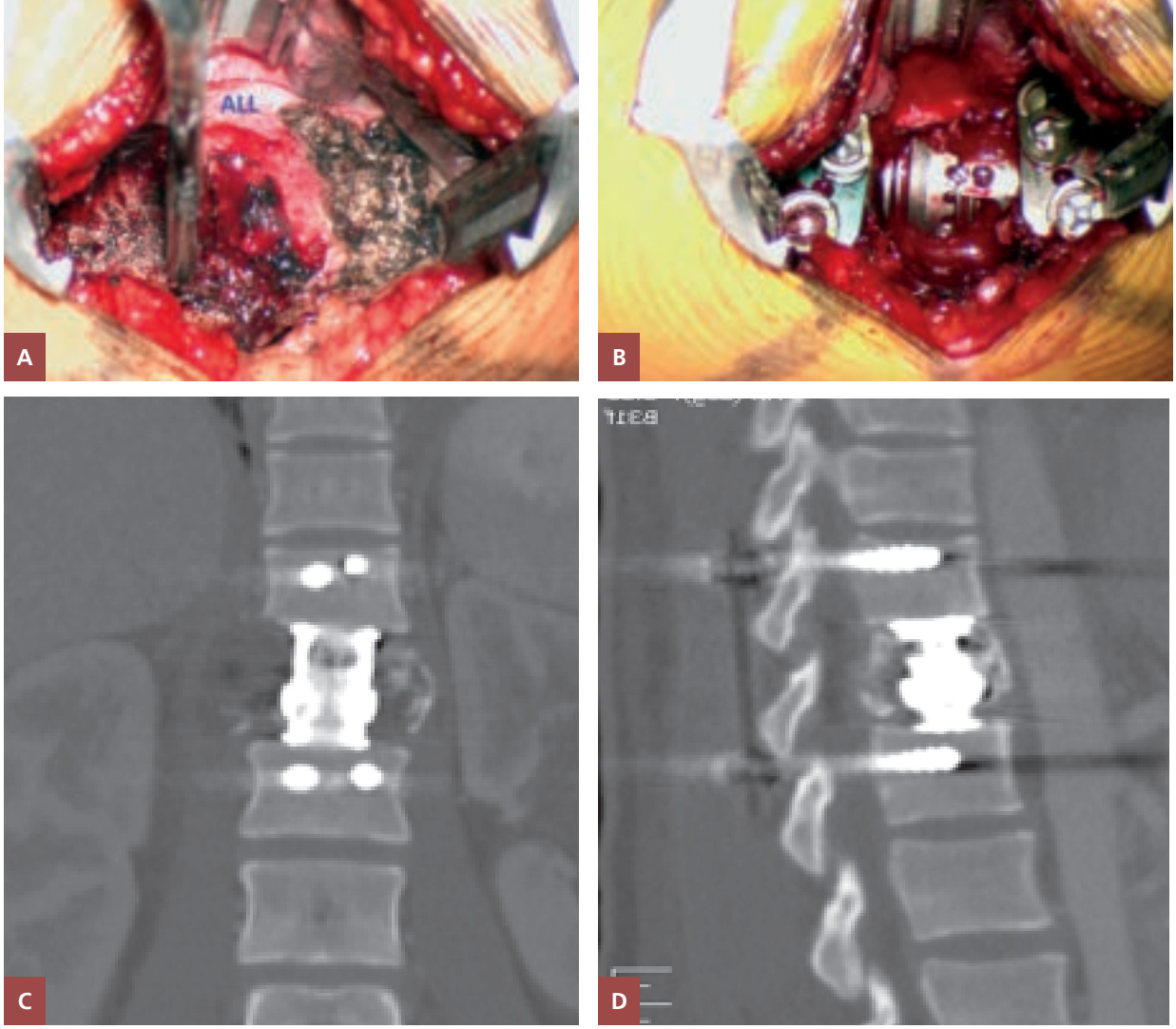


FIGURE 3.

Case 2: A patient undergoing minimally invasive corpectomy and reconstruction using an expandable titanium cage. **A,** Intraoperative photograph demonstrating discectomy and corpectomy being performed with a high-speed burr. The lateral vertebral body has been removed and tumor is present within the corpectomy site. The anterior longitudinal ligament (ALL) is intact at the top of the photo. **B,** Photograph demonstrating the expandable titanium cage after it is inserted into the defect and expanded to restore sagittal and coronal balance. **C,** Postoperative coronal CT image demonstrating reconstruction of the resected vertebral body using an expandable cage. **D,** Sagittal CT image demonstrating correction of thoracic alignment.

hospitalization days (5.0 vs 8.6 days).[14] Furthermore, the rate of complications (intercostal neuralgia, urinary retention, durotomy, lower-extremity weakness, atelectasis, pleural effusion, pneumonia, and posterior hardware infection) was significantly lower in the MIS group, at 15.0% compared to 36.7% for the open group.

CONCLUSION

The minimally invasive lateral retropleural approach for thoracic discectomy and corpectomy is a safe and effective procedure for the treatment of ventral cord compression due to trauma, infection, neoplasm, and disc herniation. Traditional open posterior approaches and open thoracotomies are associated with significant morbidity and higher rates of complications. Increased blood loss, lengthy hospitalizations, and prolonged postoperative pain have been diminished by thoracoscopic approaches. However, there are still limitations to thoracoscopy including a steep learning curve and need for single-lung ventilation. With the aid of a tubular retractor system and fluoroscopy, the MIS approach offers the benefits of direct access to the ventral spine while minimizing incisional trauma and extensive tissue dissection. ■

References and financial disclosures are available online at www.rush.edu/orthopedicsjournal.

> CLINICIANS SHOULD ONLY CLEAR AN ATHLETE TO RETURN TO COMPETITION WHEN THE ATHLETE IS SYMPTOM FREE BOTH AT REST AND WITH EXERCISE, OFF MEDICATION, HAS A NORMAL NEUROLOGIC EXAMINATION, AND ANY NEUROPSYCHOLOGICAL TESTING HAS RETURNED TO BASELINE LEVELS.

The Challenge of Sports-Related Concussion for Male and Female Athletes of All Ages

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Recognition and management of sports-related concussion (SRC) is an evolving and important component of many sports medicine practices. Concussion is a medical issue that transcends medical fields, affecting primary care physicians, orthopedic surgeons, sports medicine physicians, emergency room specialists, neurologists, and neurosurgeons. The heightened awareness in the medical community, news media, general public, and sports world has made SRC a timely issue for physicians. Health professionals who care for athletes need to be proficient in current guidelines for the evaluation and management of this common, yet challenging, sports injury.

DEFINITION

Concussion represents a type of mild traumatic brain injury, or MTBI. The term *mild* is used to differentiate these types of injuries from more severe, life-threatening injuries, such as intracranial hemorrhages or skull fractures. Nevertheless, MTBIs still represent an injury to the brain and can have serious short- and long-term effects.

Panelists at the Third International Conference on Concussion in Sport (Zurich, Switzerland, November, 2008) agreed unanimously to define *concussion* as "a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces." The panel's report[1] also listed the following common features of concussion: (1) Concussion may be caused either by a direct blow to the head, face, or neck; or by a blow elsewhere on the body with an "impulsive" force transmitted to the head; (2) Concussion typically results in the rapid onset of short-lived impairment of neurologic function that resolves spontaneously. Symptoms arise and dissipate in a sequential fashion. Loss of consciousness (LOC) may occur but is uncommon; (3) Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury; therefore no abnormality on standard structural neuroimaging studies is seen in cases of concussion.

EPIDEMIOLOGY

Concussion is a common sports-related injury.[2] As many as 3.8 million concussions occur yearly in the United States.[3] Almost 9% of all athletic injuries at the high-school level are concussions, and the rates of concussion among high school girls are higher than among boys in the same sports.[4] In the last decade emergency room visits have increased 60% for sports- and recreation-related traumatic brain injury in children and adolescents.[5] Most of these visits are related to bicycling, football, basketball, soccer, and playground activities.[5]

PATHOPHYSIOLOGY

Concussion is typically caused by a direct blow to the head, neck, or face.

Concussion may also be caused by an indirect trauma such as a blow to the body with forces then transmitted to the head and, therefore, the brain (Figure 1). The forces that cause concussion include linear (acceleration-deceleration) as well as rotational (angular) forces or, more likely, a combination of both types. The traumatic event triggers mechanical injury and a metabolic cascade resulting in a disruption of cellular processes that can last for days or weeks.

The pathophysiology of concussion in humans has been postulated based mostly on animal studies.[6] Following a traumatic event, sudden extensive release of neurotransmitters and rampant fluxes in ion concentration occur. Trauma results

FIGURE 1.

In addition to a direct blow to the face or head, concussion can occur from indirect, acceleration/deceleration forces transmitted to the head and neck, as suggested here in a female professional American football player, whose head is thrust forward as she sustains posteriorly directed contact to the chest and abdomen.

in disruption of neuronal membranes as well as stretching or shearing of the axonal projections from the nerve cells. Axons extend from white matter to gray matter in the brain and propagate impulses to adjacent neurons. Traumatic injury to the axons initiates a metabolic cascade in the nerve cells in the affected area of the brain.

Axonal injury induces opening of potassium channels, increasing the extracellular potassium concentration and triggering neuronal depolarization. The release of excitatory neurotransmitters, such as the amino acid glutamate, induces further potassium efflux and further depolarization, resulting in suppression of neuronal activity. Calcium, which is toxic to the cell, accumulates inside the neuron, impairing mitochondrial metabolism and slowing neuronal connectivity.

To restore ion homeostasis, sodium-potassium channels are activated to pump potassium back into the neuron. The energy required to operate the pumps is derived from adenosine triphosphate (ATP) and ultimately from glucose, producing lactate as a byproduct. Lactate accumulation induces intracellular acidosis, alterations in blood-brain barrier permeability, and cerebral edema. This substantial increase in glycolysis leads to an increased glucose demand in the face of decreased supply, which is due to diminished cerebral blood flow. This supply-demand disparity is, in effect, an "energy crisis" that may take days to resolve in animals and weeks in humans[6] (Figure 2).

The time period after a concussion when the body is attempting to restore the brain's homeostasis is often referred to as the "period of vulnerability." A second injury to the brain during this time period may result in impaired cerebral autoregulation, neuronal death, and cerebral edema. This hypothesis may explain the phenomenon known as second-impact syndrome (see the section titled Complications).

SYMPTOMS

Symptoms associated with concussion can be divided into 4 broad categories: physical, cognitive, mood, and sleep disturbances. Physical symptoms can include headache; blurred vision; dizziness; LOC; nausea and vomiting; poor balance; and sensitivity to light or noise, and fatigue. Cognitive symptoms include being easily distracted, long- and short-term memory problems,

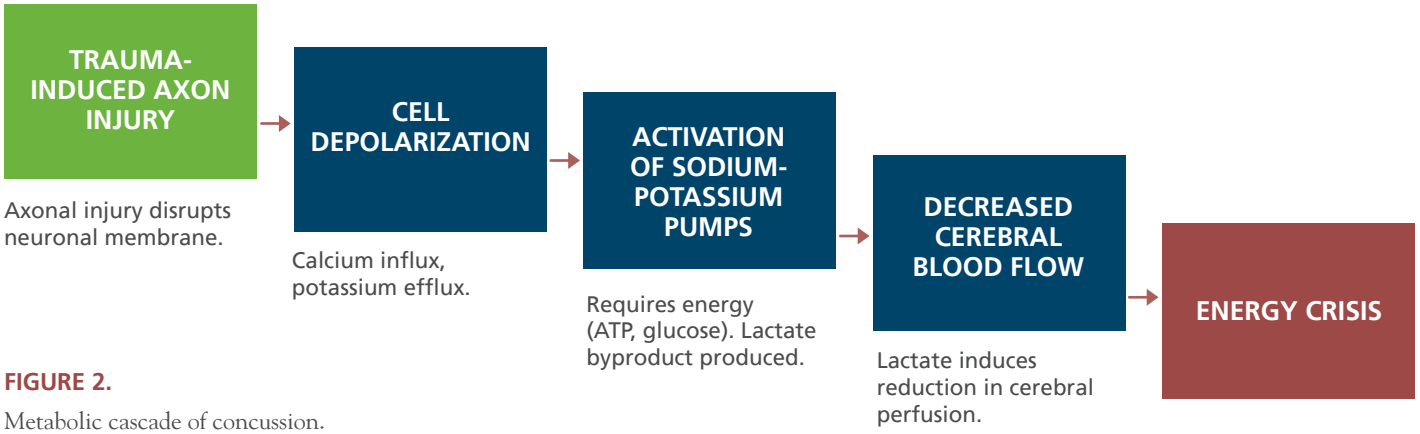

FIGURE 2.

Metabolic cascade of concussion.

and poor concentration. Mood symptoms include irritability, increased emotionality, sadness, nervousness, or anxiety. Sleep disturbances can include increased or decreased sleep time, insomnia, and difficulty falling asleep. Headache is the most commonly reported symptom. Dizziness and confusion are common as well but are reported less often.[7,8] LOC occurs in only 10% of concussions. Although LOC has been traditionally thought to be a sign of more severe head injuries, LOC does not appear to indicate injury severity or predict concussion recovery time.[9,10]

Epidural Hematoma

If there is an acute worsening of signs or symptoms within minutes to hours after a head injury, the possibility of an epidural hematoma should be considered. Epidural hematomas are often associated with skull fractures. These occur from an injury to a cerebral artery, most often the middle meningeal artery. Clinical exam findings suggestive of a skull fracture include otorrhea, rhinorrhea, and posterior auricular and periorbital hematomas.

An athlete with an evolving epidural hematoma may appear to be improving symptomatically after the injury and may have minimal clinical complaints or findings (the so-called "lucid interval"). However, a rapid clinical decline may occur within minutes to hours as the hematoma expands and intracranial pressure increases. The increased intracranial pressure results in an altered state of consciousness and, without treatment, may result in death. If the athlete has a change in neurological status or if there is a concern for an associated injury such as a skull fracture, cervical injury, or intracranial hematoma, the athlete should be immediately transferred to the hospital for evaluation and treatment.

An individual who has sustained a suspected concussion should be observed closely following the event. In most cases concussive symptoms develop immediately following the inciting force, but they may be delayed in onset or evolve over time. Symptoms may worsen within minutes to hours of the event. Some athletes may not recognize or volunteer that they are having difficulty. It is important that coaches, athletes, and family members be educated on the signs and symptoms of a concussion. The importance of prompt reporting of these symptoms should be encouraged.

Fortunately most patients with concussions recover quickly and fully. The National Football League (NFL) and National Hockey League (NHL) report that concussions typically resolve in 7-10 days. In some individuals the symptoms can last for days, weeks, or longer. Multiple studies demonstrate that recovery of full cognitive function is slower to occur in children and adolescents than in older adults. In fact, many believe that children may take twice as long as adults to completely recover from concussion. Individuals that have had a prior concussion are at higher risk of sustaining another concussion, and recovery may be longer in these individuals.[11,12]

ON-FIELD AND SIDELINE EVALUATIONS

An initial assessment of an athlete suspected of having a concussion should follow a standard protocol similar to evaluating other injuries. An on-field evaluation should begin with an assessment of the level of consciousness, airway, breathing, and circulation. If the athlete is unconscious, the clinician should suspect a possible cervical spine injury.

If the athlete regains consciousness and more severe injuries have been ruled out, the athlete should be taken to the sideline for evaluation. If the athlete remains unconscious or a more severe injury is suspected, the athlete should be transported to an emergency room for further evaluation and treatment.

Once on the sideline, the athlete should be questioned regarding the events prior to and following the injury to establish retrograde or anterograde amnesia (Tables 1 and 2).

These questions also allow the examiner to establish level of consciousness and appropriateness of responses given by the athlete. The questions should also evaluate for the presence of concussion-related symptoms such as headache, dizziness, light-headedness, and nausea. Additional tests to assess postural control, cranial nerve integrity, and neurocognitive status should be performed. There are many available

TABLE 1.

Modified Maddocks Questions

What venue are we at today?
Which half is it now?
Who scored last in this match?
What team did you play last?
Did your team win the last game?

TABLE 2.

Standardized Assessment of Concussion (SAC)

What month is it?
What is the date today?
What is the day of the week?
What year is it?
What time is it right now?

tools, and most teams have a preferred standard assessment tool to evaluate the athlete. The National Athletic Trainers' Association recommends the use of the Graded Symptom Checklist while others use the Sideline Concussion Assessment Tool (SCAT2)® or Standardized Assessment of Concussion (SAC).[13,14]

Asymptomatic athletes suspected of having a concussion should undergo functional testing prior to going back into play. This sideline activity progressively introduces physical activity to the athlete and evaluates if he or she shows concussive symptoms. The activity is progressed from activities such as push-ups or sit-ups to jogging/short sprints to more sports-specific activities to achieve the necessary level of play to return to the game. If an athlete suspected of having a concussion is still symptomatic and/or does not pass this battery of sideline tests, he or she should be not be allowed to return to play in that competition. Although many athletes will complain immediately of some concussive symptoms, others may not. It is always better to take additional time, use caution, and observe the athlete on the sideline because concussive symptoms can develop over time.

MANAGEMENT

Because of the variable presentation of symptoms and symptom severity, physicians should individualize treatment for each concussion case. The treating physician should take into consideration the history of the event, the associated signs and symptoms, and the athlete's previous concussion history. A physician experienced in the management of concussion should guide the treatment, management, and eventual clearance to return to play.

Despite the variable presentation of signs and symptoms, the mainstay of treatment is physical and cognitive rest until there has been complete resolution of symptoms. The doctor should emphasize strict physical rest, including physical education and recreational activities, in the treatment discussion. Cognitive rest includes limiting or refraining from activities requiring concentration and attention. This could include schoolwork, tests, and quizzes or screen time, such as television, computers, videogames, or cell phones. Participation in such activities may cause a worsening of symptoms and delay ultimate recovery.

Physicians occasionally use pharmacologic therapy in the management of concussions. In the acute setting, pharmacologic treatment is not usually necessary, because the majority of concussions will resolve in a short period of time. Physicians may use medications to treat specific symptoms, such as headache. In generally they avoid nonsteroidal anti-inflammatory drugs (NSAIDs) because of the effect on platelet aggregation and the risk of bleeding, especially in instances of intracranial hemorrhage. Acetaminophen is usually first-line treatment for headache. Doctors should avoid stronger narcotic pain medication in the acute setting to avoid masking progressively worsening symptoms. Corticosteroid use in head injury is not advocated at this time. A large, multicenter double-blinded, placebo-controlled trial looked at corticosteroid use in severe head injury. This study showed an 18% increase in mortality in the corticosteroid arm. Due to the results of this study, "corticosteroids should not be used to treat head injuries of any severity."[15]

Doctors typically use pharmacologic treatment in the treatment of specific, prolonged symptoms such as anxiety, sleep disturbance, or depression, or to modify the underlying pathophysiology in an effort to shorten the duration of symptoms. Only those clinicians experienced in the management of concussions should resort to pharmacotherapy.

Medical professionals should closely monitor the concussed athlete for progressive improvement in symptoms. Once the athlete has noted complete resolution of symptoms, the clinician can initiate a discussion on returning to play. The clinician must take into consideration the usage of any medications or agents that may mask or modify the symptoms of concussion when determining complete resolution of symptoms. The clinician must give careful consideration to whether to return an athlete to play while he or she continues to take medications for depression, anxiety, and sleep disturbance.[8,16-18]

According to the Zurich guidelines, the application of neuropsychological (NP) testing in concussion has been shown to be of clinical value and continues to contribute significant information in concussion evaluation. Neuropsychological testing becomes especially important because there may be a delay in cognitive recovery despite the athlete noting complete symptom recovery. Formal NP testing, done by a neuropsychologist, and computer-based testing are 2 options. Because of the ease of use and availability of computer-based testing, many organizations at the professional, collegiate, high school, and youth levels use this kind of testing. These tests have the most utility when the testing done after a concussion can be compared to a baseline test that is typically performed at that start of the season. NP testing can provide valuable information but should never be used as the sole determinant in deciding whether a concussed athlete may return to play. It should be viewed as a piece of additional information to further enhance clinical decision making. Despite appropriate management, symptoms and dysfunction may persist, or long-term complications may occur.

COMPLICATIONS

Most athletes with concussion have complete resolution of symptoms in 1 to 2 weeks and are able to return to their previous level of academic and athletic functioning. Still, some studies suggest short-term deficits in cognitive processing even after symptoms have abated. In a minority of patients, usually children or teenagers, symptoms may persist for weeks. An even smaller group may have symptoms that last for months, a condition known as postconcussive syndrome (PCS), described below. Despite all the research into concussions in the last decade, questions still exist regarding their long-term consequences.

Short-Term Sequelae

Concussion can result in cognitive deficits, such as poor short-term memory and impaired concentration, which may persist for up to a week. College athletes who suffered concussions have been shown to have concussion symptoms that lasted 7 days. Cognitive functioning improved to baseline levels within 5 to 7 days, and balance deficits dissipated within 3 to 5 days after injury.[17] Frequently symptoms resolve before complete resolution of cognitive dysfunction occurs.

