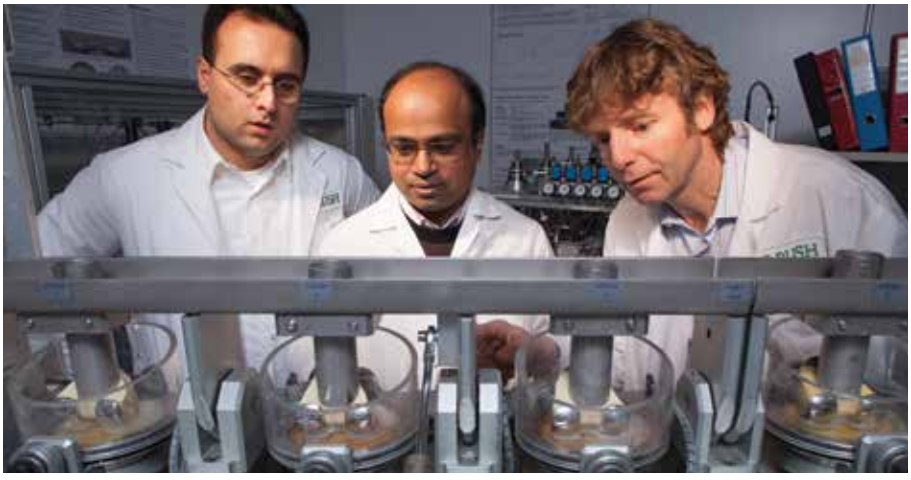
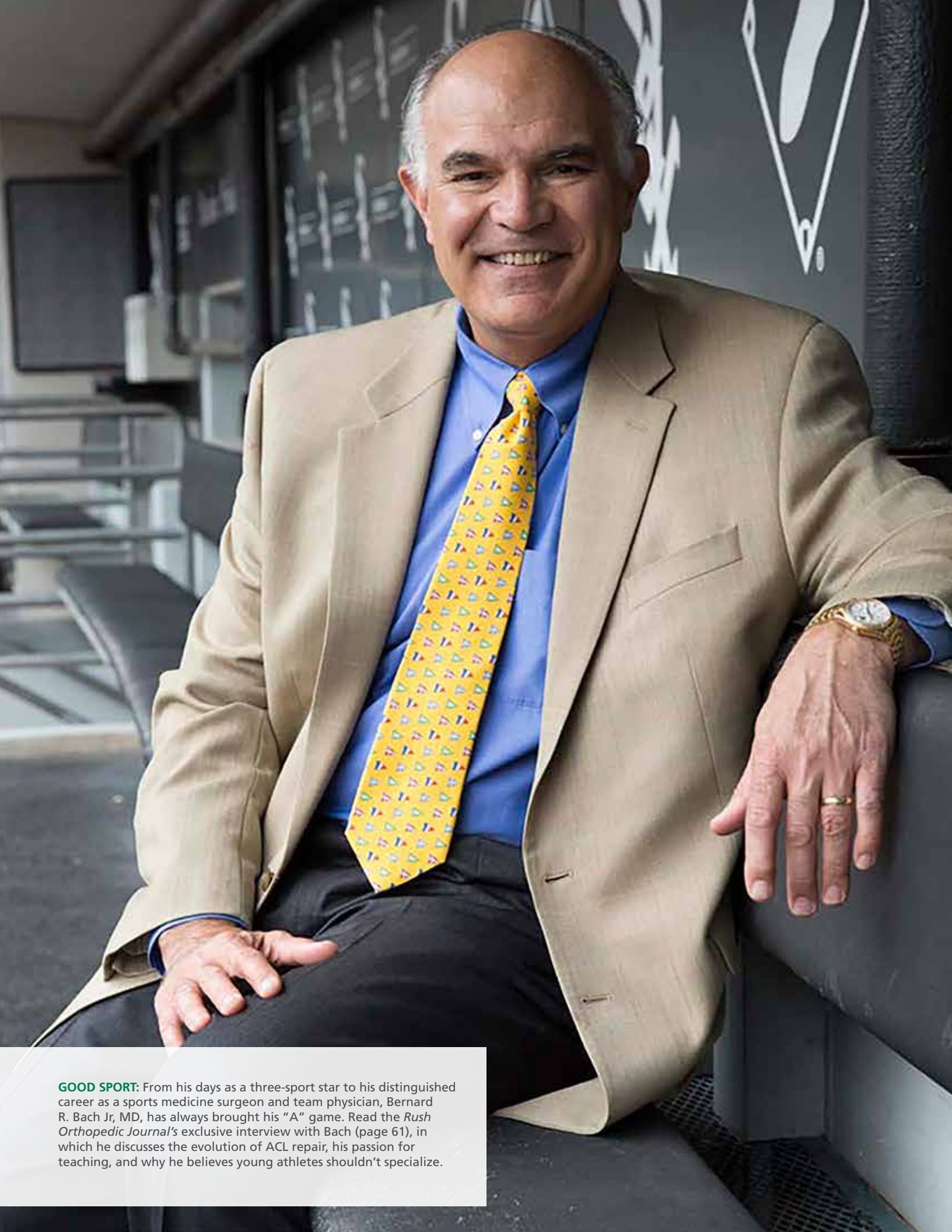


2014 RUSH

Orthopedics Journal

RUSH UNIVERSITY MEDICAL CENTER



GOOD SPORT: From his days as a three-sport star to his distinguished career as a sports medicine surgeon and team physician, Bernard R. Bach Jr, MD, has always brought his "A" game. Read the *Rush Orthopedic Journal's* exclusive interview with Bach (page 61), in which he discusses the evolution of ACL repair, his passion for teaching, and why he believes young athletes shouldn't specialize.

To view the **2014 *Rush Orthopedics Journal* online or to view past issues of the journal,** please visit the Rush website at **www.rush.edu/orthopedicsjournal.**

> RESEARCH HAS BEEN A PRIORITY FOR OUR DEPARTMENT SINCE ITS INCEPTION, AND THE *RUSH ORTHOPEDICS JOURNAL* REFLECTS THAT STEADFAST COMMITMENT.

Chairman's Letter

Since the first *Rush Orthopedics Journal* was published in 2009, I have had the privilege of introducing the journal and highlighting the achievements of our faculty in the Department of Orthopedic Surgery. While much has changed since that first edition, one thing has remained consistent: the breadth and quality of the articles submitted by our faculty, fellows, and residents.

Research has been a priority for our department since its inception, and the *Rush Orthopedics Journal* reflects that steadfast commitment. At any given time, we are involved in dozens of studies across almost every orthopedic specialty, as well as collaborative research with other departments at Rush and other institutions worldwide.

While I don't usually call attention to specific projects, it's worth noting that in 2013, our research program received tremendous support from the National Institutes of Health (NIH) at a time when competition for funding is at an all-time high. The following research faculty received sizeable NIH grants to fund projects related to prevalent orthopedic problems:

- **Tibor Glant, MD, PhD,** to map the genome for disease-promoting genes in ankylosing spondylitis
- **Nadim J. Hallab, PhD,** to look at a potential target for mitigating aseptic osteolysis
- **Nozomu Inoue, MD, PhD,** to gain a better understanding of the effects of spinal manipulation therapy on low back pain
- **Katalin Mikecz, MD, PhD,** to characterize the role of citrullinated PG—a molecule linked to rheumatoid arthritis—in provoking immune attacks against the joints
- **Tibor A. Rauch, PhD,** to explore the epigenetic factors and genes involved in the regulatory network that controls the inflammatory process in joints
- **Vincent M. Wang, PhD,** to develop therapeutic mechanobiologic approaches to eliminate or suppress pathologic chondroid (cartilage) accumulation in tendons, and to adapt these approaches to treat human tendinopathic issues

By furthering our understanding of these and other conditions, we will continue to make progress toward developing more effective and efficient treatments. This, in turn, will improve the quality of life not only for the patients we treat at Rush, but also for the millions of people globally with musculoskeletal diseases.