PCS is a complex condition in which concussive symptoms last weeks, months or even years after the initial injury. As of today, there is no consensus regarding an exact definition of PCS. Commonly

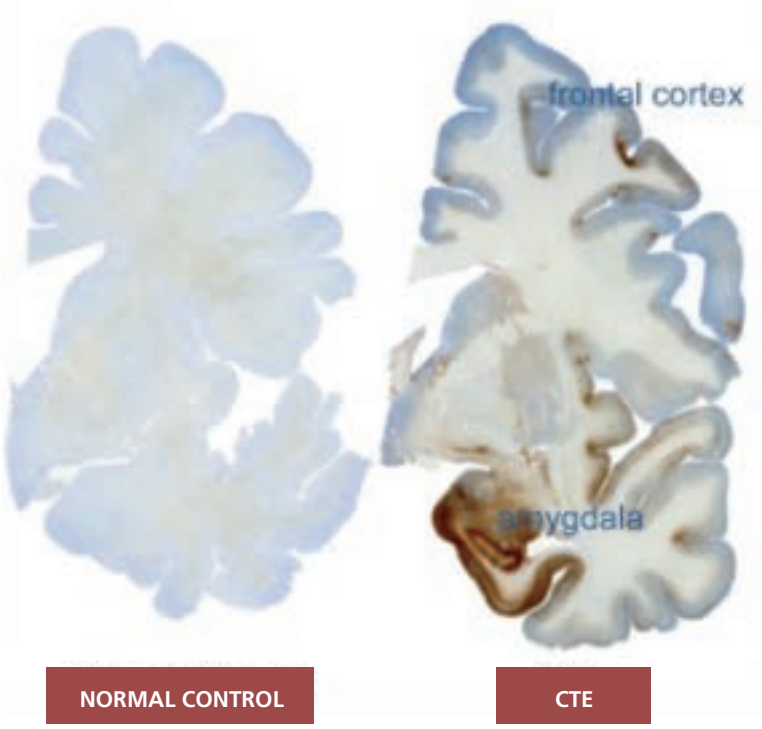

FIGURE 3.

Chronic traumatic encephalopathy (CTE) is diagnosed postmortem by examining brain tissue grossly and microscopically. The image on the left shows normal brain tissue. The image on the right shows a brain with dark staining of tau protein consistent with CTE.

reported symptoms include headache, dizziness, fatigue, irritability/depression, difficulty with concentration, impaired memory, insomnia, and reduced exercise tolerance. The exact relation of a concussion to PCS is poorly understood. There is no proven correlation between the severity of a concussive event and the development of PCS.[19] Physicians focus treatment on improving both symptoms and the patient's ability to function at school or work. Medications such as tricyclic antidepressants, antiepileptics, and sleep aids are often used to ameliorate symptoms. Cognitive therapy, neurorehabilitative programs, and progressive exercise programs may help decrease symptoms.[19] PCS appears to resolve over many months, but the process is slow and often frustrating for the athlete.

Second-Impact Syndrome

Second-impact syndrome (SIS) occurs when an athlete who has suffered a concussion and is still experiencing symptoms receives a second head injury.[20] The presumed pathophysiology includes loss of cerebral vascular autoregulation, leading to rapid cerebral edema, increased intracranial pressure, herniation, and ultimately coma or death. Those athletes who survive are neurologically devastated almost universally. Children and adolescents seem to be especially susceptible to this rare condition. All reported cases of SIS have occurred in athletes under the age of 20.[21]

Long-Term Sequelae

There are serious concerns regarding long-term effects of repetitive head trauma. Repeat concussions may increase a person's risk in later life for dementia, Parkinson's disease, and depression.[22] The term *dementia pugilistica* was coined to describe boxers who developed dementia after years of the sport and is now thought to actually represent a condition called chronic traumatic encephalopathy (CTE).

CTE is a progressive, degenerative neurological disease found in individuals such as professional hockey and football players, professional wrestlers, boxers, and soldiers who have been subjected to repetitive traumatic head injuries. CTE is diagnosed postmortem by examining brain tissue grossly and microscopically. The most striking findings include cerebral atrophy and tau protein deposition, findings similar to those in Alzheimer's disease (Figure 3). In their final years, these former athletes suffer from depression, headaches, aggressive behaviors, memory loss, confusion, and poor judgment. Death usually occurs by suicide, often by age 50.

CHILDREN

Children and younger athletes appear to be particularly susceptible to concussions and complications from traumatic brain injuries. The reason for this increased susceptibility remains unclear. The physiology of the developing brain differs from that of the adult brain with respect to degree of myelination, cerebral blood flow and blood volume, number of neuronal synapses, and brain water content. Children also have larger head size with respect to their body and less developed neck musculature than adults.[23]

Although recovery patterns have not been adequately studied in young athletes, most experts agree that concussion symptoms take longer to resolve in children than in older athletes—many times twice as long. On computerized neuropsychological testing, high school athletes with concussion scored significantly worse than age-matched controls 7 days after injury, while college students' scores returned to normal by 3 days post-concussion.[11] These findings suggest that when managing concussions in younger athletes, physicians should proceed cautiously because cognitive impairments may persist beyond symptom resolution.

As mentioned above, SIS appears to be a phenomenon that affects young athletes because no cases have been described in athletes older than 21 years of age. Debate exists as to whether SIS actually represents a separate form of diffuse cerebral swelling, malignant brain edema, seen in children.[24] Regardless whether SIS exists as a discrete condition, the severity of this outcome

Rehabilitation Stage	Functional Exercise Performed	Objective
1. No Physical Activity	Physical and cognitive rest	Recover
2. Light Aerobic Exercise	Stationary bike at 70% intensity	Elevate heart rate
3. Sport-Specific Exercise	Increased-intensity activity, sports-related training drills	Elevate heart rate and add movement
4. Noncontact Training Drills	Progression to more complex sports-specific drills. May begin resistance training.	Progress exercise tolerance and coordination
5. Full Contact Practice	Normal training activities when cleared	Instill confidence and allow assessment of skills
6. Full Return to Play	Normal game play	

TABLE 3.
Return-to-Play Table

in the period after a concussion warrants prudence and caution with respect to return-to-play decisions in concussed young athletes.

RETURN TO PLAY

Because of the subjectivity of symptoms and the lack of objective measures of brain function, a determination of symptom resolution and guiding safe return to activity can be challenging. Once the athlete has noted complete resolution of symptoms for at least 24 hours, the clinician can consider beginning the athlete on a graduated return-to-play protocol.

The graduated return-to-play protocol is a gradual, stepwise progression in physical activity over a 1-week period, with the eventual goal of return to full sports activities (Table 3). When noting complete resolution of symptoms, it is important to inquire about the use of any medications that may mask or alter the symptoms of a concussion. In addition to symptom resolution, the athlete should have a normal neurologic examination and, if utilized, neurocognitive testing should be at baseline.

Return to play should be considered and managed on a case-by-case basis. As a rule, athletes should not be returned to play on the same day as their concussion. This is especially important in management of child, adolescent, and collegiate athletes. Multiple studies have shown that athletes of these ages will often exhibit neuropsychological deficits in the absence of signs and symptoms and therefore should not be allowed same day return to play. Moreover, younger athletes have been shown to have delayed onset of symptoms and would be placed at high risk of subsequent injury, recurrence, or SIS if allowed to return to play on the same day.

Upon return to baseline, the athlete can begin a graduated return-to-play protocol.

CONCLUSION

SRCs affect men and women, boys and girls, young and old. A concussion can occur in any collision sport, but the highest-risk sports appear to be football, hockey, soccer, and basketball. Symptoms usually include headache, dizziness, trouble concentrating, memory deficits, and mental fogginess, however LOC is uncommon.

Doctors base diagnosis primarily on the presence of symptoms, and, because athletes may be reluctant to report symptoms, clinicians need to maintain a high index of suspicion. The mainstay of treatment is physical and mental rest. The athlete needs to remain out of sports until all symptoms resolve and then should begin a graded exercise program. Clinicians should clear an athlete to return to competition only when the athlete is symptom free both at rest and with exercise, is off medication, has a normal neurologic examination, and demonstrates neuropsychological testing that has returned to baseline levels

Clinicians who care for athletes of all levels should stay current with guidelines for concussion management, educate their athletes and coaches on the potential dangers of concussion, and develop a concussion safety plan with their school or sports organization. ■

*Regarding use of the SCAT 2: In November 2012, a group of international experts at the Fourth International Consensus Meeting on Concussion in Sport in Zurich, Switzerland, devised the SCAT 3, which is to be used in athletes aged from 13 years and older. This will take the place of the prior SCAT forms. Children 12 years or younger should use the Child SCAT3. The SCAT3 tool can be downloaded the website of the *British Journal of Sports Medicine*.

References and financial disclosures are available online at www.rush.edu/orthopedicsjournal.

"UTILIZING A NOVEL METHOD OF OBJECTIVE 3D CT ANALYSIS, WE HAVE BEEN ABLE TO ANALYZE DIFFERENT ASPECTS OF CAM DEFORMITIES BASED ON GENDER."

Gender Differences in Cam Deformities in Patients with Femoroacetabular Impingement

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Femoral acetabular impingement (FAI) includes pathology involving abutment of the femoral neck and the acetabular rim at the extremes of range of motion.[1] FAI lesions can be primarily related to the acetabulum (pincer), femoral neck (cam), or most commonly, a combination of the two. FAI can lead to labral injury, osseous changes, and even osteoarthritis.[2,3] Ideally, patients that are currently symptomatic or at risk for further disease progression can be identified and stratified for intervention. Unfortunately, FAI remains difficult to diagnose, though significant work has been done to improve imaging-based diagnosis.

When diagnosing cam deformities, it has been shown that differences in baseline anatomy exist with regard to gender. These evaluations typically utilize the alpha angle, which was originally described utilizing magnetic resonance imaging (MRI).[4] Studies utilizing the radiographic measurement of the alpha angle have suggested that normal femoral head sphericity is less than 68° for men (normal average, 59°) and less than 50° for women (normal average, 46°).[5,6] Abnormalities in these measurements correlate with increased risk of cartilage damage,[4,7] increased delayed gadolinium-enhanced MRI of cartilage (dGEMRIC) index,[8] and labral damage.[9] However, radiographic measurements have demonstrated poor interobserver reliability[10] and inconsistency.[11] Even using specific methods, such as the Dunn view, the extent of a given cam deformity can be difficult to quantify.[12] These difficulties are secondary to the attempt of planar description of a complex 3-dimensional (3D) lesion. Due to difficulty in measuring these lesions on radiographs and gender differences, studies evaluating gender differences in cam deformities based on computed tomography (CT) evaluation are needed.

We set out to quantify the 3D differences between male and female cam deformities utilizing CT scans. Understanding these differences in gender may help surgeons diagnose more subtle deformities in female patients, along with aiding surgeons in planning cam resection.

MATERIALS AND METHODS

We conducted a retrospective study to analyze 23 femurs in 21 patients with an average age of 41 years (range, 25-55 years). After the institutional review board (IRB) approved the investigation, all patients signed the appropriate consent for enrollment. The senior author saw all patients in clinic and determined that all had a symptomatic cam deformity. We confirmed this determination with radiographs and CT scan imaging with 3D reconstructions. Scan-slice thickness varied from 0.63 to 3 mm. This group of patients subsequently underwent arthroscopic osteochondroplasty to treat the symptomatic deformity. After the patients met the criteria, we segmented their Digital Imaging and Communications in Medicine (DICOM) data from the operated hip utilizing Mimics 13.1 software (Materialise; Leuven, Belgium) and predetermined Hounsfield units (> 226 for bone). We converted the resulting 3D femoral and acetabular models to point-cloud data, which we then analyzed with a custom written program created in Microsoft Visual C++ with Microsoft Foundation Class (MFC) programming environment (Microsoft Corp., Redmond, WA) (Figure 1). To find the gravity center of the femoral head, we assumed it to be a perfect sphere. From this, a virtual point near the gravity center was moved ± 5 mm in x, y, and z directions in 1.0-mm increments until the standard deviation of the distance to each point on the surface became the smallest. We repeated this procedure within a search range of ± 0.5 mm in 0.1-mm increments. From this point, a virtual sphere was created that mimicked the contour of the femoral head. Then we quantified the cam deformity three dimensionally, using the distance from the cam deformity surface points to the centroid of the femoral head. These

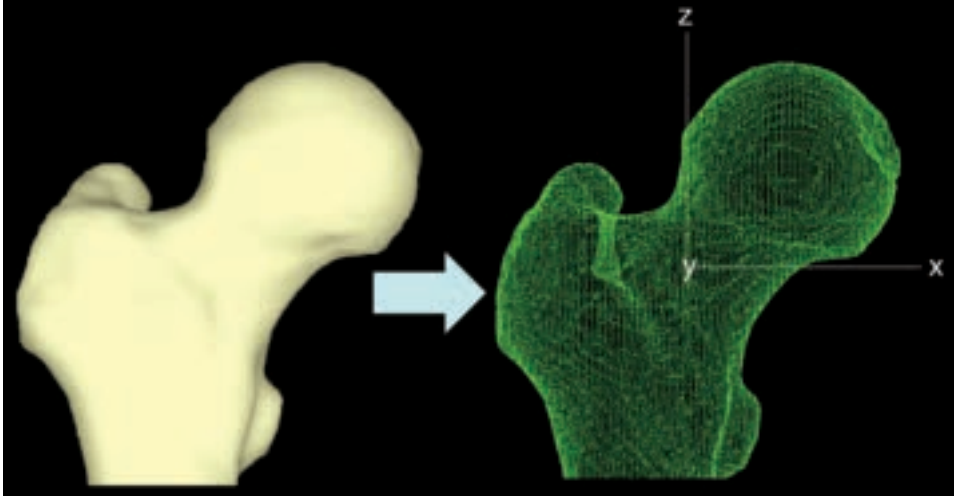

FIGURE 1.

Three-dimensional CT data are converted into point cloud data, which are rendered using Mimics software.

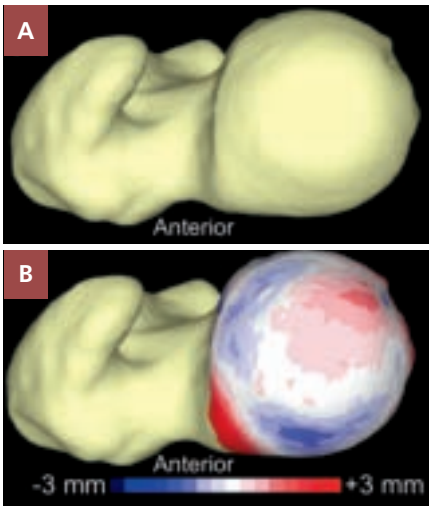

FIGURE 2.

An axial view facing caudad demonstrates the segmented femur: **A,** before analysis and **B,** after analysis. Areas in red demonstrate regions that are proud, whereas blue regions are recessed relative to the sphere of best fit.

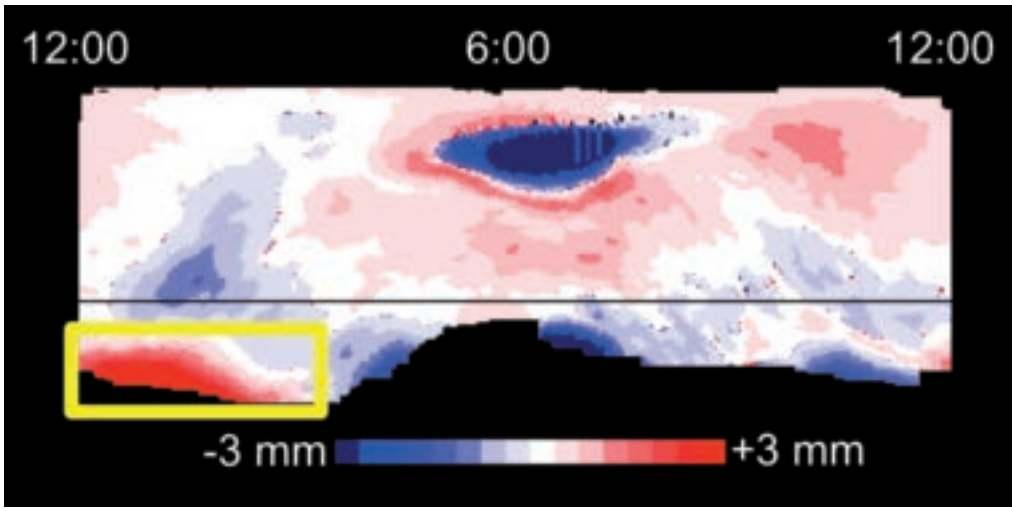

FIGURE 3.

The map projection of the globe view demonstrates the lesion (yellow rectangle), which can be analyzed for height and volume characteristics.

data could then be visualized in 1 of 3 methods: (1) globe view—3D point-cloud model with a color spectrum representing relative valleys and prominences (Figure 2); (2) atlas view—planar projection of the entire femoral head with the same color spectrum (Figure 3); and (3) histogram—distances from the optimized center to surface points. We then applied the modified Ilizaliturri zoning method[13] to the data, with zones 1-6 being contained within the acetabulum and zones 7-9 being lateral to the acetabular rim. We omitted zone 6 from the analysis because it corresponds with the insertion of the ligamentum teres. Zones 1, 2, and 7 are anterior, while 8 is midlateral (Figure 4). We calculated the mean bump height, volume, location on the clock face, and relative zone prominence and analyzed gender differences regarding these outcomes. We analyzed zonal differences using analysis of variance (ANOVA) with posthoc Tukey test with significance set at $P < .05$. Finally we completed the remaining comparisons utilizing an unpaired t test. We performed all calculations in Microsoft Excel with XLSTAT (Addinsoft; New York, New York).

RESULTS

Patients were an average age of 41 years old with a range from 25 to 55 (male: 12, female: 11). Of the 21 patients, 10 had a symptomatic left hip, and 13 complained of right hip pain, with 1 patient having bilateral disease. Average CT slice thickness was 1.6 mm, and the mode was 0.63 mm. The average lesion height was 1.34 mm (range, 0.5-2.42 mm) from the assumed spherical femoral head. Cam volume was 535 mm on average, ranging from 125 to 1381 mm. Male lesion height (1.54 ± 0.34 mm) was larger than the female lesion height (1.11 ± 0.52 mm, $P < .03$). Similarly, male cam volume (709 ± 338 mm^3) was larger than female lesion volume (344 ± 331 mm^3, $P < .02$). The average distribution on the clock face was from 1:09 hours ± 2:51 to 3:28 hours ± 1:59, with an average span of 3:06 hours ± 1:29. The zone with the largest average height was most commonly zone 8 (10 patients), followed by zone 7 (7 patients). When we compared zones, the ANOVA results demonstrated the zone that was most prominent in any given patient was significantly larger than all other zones in all cases. However, the location of the zone did not demonstrate a predilection based on gender ($P = .299$)

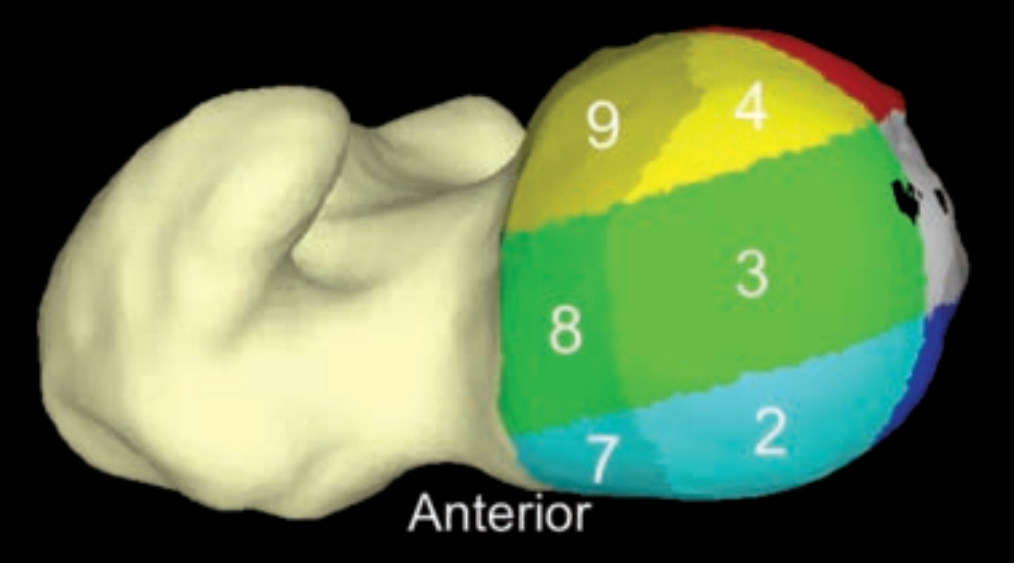

FIGURE 4.

The modified Ilizaliturri zoning system applied to this model demonstrates the distal anterior (7) and central (8) zones.

DISCUSSION

Currently, gender differences in cam deformities have been described radiographically, suggesting differences in alpha angles for male and female patients. Generally a normal alpha angle has been reported to be less than 45°, and an alpha angle greater than 50° has been associated with cam deformity.[4] However, due to variability in the 3D morphology of the proximal femur, the size and location of the cam deformity has been difficult to describe objectively. Specifically, current methods have not been able to elucidate gender specific differences of cam deformity. Utilizing a novel method of objective 3D CT analysis, we have been able to analyze different aspects of cam deformities based on gender.

The location of the cam deformity did not vary based on gender. This is in contrast with data from Ito et al, which demonstrate that lesions in young males were more lateral-to-anterior compared to lesions in older females being anterolateral to anterior, based on offset measurements.[14] Our study was not powered to analyze differences based on both age and gender, which may have limited our ability to detect location differences. As stated previously the average distribution on the clock face was from 1:09 to 3:28 with an average span of 3:06 hours, and the zone with the largest height was in zone 8. Depending on their precise location, the cam lesions, especially those centered at 3:00, may not be detected on plain radiographs or may be subtle enough to be missed on imaging studies. With this, the more subtle nature of female cam lesions may explain some of the decreased reliability of 2D evaluation of these lesions.

The volume and height of cam lesions vary significantly based on gender, with male lesions being significantly larger. Differences in head sphericity averaged 1 mm between genders, with female lesions being about 50% of the volume of male lesions. Importantly, these aspects may lead to underdiagnosis of female lesions compared to male lesions. Similarly, the amount of resection performed during cam osteochondroplasty may vary depending on lesion height, length, volume, and gender.

Jung et al recently published a 2:1 ratio of cam lesions in symptomatic males compared to females.[6] These data were based on anteroposterior scout views from CT scans using the cutoffs suggested by the Copenhagen Osteoarthritis Study.[5] Our study suggests that the true incidence in females may be larger than appreciated based on the smaller nature of female cam lesions. Similarly, Chen et al published increased incidence of cam lesions in Chinese patients with idiopathic hip pain.[15] However, when using a lower cutoff for radial height/radius ratio measurements on anteroposterior radiographs, they detected a stronger correlation in patients with symptoms having lesions. Therefore, lesions may be subtler in the Chinese populations, demonstrating the inadequacy of the alpha angle. Masjedi et al analyzed symptomatic patients with FAI and demonstrated a significant difference in male and female patients with regards to the head-neck surface area ratio.[16] Specifically, male deformities were noted to be larger (ratio, 2.59-2.91) compared to females (ratio, 2.56-2.66). Based on these data, we believe patients that demonstrate clinical signs of impingement with negative radiographic findings require more detailed evaluation, such as quantitative 3D analysis.

This study has several limitations, including a small sample size. Increasing the number of subjects may allow for further subgroup analysis, such as age differences. We did not collect the CT scans from the same CT scanner, and therefore, the thickness of slices was variable. The quality of the segmentation was dependent on the slice thickness. The most notable limitation of our methods was the lack of analysis of the femoral neck. With this, we believe the characterization of the femoral head to be an important initial step to further and more complete analysis. The end goal involves creating more accurate diagnosis and a language to describe cam deformities that can help direct both clinical and surgical decision making. Lastly, our study does not draw any correlation between the alpha angle and CT data, which would be useful to compare CT scans to plain radiographs.

CONCLUSIONS

The quantification of cam deformities is critical for an improved understanding of the pathology in patient populations with more subtle lesions. Similarly, guidance for resection will also lead to improved understanding of the amount of surgical intervention necessary. ■

References and financial disclosures are available online at www.rush.edu/orthopedicsjournal.

"OUR RESULTS SUGGEST THAT . . . JUDICIOUS CAPSULAR MANAGEMENT IS INDICATED DURING ARTHROSCOPIC HIP PROCEDURES."

The Effect of Capsulotomy on Hip Stability

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Hip arthroscopy is an increasingly popular procedure with steadily expanding utility and broadening indications. In order to execute arthroscopic procedures of the hip, a capsulotomy is often performed because it aids the surgeon's ability to achieve intra-articular visualization, facilitates instrument exchange, and enhances maneuverability within a highly constrained joint. As such, an appropriate capsulotomy is a necessity when performing arthroscopic procedures in the central compartment. Additionally, capsulotomy improves visualization of the peripheral aspect of the hip for the treatment of cam lesions and other extra-articular pathologies. Consequently, interest in the contribution of capsular structures to native hip stability has increased.

The role of the hip joint capsule in stability or kinematics is relatively unknown. Martin et al performed an initial biomechanics study investigating the role of the iliofemoral, ischiofemoral, and pubofemoral ligaments.[1] The triangular-shaped iliofemoral ligament (Y-ligament of Bigelow) is the strongest of the capsular ligaments. It arises from the anterior inferior iliac spine of the pelvis and extends distally and laterally along the femoral neck to attach to the intertrochanteric line of the anterior femur. The iliofemoral ligament is taut in a position of extension and external rotation of the hip and loose in flexion and internal rotation.[2] Martin et al reported that the iliofemoral ligament was important in resisting anterior translation of the femoral head from within the acetabulum, with its lateral arm limiting internal rotation in extension. The pubofemoral ligament was shown to control external rotation in extension. The ischiofemoral ligament was found to be the most significant resistor of internal rotation forces of the hip, as well as resistor to adduction forces.[1]

In this in vitro model, we attempt to further understand the effect of a transverse capsulotomy, as is often performed during hip arthroscopy, (1) on the rotational kinematics of the hip and (2) on the translational kinematics of the hip. We hypothesized that both increased rotational and translational femoro-acetabular motion would be observed after capsulotomy.

METHODS

Thirteen fresh frozen cadaveric hip specimens consisting of the hemipelvis, femur, and overlying soft tissues were obtained. All specimens were screened by computed tomography (CT) examination to assess acetabular and femoral version and to confirm the absence of bony pathology. Inclusion criteria included hips with a center edge angle less than or equal to 25° and a Tönnis grade 1 or less. Exclusion criteria included hips with a center edge angle greater than 25° and a Tönnis grade greater than 1. After thawing each specimen for 24 hours, we removed all muscle and soft tissue from each specimen by careful dissection, leaving the hip capsule and labrum intact. The femur was transected at the junction of the proximal and distal thirds to allow for potting in polymethyl methacrylate (PMMA) in a cylindrical polyvinyl chloride (PVC) mold. The iliac wing of the hemipelvis was placed in a 10.6 × 10.6-cm mold to allow for potting in PMMA. The acetabular seal was vented by placing a 20-gauge needle between the labrum and the bony acetabulum. Each specimen was placed into a modified version of the loading apparatus described by Provencher.[3] The apparatus allowed for adjustment of flexion, extension, and axial rotation of the femur around a static ilium and acetabulum.[3]

A set of 6 reflective markers was rigidly attached to the specimens to allow for 3D position tracking. A motion tracking system (Eagle-4 cameras and EVaRT analysis software; Motion Analysis Corp., Santa Rosa, CA) was used to record the experimental kinematics, postprocess translation, and rotation data. The loading apparatus holding each specimen was mounted on an *x-y* displacement table (a modified Provencher frame). An external rotation torque of 0.588 Nm was applied via static load and held while data were recorded for 10 seconds

for each loading condition. This torque magnitude was chosen because our pilot testing demonstrated that 0.588 Nm was sufficient to cause full external rotation of the femur without causing impingement of the greater trochanter on the acetabulum at terminal rotation.

Each hip was tested under 4 conditions: (1) neutral flexion with capsule intact; (2) neutral flexion with transverse capsulotomy; (3) maximum flexion with capsule intact; (4) maximum flexion with transverse capsulotomy. The transverse capsulotomy was performed on the anterior aspect of the femoral neck, 1 cm from acetabular rim. It was continued distally, parallel to the labrum, involving the entire iliofemoral ligament.

The 3-dimensional (3D) position of the markers in space was analyzed using the Euler angle calculation in order to obtain translational and rotational parameters. A CT scan was obtained of each specimen, and a virtual model was segmented utilizing Mimics software (Materialise; Leuven, Belgium). In the model, the femoral and pelvic bones were separately extracted at the neutral position. These were superimposed over the images of each different position, using voxel-based registration to evaluate femoral head translation after the application of the external rotation torque. Differences between experimental groups were assessed with both analysis of variance (ANOVA) and nonparametric analysis. The level of significance was set at $P < .05$.

RESULTS

We compared femoral-acetabular motion caused by an applied external rotation torque for each testing condition in terms of translation and rotation. The vector components of the rotation observed for each applied torque were analyzed in the x, y, and z axes. These equated to flexion, abduction, and external rotation components, respectively. When testing in neutral flexion, there was no significant difference in rotation in any plane between the hips with intact capsules and hips with capsulotomy (Figure 1). However, for hips tested in maximum flexion, there was a significant difference in rotation found in the y and z axes after capsulotomy (y axis = 0.4° before capsulotomy, 1° after capsulotomy; $P = .01$) (z axis = 0.30° before capsulotomy, 1.20° after capsulotomy; $P = .02$). This equated to a 0.6° increase in abduction after capsulotomy and a 0.9° increase in external rotation after capsulotomy (Figure 2).

There were no statistically significant differences for displacement of the femoral head after torque application (Table 1). However, displacement vectors were plotted in a Cartesian coordinate system to visualize any changes in femoral head translation after torque application in the distal/proximal, anterior/posterior, and medial/lateral planes (Figure 3). Several qualitative changes in directionality were observed. Femoral head translation for hips tested in neutral was likely more distal, anterior, and lateral. This was true for hips with intact capsules and hips after capsulotomy. Femoral head translation for hips tested in flexion was inclined mostly to be distal, posterior, and lateral. However, after capsulotomy, hips tested in flexion leaned towards distal, posterior, and medial translation (Figure 4).

To better understand these qualitative observations, the mean displacement of all specimens in each testing condition was also plotted. Again, there was no statistical significant difference found in displacement of the femoral head after applied torque. Only qualitative directional tendencies were observed. In general, specimens tested in neutral rotation were likely to demonstrate anterior displacement of the femoral head both before and after capsulotomy. This anterior displacement was greater after capsulotomy (0.17 mm anterior before capsulotomy, 0.22 mm anterior after capsulotomy). Specimens tested in flexion had a predisposition to demonstrate posterior displacement of the femoral head both before and after capsulotomy. This posterior displacement was also greater after capsulotomy (0.23 mm posterior before capsulotomy, 0.61 mm posterior after capsulotomy) (Table 1).

There was increased qualitative distal displacement after capsulotomy in neutral and flexed hips, with a more marked difference for hips tested in flexion (neutral: 0.20 mm before capsulotomy, 0.87 mm after capsulotomy; flexed: 0.23 mm before capsulotomy, 1.05 mm after capsulotomy).There was an observation of greater qualitative lateral displacement after capsulotomy in specimens tested in neutral (1.64 mm laterally before

FIGURE 1. Rotation in neutral flexion.

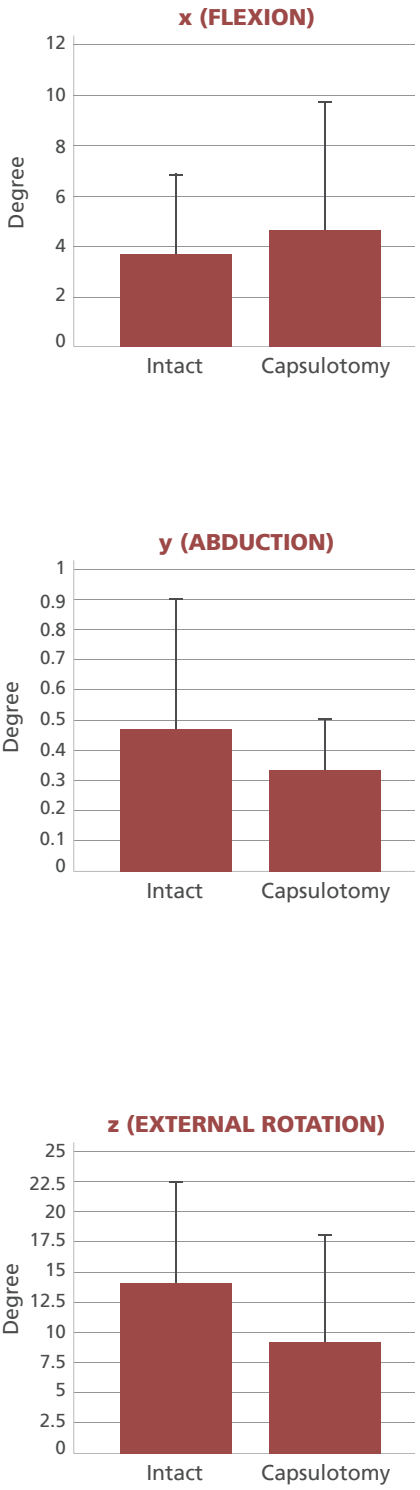


FIGURE 2. Rotation in maximum flexion.

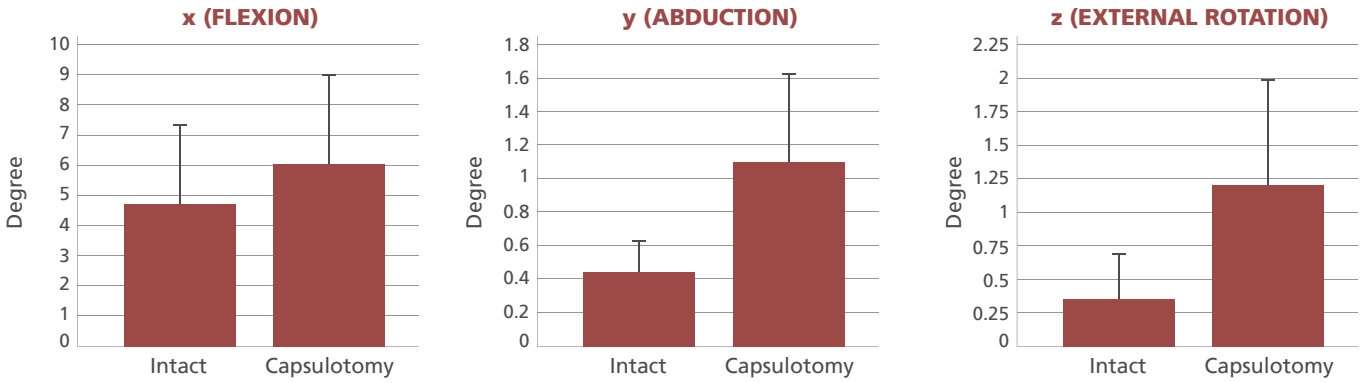


TABLE 1. Displacement

		MEAN		
		ML	AP	PD
Neutral	Intact	1.64 (0.75)	0.17 (1.11)	–0.20 (0.48)
Neutral	Caps	1.68 (1.81)	0.22 (0.73)	–0.87 (1.29)
Flexed	Intact	0.09 (1.14)	–0.23 (0.55)	–0.23 (1.50)
Flexed	Caps	–0.75 (2.11)	–0.61 (1.95)	–1.05 (1.32)

Abbreviations: Caps, capsulotomy; ML, medial negative, lateral positive; AP, anterior positive, posterior negative; PD, proximal positive, distal negative. All units are in mm. Standard deviations are in parentheses.

capsulotomy, 1.68 mm laterally after capsulotomy). Similarly, there was greater qualitative medial displacement after capsulotomy for hips tested in flexion (0.9 mm laterally before capsulotomy, 0.75 mm medially after capsulotomy).

DISCUSSION

Recently, the capsular structures of the hip have received increased attention as the pervasiveness of nonarthroplasty intra-articular hip procedures continues to increase rapidly. The question of how the associated capsular sectioning required to perform these procedures affects femoro-acetabular mechanics becomes increasingly more relevant, and several reports of hip instability after hip arthroscopy have been described.[4,5] Consequences of this instability may be as severe as femoro-acetabular dislocation.[4,5] Few studies exist examining the contribution of the capsule to the stability of the hip joint.[1,6,7,8]

The objective of this experimental work was to demonstrate the effect of a transverse capsulotomy on hip stability by evaluating its effect on rotational and translational hip kinematics. In a study by Myers et al, the investigators observed increased rotation after iliofemoral ligament sectioning for hips experiencing torque while in a flexed position.[8] The larger increase in external rotation (12.9° ± 5.2°)[9] observed in that study is likely due to the larger torque used (5 N·m in our study vs 0.588 N·m used in Myers et al). There is no consensus on what loads should be used for this type of cadaver study.

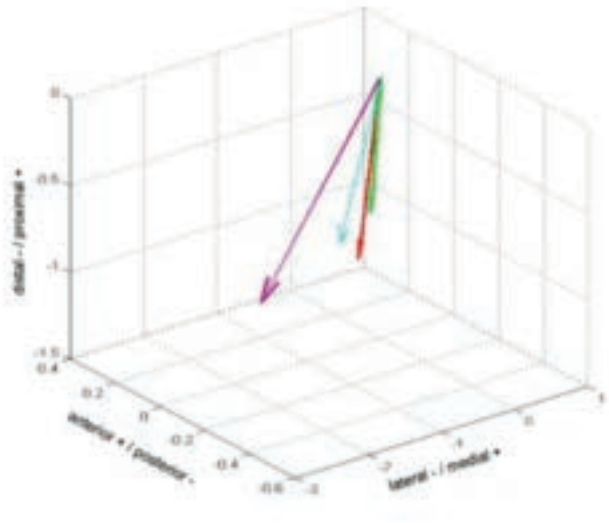
Transverse capsulotomy may also permit greater distal, lateral, and anterior displacement of the femoral head within the acetabulum in neutral and increased medial, posterior, and distal translation of the femoral head in flexion. Data suggest that the overall trend in motion after the application of an external rotation torque is such that after capsulotomy, hips that sustain the torque in neutral rotation tend to experience greater translation than rotation. Contrastingly, hips that sustain the torque in flexion trend towards greater rotation than translation (Figure 3). We believe that these observations are largely attributable to the relationship between the femoral head and acetabulum with certain range of motion and the anatomic location of transverse capsulotomy.

In this study, we attempted to duplicate the capsulotomy generally utilized for hip arthroscopy. The arthroscopic transverse capsulotomy typically begins 1 cm from the acetabular rim and continues parallel to the labrum, connecting the anterior and anterolateral portals. It principally involves the iliofemoral ligament.

In addition to contributing to the resistance of internal and external rotation in extension, the lateral arm of the iliofemoral ligament has also been shown to limit external rotation in flexion.[1] It is this latter function that we believe explains the increase in rotation observed after the application of torque for hips tested in flexion. With transverse capsulotomy, the iliofemoral ligament loses its ability to resist rotation in flexion, and an increase in external rotation is observed.

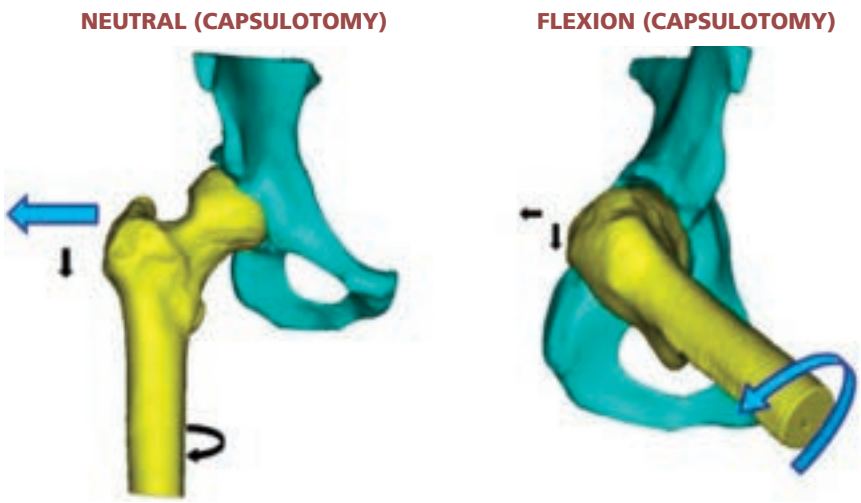
Perceived increases in anterior displacement of the femoral head after capsulotomy in neutral are also likely due to iliofemoral ligament compromise, because it has been shown to resist anterior translation of the femoral head.[1] The inability of the ligament to perform this function after capsulotomy ostensibly permits greater anterior translation of the femoral head from within the acetabulum and explains our observed trends.

FIGURE 3. Mean displacement.



LEGEND
Neutral (intact) - light blue
Neutral (capsulotomy) - purple
Flexed (intact) - green
Flexed (capsulotomy) - red

FIGURE 4. Overall motion trends after capsulotomy.



After capsulotomy, the increased qualitative lateral and distal displacement in neutral and increased qualitative medial and distal displacement in flexion is likely a result of the bony congruency of the femoro-acetabular joint. This translation represents the normal path that the femoral head traverses when moving against the inferior aspect of the acetabulum with rotation. Without the contribution of the iliofemoral ligament to anterior capsular restraint, the femoral head set into motion after an external rotation torque presumably follows the contours of the acetabulum as it rotates. As it continues, it probably translates laterally and distally when the hip is in neutral and medially and distally when the hip is in flexion.