Some of our recent findings are featured in the pages that follow. I hope you enjoy reading about them as much as I did. ■

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

JOINT EFFORT: Joshua J. Jacobs, MD (center), shown here with fellow adult reconstructive surgeons Richard A. Berger, MD (left), and Craig J. Della Valle, MD, is engaged in groundbreaking research on biocompatibility, implant wear, and candidate biomaterials for joint reconstruction. His team studies implants and tissues obtained from patients at revision surgery and as part of the world's largest postmortem retrieval program for joint replacement.

Orthopedic Faculty and Fellows

ADULT RECONSTRUCTIVE SURGERY

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

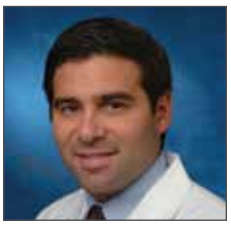
Chairman and professor, Department of Orthopedic Surgery

Richard A. Berger, MD

Assistant professor, Department of Orthopedic Surgery

Brett Levine, MD, MS

Assistant professor, Department of Orthopedic Surgery



Craig J. Della Valle, MD

Professor, Department of Orthopedic Surgery

Director, Adult Reconstructive Orthopedic Surgery Fellowship Program

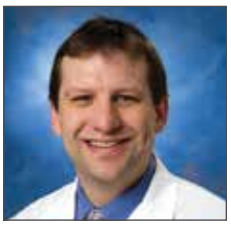
Wayne G. Paprosky, MD

Professor, Department of Orthopedic Surgery

Jorge O. Galante, MD, DMSc

The Grainger Director of the Rush Arthritis and Orthopedics Institute

Professor, Department of Orthopedic Surgery



Scott M. Sporer, MD, MS

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Associate professor, Department of Orthopedic Surgery

FELLOWS

Herman Botero, DO
Medical school – New York College of Osteopathic Medicine
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Ryan Caufield, MD
Medical school – Georgetown University School of Medicine
Residency – Georgetown University Hospital

Jonathan Christy, MD
Medical school – West Virginia University School of Medicine
Residency – The Ohio State University Wexner Medical Center

Elizabeth A. Dailey, MD
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Residency – University of Washington - Seattle

Joshua Murphy, MD
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Residency – University of Michigan

Muthana Sartawi, MD
Medical school – Arabian Gulf University
Residency – Dalhousie University, Halifax, Nova Scotia

Roshan Shah, MD, JD
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Residency – Hospital of the University of Pennsylvania

ELBOW, WRIST, AND HAND SURGERY

Mark S. Cohen, MD

Director, Section of Hand and Elbow Surgery

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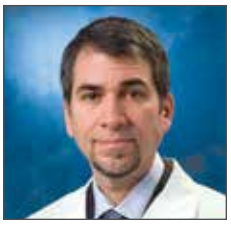
Robert W. Wysocki, MD

Assistant professor, Department of Orthopedic Surgery



John J. Fernandez, MD

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FOOT AND ANKLE SURGERY

George Holmes Jr, MD

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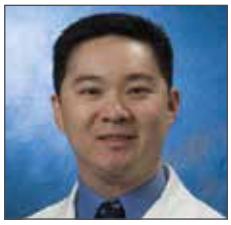
Associate Professor, Department of Orthopedic Surgery



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Assistant professor, Department of Orthopedic Surgery

Johnny L. Lin, MD

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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HAND, UPPER EXTREMITY, AND MICROVASCULAR FELLOWSHIP

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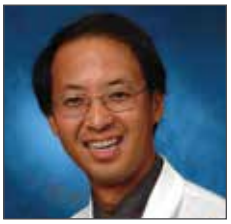
PEDIATRIC ORTHOPEDIC SURGERY

Monica Kogan, MD

Director, Section of Pediatric Surgery

Assistant professor, Department of Orthopedic Surgery

SPINE SURGERY



Howard S. An, MD

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The Morton International Chair of Orthopedic Surgery

Professor, Department of Orthopedic Surgery

Director, Spine Surgery Fellowship Program

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Kim W. Hammerberg, MD

Assistant professor, Department of Orthopedic Surgery

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Kern Singh, MD

Associate professor, Department of Orthopedic Surgery

SPINE SURGERY FELLOWS

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Residency – William Beaumont Army Medical Center

Eric Sundberg, MD

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Residency – Stanford University Medical Center

SPORTS MEDICINE, SURGERY

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

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Professor, Department of Orthopedic Surgery

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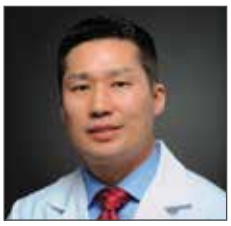

Shane J. Nho, MD, MS

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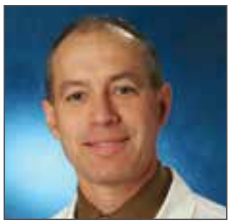

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Professor, Department of Orthopedic Surgery

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Assistant professor, Department of Family Medicine and Department of Orthopedic Surgery

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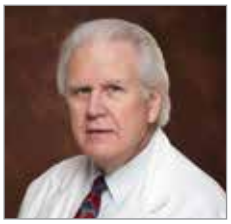

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Anastasia Skipor, MS

Instructor, Department of Orthopedic Surgery

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Director, the Joan and Paul Rubschlager Tribology Laboratory

Professor, Department of Orthopedic Surgery

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Professor, Department of Orthopedic Surgery

Katalin Mikecz, MD, PhD
Professor, Department of Orthopedic Surgery

Not pictured:
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Qiping Zheng, PhD
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Alejandro A. Espinoza Orías, PhD
Instructor, Department of Orthopedic Surgery

SPINE BIOMECHANICS; CAD/COMPUTER ANALYSIS

Raghu N. Natarajan, PhD
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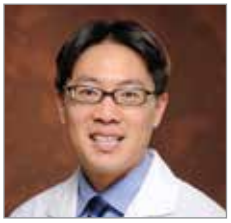
SPORTS MEDICINE RESEARCH LABORATORY



Vincent M. Wang, PhD

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Professor, Department of Orthopedic Surgery

THE JOAN AND PAUL RUBSCHLAGER TRIBOLOGY LABORATORY

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Assistant professor, Department of Orthopedic Surgery

Alfons Fischer, PhD

Visiting professor, Department of Orthopedic Surgery

Mathew T. Mathew, PhD

Assistant professor, Department of Orthopedic Surgery

Not pictured:

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Michel Laurent, PhD, scientist, Department of Orthopedic Surgery

Department of Orthopedic Surgery Residents

CLASS OF 2014

Sanjeev Bhatia, MD
Medical school – Northwestern University Feinberg School of Medicine

Debdut Biswas, MD
Medical school – Yale University School of Medicine

Christopher E. Gross, MD
Medical school – Harvard Medical School

Andrew R. Hsu, MD
Medical school – Stanford University School of Medicine

Kevin U. Park, MD
Medical school – Tulane University School of Medicine

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Medical school – Medical College of Wisconsin