After torque with the hip in flexion, there is more rotation and less translation because the femoral head is more engaged in the acetabulum. In neutral position, the femoral head is less engaged, and more translation compared to external rotation is observed.

Of note, specimens tested in flexion qualitatively demonstrated posterior displacement of the femoral head both before and after capsulotomy. This may explain the "contrecoup" pattern of cartilage damage (posteroinferior lesion of femoral head and acetabulum believed to be due to subtle subluxations of the hip) over the femoral head and corresponding acetabulum often observed during hip arthroscopy. Flexed and rotated hips in the presence of capsular or labral damage may lever off the anterior acetabulum and impact the posterior acetabulum causing posteroinferior acetabular cartilage injury.

We describe a novel approach for the analysis of native, cadaveric hip motion. The accurate quantification of positional changes of cadaver tissue in capsular studies is difficult.[10,11] Previous work ranges from the use of the photoelastic coating method to measure strain in cadaver knee ligaments[10] to elaborate roentgen stereophotogrammetry (RSA) models that need artificial nominal-strain states that might not be physiological but that are warranted from an engineering testing perspective.[11] More recently, the use of biplanar fluoroscopy with RSA to study cadaver femoro-acetabular motion has provided some initial results, but there are concerns regarding tissue quality, due to the extensive number of repetitions for each condition (n = 20).[8] In their study, Myers et al also observed increased rotation in flexion after iliofemoral ligament sectioning.[8]

To the best of our knowledge, this is the first study to analyze hip kinematics after capsular sectioning, using motion capture analysis. Crawford et al evaluated the kinematics of the hip after labral venting and sectioning utilizing motion capture analysis.[12] Based on their data, they concluded that a breach in labral integrity decreases femoral stability.[12] The biomechanics laboratory at Rush has expertise in using motion capture methodology to quantify joint mechanics in cadaver tissue. [13,14, 15] The postprocessing method developed in our laboratory makes use of CT-based models to accurately determine the spatial relationships between marker position and bone geometry to define the trajectory of the rigid body centroid that is tracked by the motion analysis system, making it a more robust approach. Conversely, biplanar fluoroscopy and RSA are dependent on accurate calibrations in order to remove distortion artifacts, and frequently, image registration is carried out by hand to evaluate changes in rigid body kinematics. As such, motion capture analysis may theoretically minimize human error.

Furthermore, this is the first capsular hip motion study to analyze native femoral head rotation as a consequence of torsion in its component axes (abduction, flexion, and external rotation) and to examine the direction of translation of the native femoral head in its component vector planes (anterior/posterior, medial/lateral, proximal/distal).

This study, unfortunately, is not without limitations. One such limitation is its small sample size. Prior cadaveric hip motion model studies have used between 6 and 24 hips.[1,8,12] Related to this limitation is the fact that only qualitative tendencies, not statistical differences in translation, were observed after capsulotomy. Larger studies are warranted to further examine how capsular integrity affects hip stability. Another limitation of this study is the effect of bony morphology on hip kinematics. We attempted to minimize variability by controlling for hips without evidence of acetabular dysplasia or arthritis. However, it is unknown how variations in hip morphology and orientation may affect rotational and translational motion. Finally, this is a cadaveric study, and as such we are only able to evaluate the kinematics of the femoro-acetabular joint in vitro. Presently the clinical significance of our observed differences in rotation and qualitative observational tendencies in translation are unknown. We evaluated only the static stabilizers of the hip, and so this study does not account for dynamic sources of stability including the surrounding musculature. Furthermore, we are able to evaluate only data immediately after the capsulotomy. Behavior of the hip joint over time after capsulotomy is unknown for the time being. Simulation studies may be able to provide an answer if the appropriate constitutive equations are developed. The literature shows very few studies on hip capsule instability and even fewer analytical models. A May 2012 Pubmed search showed only 52 articles with the search terms "hip capsule instability." The vast majority of those articles were clinical reports. The only finite element study shown in that search, Elkins et al, has innovated in this area, but the focus of their instability investigation was on a total hip arthroplasty (THA) model and not on femoro-acetabular impingement (FAI).[6] As such, the capsular ligament elastic anisotropy and spatial variation in capsule tissue thickness were not included in the model due to the added computational expense and complexity.[6] Knowledge of these properties will also help to describe how the model's kinematics are affected by capsular healing. However, we believe that the observations made in this study are motivating and warrant further investigation. Given the possibility of increased femoral head translation with capsulotomy, we believe that judicious capsular management is indicated during hip arthroscopy.

CONCLUSION

Our results suggest that the presence of transverse capsulotomy appears to permit increased rotation in maximum flexion compared to hips with intact capsules. Capsulotomy may also allow greater translation of the femoral head in both neutral and flexion. As such, we believe that judicious capsular management is indicated during arthroscopic hip procedures. ■

References and financial disclosures are available online at www.rush.edu/orthopedicsjournal.

“THE CURRENT STUDY ADDS TO OUR UNDERSTANDING OF IMPINGEMENT-INDUCED HIP DEGENERATION WITH THE COMPARISON OF ARTHROPLASTY PARTICIPANTS IN TREATMENT-MATCHED COHORTS BELOW 50 YEARS AND THOSE 50 YEARS AND OVER.”

Age-Related Differences in Radiographic Parameters for Femoroacetabular Impingement in Hip Arthroplasty Patients

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There are multiple etiologies that can lead to end-stage degenerative joint disease of the hip in young patients. Dysplasia, osteonecrosis, Perthes disease, posttraumatic deformity, and inflammatory arthropathies are among the common causes; however, there are a large number of young patients who appear to have a primary arthritic process. Femoroacetabular impingement (FAI) has been proposed as a significant cause of this premature hip degeneration in patients who are less than 50 years old[1-5] (Figures 1 and 2). Advancements in the diagnosis and understanding of FAI have led to the development of new treatment algorithms and modalities. This is clearly seen in the exponential increase of arthroscopic hip procedures that are performed in contemporary orthopedic practice.[6] However, the questions still remain regarding the cause, effect, and relationship of FAI to early degeneration of the hip.

Clohisy et al[4] recently reported that 33% of participants under the age of 50 who underwent a total hip arthroplasty had radiographic evidence of FAI. The authors also found a 73% rate of arthroplasty or advancement in osteoarthritis grade in the contralateral hip with bilateral findings of FAI. These results suggest a distinct relationship between impingement and end-stage hip degeneration. However, Laborie et al[7] found a relatively common prevalence of cam deformities (in 46.2% of males and in 12.1% of females) as well as pincer deformities (in 38% of males and in 15.9% of females) in a prospective study of asymptomatic volunteers. This calls into question the pathologic relationship by demonstrating

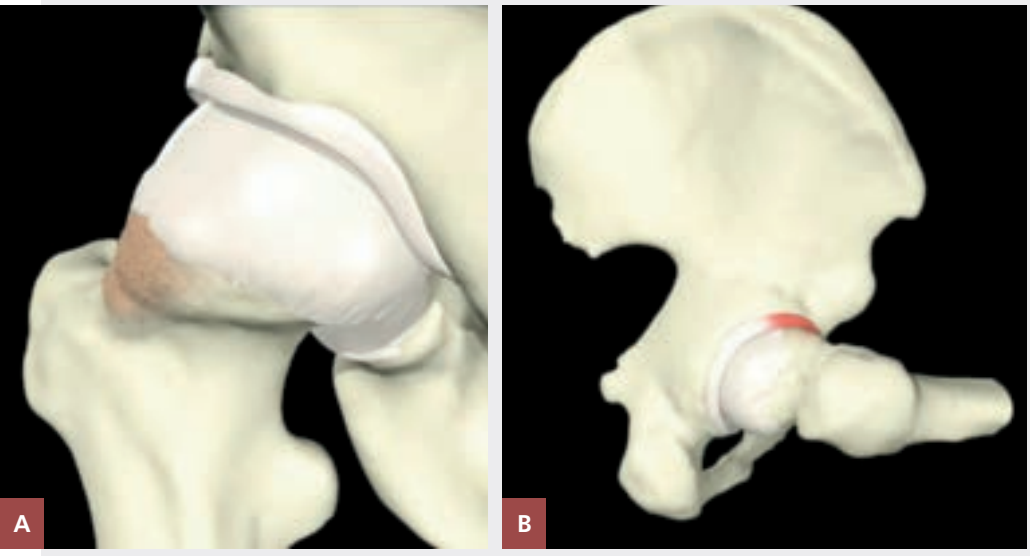

FIGURE 1.

Illustration of cam-type impingement in the hip joint. The convexity on the anterosuperior femoral head-neck junction contacts the anterosuperior acetabulum during normal hip motion. **A,** Cam lesion illustrated on anterosuperior femoral neck. **B,** Cam lesion contacting the acetabulum. (Anatomy images courtesy and copyright of Primal Pictures Ltd – www.primalpictures.com)

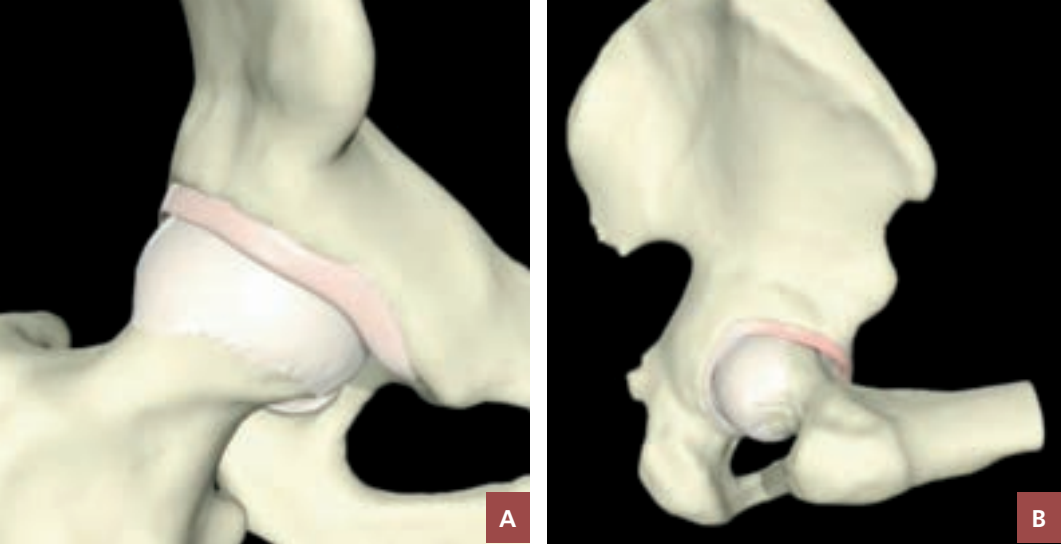

FIGURE 2.

Illustration of pincer-type impingement in the hip joint. The overcoverage of the acetabulum contacts the femoral neck resulting in impingement. **A,** Pincer overcoverage of the acetabulum on the femoral head. **B,** Impingement of the femoral neck on the pincer deformity. (Anatomy images courtesy and copyright of Primal Pictures Ltd – www.primalpictures.com)

a high rate of impingement findings in participants with no hip symptoms. Ganz et al[2,5,8] repeatedly proposed that structural abnormalities associated with cam and pincer impingement can lead to advanced arthrosis. Nonetheless, a direct relationship between FAI and end-stage hip degeneration has not been established and represents the crux of current hip research in the young patient.

The purpose of the current study was to compare the prevalence of femoroacetabular impingement findings between 2 populations of participants who had undergone total hip arthroplasty, 1 group under the age of 50 and the other group 50 years and over. More specifically, we measured and compared radiographic indices for FAI for each study population. We hypothesized that FAI that eventually leads to hip arthroplasty would be significantly more common in a participant population under the age of 50 years than in an older participant subset.

METHODS

We retrospectively identified patients who had undergone a total hip arthroplasty between January 2007 and June 2009 by using a facility repository database from multiple surgeons. We randomly selected 100 participants, 50 from each of 2 subgroups, and collected all preoperative radiographs. One subgroup consisted of participants who were less than 50 years of age (mean, 43 years; standard deviation, 5.6 years), while the other subgroup contained participants who were 50 years of age or over (mean, 68 years; standard deviation, 8.3 years).

We interviewed both subgroups with specific inclusion/exclusion criteria in mind. Exclusion criteria included the presence of osteonecrosis, developmental dysplasia of the hip, inflammatory arthritides, and posttraumatic arthritis. Inclusion criteria included adequate available radiographs with appropriate pelvic tilt and rotation. We assessed the rotation qualitatively, by observing the obturator foramen for symmetry and quantitatively by drawing a plumb line from the lumbar spinous processes through the pelvis. We considered a distance between the plumb line and the pubic symphysis of less than 16 mm to be adequate. We determined pelvic tilt by measuring the distance between the sacrococcygeal joint and the pubic symphysis. An acceptable range for males was between 8 and 50 mm and for females, between 15 and 72 mm.[9]

Two blinded independent observers analyzed the calibrated digital preoperative radiographs and performed measurements on the anterior-posterior (AP) pelvis and frog-leg lateral of the affected hip. They determined the following parameters[10,11] after they identified the center of the femoral head with the use of Mose circles[12]: angle on the AP and lateral, the neck-shaft angle, the Tönnis angle, the center-edge angle, Sharp's angle,[13] osteophytes, medial/lateral joint space, congruency, and herniation pits (Figures 3 and 4). They also assessed the radiographs for the presence of localized

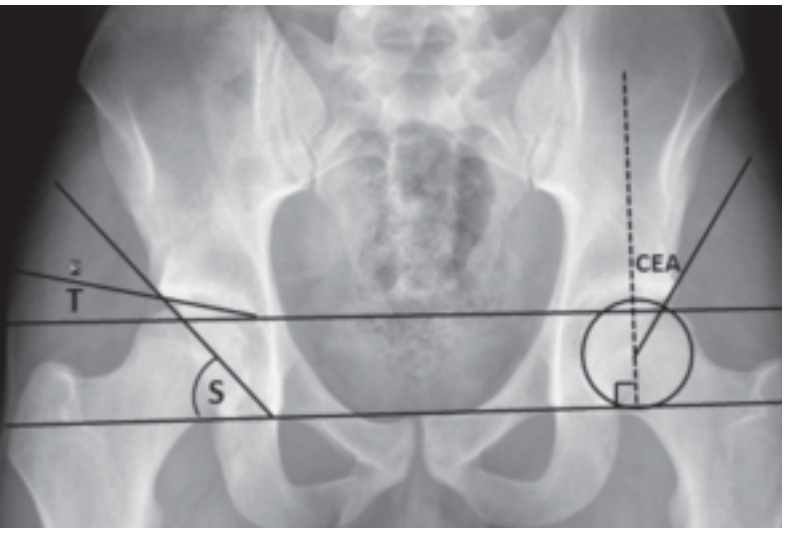

FIGURE 3.

Standing AP (anterior-posterior) pelvis radiograph. The center-edge angle (CEA) is calculated by first drawing a line connecting the inferior tips of the radiographic pelvic teardrops. Then a vertical line, perpendicular to the first line, is drawn through the center of the femoral head (via use of Mose circle). Next, a line is drawn through the center of the femoral head and the lateral acetabular edge. The CEA is the angle subtended by the vertical line and the latter. Sharp's angle (S) is calculated by first drawing a line connecting the inferior tips of the radiographic pelvic teardrops. Next, a line is drawn connecting the inferior tip of the teardrop to the lateral edge of the acetabulum. Sharp's angle is the angle subtended by these two lines. The Tönnis angle is calculated by first drawing a line parallel to the transverse line connecting the radiographic teardrops. Next, a line is drawn connecting the inferior and lateral margins of the sourcil. The Tönnis angle is the angle subtended by these two lines.

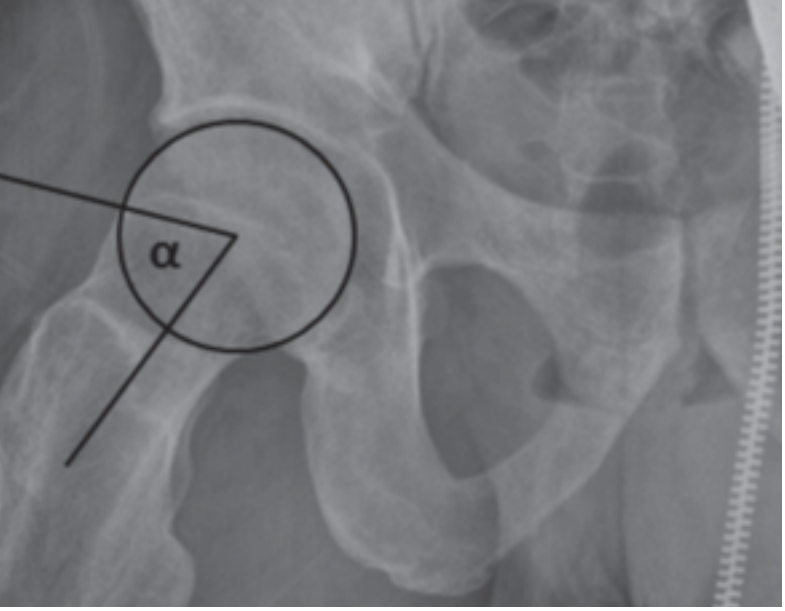

FIGURE 4.

Frog-leg lateral right hip radiograph. The α angle is calculated by first drawing a Mose circle best-fitting the femoral head. Next, a line is drawn connecting the center of the femoral head and the femoral neck. Then, a line is drawn from the center of the femoral head to the point at which the femoral head loses its sphericity. The α angle is the angle subtended by these two lines.

overcoverage, shown radiologically by the cross-over and posterior wall signs, or generalized overcoverage of the femoral head demonstrated in the femoral head extrusion ratio.

Intra- and interobserver repeatability were evaluated with K statistics for categorical variables and intraclass correlation coefficients (ICC) for continuous variables. Statistical analysis was completed using SPSS software (Chicago, Illinois). For categorical variables, such as the presence of cross-over and posterior wall signs, the intra- and interobserver agreement K statistics were ≥ 0.80. For continuous variables, the intra- and interobserver ICCs were ≥ 0.90. We used an independent *t* test to compare continuous variables, a chi-square analysis for discrete variables, and a *z*-ratio to analyze proportions.

RESULTS

Detailed radiographic analysis of the osteoarthritic hips demonstrated a high prevalence of structural abnormalities associated with FAI in the participant population under 50 years of age, as described in Table 1. In the participant group under 50, the average age was 43 years (± 5.66 years) with 78% male/22% female. This significantly contrasted with the 50-and-over group, which had an average age of 68 years (± 8.34 years) and was 36% male/64% female, ($P < .05$ and $P = .0002$, respectively).

There was no significant difference in lateral joint space, but the under-50 group had a significantly larger medial joint space (1.9 ± 2.5 mm vs 3.28 ± 1.8 mm, $P = .0022$). There was also a significant difference in the Tönnis and Sharp's

TABLE 1. Summary of Results

MEASUREMENT	≥ 50 YRS	< 50 YRS	SIGNIFICANCE (*P* VALUE)
Age	67.8 (± 8.34)	43.0 (± 5.66)	$P < .05$
Gender	36% male, 64% female	78% male, 22% female	$P < .0002$ (z-ratio)
Lateral Joint Space (mm)	1.2 (± 1.59)	1.4 (± 1.53)	$P = .50$
Medial Joint Space (mm)	1.9 (± 2.49)	3.28 (± 1.84)	$P = .0022$
Tönnis	2.2 (± 0.71)	2.3 (± 0.61)	$P = .98$ (chi-square)
Osteophytes	2.3 (± 0.58)	2.1 (± 0.61)	$P = .095$ (chi-square)
Cross-over Sign	18% yes	24% yes	$P = .46$ (*z*-ratio)
Ischial Spine on AP	20%	20% yes	$P = 1.00$
Ilioischial – fossa	54% medial, 28% touching, 18% lateral	74% medial, 20% touching, 6% lateral	$P = .073$ (chi-square)
Tönnis Angle	4.95 (± 4.78)	7.9 (± 3.45)	$P < .001$
Sharp's Angle	33.81 (± 5.23)	37.2 (± 4.11)	$P = .0084$
CEA	45.78 (± 12.00)	34.81 (± 7.69)	$P < .001$
Congruency	36% (non)	34% (non)	$P = .834$ (*z*-ratio)
Femoral Head Extrusion (mm)	0.11 (± 0.11)	0.18 (± 0.10)	$P < .001$
AP Head-neck Offset (mm)	46.9 (± 6.65)	47.7 (± 5.66)	$P = .10$
COR-Troch (varus, valgus)	36% valgus	52% valgus	$P = .107$ (*z*-ratio)
Alpha Angle	46.2 (± 8.90)	62.3 (± 8.95)	$P < .001$
NSA	130.3 (± 5.97)	134.3 (± 4.22)	$P = .517$
Herniation Pits	20% yes	10% yes	$P = .162$ (*z*-ratio)
Frog-leg Lateral			
Lateral Alpha Angle	47.8 (± 12.78)	59.8 (± 13.88)	$P < .001$
Lateral Head-neck offset (mm)	17.2 (± 4.08)	13.1 (± 3.62)	$P < .001$

Abbreviations: AP, anterior-posterior; CEA, center-edge angle; COR, center of rotation; NSA, neck-shaft angle.

angles, with the under-50 group having significantly increased values ($P < .001$ and $P = .0084$, respectively). These findings also correspond with a substantial decrease in the center edge angle for arthroplasty participants under 50 (45.78 ± 12.00° vs 34.81 ± 7.69°, $P < .001$). The femoral head extrusion index was also increased in these young participants (0.11 ± 0.11 vs 0.18 ± 0.10, $P < .001$). As predicted, there was a significantly higher alpha angle on both the AP and lateral radiographic views in the participants under 50 years of age (46.2 ± 8.90° vs 62.3 ± 8.95°; 47.8 ± 12.78° vs 59.8 ± 13.88°, respectively). Interestingly, there was no difference in the head-neck offset on the AP view, but there was a significant decrease in offset on the frog-leg lateral view in participants under 50 (17.2 ± 4.08 mm vs 13.1 ± 3.62 mm, $P < .001$).