Peter N. Chalmers, MD
Medical school – Columbia University College of Physicians and Surgeons

Jonathan M. Frank, MD
Medical school – University of California Los Angeles Geffen School of Medicine

William Slikker III, MD
Medical school – Stanford University School of Medicine

David M. Walton, MD
Medical school – Case Western Reserve University School of Medicine

CLASS OF 2016

Nicholas M. Brown, MD
Medical school – Columbia University College of Physicians and Surgeons

Rachel M. Frank, MD
Medical school – Northwestern University Feinberg School of Medicine

Bryan D. Haughom, MD
Medical school – University of California San Francisco School of Medicine

Michael D. Hellman, MD
Medical school – Jefferson Medical College of Thomas Jefferson University

Andrew J. Riff, MD
Medical school – Georgetown University School of Medicine

CLASS OF 2017

Gregory L. Cvetanovich, MD
Medical school – Harvard Medical School

Brandon J. Erickson, MD
Medical school – Tufts University School of Medicine

Yale A. Fillingham, MD
Medical school – Rush Medical College

David M. Levy, MD
Medical school – Columbia University College of Physicians and Surgeons

Nathan G. Wetters, MD
Medical school – University of Illinois College of Medicine at Rockford

CLASS OF 2018

Bonnie P. Gregory, MD
Medical school – University of Louisville School of Medicine

Molly C. Meadows, MD
Medical school – Columbia University College of Physicians and Surgeons

Bryan M. Saltzman, MD
Medical school – Rush Medical College

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

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

CLASS OF 2019

Joshua Bell, MD
Medical school – Medical College of Georgia at Georgia Regents University

Kevin Campbell, MD
Medical school – University of Wisconsin School of Medicine and Public Health

Philip Louie, MD
Medical school – University of Washington School of Medicine

Timothy Luchetti, MD
Medical school – Columbia University College of Physicians and Surgeons

Allison Rao, MD
Medical school – Stanford University School of Medicine

"TREATMENTS THAT BIOLOGICALLY REPAIR OR REGENERATE THE INTERVERTEBRAL DISC TISSUES HOLD GREAT PROMISE FOR THE TREATMENT OF DISCOGENIC LOW BACK PAIN."

Cell Therapy with Human Dermal Fibroblast for Intervertebral Disc Repair

ANA CHEE, PHD / PENG SHI, DDS, PHD / THOMAS CHA, MD, MBA / TING-HSIEN KAO, MD / SHU-HUA YANG, MD, PHD / DING CHEN, MD / YEJIA ZHANG, MD, PHD / HOWARD S. AN, MD

AUTHOR AFFILIATIONS

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CORRESPONDING AUTHOR

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Spinal disorders present a major burden on medical, social, and economic structures of developed countries. Non-operative and operative treatments, mostly not evidence based, carry an enormous socio-economic burden in today's aging populations. Targeted biological therapies may be less invasive and provide patients better relief from spinal pain.

The intervertebral disc (IVD) has a tough outer ring called the annulus fibrosus (AF) and a gelatinous inner core, called the nucleus pulposus (NP). The AF and the endplates of the vertebrae enclose the NP. The NP maintains fluid pressure by means of negatively charged proteoglycans, which attract sodium ions (Na+) and water molecules. Studies have implicated the progressive loss of the proteoglycan content in the NP in the pathogenesis of IVD degeneration. The imbalance of anabolic and catabolic activities results in the loss of homeostasis. In degenerative IVDs, the number of proliferating cells eventually decreases as the number of senescent cells increases.[1]

The understanding of molecular mechanisms of disc degeneration and development of animal models spur advancements in designing and testing targeted biological treatments of diseases associated with disc degeneration. The rabbit disc degeneration model has been beneficial in studying biological mechanisms of disc degeneration and testing therapeutics for disc regeneration. After annulus needle puncture and aspiration of the nucleus, rabbit discs slowly and progressively degenerate. The degeneration can be quantitatively assessed through conventional radiography, magnetic resonance imaging (MRI), and histology.[2] Treatments that biologically repair or regenerate the IVD tissues hold great promise for the treatment of discogenic low back pain. Injections with osteogenic protein-1 (OP-1) or recombinant human growth and differentiation factor-5 (rhGDF-5) into degenerating rabbit discs have restored disc height and resulted in improvements in MRI and histology grades, validating these molecules as potential therapeutics for disc regeneration.[3-6]

Introducing viable cells into the degenerating IVD may promote matrix repair and restore physiological function. Gruber et al demonstrated long-term survival of transplanted autologous IVD cells embedded in a collagen matrix in sand rats.[7] In a canine model, transplanted autologous IVD cells survived for at least 1 year.[8] Autologous human IVD cells derived from a therapeutic discectomy have also been tested in a clinical setting.[9,10] Autologous bone marrow mesenchymal stem cells have survived and replicated in rabbit degenerative discs 48 weeks after transplantation.[11] Murine embryonic stem cells have survived in rabbit degenerative discs 8 weeks after transplantation in vivo without rejection.[12] Yoshikawa et al reported 2 case studies where bone marrow mesenchymal stem cell transplantation restored disc height and function and decreased symptoms.[13]

We used the rabbit disc degeneration model to explore cell therapy with neonatal human dermal fibroblasts (nHDFs). Because nHDFs can differentiate into chondrocytes,[14] we hypothesized that nHDFs would likely be a promising cell therapy for degenerating discs. Dermal fibroblasts can transdifferentiate into fat-, cartilage-, and bone-like cells, demonstrating their multilineage potential.[15] Preclinical and clinical studies have shown that nHDFs embedded in human collagen-based extracellular matrix helps heal surgical wounds.[16,17]

Our aim was to determine the effects of intradiscal transplantation of nHDFs on the progression of IVD degeneration by measuring radiographic, MRI, biochemical, histological, and gene expression changes in the rabbit disc degeneration model.

MATERIALS AND METHODS

NEONATAL HUMAN DERMAL FIBROBLASTS (NHDFS)

The nHDFs isolated from human foreskin were purchased from Invitrogen Life Technologies (Carlsbad, California). The cells in monolayer were expanded and cultured in Dulbecco's Modified Eagle medium (DMEM, high glucose), 1% penicillin-streptomycin (PS), 25 mM 4-(2-hydroxyethyl)-1-piperazineethanesulfonic acid (HEPES), (all from Invitrogen Life Technologies, Carlsbad, California), and 10% fetal bovine serum (Omega Scientific, Tarzana, California) under normal atmospheric oxygen conditions (~20% O_2). On the day of injection, we labeled nHDFs with infrared dye for cell tracking. We detached cells from the monolayer with trypsin, labeled them with CellVue® NIR815 fluorescent dye (LI-COR Biosciences, Lincoln, Nebraska) and resuspended them at a concentration of 1.0×10^7 cells per mL.

SURGICAL METHODS FOR RABBIT DISC DEGENERATION AND CELL TREATMENT

For this study, we used New Zealand white rabbits (n = 16) (Myrtle's Rabbitry, Thompson Station, Tennessee) weighing 2.5-3 kg, with the approval of the Institutional Animal Care and Use Committee. We cared for and maintained the rabbits in accordance with National Institute of Health guidelines. One day before surgery, we administered meloxicam (0.2 mg/kg) subcutaneously as pre-emptive analgesia. Immediately before surgery, we took X-ray images of the sedated rabbits. Under general anesthesia and using aseptic conditions, we made a left abdominal incision and exposed the ventral surfaces of 4 consecutive lumbar IVDs (L2/3, L3/4, L4/5, and L5/6). Using an 18-gauge needle, we punctured the ventral AF to a depth of 5 mm into the NP; rotated the needle 360 degrees; and applied suction through a 5-mL syringe for 10 seconds to denucleate the IVD at all 4 levels. We placed a staple and a suture on the psoas muscle at the L4/5 level as a marker. The surgical wound was closed in layers. We took a postoperative X-ray to confirm the level of puncture. The rabbits were returned to their cages and mobilized ad lib. As postsurgical analgesics, we administered meloxicam for 2-3 days. Buprenorphine HCl (0.01-0.03 mg/kg) was given up to twice daily for 2-3 days, when needed, in consultation with the veterinary staff.

Four weeks postoperatively, we took an X-ray to confirm degeneration of discs. Then, we made right abdominal incisions to expose the ventral surfaces of the L2/3, L3/4, L4/5, and L5/6 IVDs. We randomized treatments, using either nHDFs or saline control, and injected 8 µL of solution into the degenerated rabbit IVDs with an Exmire syringe (Ito Corporation, Shizuoka, Japan) and 27-gauge needle. We took X-ray images again 8 weeks posttreatment.

RADIOGRAPHIC ANALYSIS

Using the X-ray images we took before the first and second surgeries, and after treatment, we calculated disc height indexes with digitized radiography. Three orthopedic researchers who were blinded to the treatment groups independently interpreted all X-ray images. They analyzed vertebral body height and IVD height, using the custom program for MATLAB software (Mathworks Inc., Natick, Massachusetts). We exported the data to Microsoft Excel software (Microsoft Corporation, Redmond, Washington) and expressed the IVD height as the disc height index (DHI), using the method of Lu et al[18], with a slight modification (DHI = IVD height / adjacent IVD body height). We calculated average IVD height by averaging measurements from anterior, middle, and posterior portions of the IVD and dividing that by the average of adjacent vertebral body heights. Changes in the DHI of injected discs were expressed as %DHI and normalized to the measured preoperative IVD height (%DHI = postoperative DHI / preoperative DHI ×100) as previously described.[3]

MRI ANALYSIS

At 8 weeks after nHDF treatment, we euthanized the rabbits with pentobarbital and removed the spines en block. We performed MRIs on the rabbit spines using a 1.5 Tesla MRI machine (Siemens, Malvern, Pennsylvania) with a hand coil. We obtained transverse relaxation time (T2)-weighted sections in the sagittal plane in the following settings: fast-spin echo sequence with time to repetition (TR) of 4000 milliseconds; time to echo (TE) of 97 milliseconds; slice thickness of 3.5 mm; field of view (FOV) 250, and matrix of 320 (base)/80 (phase). The Dicom-formatted image data (Dicom 3.0, National Electrical Manufacturers Association, Rosslyn, Virginia) were transferred to pictures using Image J software (National Institutes of Health, Bethesda, Maryland). Five blinded independent orthopedic researchers graded the MR images according to the modified Pfirrmann scale and used the images to determine MRI indexes.

We calculated MRI indexes as described by Sobajima et al.[19] Briefly, to define the region of interest, we outlined the NP of each IVD, using a computer mouse. Using the Image J software, we calculated the high signal intensity area, highest signal intensity, and average signal intensity of this region of interest for each IVD. We defined the MRI index as the product of the high signal intensity area and average signal intensity. Then we normalized the MRI indexes to the uninjured intact discs in the same animal.

CELL TRACKING

We removed remaining muscles surrounding the spines and scanned the spine segments with an infrared imager (LI-COR Biosciences, Lincoln, Nebraska) to detect signals at the 700- and 800-nm wavelengths. Using the imaging software, we determined infrared fluorescence intensity counts per mm^2 of the individual discs and exported the data to Microsoft Excel. We then averaged the infrared fluorescence intensities for each time point.

INTERVERTEBRAL DISC TISSUE PREPARATION FOR HISTOLOGY

For histological analysis, we isolated intervertebral discs with approximately one-third of the adjacent bony vertebral body and fixed them with 10% formaldehyde for 1 week. We decalcified the disc-endplate segments with a solution containing 10% citric acid and 20% formic acid, which we changed daily until the bony portion was completely decalcified. We embedded the tissues in paraffin and sectioned them to 5-µm thickness. We then deparafinized the tissue sections, stained them with 1% Alcian blue solution (Poly Scientific R&D Corp., Bay Shore, New York) for 30 minutes, followed with haematoxylin for 5 minutes and eosin (both from VWR, Radnor, Pennsylvania) for 20 seconds. Figure 1 shows representative images of the sections.