There were no significant differences in the presence of osteophytes, ischial spine on the AP radiograph, position of the ilioischial line, or a cross-over sign. In the 50-and-over group, 18% of participants had a positive cross-over sign, and 24% of the under-50 group had the same findings. There were also no significant differences in the femoral head congruency, varus/valgus position, neck shaft angle, or presence of herniation pits.

DISCUSSION

The natural history of impingement and associated pathology is poorly understood. A variety of theories have been proposed but with limited scientific evidence. However, the concept that FAI can lead to premature degeneration of the hip has been supported by many authors.[2-5,14,15] In fact, this is not a new concept. In 1965 Murray[16] described a "tilt deformity" of the femoral head with the formation of excess bone and buttressing of the femoral neck. He linked this phenomenon to premature osteoarthritis in the lateral third of the joint. Early descriptions of the "pistol-grip" deformity in the hip also contributed to our initial understanding of how hip morphology contributes to early degeneration. Ganz and Harris were some of the first researchers to revisit this concept and have eloquently summarized the relationship of impingement to early hip degeneration in multiple publications.[2,17] The theory that FAI can lead to early arthritis of the hip has been proposed by multiple authors but not directly established in the contemporary literature. Furthermore, it remains to be seen if this early degeneration leads to end-stage destruction that requires arthroplasty. The current study indirectly supports the relationship between FAI and total hip replacement through the evaluation of radiographic abnormalities found in an arthroplasty population under 50 years of age as compared with participants of 50 years of age and over.

Clohisy et al[4] has recently reported on the results of radiographic evaluation of 710 participants who received a hip arthroplasty before the age of 50. Overall, 17% of the participants were diagnosed with osteoarthritis of "unknown etiology" and received a comprehensive radiographic evaluation. Of these, 98% were diagnosed with cam, pincer, or combined pathologies. These participants were also more likely to be male. In comparison with an asymptomatic participant population, Clohisy et al found that there was a significant decrease in the head-neck offset ratio and femoral head sphericity in the affected hips. Furthermore, female participants had an increased acetabular inclination, and participants with pincer lesions had an increased lateral center edge angle. Interestingly, all members of a subgroup of 70 participants with contralateral imaging also had bilateral structural abnormalities, and 37% of these participants underwent contralateral total hip arthroplasty at an average of 5.4 years, and 36% of them had an increase in Tönnis grade at an average of 8.4 years. This was a large study with great insight into the concomitant pathologies of participants who require total hip arthroplasty at an early age. An asymptomatic comparison group provided a good reference frame; however, this study did not provide an older cohort that also underwent total hip replacement to determine the increased, decreased, or similar prevalence of structural abnormalities.

The relationship between radiographic findings and articular damage is also supported by Nepple et al[18]. The authors studied 355 arthroscopic hip procedures with 67% of the participants having some degree of acetabular chondromalacia. More specifically, male sex, age, and an alpha angle >50° on the frog-leg lateral were independent risk factors for more advanced articular damage, with an odds ratio greater than 3.0. Pincer-type impingement was not associated with an increased rate of degeneration. Johnston et al[19] also described a correlation between radiographic findings and arthroscopic acetabular degeneration. In their study, a higher offset alpha angle was associated with acetabular rim chondral defects and full thickness delamination of the acetabulum.

To further define the degeneration associated with FAI, Beck et al[15] reviewed the results of 244 hips that were treated with an open dislocation. They identified 26 hips that had isolated cam impingement and 16 with only a pincer lesion. The authors found that the hips with cam impingement had focal damage to the articular cartilage in the anterosuperior region of the acetabulum with separation of the cartilage and the labrum, whereas the hips with pincer lesions had a circumferential thin zone of injury to the articular surface. Anderson et al[14] also found a close association with cam-type impingement and acetabular delamination. In a retrospective review of 64 surgical dislocations for impingement, they found 44% of participants with acetabular cartilage delamination. Interestingly, male sex and cam lesions were strongly associated with the delamination, but acetabular overcoverage was not.

However, results of the referenced studies must also be regarded in the context of an intriguing study completed by Laborie et al.[7] In a prospective population-based radiographic analysis of asymptomatic hips, they found a 35% rate of radiographic cam impingement in males with an accompanying 34% rate of pincer lesions. However, females had a 10% rate of cam lesions and a 17% rate of pincer findings. Thus, the question remains as to whether the 35% of asymptomatic participants with impingement lesions in the study by Laborie et al will develop symptoms, or if the impingement signs in 33% of arthroplasty participants in the study by Clohisy et al are merely incidental findings.

The current study does provide some insight into the complex interactions between femoroacetabular impingement and early end-stage osteoarthritic changes. Similar to other studies, there was a much higher percentage of male participants in the arthroplasty group under 50 years of age. These young participants also showed signs of decreased lateral joint space with relatively maintained medial joint space. This would support the theory that impingement progresses from a lateral to medial direction due to the forces applied on the acetabular cartilage from

the impingement lesion. Furthermore, the participants under the age of 50 years also had a significantly increased alpha angle on both the anteroposterior view and the frog-leg lateral. However, there was also a significant increase in the Tönnis and Sharp angles with a significant decrease in the center-edge angle. These findings support the conclusions of Beck et al[15] and Anderson et al[14] that cam lesions are more detrimental to the acetabular articular cartilage than pincer deformities. In the current study, impingement lesions were analyzed based on a continuous distribution as opposed to the presence or absence of a deformity. The authors believe that there may be a spectrum of deformities and that reporting deformities as either "present" or "absent" overlooks the influence of magnitude on the degeneration of the hip.

There are limitations to the current study. We selected the 100 participants randomly from a larger pool for radiographic analysis. The possibility of sampling error does exist, but a power analysis revealed that 50 participants in each group would be sufficient for comparison, and it was assumed that the 50 participants would be sampled from a normally distributed population. There is also the possibility of a large degree of variability in many of the measurements taken. However, the interobserver reliability was acceptable, and the statistical testing would have taken into account any variability of results. There is also a significant difference in the number of males and females between the 2 groups. This is to be expected with a random selection from a larger pool of participants. It is possible that younger males have a higher predilection for FAI than females.[1] However, it is also important to note that the 2 groups are very similar with respect to the Tönnis scale. Lastly, this is a retrospective review, and we cannot establish a direct causal relationship based on radiographic findings.

It is commonly believed that impingement can lead to end-stage degeneration through mechanical wear and abutment due to morphologic problems with the hip joint. However, these theories have not been robustly defended in the contemporary orthopedic literature. The current study adds to our understanding of impingement-induced hip degeneration with the comparison of arthroplasty participants in treatment-matched cohorts below 50 years and those 50 years and over. The young participants did have more significant cam findings, decreased acetabular coverage, and maintenance of medial joint space. These findings would seem to support a degenerative mechanism that is more reliant on the deformity of the proximal femur and that progresses from lateral (or anterosuperior) to medial on the anteroposterior imaging.

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References and financial disclosures are available online at www.rush.edu/orthopedicsjournal.

"THIS STUDY'S RESULTS SUGGEST THAT AN FCU MUSCLE FLAP CAN BE USED TO EFFECTIVELY COVER SOFT-TISSUE DEFECTS OF THE POSTERIOR ELBOW WITH LOW MORBIDITY TO THE WRIST IN TERMS OF PAIN OR DISABILITY."

Posterior Elbow Soft-Tissue Reconstruction Using a Flexor Carpi Ulnaris Muscle Turnover Flap

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Management of soft-tissue defects at the elbow remains a challenge. The posterior aspect of the elbow is particularly predisposed to skin coverage difficulty due to tension placed on the skin during elbow flexion and the paucity of subcutaneous adipose tissue and muscle in this region. Complex wounds, particularly those involving exposed bone, tendon, or orthopedic implants, along the posterior aspect of the elbow usually require local or distant flap coverage. Traditional methods of posterior elbow soft-tissue reconstruction involve the use of rotational flaps, fasciocutaneous flaps, regional flaps, or free tissue transfer.[1,2] The most commonly used rotational flaps include anconeus,[3,4] brachioradialis,[5] and extensor carpi radialis muscles.[6] However, these flaps are associated with either potential donor site morbidity or limited coverage.[7]

The flexor carpi ulnaris (FCU) flap has been described anatomically as a reliable option for soft-tissue reconstruction of the posterior elbow.[8-10] The FCU consists of two heads: the humeral head, which originates from the medial epicondyle, and the ulnar head, which originates from the posterior border of the ulna and medial border of the olecranon. Initially, the FCU flap was suggested for exceptional cases such as osteomyelitis within a large soft-tissue defect.[8] Linjaraj and colleagues proposed splitting of the FCU and use of the larger ulnar compartment while maintaining the humeral compartment to preserve wrist flexion and deviation.[11]

The FCU flap is remarkable due to its ease of elevation, adequate surface area to cover moderate-sized defects, the unique bipennate nature of the muscle, minimal donor morbidity, and consistent vascular supply.[12-14] Our recent cadaveric study suggests that the FCU flap can provide an excellent solution to posterior-elbow soft-tissue reconstruction, especially in the posteromedial area of the elbow.[12] In this study, we report the outcomes for patients who have undergone FCU flap for the management of posterior-elbow soft-tissue defects associated with a spectrum of conditions.

METHODS

We obtained institutional review board (IRB) approval and informed consent for this study. A total of 7 patients who developed a soft-tissue defect on the posterior aspect of the elbow (Figure 1) were treated with an FCU flap between the years of 2003 and 2011. Of the 7 patients, 5 were female and 2 were male. The average age was 57.8 years (range, 25-85 years). See Table 1 for patient demographics. In 6 patients, the soft-tissue defect was the result of poor wound healing or chronic infection after previous surgical intervention. Index surgical procedures included fracture reduction and internal fixation or total elbow arthroplasty. In 1 patient the soft-tissue defect was the result of same-procedure soft-tissue removal for tumor resection (Table 2).

Two surgeons performed all of the FCU operations at a single center. The following summarizes our procedure: after administering a long-acting regional block or general anesthesia, we made a longitudinal incision along the line between pisiform and medial epicondyle of the elbow. Then we dissected the skin and subcutaneous tissues, identifying and protecting branches of medial antebrachial cutaneous nerve. The surgeon then identified the FCU tendon and sharply incised it transversely, 2 cm from its insertion on the pisiform. The surgeon then elevated the FCU muscle sharply from distal to proximal. We took care to either

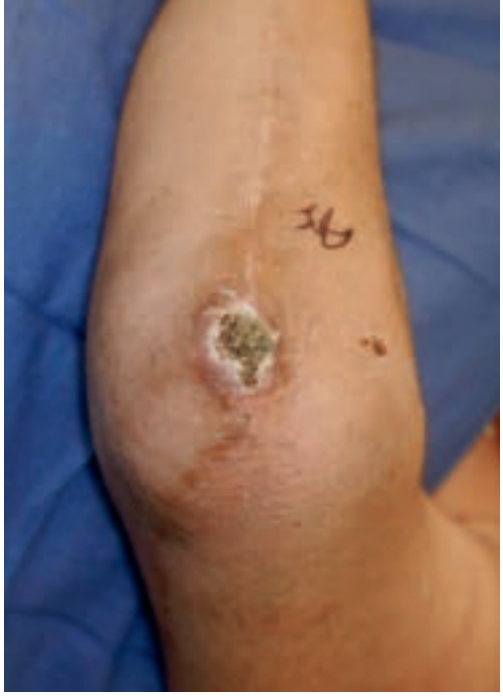

FIGURE 1. A 49-year-old patient with a wound dehiscence after several operations for a Monteggia fracture/dislocation. Below the pictured scab is full-thickness tissue loss with exposed bone.

PATIENTS (NUMBER)	EXTREMITIES (NUMBER)	SEX	AGE	FOLLOW-UP
7	7	5 female/2 male	avg 57.8 y (range, 25-85 y)	Follow-up: avg 50.4 mo (range, 17-108 mo)

TABLE 1. Patient Demographics

PATIENT	CAUSES	SIZE OF DEFECT
1	Chronic infection after TEA for distal humerus fx	Data not available
2	Sarcoma removal	10.0 cm × 8.0 cm
3	Chronic infection after olecranon ORIF	1.5 cm × 1.5 cm
4	Wound dehiscence after ORIF of proximal radius/ulna fx	3 cm × 3 cm
5	Wound dehiscence after ORIF of olecranon fx	5 cm × 1 cm
6	Wound dehiscence after ORIF of olecranon ulna fx	0.8 cm × 1.2 cm
7	Wound dehiscence after ORIF of distal humerus fx	Not noted in chart

Abbreviations: TEA, total elbow arthroplasty; ORIF, open reduction and internal fixation.

TABLE 2. Patient Defect Descriptions

(1) identify and protect the entry point of the dominant pedicle on the muscle's undersurface or (2) more commonly terminate the proximal dissection distal to the known location of the dominant pedicle (located 5 cm distal to the olecranon tip) when sufficient length was achieved to rotate the flap proximally and cover the defect.[12] Next the surgeon performed longitudinal sectioning of the FCU fascia to gain additional width if needed. The surgeon then folded the muscle upon itself and placed it directly over the olecranon process without tension (Figure 2). Then the surgeon sutured the muscle to the adjacent soft tissue under light tension with interrupted sutures 1 cm apart on both sides of the muscle. After suturing, the surgeon closed the skin primarily where possible under minimal tension with the elbow flexed (Figure 3) and covered the remaining exposed muscle flap with a split thickness skin graft (Figure 4). After surgery, the surgeon applied a long arm splint. At 1 to 2 weeks postoperative, the patient began range-of-motion exercises of the elbow after the splint is discontinued.

We evaluated all patients by follow-up appointment. Mean follow up was 20 months (range, 7-39 months). At the time of final follow-up, 4 patients were available for physical exam, and 1 patient was available for phone interview. One patient was deceased, and 1 patient was lost to follow up. We measured elbow and forearm range of motion of both the affected and unaffected extremities, using a standard goniometer. We evaluated wound healing; grip strength; isokinetic dynamometry; visual analogue scores (VAS); disabilities of the arm, shoulder, and hand (DASH) score; and Mayo Elbow Performance Scores (MEPS), including pain, motion, stability, and function. The DASH and MEPS questionnaires are upper-extremity specific outcome measures that have been used to detect and differentiate small and large changes of disability after surgery in patients with upper-extremity musculoskeletal disorders.[15]

At the final follow-up examination, we evaluated the patients for isokinetic

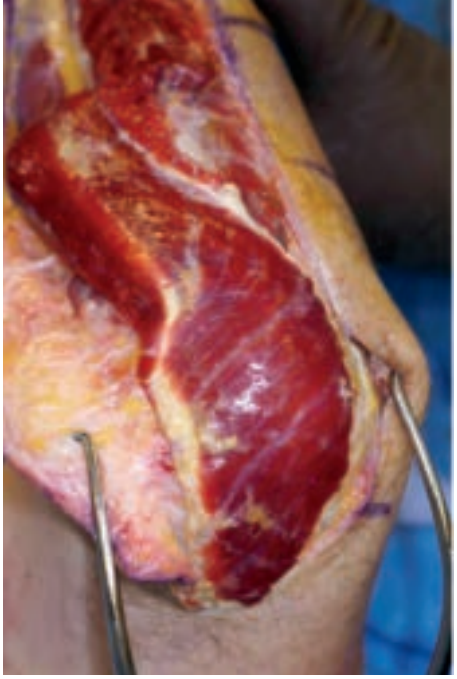

FIGURE 2. All diseased tissue has been excised, and the flexor carpi ulnaris (FCU) is mobilized and folded over itself to cover the defect over the olecranon.

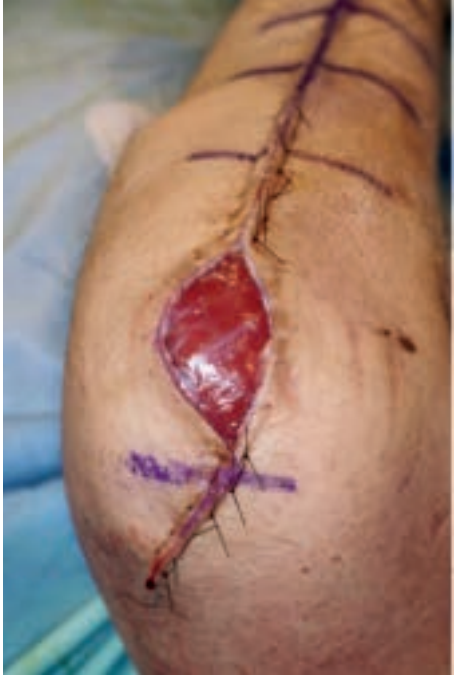

FIGURE 3. The proximal and distal skin is closed under minimal tension, leaving the remaining exposed FCU for split-thickness skin grafting. Closure over mobilized FCU flap, showing distal skin closure and exposed FCU muscle covering olecranon defect.

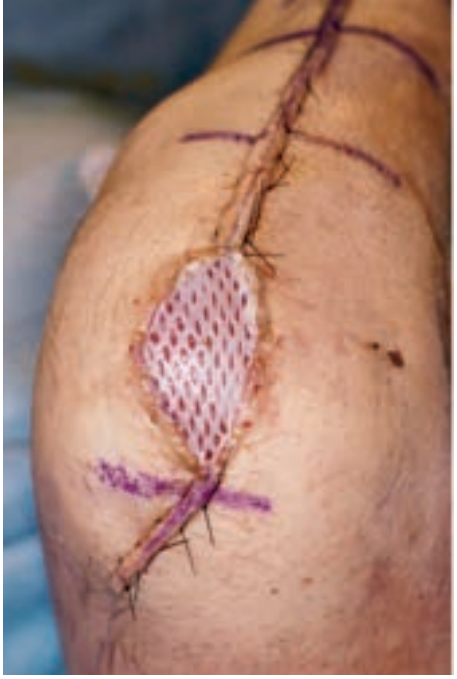

FIGURE 4. A full-thickness skin graft has been applied to the FCU flap.

strength in wrist flexion and extension of the affected and unaffected arm, using the Biodex II dynamometer (Biodex Medical Systems; Shirley, New York). We instructed patients to avoid workouts or activities that would stress the wrist for 48 hours prior to the evaluation. The patients sat upright with the elbow stabilized for forearm flexion and extension. We aligned the machine's axis of rotation with that of the elbow joint and the forearm, respectively. Because there was no clear consensus regarding the ideal testing parameters for this population, we selected a protocol based on the parameters most commonly used in the literature. Prior to each velocity and position change, we permitted 5 low-intensity repetitions to allow the patient to become familiar with the angular velocity. We instructed patients to use maximal exertion and speed on each repetition of testing. The protocol consisted of 1 set of 3 repetitions at an angular velocity of 30° per second, from which the value for the single highest peak torque was recorded, and a second set of 15 repetitions at an angular velocity of 60° per second, from which we calculated a fatigue percent to estimate endurance. The fatigue percent is the percentage of decline in work done during the last third (in this protocol, 5 repetitions) as compared to the first third of the set. The result is calculated by the formula $100 \times [1 - (\text{work done during last third} \div \text{work done during first third})]$. Positive values reflect a decrease in work done over time, whereas negative values reflect an increase in work. The patient always performed the peak torque set for each exercise prior to the fatigue set. We gave subjects 45 seconds rest after each set.

RESULTS

Of the 7 cases, all wounds healed after the FCU flap surgery with no reoperations. Outcome measures, performed at the patients' last follow-up (range, 17-108 mos) after surgery, showed an average VAS of 2.3 in the affected wrist, an average DASH score of 35, and an average MEP score of 80. Average elbow range of motion and forearm rotation included extension: lacking 11°; flexion: full (< 140°); pronation: 70°; supination: 73°.