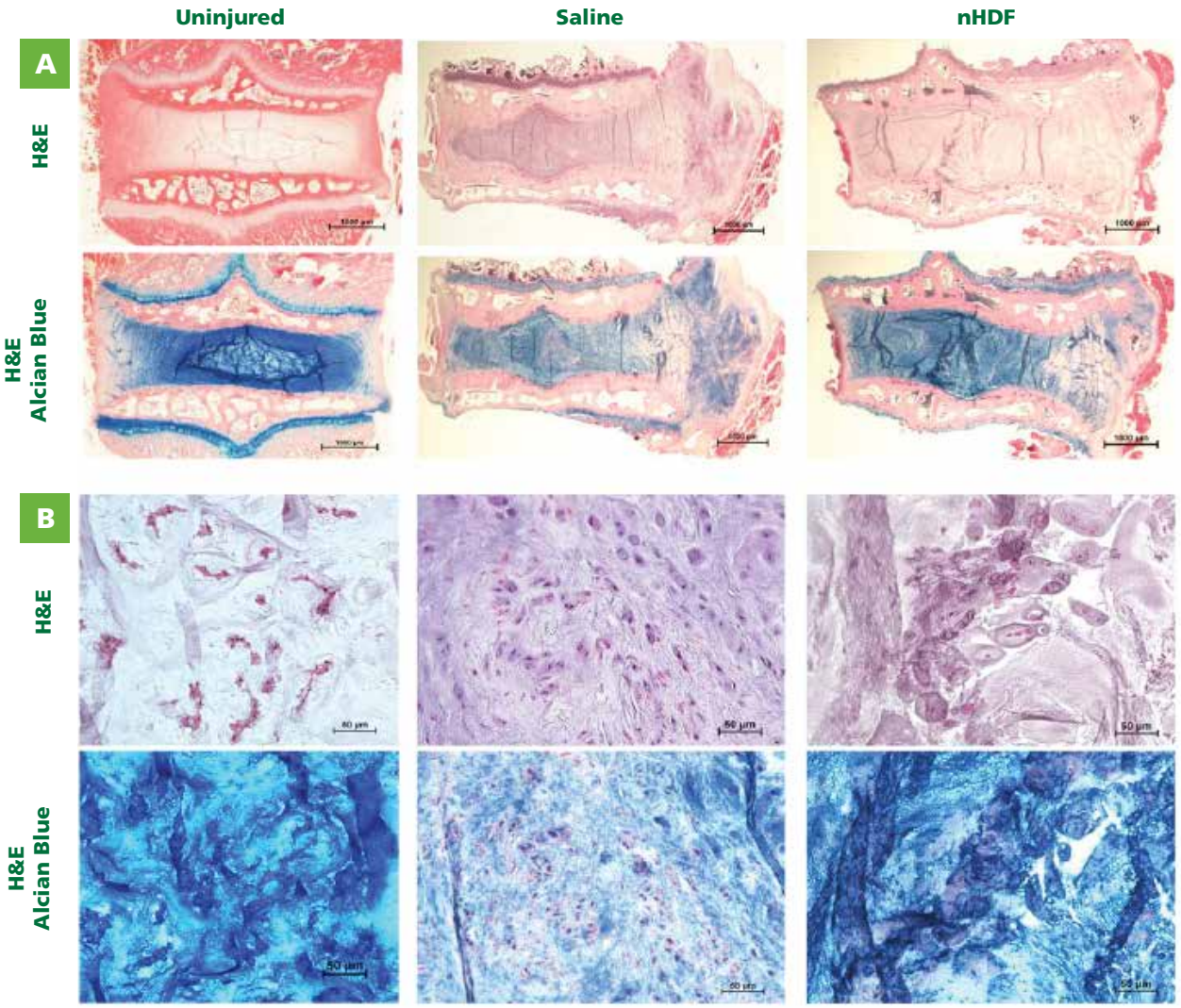


FIGURE 1. Histological changes 8 weeks after treatment. At 4 weeks after disc injury, we treated the degenerated rabbit discs with neonatal human dermal fibroblast (nHDF) or saline. At 8 weeks after treatment, we fixed, decalcified, and sectioned the rabbit discs, and stained them with haematoxylin and eosin (H&E) or Alcian blue and H&E. Histological images of an uninjured intact rabbit disc (left column), an injured saline-treated rabbit disc (middle column), and an injured nHDF-treated rabbit disc (right column). **A,** 10×magnification, scale bar = 1000 µm; **B,** 200×magnification, scale bar = 50 µm).

HISTOLOGICAL SCALE FOR INTERVERTEBRAL DISC

We used a numeric scale, based on previous studies, to quantify the degree of degeneration and to which we added cell proliferation scores to better suit this cell therapy study. We analyzed the sections using 4 categories: NP, AF, border between the NP and AF, and the status of the extracellular matrix (Table 1, Part 1). On a separate scale, proliferation of cells was also graded (Table 1, Part 2). Four blinded orthopedic researchers evaluated the tissues, and we averaged scores from each of the categories and compared the nHDF-treated, saline-treated, and uninjured untreated control groups. We obtained total morphological scores by adding the average scores from each of the 4 categories.

BIOCHEMICAL ASSAYS

Using a microbalance with a readability of 1 µg (Mettler-Toledo, Columbus, Ohio), we obtained accurate wet and dry weights of NP and AF tissues. We used papain to digest the tissues and determined the contents of DNA and proteoglycan as we described previously.[20,21] To analyze the DNA in the digest, we used a fluorometric DNA assay and the bisbenzimidozole fluorescent dye method (Hoechst 33258; Polysciences, Inc., Warrington, Pennsylvania). We then analyzed the total sulfated proteoglycans, using the dimethyl-methylene blue (Polysciences, Inc., Warrington, Pennsylvania) dye binding method.

GENE EXPRESSION ASSAYS

We isolated total recombinant nucleic acid (RNA) from NP and AF tissues of each rabbit disc and measured mRNA levels of specific genes with real-time polymerase chain reaction (PCR). We homogenized the NP and AF tissues, extracted the RNA with Trizol reagent (Invitrogen

TABLE 1. Definition of Histological Scale

PART 1. MORPHOLOGICAL SCORE

I. Annulus fibrosus

Grade:

1. Normal, pattern of fibrocartilage lamellae (U-shaped in the posterior aspect and slightly convex in the anterior aspect) without ruptured fibers and without a serpentine appearance anywhere within the annulus
2. Ruptured or serpentined patterned fibers in less than 30% of the annulus
3. Ruptured or serpentined patterned fibers in more than 30% of the annulus

II. Border between the annulus fibrosus and nucleus pulposus

Grade:

1. Normal
2. Minimally interrupted
3. Moderately/severely interrupted

III. Matrix of the nucleus pulposus

Grade:

1. Normal gelatinous appearance
2. Slight condensation of the extracellular matrix
3. Moderate/severe condensation of the extracellular matrix

IV. Cellularity of nucleus pulposus

Grade:

1. Normal or increased cellularity with large vacuoles in the gelatinous structure of the matrix
2. Slight decrease in the number of cells and fewer vacuoles
3. Moderate/severe decrease (>50%) in the number of cells and no vacuoles

PART 2. PROLIFERATION SCORE

Grade:

1. No evidence of proliferation.
2. Limited Proliferation: proliferation can be found only in limited areas; proliferation cells are more fibroblast-like.
3. High Proliferation: proliferation (cloning) can be found in multiple areas; proliferation cells are more chondrocyte-like.

Life Technologies, Carlsbad, California), and then further purified the tissues using an RNeasy Kit (Qiagen, Germantown, Maryland). We reverse transcribed 50 ng of total RNA into complementary deoxyribonucleic acid (cDNA) with random primers, using the High Capacity RNA to cDNA Kit from Applied Biosystems. We performed quantitative real-time PCR using Taqman PCR Master Mix, Taqman Gene expression Assays for specific genes and a spectrofluorometric thermal cycler (7300 Real-time PCR System, all from Applied Biosystems, Foster City, California). To determine the expression levels of phenotypic markers of disc regeneration and repair (collagen type I, collagen type II, and matrix metalloproteinase 13), we used Taqman Gene Expression Assays: Oc03396113_m1, Oc03396134_m1, Oc03396896_m1 (Applied Biosystems). To standardize mRNA levels, we amplified the rabbit 18S ribosomal RNA (a structural RNA that is a component of eukaryotic ribosomes) using the available Taqman Gene Expression Assay (Applied Biosystems).

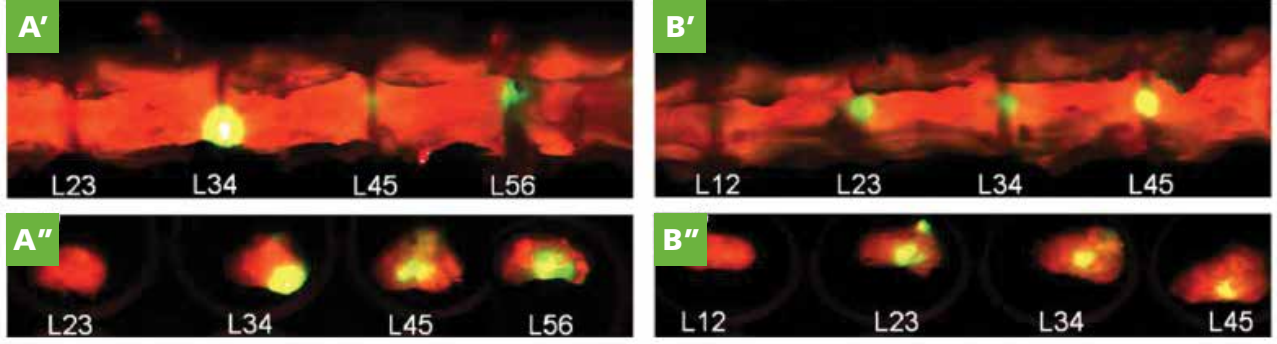
STATISTICAL METHODS

We analyzed significant differences by using ANOVA and Fisher PLSD as a post hoc test (biochemical analysis). We expressed the data as the mean +/- standard error. Using the Kruskal-Wallis test and Mann-Whitney test we analyzed nonparametric data (MRI and histological grading) for the effect of treatment. The Spearman rank correlation test assessed changes in disc height index. Using the student *t* test, we compared the MRI indexes between the treatment groups. We considered differences to be significant in all analyses when the *P* value was equal to or below .05.

RESULTS

CELL TRACKING NHDFS IN VIVO

To determine if the cells transplanted into the degenerating IVD remained in the IVD, we treated the rabbit degeneration model with nHDFs labeled with infrared dye. We transplanted 8 µL of nHDFs (1.0×10^7 cells per mL) into the degenerating rabbit IVDs. At 2 and 8 weeks after transplantation, we euthanized the rabbits and used an infrared scanner to examine the isolated spines and individual IVDs. As shown in Figure 2, the rabbit spine and disc contours appear in red, using the 700-nm wavelength channel. We detected the injected nHDF cells using the 800-nm wavelength channel: they are represented in green. When the 800-nm



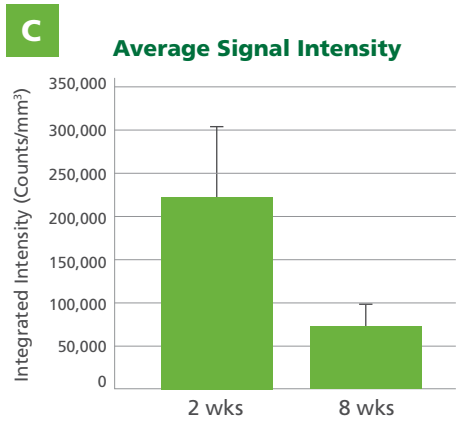


FIGURE 2. Detection of neonatal human dermal fibroblasts (nHDFs) after transplantation into the degenerating intervertebral rabbit disc. Four weeks after disc injury, we injected the degenerated rabbit discs with nHDFs labeled with infrared dye and imaged them with an infrared scanner at 2 and 8 weeks posttreament. Coronal images of the spine and transverse images of the individual discs at 2 weeks postinjection **(A′ AND A″)** and 8 weeks postinjection **(B′ AND B″)**. **C,** Average integrated signal intensity of the infrared fluorescence in the 800-nm wavelength of the IVDs. Error bars represent the standard error of the mean.

wavelength signal from the nHDF cells overlapped with the 700-nm wavelength signal from the spine, the resulting signals appear in yellow in the scan. At both 2 weeks (Figures 2A' and 2A") and 8 weeks (Figures 2B' and 2B") after transplantation, we detected cells labeled with infrared dye in the spines and individual IVDs. The average intensity of the IVDs was 226,555 counts at 2 weeks postinjection (n = 2 rabbits) and 75,239 counts at 8 weeks postinjection (n = 3 rabbits). Because variation in cell fluorescence may differ between sets of cells during the time of labeling, we injected these 5 rabbits on the same day with the same set of cells. We excluded from this analysis the rabbits that we injected on a different day. Although there was a 3-fold decrease in signal intensity from 2 to 8 weeks, these data suggest that some of the cells injected into the IVD remained there for up to 8 weeks.

MAGNETIC RESONANCE IMAGING ASSESSMENT 8 WEEKS POSTTREATMENT

We used T2-weighted MRIs to evaluate the integrity of the intervertebral discs by detecting the water content in the NPs. At 8 weeks posttreatment, rabbit spines underwent MR imaging. We injured and treated 4 levels (L2/3, L3/4, L4/5, and L5/6), while leaving the other levels (L1/2 and L6/7) uninjured and intact. Using the Modified Pfirrmann Grading Scale, 5 independent orthopedic researchers determined MRI grading. The NPs in the uninjured intact discs showed lower average MRI grades and better MRI signal intensities than those in the injured nHDF-treated or injured saline-treated discs (Figure 3A). The average MRI grade for the injured nHDF-treated discs was 3.5, while the average for the injured saline-treated discs was 2.8. The nHDF-treated discs had higher MRI grades and lower signal intensities than the saline-treated IVDs, but this difference was not significant ($P = .23$).

Because MRI grades can be subjective, we calculated MRI indexes from values for highest signal intensity, average signal intensity, and high signal intensity area (Figures 3C-3F). We normalized the MRI index (defined as the NP area ×NP average signal intensity) of each treated disc to the intact disc in the same animal. The MRI indexes showed that saline-treated IVDs had higher signal intensities than nHDF-treated IVDs, but this difference was not significant ($P = .44$).

RADIOGRAPHIC ASSESSMENT AND DISC HEIGHT INDEX 8 WEEKS POSTTREATMENT

We performed disc injury in both the nHDF- and saline-treated groups. Four weeks after injury, a narrowing of the disc height occurred due to injury-induced degeneration. Representative lateral radiograms are shown in Figure 4A. Disc height indexes at 4 weeks after injury had decreased by about 30% compared to the initial disc height index taken before surgery. Eight weeks after saline treatment, there was a small increase in the disc height index, but this increase was not significant. In the discs that were treated with nHDFs, there was about a 10% restoration in the disc height index. This difference was significant from the 4-week time point ($P \leq .05$) (Figure 4B).

GENE EXPRESSION ANALYSIS 2 AND 8 WEEKS POSTTREATMENT

We analyzed RNA from rabbit IVDs treated with nHDFs and saline and uninjured intact controls. At 2 and 8 weeks after treatment, gene expression of phenotypic markers of disc regeneration and repair (collagen type II, collagen type I, and matrix metallopeptidase 13) increased when compared to the uninjured, intact controls (Figures 5A-5C). We calculated the ratios of collagen II and collagen I gene expression in the treated samples and normalized them to the ratios of the saline treated samples. At 2 weeks posttreatment, the nHDF (0.84) and saline (1.00) treatment groups showed similar ratios of collagen II/collagen I gene expression (Figure 5D). At 8 weeks posttreatment, the ratio was higher in the IVDs treated with nHDFs (2.71) compared to those that were treated with saline (1.00).

BIOCHEMICAL ANALYSIS 8 WEEKS POSTTREATMENT

We calculated the average proteoglycan/DNA (μg/μg) ratio of the AF tissues for intact discs (441.84), injured nHDF-treated discs (386.11), and injured saline-treated