Biomechanical testing of wrist muscle strength showed that on average the patients' operative arm had 97% of the grip strength of the nonoperative arm. Using a Biodex II dynamometer, we found the average peak torque produced by wrist flexion of the operative arm to be 87% of the nonoperative arm. The fatigue percentage for wrist flexion was 29% for the nonoperative arm and 7% for the operative arm. The results are summarized in Table 3.

DISCUSSION

Soft-tissue defects of the posterior elbow are common and challenging to treat. Trauma, infection, wound dehiscence, burns, radiation, decubitus ulceration, chronic inflammation, and bursa excision are typical causes. The FCU flap is an attractive local pedicle flap, given its ease of elevation and adequate surface area to cover small- to moderate-sized defects. In this study population, the FCU flap was able to cover wounds of up to 10 cm × 8 cm with 100% flap survival.

However, as with any flap procedure, there are some concerns over donor-site

PARAMETER	VAS	DASH	MEPS	ELBOW FLEXION	ELBOW EXTENSION	PRONATION	SUPINATION	GRIP STRENGTH	PEAK TORQUE IN WRIST FLEXION	FATIGUE %, NONOPERATIVE ARM	FATIGUE %, OPERATIVE ARM
Average	2.3	35	80	Full	Lacking 11°	70°	73°	97%	87%	22%	7%

Abbreviations: VAS, visual analog scale; DASH, Disabilities of the Arm, Shoulder, and Hand; MEPS, Mayo Elbow Performance.

TABLE 3. Summary of Results

morbidity with the flap harvest, which in the case of the FCU is the loss of wrist flexion and ulnar deviation strength, which are known to make an important contribution to power grip. In this study, biomechanical testing of grip strength, peak torque in flexion, and fatigue-index flexion demonstrated minimal effect when compared to the control side at the latest follow-up examination. Interestingly, the amount of wrist flexion fatigue was actually less on the operative wrist than the nonoperative wrist.

Follow-up on patients who underwent the FCU flap procedure shows a minimal amount of daily discomfort and good to excellent outcome scores in regards to function. In addition, the patients retained good range of motion at the elbow and wrist, which allowed them to undertake almost all activities of daily living. The major limitation to this study's outcomes is the very small patient population, which can be attributed to infrequency of the procedure. Further studies are certainly needed to confirm and reproduce these findings. However, with the limited data currently available on the topic, this study demonstrates encouraging results for the outcomes of the FCU rotational flap for coverage of soft-tissue defects of the posterior elbow.

CONCLUSION

In this small study population, patients undergoing an FCU flap were found to have little pain as measured by the VAS, good functional outcomes as measured by the DASH and MEP, and largely preserved grip strength and wrist flexion strength on biomechanical testing. This study's results suggest that an FCU muscle flap can be used to effectively cover soft-tissue defects of the posterior elbow with low morbidity to the wrist in terms of pain or disability. ■

References and financial disclosures are available online at www.rush.edu/orthopedicsjournal.

"THE RESULTS FROM THIS STUDY MAY BE USED TO ASSIST WITH PATIENT SELECTION FOR SLAP SURGERY AND CAN HELP PREDICT WHICH PATIENTS MIGHT BENEFIT FROM SLAP REPAIR..."

Retrospective Analysis of Prognostic Factors Associated with Successful Arthroscopic Superior Labrum Anterior to Posterior (SLAP) Repair

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Superior labrum anterior to posterior (SLAP) tears occur more frequently in overhead athletes, manual workers, and military personnel.[1-10] The orthopedic literature thoroughly discusses the classification, mechanisms of injury, and surgical treatment of these patients. However, significant controversy exists regarding diagnosis and operative indications. Clinical outcomes following SLAP repair have been reported as good to excellent in 63% to 100% of patients, and thus up to approximately one-third of patients are still dissatisfied after SLAP repair.[11-18] Factors that prognosticate poor outcomes after SLAP repair have not been fully delineated. This study evaluates potential prognostic factors that may have a significant effect on clinical outcomes following arthroscopic repair of type II SLAP tears.

MATERIALS AND METHODS

Our team retrospectively identified patients undergoing arthroscopic repair of type II SLAP tears (labral fraying with detached biceps tendon anchor) at a single institution. All patients had a minimum of 2-year follow-up. We did not exclude patients for undergoing concomitant procedures, including rotator cuff repair, biceps tenodesis, subacromial decompression, or acromioplasty. Our institutional review board (IRB) approved the study proposal, and we obtained informed consent for all patients prior to data collection.

We invited patients meeting study criteria to return for follow-up examination and questionnaires. Clinical evaluation included the simple shoulder test (SST), American Shoulder and Elbow Surgeons (ASES) score, single assessment numeric evaluation (SANE) score, visual analog scale (VAS), and the Short-Form 12 health status survey (SF-12) (Table 1). A single orthopedic research fellow performed a bilateral physical examination, independent of the operating surgeon. The fellow obtained goniometric measurements for active and passive range of motion. Specific provocative examination included the O'Brien's test,[19] Kibler test,[20] Speed's test,[21] Yergason's test,[22] compression-rotation test,[2] apprehension test,[23] and relocation test.[24] The fellow also tested strength measurements of forward elevation and external rotation at the side using a JTech Commander Muscle Tester dynamometer (JTech Medical; Salt Lake City, Utah).

We collected intraoperative data including labral pathology (location and size), number and type of anchors used, concomitant procedures, chondral lesions (location, size, and depth), and biceps pathology (none, incomplete, or complete tear).

Surgical technique

Patients underwent repair with general anesthesia and an interscalene block in the beach chair or lateral decubitus position. We performed diagnostic arthroscopy from a standard posterior viewing portal using a 30° arthroscope. After needle-localizing a high rotator interval portal, we placed an 8.25-mm cannula for instrumentation. In this case, after the type II SLAP tear was confirmed, we used a hooded arthroscopic burr to debride the superior glenoid to bleeding cancellous bone. We then introduced BioComposite 3.0-mm suture anchors (BioComposite SutureTak; Arthrex, Inc., Naples, Florida) through incisions at the lateral acromion edge, aiming toward the superior glenoid tubercle (12 o'clock). We shuttled the sutures through the labrum using a 45° curved suture shuttle device (Spectrum; Linvatec, Key Largo, Florida) loaded with a no. 1 polydioxanone (PDS) suture through the anterior portal. Arthroscopic knot tying was performed using 5 reverse half-hitches with alternating posts, with the post limb being further from the articular surface. In the same manner, a second suture anchor may be placed at the 10 o'clock position (Figure 1).

FACTOR	POSSIBLE RESPONSES
Age	Years
Tobacco History	*Yes* or *no*
Preoperative Pain	*Yes* or *no*
Anti-inflammatory Use	*Yes* or *no*
Narcotic Use	*Yes* or *no*
Extremity	*Right* or *left*
Dominant Extremity	*Yes* or *no*
Trauma	*Yes* or *no*
Mechanism or Injury	*Sports, motor vehicle accident, fall, traction, insidious*
Sport	
Level of Sports Participation	*Professional, collegiate, high school, recreational, none*
Thrower	*Yes* or *no*
Overhead Athlete	*Yes* or *no*
Collision Sport	*Yes* or *no*
Level of Work	*Very heavy, heavy, medium, light, sedentary*
Worker's Compensation	*Yes* or *no*
History of Dislocation	*Yes* or *no*
History of Subluxation	*Yes* or *no*
Preoperative O'Brien Test	*Positive, negative, equivocal*
Preoperative Biceps Load II Test	*Positive* or *negative*
Preoperative Compression-Rotation	*Positive* or *negative*
Preoperative Kibler Test	*Positive* or *negative*
Preoperative Bicipital Groove Tenderness	*Yes* or *no*
Preoperative Speed's Test	*Positive* or *negative*
Preoperative Yergason's Test	*Positive* or *negative*
Preoperative Apprehension Test	*Positive* or *negative*
Preoperative Relocation Test	*Positive* or *negative*

TABLE 1. Prognostic Factors: Data Collected

Postoperative rehabilitation protocol

All patients followed the same standardized rehabilitation protocol postoperatively. For the first 6 weeks the shoulder was immobilized, with passive- and active-assisted range of motion permitted; including motion up to 40° of external rotation and 140° of forward flexion. From 6 to 12 weeks, the patient was advanced to active range of motion. The final 12 weeks focused on rotator cuff strengthening and conditioning. All patients were released to full activity after 6 months.

Definition of surgical failure

Patients with any of the following were considered failures: revision surgery on the ipsilateral shoulder related to the capsule and/or labrum, ASES score less than 50,[25] complications (stiffness, instability), and/or poor patient satisfaction.

Statistical analysis

We performed statistical analysis using SPSS software (SPSS; Chicago, Illinois). Descriptive analysis consisted of frequencies and percentages for discrete data and means and standard deviations for continuous data. We used paired *t* tests to compare preoperative to postoperative measures at final follow-up. To identify correlations between potential risk factors and outcome measures, we performed a contingency table analysis using Fisher's exact test. Significance was set at $P < .05$ for all tests.

RESULTS

A total of 100 patients met the original inclusion criteria, and 62 of the patients were available for follow-up. The average age was 36 years (range, 15.2-64.3 years) with average follow-up duration at 3.3 years (range, 2.0-5.0 year). Of the 100 patients, 46 (74%) were male, and 16 (26%) were female. Eleven patients reported either active or prior tobacco history. In addition to SLAP repair, several patients also underwent concomitant Bankart repair (n = 9), rotator cuff tear repair (n = 10), acromioplasty (n = 8), distal clavicle resection (n = 2), and biceps tenodesis (n = 9).

There were statistically significant improvements in the following average scores: ASES (preop, 64.8 ± 19; postop, 83.9 ± 18.3;

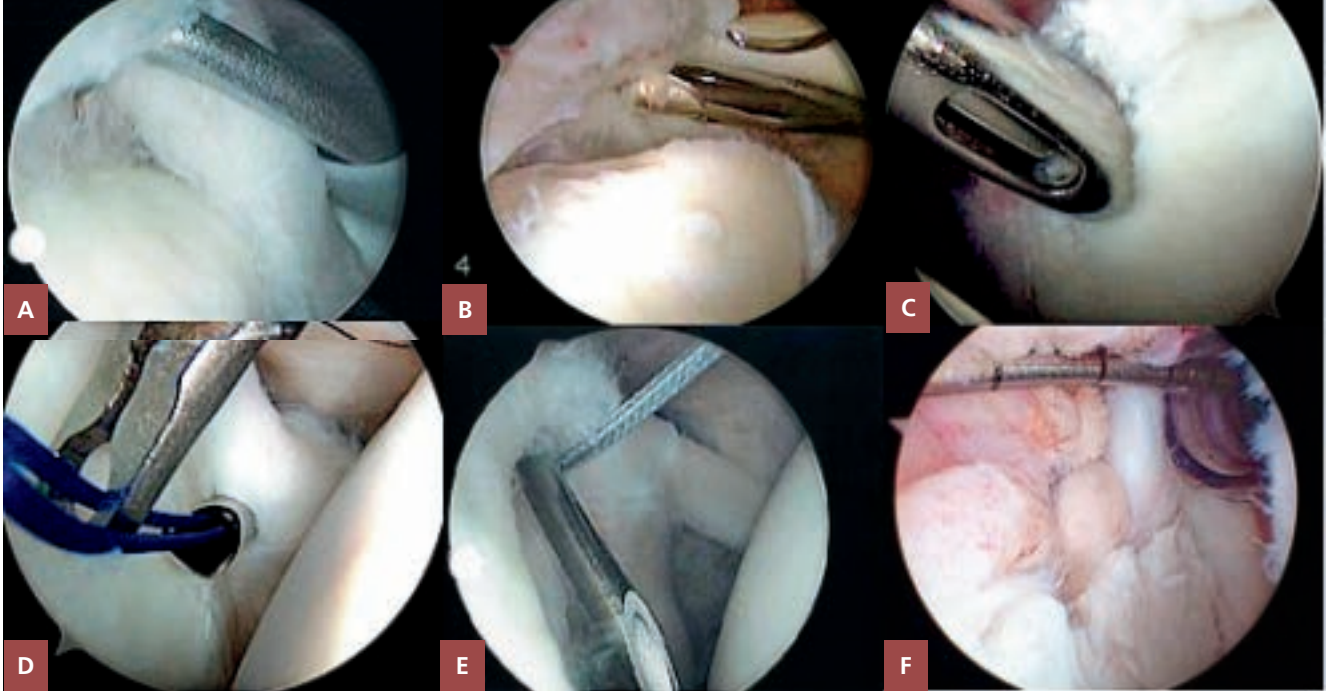

FIGURE 1. Arthroscopic figures demonstrating surgical technique of superior labrum anterior to posterior (SLAP) repair. **A,** Identification of SLAP lesion. **B–C,** a hooded arthroscopic burr is used to debride the superior glenoid to bleeding cancellous bone to facilitate labral healing. **D,** Passage of no. 1 PDS suture posterior to the biceps tendon and underneath the labrum is shown. **E,** Passage of heavy suture for SLAP repair. **F,** Final SLAP repair is seen.

$P < .001$), SST (preop, 8.6 ± 2.9; postop, 10.3 ± 2.3, $P = .004$), and VAS (preop, 3.3 ± 2.3; postop, 1.6 ± 1.9, $P < .001$). The mean postoperative SANE score, indicating the patient's overall assessment of shoulder function, was 86.9 ± 16.4.

There was a statistically significant improvement in average forward flexion (preop, 156° ± 34°; postop, 172° ± 14°, $P = .005$). However, external rotation (preop, 66° ± 19°; postop, 70° ± 12°, $P > .05$) and abduction (preop, 155° ± 34°; postop, 169° ± 66°, $P > .05$) improvements were not significant.

There were 5 total patients (8.1%) with a postoperative ASES score less than 50. In addition, a total of 5 other patients (8.1%) went on to receive revision shoulder surgery. Revision surgery included capsular release (softball injury), 270° repair to SLAP after traumatic retear of labrum (baseball injury), revision SLAP repair (baseball injury), revision SLAP repair (basketball dunking injury), and debridement to SLAP repair (wrestling injury). Of the patients under 20 years old, 2 out of 3 patients were revised, as compared to 3 out of 35 patients 20 years of age and older. Of the patients who were throwers, 4 out of 10 were revised, as compared to 0 out of 29 nonthrowing patients. We identified a significant correlation between patients requiring revision surgery and age less than 20 years ($P = .035$) as well as preoperative participation in throwing activities ($P < .001$). We classified patients as overhead throwers if they use their arms in an overhead position, including, but not limited to baseball players (especially pitchers), football players, swimmers, and tennis players.

Scoring below 50 on the ASES score was associated with age greater than 40 years ($P = .005$), alcohol use ($P = .033$), tobacco use ($P = .002$), and diabetes ($P < .001$). With ASES scores less than 50, we also observed associations between physical examination components, including pain in the bicipital groove on examination ($P < .001$), positive O'Brien's test ($P = .002$), positive Speed's test ($P < .001$), and positive Yergason's test ($P = .015$). Finally, there was a significant correlation between ASES less than 50 and high levels of lifting required at work ($P = .004$).

DISCUSSION

Although the technical aspects of arthroscopic repair of type II SLAP tears have been well described, the clinical decision making may not be as apparent. There may be a certain subset of patients who have suboptimal clinical outcomes after surgical fixation unstable SLAP lesions. The present study suggests that arthroscopic repair of type II SLAP tears results in a significant improvement in shoulder functional outcome and range of motion; however, there are a number of prognostic factors that may have a higher association with clinical failure. The principle findings of this study include (1) when using revision surgery as an indicator of failure, the prognostic factors most associated with failure were overhead throwers and age less than 20 years; and (2) when using ASES score less than 50 as an indicator of failure, the prognostic factors most associated with failure were age greater than 40 years, heavy labor as an occupation, tobacco and/or alcohol use, diabetes, and/or patients who present with persistent anterior shoulder pain (symptoms consistent with persistent SLAP lesion or bicipital groove tenderness).

Using a poor ASES score (< 50) as a reflection of overall poor shoulder function, the results from the present study suggest that patients more likely to fail SLAP repair are older than 40 years old, heavy laborers, users of tobacco and/or alcohol, diabetics, and/or patients who present persistent SLAP or bicipital groove pain (tenderness over the long head of the biceps tendon, positive O'Brien's test, positive Speed's test, and/or positive Yergason's test). These are the type of patients that one might expect

AUTHORS (REFERENCE)	NUMBER OF PATIENTS	CLINICAL OUTCOME MEASURES	OUTCOMES	POTENTIAL FACTORS
Katz et al, 2009	40 shoulders (39 pts)	SST, pt satisfaction	71% of those with poor outcome dissatisfied with conservative treatment	Not discussed
Brockmeier et al, 2009	47	ASES, L'Insalata	87% good to excellent	Higher outcomes after traumatic etiology
Boileau et al, 2009	10 (15 others with BT)	Constant, pt satisfaction	• Constant score: 65 → 83 • 60% dissatisfied • 4 overall failures converted to BT	Not discussed
Yung et al, 2008	16	UCLA, physical exam	31% excellent, 44% good, 25% poor	Overhead athletes required longer time to RTP
Park et al, 2008	24	UCLA, VAS	• UCLA: 22.7 → 29.9 • VAS: 6.4 → 2.1	Mechanism of injury did not impact outcomes
Oh et al, 2008	25 (58 total in study, only 25 with isolated SLAP lesions)	ASES, L'Insalata	Significant improvements: • VAS pain: 1.8 • ASES: 84.1 • UCLA 32.6 • SST: 94.7 • VAS: 8.9	Not discussed
Voos et al, 2007	30 (combined RCT with SLAP or Bankart)	Modified Rowe	• 90% good to excellent • 77% return to play • 2 recurrent RCT	Not discussed
Funk et al, 2007	18	UCLA	89% satisfaction	Isolated SLAP lesions had quickest RTP
Enad et al, 2007	27 (15 with isolated tears), military population	ASES, L'Insalata	• Excellent in 4, good in 20, fair in 3 • 96% return to duty	Higher outcomes scores in pts with concomitant diagnosis
Coleman et al, 2007	50 (16 with concomitant acromioplasty)	VAS, ASES, UCLA, SST, Constant	• 65% good to excellent in SLAP only group • 81% good to excellent in acromioplasty group	Not discussed
Cohen et al, 2006	39	ASES, L'Insalata	• 71% satisfied • 41% with continued night pain	Athletes and pts with rotator cuff piercing with worse outcomes
Ide et al, 2005	40, all overhead athletes	Pt satisfaction, time to RTP	• Rowe: 27.5 → 92.1 • 75% return to preinjury level of activity	Traumatic etiology with better return to activity than overuse etiology
Kim et al, 2002	34	ASES, UCLA	• 94% satisfied • 91% return to preinjury level	Overhead sports with lower ASES (P = .024) and lower return to preinjury level (P = .015)
O'Brien et al, 2002	31	ASES, L'Insalata,	• 52% return to preinjury level • L'Insalata: 87 • ASES: 87.2	Not discussed

Abbreviations: ASES, American Shoulder and Elbow Society; BT, biceps tenodesis; pt, patient; RCT, rotator cuff tear; RTP, return to play; SLAP, superior labrum anterior to posterior; SST, simple shoulder test; UCLA, University of California Los Angeles; VAS, visual analog scale.

Note that → is placed between preoperative and postoperative scores.

TABLE 2. Outcomes and Potential Contributing Factors Following Arthroscopic SLAP Lesion Repair

to have a poor outcome due to persistent bicipital symptoms, and not necessarily due to the SLAP tear or repair itself. In patients over 40 years old with a type II SLAP lesion, the decision of whether or not to perform a SLAP repair and/or biceps tenodesis remains unclear because the true etiology of symptoms in this specific patient population is extremely difficult to determine clinically. Although the present study evaluated only patients who have had SLAP repairs, these patients may have had improved shoulder functional outcome with a biceps tenodesis with or without a SLAP repair.[26,27] Boileau et al[26] recently studied the clinical outcomes following arthroscopic biceps tenodesis, using interference screws as an alternative to repair of isolated unstable type II SLAP defects. The authors found that patients were subjectively more satisfied and had a significantly higher rate of return to previous level of activity in the biceps tenodesis group, as compared to the

SLAP repair group, including patients participating in overhead-throwing sport. Interestingly in this study the patients in the biceps tenodesis group were significantly older, with a mean age of 52 years (range, 28-64 years) compared to the SLAP repair group with a mean age of 37 years (range, 19-57 years) ($P < .001$), which clearly may be a contributing factor to the success of the biceps tenodesis procedure in this cohort.

When using revision surgery as the definition of a failed SLAP repair, age under 20 years was a significant (negative) prognostic factor. Based on these results, it is evident that greater proportions of patients under 20 years of age had to be revised compared to their comparison groups (patients 20 years of age and older). It is possible that young patients who had SLAP repairs are less likely to tolerate these repairs, potentially due to postoperative stiffness and/or reinjury.