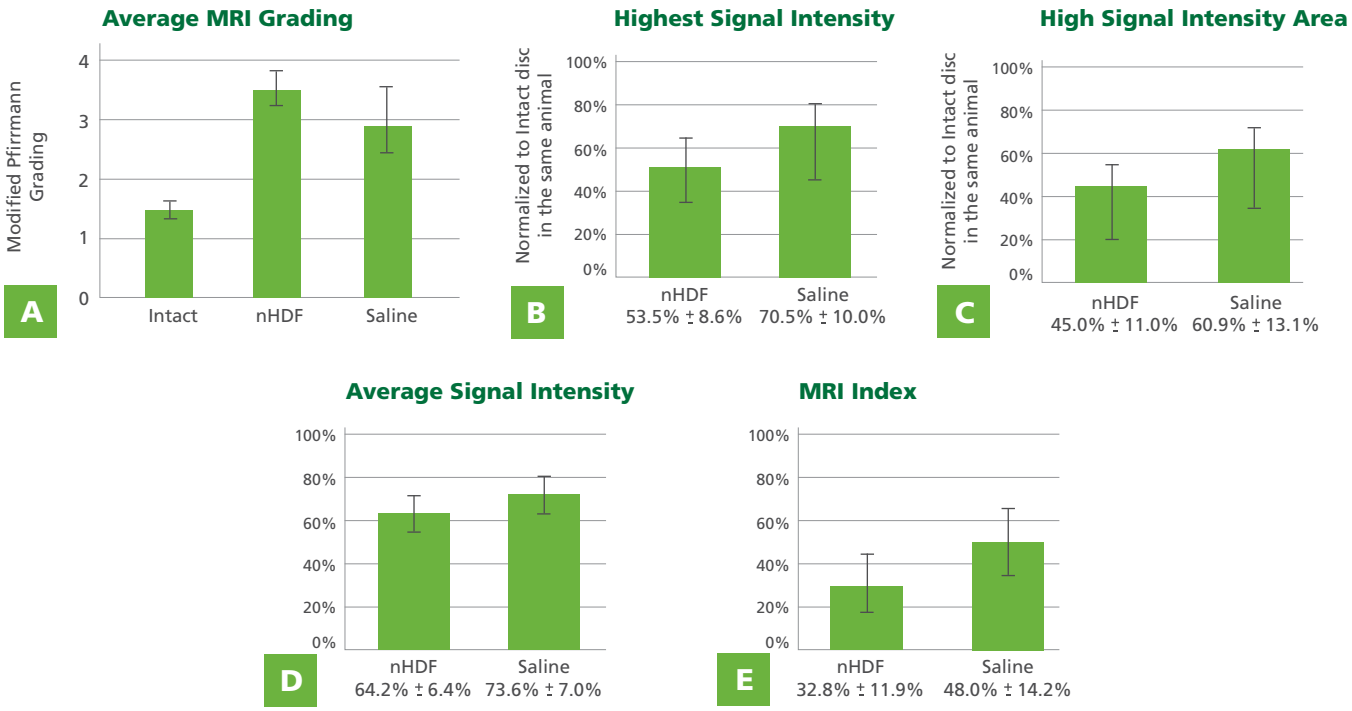


FIGURE 3. Changes in magnetic resonance imaging (MRI) grades and indexes 8 weeks after treatment. Four weeks after disc injury, the degenerated rabbit discs were treated with either neonatal human dermal fibroblast (nHDF) or saline. We obtained MRIs 8 weeks after treatment, graded them, and used them to calculate MRI indexes. **A,** Average MRI grading using the Modified Pfirrmann Grading Scale. **B-E,** Calculations of highest signal intensity, and high signal intensity area, and average signal intensity determine the MRI index (nucleus pulposus (NP) average signal intensity ×NP area).

discs (423.74) (Table 2). For NP tissues, the average proteoglycan/DNA ratio of the intact discs was 1031.77, the ratio for injured nHDF-treated discs was 769.17, and the ratio for injured saline-treated discs was 741.61 (Table 2). While disc injury did not change the proteoglycan content in the AF tissues much, it caused about a 30% decrease in proteoglycan content in the NP tissue compared to uninjured intact controls. After 8 weeks of treatment, there was a slight recovery of proteoglycan contents in the nHDF-treated NP tissues compared to the saline-treated NP tissues. This difference was promising but not significant due to the small sample size.

HISTOLOGICAL GRADING 8 WEEKS POSTTREATMENT

Eight weeks after treatment, we fixed, sectioned, and stained the IVDs with Alcian blue and haematoxylin and eosin (H&E) and evaluated them. Due to injury, the morphological scores of the all categories were worse in the injured nHDF- and saline-treated groups (total morphological scores of 7.0 and 6.8, respectively) than the uninjured intact group (total morphological score of 4.0). The proliferation scores of the NP and anterior AF suggest that there was slightly more proliferation in the injured nHDF- and saline-treated samples (range, 1.8-2.2) than the uninjured intact samples (1.0). As expected, the proliferation scores in the uninjured areas, the posterior AF and the endplate, were normal (1.0) in all 3 groups.

DISCUSSION

Cell therapy is a promising approach to help regenerate the intervertebral disc. In this study, we tested nHDF as a cell therapy for disc degeneration in the rabbit model. Although this was a small study with a short time frame, there were still some encouraging results in our disc height analysis, cell-tracking and gene-expression studies. Because we collected the samples at 8 weeks posttreatment, there was not a significant difference in proteoglycan content in the nHDF-treated IVDs when compared to the saline-treated IVDs. In the cell therapy studies by Sakai et al, which used mesenchymal stem cells and the rabbit disc degeneration model,[11] a significant increase in proteoglycan content was seen at 48 weeks after mesenchymal stem cell transplantation. In the cell therapy study by Hiyama et al, which used mesenchymal stem cells and the canine disc degeneration model,[22] a significant difference in proteoglycan content was seen at 12 weeks after mesenchymal stem cell transplantation. These studies suggest that increasing the time frame after transplantation may yield a better biochemical outcome measure and give us a better indication of the therapeutic effects of nHDF cell therapy.

When we labeled the nHDFs with infrared dye in our cell tracking studies, the infrared signal was detectable in the IVD at both 2 and 8 weeks postinjection. There was a 3-fold decrease in signal intensities when comparing the discs at 2 and 8 weeks after injection. It is unclear if this decrease is due to cell death or to a natural decrease in signal intensity. Cell death may occur if the transplanted cells cannot survive in the conditions of the new environment. Singh et al[14] generated an in vitro environment to mimic the oscillating pressures in the disc

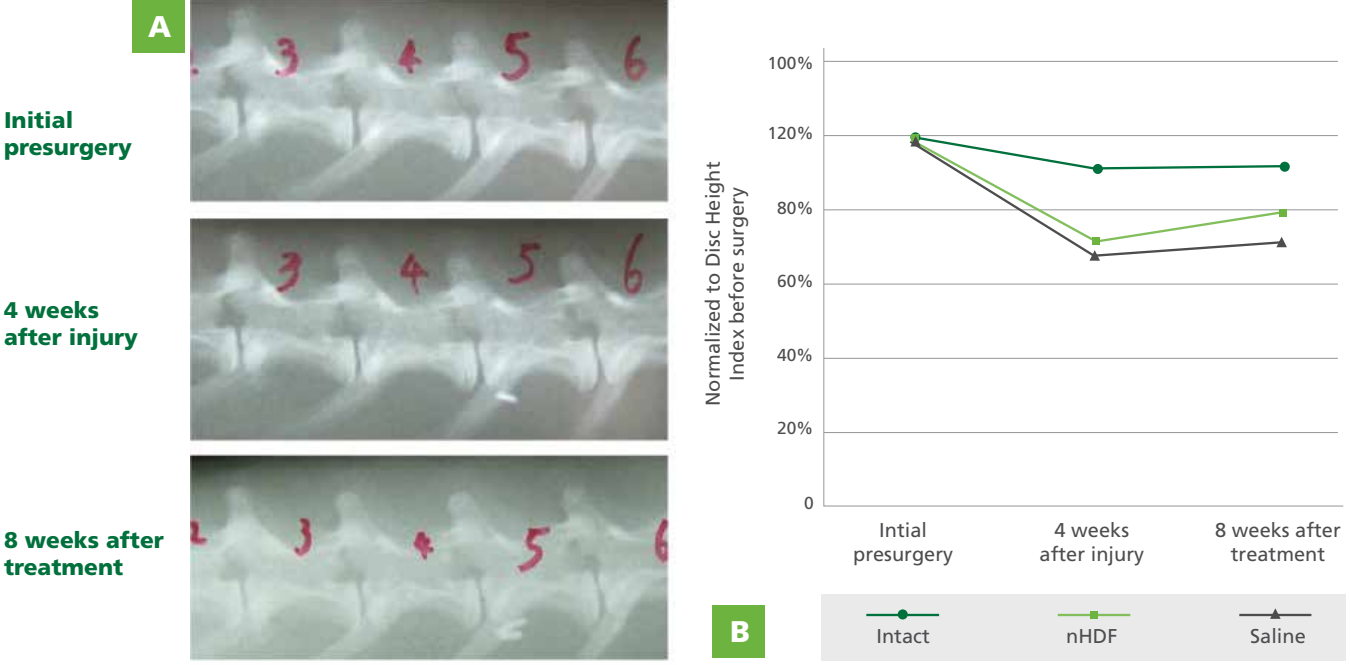


FIGURE 4. Changes in disc height indexes after injury and treatment. **A,** Representative lateral radiographs of a lumbar spine of a rabbit before disc injury surgery (initial presurgery), 4 weeks after injury, and 8 weeks after neonatal human dermal fibroblast (nHDF) or saline treatment. **B,** Average disc height indexes of intact discs, injured nHDF-treated discs, and injured saline-treated discs, calculated at each of these time points and normalized to the presurgery disc height index.