As proposed by Burkhart et al[28] in 2001, it is possible that the mechanism of SLAP injury in overhead throwers, notably baseball pitchers, is actually related to the acceleration phase of throwing when the shoulder is in a position of extreme abduction and external rotation. Overhead athletes with high pitching/throwing volumes may develop posteroinferior shoulder stiffness, causing a deficit in internal rotation range of motion and subsequent stiffness, also known as the dead arm syndrome, as coined by Rowe.[29] This becomes problematic when the athlete acquires a SLAP lesion, such as when the patient is unable to compensate for their internal rotation deficit with a gain of external rotation, unlike in a healthy shoulder. In an outcomes study of SLAP repairs comparing overhead athletes to nonoverhead athletes, Kim et al[14] found that nonoverhead athletes had significantly better outcomes when using UCLA scores and return-to-preinjury level of activity as outcomes assessment tools. Specifically, the authors reported that only 22% of overhead athletes returned fully to their preinjury level of activity, as compared with 63% of the nonoverhead athletes. Interestingly, Pagnani et al[30] found that 12 out of 13 athletes (92%) were able to return to their preinjury level of overhead activity following SLAP repair. Ide et al[31] found that 36 of 40 (90%) overhead athletes were satisfied with their SLAP repair, with 75% of the athletes returning to their preinjury level of competitiveness. Finally, Yung et al[32] recently found that overhead athletes required a longer duration of therapy or rehabilitation in order to return to their preoperative level of activity following SLAP repair. Thus, the results represented in the literature are inconsistent, and the reason explaining why overhead athletes may be more likely to be less satisfied or take longer to return to activity after SLAP repair remains largely unknown.

Recently, Katz et al[33] performed an analysis of patients with poor outcomes following SLAP repair, with a focus on outcomes following subsequent treatment after the initial poor outcome. Overall, the authors reported that while 68% of their patient cohort was satisfied after initial SLAP failure followed by either surgical or nonoperative therapy, 32% continued to have a suboptimal response. While the authors commented on the number of patients who used tobacco ($n = 4$) and had a history of diabetes ($n = 2$), no statistical analysis was performed in attempt to correlate these and other similar demographic and social factors with a potential prognostic significance. In addition, there are a number of additional studies available that report on outcomes following SLAP repair, several of which report associations between poor outcomes and specific factors (Table 2)[11,12,14-18,26,31-36].

This study had several limitations, most notably its retrospective nature, lack of control group, and follow-up rate of 62%. We made multiple attempts to contact all of the 100 consecutive patients in the initial cohort, and unfortunately due to missing and/or incorrect contact information, we were unable to reach several patients. Another limitation is the number of concomitant procedures performed in our patient population. One major difficulty with treating shoulders with multiple injuries is understanding which lesions are truly symptomatic and which are simply incidental. As discussed by Boileau et al[26] and Kim et al,[37] it is impossible to know if patients who had a successful outcome following, for example, both SLAP and rotator cuff repair, benefited more from one repair versus than from the other or if both were truly needed to produce a successful outcome. Subgroups of patients undergoing concomitant procedures could not be statistically analyzed secondary to the small number of patients undergoing these procedures as well as the overlap between patients undergoing more than 1 concomitant procedure. Another limitation is the lack of follow-up imaging, which would have provided another objective outcome as to whether or not the SLAP repairs remained intact.

This study also had several strengths. To our knowledge, this is the first study that discusses the prognostic factors affecting the clinical outcome after SLAP repair. All patients of this relatively large cohort completed questionnaires utilizing validated, shoulder-specific outcomes surveys. Additionally, all patients were examined by a single, blinded orthopedic research fellow. There were 4 fellowship-trained orthopedic surgeons, in either sports medicine or upper extremity surgery, performing all procedures at a single institution, allowing the results to be generalizable to other surgeons who focus on the shoulder. Finally, we utilized a standardized rehabilitation protocol for all patients.

CONCLUSION

Overall, patient selection in SLAP repairs can be difficult, and the results from this study may be used to assist with patient selection for SLAP surgery and can help predict which patients might benefit from SLAP repair and which are less likely to experience significant improvement. Further long-term studies are necessary to determine the natural history of SLAP repair and to further evaluate factors that may be associated with improved surgical outcomes. ■

References and financial disclosures are available online at www.rush.edu/orthopedicsjournal.

"THE GENERAL EQUIVALENCE OF CLINICAL OUTCOMES INDICATES THAT JOINT-SPARING TECHNIQUE OF STAPLE FIXATION OF LISFRANC INJURIES SHOULD BE CONSIDERED, ESPECIALLY WHEN THERE ARE SIGNIFICANT CONCERNS REGARDING SOFT-TISSUE STATUS AND HEALING."

A Description of the Technique and Clinical Results of Staple Fixation of Lisfranc Injuries

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Lisfranc injuries to the midfoot are tarsometatarsal dislocations and fracture dislocations that result in disruption of the Lisfranc ligament, which is between the second metatarsal and the medial cuneiform (Figure 1). The mechanism is often axial load and rotation through a plantar flexed forefoot, as sometimes seen after a motor-vehicle accident, athletic activity, or fall. Lisfranc injuries may result in significant morbidity if not appropriately treated.[1] The Lisfranc joint is inherently unstable, and the maintenance of a closed reduction may be difficult. Untreated injuries may lead to midfoot collapse.[1] As such, in all but the mildest variants, nonoperative treatment is typically not advised.[2]

Numerous treatment modalities have been described, including open reduction and internal fixation with various forms of instrumentation, as well as primary arthrodesis.[2,3,4] However, all of the commonly proposed methods have significant limitations with regard to the treatment goals of (1) anatomic reduction of the tarsometatarsal joint, (2) stable fixation to allow ligamentous healing, (3) preservation of the articular cartilage of the Lisfranc joint, (4) maintenance of physiologic Lisfranc joint motion so as not to overload adjacent joints, and (5) use of fixation that does not irritate adjacent soft tissues and nerves and that is also robust enough to resist complications related to failure and breakage.

In an effort to balance these often competing goals of surgical management

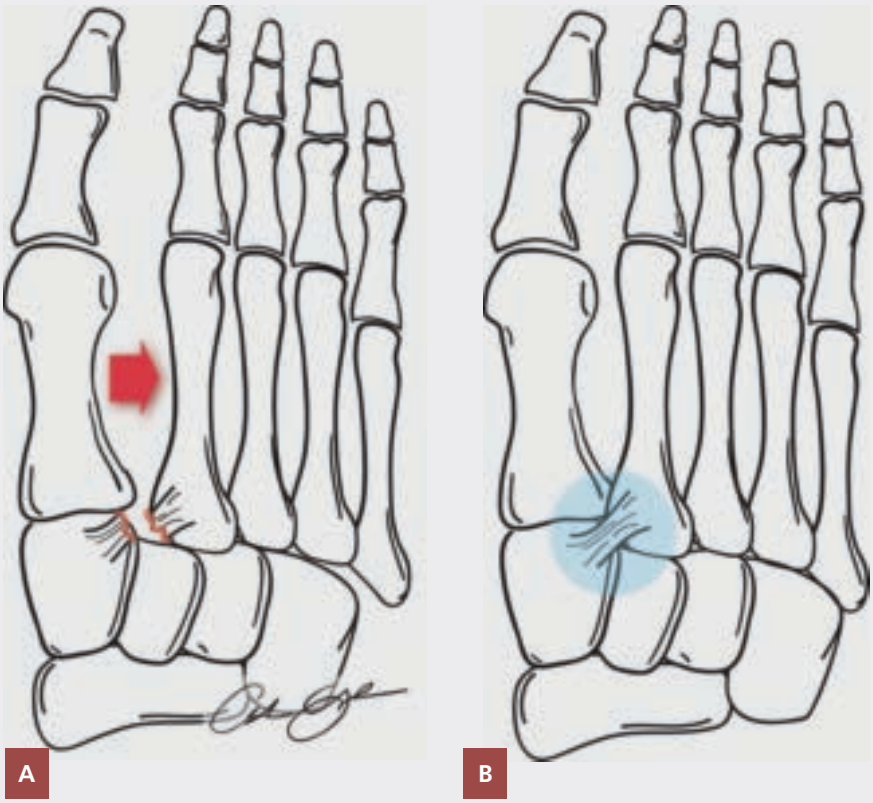

FIGURE 1. Anatomic drawing of Lisfranc joint and ligamentous structure. Image courtesy of Dr Adam Yanke, Department of Orthopedic Surgery, Rush University Medical Center.

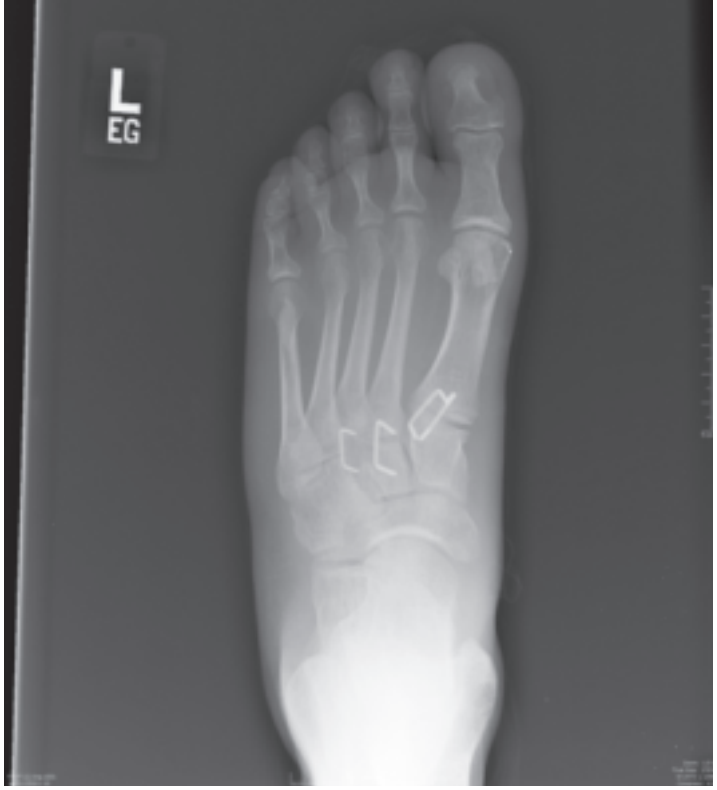

FIGURE 2. Anterior-posterior (AP) radiograph of a foot demonstrating staple fixation of the Lisfranc joint.

of the injuries, the senior author began using staple fixation with a pneumatic staple device (Stapilizer, Linvatec; Largo, Florida). It has been our experience that staple fixation allows enough rigidity for ligamentous healing without violating the articular surfaces and without limiting all motion, which would in turn, increase the motion through adjacent joints, potentially hastening the development of arthrosis in other locations. Further, in contrast to dorsal plating, the staples have a low profile, which seldom becomes symptomatic enough to necessitate removal.

This study details our technique and experience to date.

METHODS

We identified 13 consecutive patients with Lisfranc injuries who were treated operatively using pneumatic staple fixation between 2004 and 2009. Of these patients, 7 were treated with staple fixation alone; 2 with a combination of staples and a minisuture button; 1 with screw, staple, and minisuture button; and 3 with a combination of screws and Kirschner wires (K-wires).

In order to evaluate postoperative functional health and well-being, an investigator not involved in the surgical procedure administered via telephone interview both the SMFA[6] and SF-36[7] questionnaires. Our investigators carried out a literature search to identify all previous studies examining SMFA and SF36 results after operative fixation of Lisfranc injuries. We compared our questionnaire results to the results found in the literature.

Surgical technique for staple fixation

We used preoperative weight-bearing (AP) radiographs as well as clinical examination and history to make the diagnosis. We identified Lisfranc injury as pain over the dorsal midfoot and greater than 1 mm of widening between the medial and middle cuneiforms on weight-bearing AP radiograph when compared to the contralateral side.

We prepared the lower limb, draped it sterilely, and exsanguinated it with an Esmarch tourniquet. We made a longitudinal incision over the interspace between the first and second tarsometatarsal joints. We carried dissection down to the level of the joint capsule, taking care to protect the extensor tendons and neurovascular bundle.

Under fluoroscopy, we reduced the Lisfranc joint by holding the foot in neutral dorsiflexion and applying compression in a medial-lateral plane. While verifying reduction, the joint was fixated using one or more Stapilizer staples (Figure 2). The staples averaged 15 mm in length and 13 mm in width.

We took care to impact the staple several times to ensure that it was not prominent. We confirmed the reduction under fluoroscopy, copiously irrigated the wound, and closed it in an interrupted fashion. We applied a bulky, compressive dressing with a U-shaped plaster sugar tong. We instructed the patients to be non-weight-bearing for a total of 8 weeks and converted to a posterior mold splint at 2 weeks when we removed the sutures.

We administered the Short Musculoskeletal Functional Analysis (SMFA) and Short Form 36 (SF-36) questionnaires via telephone interview at a minimum of 24 months follow-up (average, 47.8 months).

We searched the PubMed literature database to locate all studies utilizing the SMFA and SF-36 questionnaires to evaluate postoperative Lisfranc patients. The keywords we used were *Lisfranc, SMFA* and *Lisfranc*, SF-36. Our search yielded 3 studies that utilized the SF-36 questionnaire (Henning, O'Conner, Schepers) and 1 study that utilized the SMFA questionnaire (Henning) to evaluate functional health and well-being in postoperative Lisfranc patients (Table 1).

We compared the mean score obtained in all categories of the SMFA and SF-36 questionnaires from our study with the mean scores found in the literature.

RESULTS

Of the 13 patients treated operatively, 8 patients were available for interview at the time of follow-up. Four patients were treated with staple fixation alone. Two were treated with a combination of staples and a minisuture button. One was treated with a combination of a combination of screws and staples, and 1 with a combination of a screw, K-wire, staple, and minisuture button.

Our literature search yielded 3 studies that utilized the SF-36 questionnaire and only 1 study that utilized the SMFA questionnaire to evaluate functional health and well-being in postoperative Lisfranc patients.[11,12,13] One study utilized both the SMFA questionnaire and the SF-36 questionnaire to evaluate functional outcome in postoperative patients,[11] and thus appears separately in both

LISFRANC STUDIES UTILIZING SMFA QUESTIONNAIRE	UTILIZED IN OUR STUDY?
Henning JA, Jones CB, Sietsema DL, et al. Open reduction internal fixation versus primary arthrodesis for Lisfranc injuries: a prospective randomized study. *Foot Ankle Int.* 2009;30(10):913-922.	No (Did not differentiate between operative and nonoperative patients)
O'Conner PA, Yeap S, Noel J, et al. Lisfranc injuries: patient- and physician-based functional outcomes. *Int Orthop.* 2003;27(2):98-102	No (Did not report SF-36 scores)
Schepers T, Kieboom BC, van Diggele P, et al. Pedobarographic analysis and quality of life after Lisfranc fracture dislocation. *Foot Ankle Int.* 2010;31(10):857-864.	Yes
LISFRANC STUDIES UTILIZING SF-36 QUESTIONNAIRE	**UTILIZED IN OUR STUDY?**
Henning JA, Jones CB, Sietsema DL, et al. Open reduction internal fixation versus primary arthrodesis for Lisfranc injuries: a prospective randomized study. *Foot Ankle Int.* 2009;30(10):913-922.	Yes

TABLE 1. Lisfranc Studies Utilizing SMFA and SF-36 Questionnaires

categories in our literature search (Table 1). Of the 3 studies that utilized the SF-36 questionnaire, 1 was not suitable for comparison because it did not report SF-36 scores.[12] A second study was not suitable because reported SF-36 scores were not reported separately for operative versus nonoperative patients (Table 1).[11]

The patients in our staple fixation group scored better in all categories on both the SMFA and SF-36 questionnaires than the patients in the studies we consulted. The patients in our study had a mean score on the SF-36 survey of 73.8 compared to 48.6 in the literature and a mean score on the SMFA of 11.8 compared to 17.3 in the literature.[11,12,13]

DISCUSSION

Staple fixation of Lisfranc ligament injuries appears to be a viable and promising treatment strategy. In our series, the average SF-36 score of 73.8 and SMFA score of 11.8 compare favorably with those reported in the literature (SF-36 of 48.6 and SMFA of 17.3). [8,9,10,11,12,13]

This study has potential limitations. It includes a relatively small number of study patients (n = 8). As such, we felt that the study did not have sufficient power to do a statistical comparison. Because the location of ligamentous injuries as well as the presence or absence of associated fractures varies throughout this series, direct comparisons are difficult. However, given that this was a preliminary, retrospective pilot study, we believe that the results are promising, because the scores for utilized postoperative outcome measures are just as good as or slightly better than those reported in the literature.

CONCLUSION

This retrospective study presents the preliminary results of postoperative functional health and well-being following the joint-sparing technique of staple fixation of Lisfranc injuries. Drawing definitive conclusions about the optimal treatment strategy for Lisfranc injuries is difficult. Not only are the injuries relatively rare, but they are seldom equivalent to one another. The general equivalence of clinical outcomes indicates that joint-sparing technique of staple fixation of Lisfranc injuries should be considered, especially when there are significant concerns regarding soft-tissue status and healing. More long-term follow-up studies, as well as comparative meta-analysis, is needed to further evaluate this promising surgical method. ■

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**This is a partial list of published works for the faculty members of the Department of Orthopedic Surgery at Rush in 2012. Works with electronic publication dates in 2012 and print publication dates in 2013 are not included in this list. Although only faculty members are cited, the department gratefully acknowledges the co-authorship of students, nurses, practitioners, therapists, residents, fellows, and colleagues at Rush. Source: PubMed.*

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SELECT RESEARCH GRANTS (2011-2012)

HOWARD S. AN, MD
Reparative, Regenerative and Anti-Inflammator Effects of E-Matrix on Bovine Pulpous Cells

BRIAN J. COLE, MD, MBA
Collecting Patient Reported Outcomes in Orthopaedic Procedures

Partial Meniscectomy vs Nonoperative Management in Meniscal Tear with OA

CRAIG J. DELLA VALLE, MD
Evaluation of Clinical Outcomes

Retrospective Comparison of Knee Arthoplasty Options for Young Active Patients

Study of Hip System in Patients with Non-Inflammatory Arthritis

TIBOR T. GLANT, MD, PHD
Mapping of Arthritis Susceptibility Genes

JOSHUA J. JACOBS, MD
Metal Ion Testing

KATALIN MIKECZ, MD, PHD
Myeloid-derived Suppressor Cells in Autoimmune Arthritis

SHANE J. NHO, MD, MS
3-D Quantification of Cam Lesion for Patients with Femoracetabular Impingement

Hip Labral Reconstruction Using an Animal Model

The Effect of Capsulotomy and Capsulectomy on Hip Stability

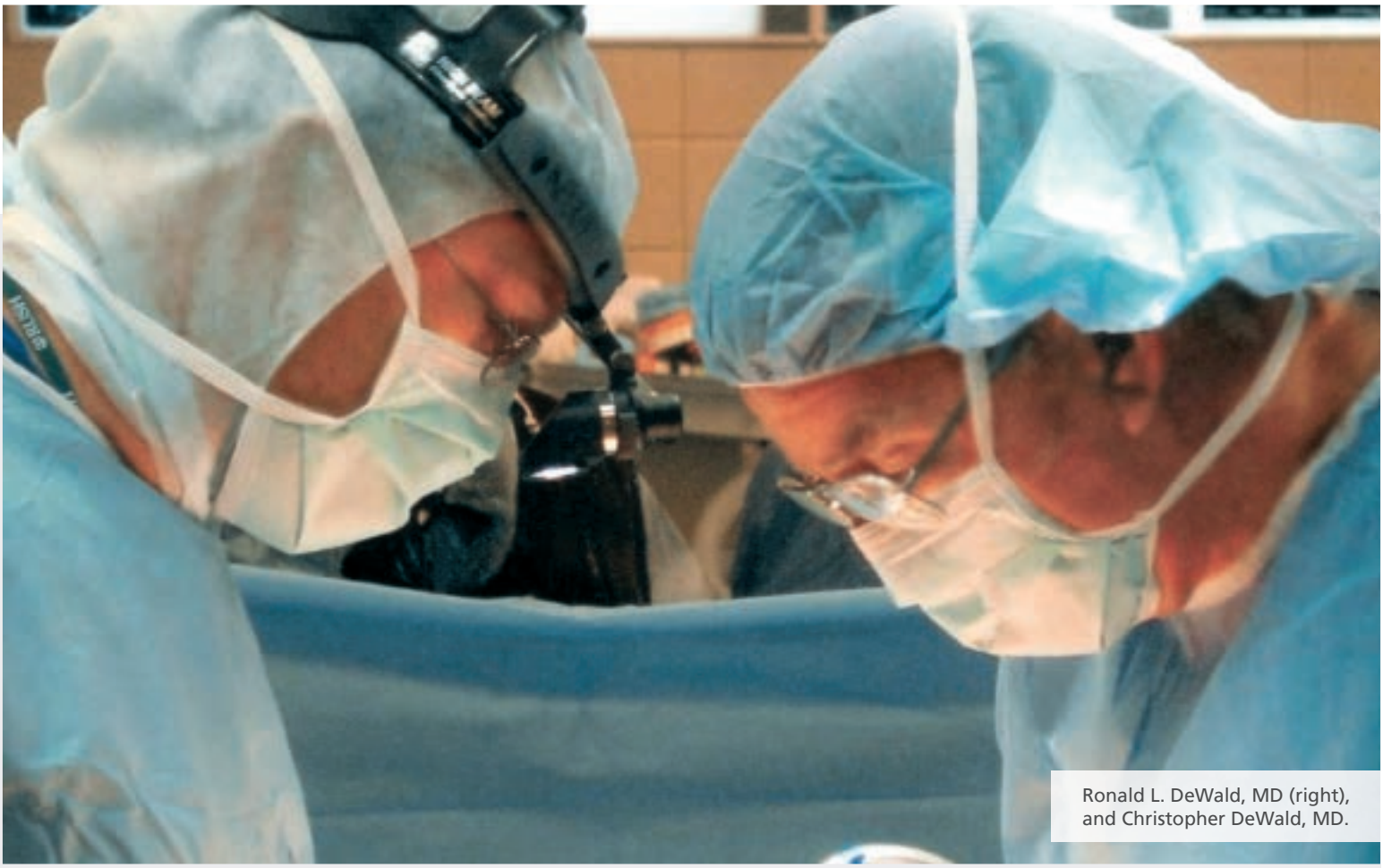
FRANK PHILLIPS, MD
Spinous Process System vs Pedicle Screw Fixation for Anterior Lumbar Fusion

KERN SINGH, MD
Facilitate Hemostasis in Subjects Undergoing Multi-level Spinal Fusions

ROBERT M. URBAN
Effect of Bisphosphonates on New Bone Formation and Strength in Critical-Size Defects

VINCENT M. WANG, PHD
MR Monitoring of Engineered Tissues

MARKUS A. WIMMER, PHD
Wear Testing for Enhanced Prediction of TKR Clinical Performance ■



Ronald L. DeWald, MD (right), and Christopher DeWald, MD.

Ahead of the Curve

An interview with one of the founding fathers of spinal deformity surgery—my father, Ronald L. DeWald, MD

BY CHRISTOPHER DEWALD, MD

At the time my father, Ronald L. DeWald, MD, began his medical training in 1955, very few orthopedic surgeons were interested in treating patients with scoliosis. Little had been written about spinal deformity, and scoliosis in particular, up to that point. And as a result of the lack of research to advance treatment, protocols were grueling for both surgeon and patient.

Never one to resist a challenge, Dad was intrigued. His interest in scoliosis—sparked during residency—grew into a passion that fueled a remarkable career and helped to transform care for spinal deformity patients. He literally wrote the book on the subject when he served as editor-in-chief of *Spinal Deformities: the Comprehensive Text*. His ideas on how to effectively treat spinal deformities, which led to the book, also served as a core curriculum for the fellowship program my father established for the Department of Orthopedic Surgery at Rush in 1972: It was the first year-long spinal deformity fellowship in the country, and I was fortunate enough to earn one of the coveted slots.

My father's lasting contributions were recognized in 2012 when the Scoliosis Research Society (SRS), of which he is a founding member and past president, honored him with its Lifetime Achievement Award. These days, after a prolific, decades-long career, Dad is enjoying a well-deserved retirement. I convinced him to take a break from playing golf to sit down with me and reflect on the career that has inspired me and so many others.

CHRISTOPHER DEWALD: You've spent almost your entire career at Rush. What brought you here initially?

RONALD DEWALD: Well, as you're aware, Rush didn't have a medical school back then. At the start of World War II, they dropped the medical school and everyone went to war. The charter was kept alive and the school reopened in 1972. I attended medical school at the University of Illinois in 1955 and graduated in 1959, which was the year of the merger between Presbyterian Hospital and St. Luke's Hospital; they closed St. Luke's and the staff came here to join the staff from Presbyterian.

> MY FATHER'S LASTING CONTRIBUTIONS WERE RECOGNIZED IN 2012 WHEN THE SCOLIOSIS RESEARCH SOCIETY (SRS), OF WHICH HE IS A FOUNDING MEMBER AND PAST PRESIDENT, HONORED HIM WITH ITS LIFETIME ACHIEVEMENT AWARD.

While I was in medical school, I worked for two years as a scrub nurse at night in the St. Luke's operating room. They paid me some money—I think it was around $12 for 12 hours. So not a lot of money, but I learned a lot. If I didn't have a case, I studied and I slept, probably in that order. I got to know some of the residents from St. Luke's and established a good camaraderie with them, and I'd done a few clerkships at Presbyterian Hospital, so I was familiar with the hospital as well as the house staff. I just felt comfortable here. When it came time to make my internship selection—in those days everyone did an internship before starting their residencies—I took a rotating internship at Presbyterian-St. Luke's Hospital.

CHRISTOPHER DEWALD: And what led you into orthopedic surgery?

RONALD DEWALD: I started my internship not really knowing what I wanted to do afterward. The rotating internship was great because you went through all of the different specialties: ob/gyn, medicine, ENT, urology, surgery, etc. I had liked orthopedics when I was a student. I did a clerkship in orthopedics for two weeks during my senior year, and it was a good duty. The attending liked the way I put on clubfoot casts. Of course, I doubt if senior medical students are allowed to do that nowadays. But I also enjoyed my radiology rotation, and I liked the radiology department at Presbyterian-St. Luke's Hospital. I applied to both programs and was accepted to both, and then I had to make a decision. I chose orthopedic surgery, and I've never looked back.

CHRISTOPHER DEWALD: Scoliosis was not a popular focus in orthopedics at that time. Why did you choose it?

RONALD DEWALD: You could say I didn't choose scoliosis, it chose me. The scoliosis cases that were being done at our hospital were very interesting, but none of the surgeons wanted to tend to these patients because treatment was extremely difficult for both patient and physician: There was an immense surgery, a long time in a cast, a lengthy hospitalization.

During my residency, we had to present a thesis at the end of each year. My first year, I had no idea what I was going to present, and then the senior resident said to me, "You're going to do scoliosis." That's what started it off, just that casual remark. It turned out I liked it so much that I asked for scoliosis again the second year, and again my third year. The more I read about it, the more interested I became. But the defining moment was in 1962, when I was a second year resident. The famous Paul Harrington, MD, a pioneer in scoliosis surgery who developed internal instrumentation called the Harrington Rod, was touring the country giving demonstrations, and he came here. Because of my interest, I was allowed into the OR. I wasn't allowed to scrub in—I was too far down the totem pole—but I got to watch him operate a patient with polio scoliosis. In about 6 hours he achieved what it was currently taking the rest of us about 9 months to accomplish. I knew I was witnessing the dawn of spine surgery, and I wanted to be part of it.

CHRISTOPHER DEWALD: So internal instrumentation caused a huge shift in scoliosis treatment?

RONALD DEWALD: It's not an exaggeration to say that it changed everything. Before, we used to take these patients in the hospital and put them in some kind of a cast to correct their spine as much as possible. And some of them were really crooked. Once the spine was as straight as we thought we could make it, we would do a spinal fusion. We'd cut a window in the back of the cast and operate through that window, with the patient still in the cast. It was grueling to do the procedure that way. Then we'd close the window, wrap the cast back up again, and nurse the patient back to health. They'd be in this cast for 6-9 months after surgery.

But the internal instrumentation changed all that. And when the Scoliosis Research Society got going in 1966 and the guys who treat spinal deformity started working together, that fueled the rapid evolution of scoliosis surgery. We went through a series of stages with instrumentation, each one improving on the last. Today, with the good instrumentation we have, scoliosis patients often don't wear a cast at all, and some doctors don't even put patients in braces postoperatively. Many patients are back home 6 days after surgery. It's just remarkable. With all these advances, now we're operating scoliosis patients who we wouldn't have operated 40 or 50 years ago. When I was a resident, we wouldn't commit to surgery until the curvature was 65 degrees. Can you imagine? Today, the standard is between 30 and 40 degrees.

CHRISTOPHER DEWALD: Besides internal instrumentation, what were the biggest changes to either surgery or medicine from the start of your career to now?

RONALD DEWALD: For one, the development of flexible tubing: endoscopes, intravenous catheters, and endotracheal and tracheostomy tubes. We did a lot more tracheostomies in those years than we do now, and we had to use metal tubes. You had to clean those tubes constantly, and a lot of the inexperienced people were afraid to take the metal tubes out because they were afraid they'd never get them back in. All of that has changed when they created the flexible tubing. Bronchoscopies are also a lot easier now. I can remember as an intern, my job was head holder. I thought, well, that can't be hard to do. But they had the patient on a frame—and you'd really have to bolt them down so their arms couldn't move—and then you'd slide the frame out and the headrest would drop down and their head would drop down, and you, the intern, would sit there and hold the patient's head. The bronchoscope—it was a solid piece—was inserted into the mouth and right down the gullet. The patient was like a sword swallower.

Anesthesia has also come a long, long way. So has blood banking. When I was a resident at Cook County Hospital [now John H. Stroger, Jr Hospital of Cook County], the blood bank worked a lot like a regular bank: You had to have credit

for a certain number of pints of blood if you wanted to do surgery. If you needed 2 pints for your operation, you had to have 2 pints in the bank. The only way to keep a positive balance was to have all of our patients, before they went home, donate a pint of blood. That's how we kept our balance positive. It didn't matter what the blood type was, you just needed the right amount so you could schedule your surgery, and then they'd find you the right type for your patient.

CHRISTOPHER DEWALD: So the doctors were the blood bankers?

RONALD DEWALD: That's right. That was at the old Cook County; obviously it's changed a lot since then. Did you operate in the amphitheater?

CHRISTOPHER DEWALD: No, that had been turned into a storage unit by the time I got there.

RONALD DEWALD: Well the amphitheater was like you see it the old black and white pictures. The operating table was in the middle of the room, and anyone could just come down in their street clothes and watch our surgery. When I was a senior resident in 1965, I would be doing a procedure and my attending surgeon would come down in his overcoat and galoshes. He'd plop right down in the front row, take off his overcoat, put it on the chair next to him, cross his legs, take his newspaper out, and start reading the paper. He'd be sitting literally 15 feet away from me. No mask on. No gown. Every so often, he'd say, "How's it going, DeWald?" And I'd reply, "Fine, sir." After awhile, he'd just fold up his newspaper, put his coat on, and walk out. Today, that kind of thing is unthinkable, but at the time it didn't raise any eyebrows. And I don't know what it was like by the time you trained there as a resident, but County wasn't air conditioned at the time, so the windows in the OR would be open, and sometimes birds would fly in. And when the electricity would fail, they didn't have a backup system, so the nurses would stand there and hold flashlights over the incision so we could see.

CHRISTOPHER DEWALD: You're kidding.

RONALD DEWALD: I kid you not. I did my first hemi-pelvectomy at County, when I was a senior resident. This was in 1965. I should clarify that I was supposed to have 2 more years of residency at this point. But as you know, I spent 2 years in the Army as an orthopedic surgeon at Valley Forge General Hospital [a former military hospital in Phoenixville, Pa.]. When I finished my duty, I applied for credit toward my residency and received 1 year of credit for my 2 years of military service. That's why I was a senior resident in 1965 instead of 1966. So the hemi-pelvectomy patient had an osteosarcoma on his pelvis, and when I finished the procedure, I got a standing ovation from my attending doctors.

CHRISTOPHER DEWALD: A hemi-pelvectomy in 1965 in an amphitheater without air conditioning?

RONALD DEWALD: That's right.

CHRISTOPHER DEWALD: Shortly after you finished your residency, the SRS was founded, and you were one of the founding members. How did that come about?

RONALD DEWALD: In 1966, there was a new scoliosis society forming, and the society had invited Claude Lambert, MD, one of the attendings at St. Luke's, to come to the first meeting. He couldn't go, but he knew of my interest in scoliosis and said, "Why don't you go in my place?" It's another one of these serendipitous things. I went to the meeting instead of Claude and was fortunate enough to become a founding member of the Scoliosis Research Society, which over the years has enhanced my fascination with and knowledge of scoliosis and the spine.

CHRISTOPHER DEWALD: How did you feel when the SRS gave you its Lifetime Achievement Award?

RONALD DEWALD: It's a significant award, but when I first found out about it, I don't think I fully appreciated what it meant to receive this kind of honor. I was taking some golf lessons in Florida, and I casually mentioned to my pro that I was going to receive a lifetime achievement award, and he was really excited about it. Probably more excited than I was. I said, "Well, it's not in golf," and he said, "Well, I could tell that." But he said no matter what your profession, when you get a lifetime achievement award, it's an incredible accomplishment. And that made me start to think about what an accomplishment it truly is. Around 27 of us founded the SRS, and I think 3 have received the lifetime achievement award, including me. Many of my fellow co-founders have gone on to focus on other areas of spine surgery, but I kept my focus on scoliosis.

CHRISTOPHER DEWALD: What inspired you to start the spinal deformity fellowship program at Rush?

RONALD DEWALD: I conceived it as a way to bring folks in who were interested in spine and who were knowledgeable. By this time, it had become obvious to me that most orthopedic residents were not interested in scoliosis or even the spine; most of them were interested in total joints or the up-and-coming sports medicine field. The residents had to rotate through my spine service whether they liked it or not, and you could tell which ones didn't like it. They would come in late; they didn't take initiative. If I was going to have any degree of competency in my house staff, I needed a fellow to help me.

We started the fellowship in 1972, and it was the first year-long spinal deformity fellowship in the country accredited by the Accreditation Council for Graduate Medical Education (ACGME). Other fellowships at that time were just for 2 or 3 months. Our first fellow, Dan Benson, MD, is now emeritus professor of orthopedic surgery at University of California-Davis, and I think we're up to about 90 fellows who have matriculated through—60 while I was director.

CHRISTOPHER DEWALD: How did you come up with the curriculum?

RONALD DEWALD: There were no models to look at, so I had to make up my own. I devised a curriculum and what I thought could be a core. What I did was look back upon my own hit or miss learning and try to formulate the curriculum along those lines. Our fellows would see the patients first and do the usual history and physical, and then I would go over it with them. If they were remiss in either their exam or their history, I would correct them. It was mandatory that they author a paper and that they submit it. It didn't matter if it was accepted, but they had to have it perfected enough that they wouldn't be embarrassed to submit it to a peer-review journal.

Our fellows were also tasked with educating the nurses. Spine patients were not part of the nursing curriculum at all,

so the fellows would give lectures and demonstrations to the nurses on how to roll the patients, how to use the different frames that we have for patients. I and my nurse, Mary Rodts, CNP, ONC, actually started the first nurse practice program at Rush because we needed a way to teach nurses how to care for spine surgery patients. That program eventually grew into Rush's nurse-practitioner program.

Anyway, that's how the fellowship kind of organized itself. In 2003, I was editor-in-chief of the textbook *Spinal Deformities: the Comprehensive Text*, which featured my thoughts and was written primarily as a core curriculum for spinal deformity fellows.

CHRISTOPHER DEWALD: Did you foresee the fellowship program becoming such a huge success?

RONALD DEWALD: I had absolutely no idea at the time, but I'm extremely proud of how the program has developed. Around 10 of our fellows have gone on to run their own spine fellowship programs, and 3 have served as president of the SRS. It's a highly competitive program, and it has a great reputation nationally for providing a top-notch educational experience. Rush now has a lot of attendings who each have their own little niche of spine surgery, so it's a wonderful opportunity to learn about the different aspects of spine surgery, from deformity to degenerative disease to tumors.

You know, there still aren't many spine fellowship programs with ACGME accreditation. I would say of the roughly 100 spine fellowships in the country, 14 are accredited by the ACGME. I've always felt strongly about having an ACGME accredited fellowship program because I was a big proponent of subspecialty certification for spinal deformity, and you have to have an accredited fellowship to move to certification. They go hand-in-hand.

CHRISTOPHER DEWALD: You fought hard to bring about subspecialty certification in spinal deformity surgery. Why didn't it come to fruition?

RONALD DEWALD: Unfortunately, I ran into a lot of roadblocks at that time, the primary one being that the pediatric orthopedic surgeons didn't want it to happen. They felt that their purview was children, and they knew how to take care of children's spines and didn't need any further qualifications or examinations. So they blocked it, and it never got anywhere. I think we really missed the boat. If we did have that recognition as a subspecialty, we would have more say so in public health policy; we would have a stronger voice. And I think it would also help the patients and be a nice core curriculum. But it wasn't to be. The only regret I have in my career is that I was unable to convince my peers that subspecialty certification in spinal deformity surgery was a worthwhile goal.

CHRISTOPHER DEWALD: And what in your career are you most proud of? I think I asked you that when you received your lifetime achievement award from the SRS.

RONALD DEWALD: You did ask me. Do you remember my answer? I said I was most proud of you, of the fact that you became a spine surgeon.

CHRISTOPHER DEWALD: I was afraid that's what you said.

RONALD DEWALD: I think every father's dream is to have his son follow in his footsteps, take over his business, his practice, his legacy. The relationship between a father and son is precious. You can encourage your child, but as they reach a certain age you can't push them too hard or it becomes negative. I always tried to encourage all of my sons to go into medicine, but you were the only one who stuck to the idea. If one of your kids goes to medical school and goes into orthopedics, you'll have that same feeling.

CHRISTOPHER DEWALD: So besides *me*, what are you most proud of?

RONALD DEWALD: Well, certainly the Ronald L. DeWald, MD, Endowed Chair in Spinal Deformities is very near and dear to me. That was established in 1996, and it's the only one of its kind in the United States. It's interesting how the chair came about. I worked with an implant company called Sofamor Danek (now Medtronic Sofamor Danek) that produced implants for spinal deformity. I helped them develop some implants over the years. Well, the company became extremely successful and told me they wanted to reward me for my work with them by giving me stock options; I decided instead to ask them to make a direct contribution to Rush toward the establishment of an endowed chair. I talked to Leo Henikoff, MD, who was president and CEO of Rush back then, and he said, "Great idea. We'll make it the Ronald DeWald Chair in Orthopedics." I said, "No. I want it to be in spinal deformity surgery." He ultimately agreed, and we were able to make it happen thanks to Sofamor Danek's gift, additional contributions, and other funding sources.

The first chair holder was John Lubicky, MD, and Gunnar B. J. Andersson, MD, PhD, has it now. I'm honored that Gunnar is holding it because his CV is bigger than the telephone book, and he's going to set a very high standard for the next person who's appointed; once you set a high standard like that you can't go backwards. It's especially pleasing to me that spinal deformity is a recognized entity now—even if we never did get subspecialty certification.

CHRISTOPHER DEWALD: What do you want your legacy to be?

RONALD DEWALD: I want my professional colleagues to remember me as a dedicated, honest, devoted physician and surgeon, a pioneer in spinal surgery, and someone they could count on in any hour of need. I'd also like to be remembered as someone who was passionate about teaching others how to care for spinal deformity patients. And, of course, I'm working on getting a third generation of DeWald orthopedic surgeons to keep the legacy going. Now that your daughter is in college, it's time to start campaigning.

CHRISTOPHER DEWALD: I can't push her.

RONALD DEWALD: But Grandpa can.

Christopher DeWald, MD, through his active practice at Midwest Orthopaedics at Rush, is widely recognized as a leader in surgical care of spinal deformities. ■

Principal photography for the 2013 *Rush Orthopedics Journal* provided by Lauren Anderson and Jason Chiou.

A Tradition of Excellence

> "MIDWEST ORTHOPAEDICS AT RUSH SPECIALISTS ARE LEADING THE WAY IN THE DEVELOPMENT AND REFINEMENT OF ADVANCED TREATMENTS THAT BENEFIT PATIENTS AT RUSH—AND AROUND THE GLOBE."

Midwest Orthopaedics at Rush (MOR) is a private practice medical group whose fellowship-trained physicians are on the faculty of Rush University Medical Center in Chicago. With MOR based primarily at Rush, our renowned surgeons and physicians have access to all the resources of a world-class academic medical center, including the state-of-the-art operating rooms in Rush's new hospital.

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3D Topographic Analysis of Lumbar Facet Joint Degeneration In Vivo

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Cutting-Edge Techniques for Minimally Invasive Thoracic Spine Surgery

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The Challenge of Sports-Related Concussion for Male and Female Athletes of All Ages

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Gender Differences in Cam Deformities in Patients with Femoroacetabular Impingement

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The Effect of Capsulotomy on Hip Stability

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Age-Related Differences in Radiographic Parameters for Femoroacetabular Impingement in Hip Arthroplasty Patients

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Posterior Elbow Soft-Tissue Reconstruction Using a Flexor Carpi Ulnaris Muscle Turnover Flap

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Retrospective Analysis of Prognostic Factors Associated with Successful Arthroscopic Superior Labrum Anterior to Posterior (SLAP) Repair

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A Description of the Technique and Clinical Results of Staple Fixation of Lisfranc Injuries

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