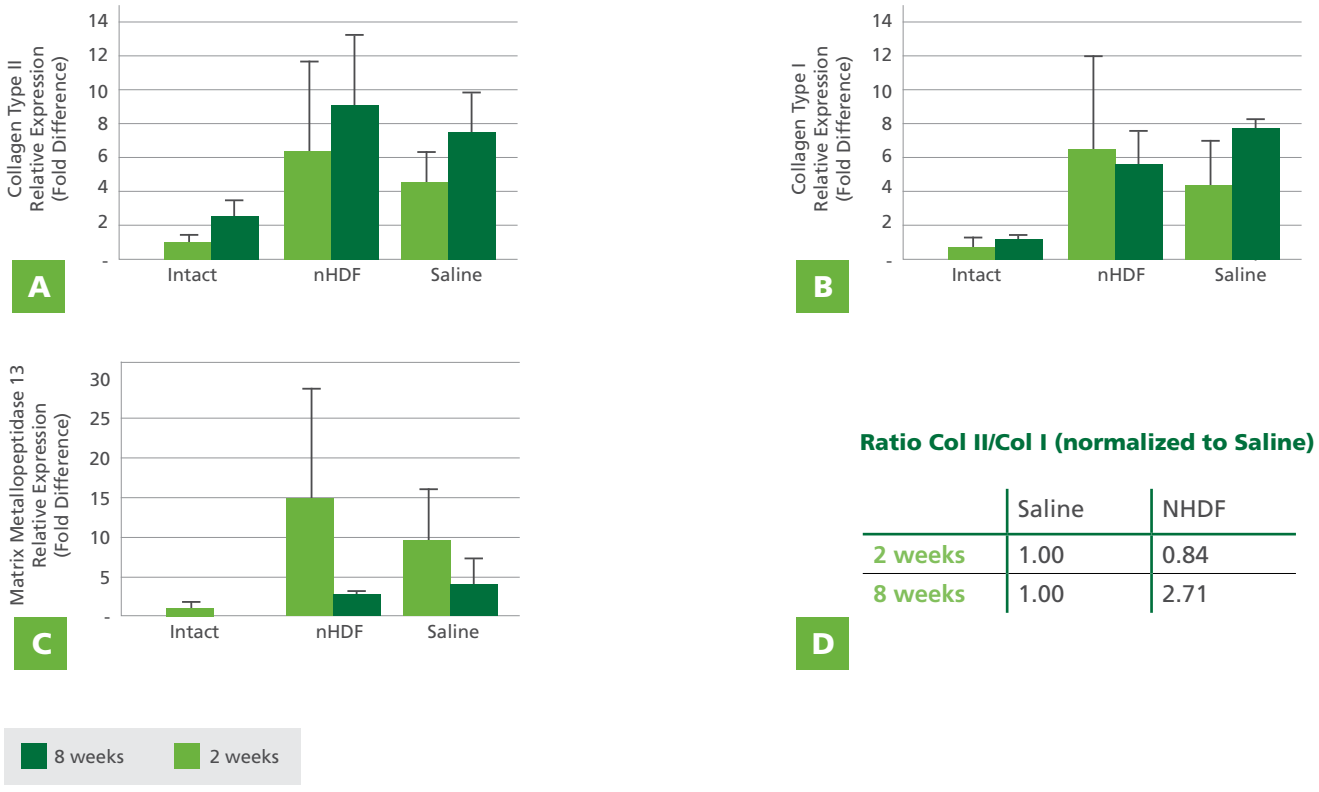


FIGURE 5. Changes in gene expression 2 and 8 weeks after treatment. At 2 and 8 weeks after treatment, we isolated ribonucleic acid (RNA) from the rabbit discs and analyzed the expression of phenotypic marker genes. **A-C,** Gene expression of collagen type II (Col II), collagen type I (Col I), and matrix metallopeptidase 13 in uninjured intact discs, injured discs treated with neonatal human dermal fibroblast (nHDF), and injured discs treated with saline. **D,** Ratio of Col II/Col I gene expression calculated and normalized to the saline-treated discs.

TABLE 2. Changes in Proteoglycan and DNA contents

	Group	Proteoglycan (µg)/wet weight tissue (mg)	DNA (µg)/wet weight tissue (mg)	Proteoglycan (µg)/DNA (µg)	*P* Value (ANOVA)
NP	nHDF (n = 11)	101.81 ± 27.82	0.14 ± 0.02	769.17 ± 277.2	
	Saline (n = 9)	97.84 ± 25.91	0.14 ± 0.04	741.61 ± 308.8	.12
	Intact (n = 14)	113.98 ± 32.54	0.12 ± 0.03	1031.77 ± 462.6	
AF	NHDF (n = 11)	75.08 ± 22.41	0.17 ± 0.03	386.11 ± 174.4	
	Saline (n = 9)	71.29 ± 16.16	0.18 ± 0.04	423.74 ± 124.9	*.71*
	Intact (n = 14)	80.03 ± 22.98	0.18 ± 0.03	441.84 ± 180.1	

Abbreviations: NP, nucleus pulposus; AF, annulus fibrosus; nHDF, neonatal human dermal fibroblast; DNA, deoxyribonucleic acid; ANOVA, analysis of variance

using a custom-made hydrostatic pressure bioreactor. In this harsh environment, nHDFs showed a 20% to 30% decrease in viability after 3 weeks. On the other hand, the nHDF cells that survived this environment expressed more collagen type II than cells grown in a static environment.[14] In the studies by Serigano et al, the researchers determined cell death after transplanting different concentrations of mesenchymal stem cells into the canine degenerative disc. Transplanting 1.0×10^7 mesenchymal stem cells in a canine degenerative disc resulted in more cell death than transplanting 1.0×10^6 cells.[23] Future studies of injecting different doses of nHDFs in rabbit IVDs in vivo would determine the optimal dosage of nHDFs to transplant. It would be also interesting to determine if the transplanted cells have transdifferentiated into chondrocyte-like cells.

In the MRI analysis, the average signal intensity of the nHDF-treated IVDs was lower than the saline-treated IVDs, although the difference between the 2 intensities was not significant. The disc height analysis showed that the disc height in the nHDF-treated IVDs did significantly increase compared to the saline controls. One explanation for the MRI signal reduction and increase in disc height may be the formation of fibrocartilage. Fibrocartilage contains less water than hyaline cartilage, resulting in a lower MRI signal intensity.[24] The ratio of proteoglycan and collagen is greater in NP tissue and lesser in both hyaline cartilage and AF tissue.[25] Depending on the water collagen content, fibrocartilage may have a similar or lower MRI signal intensity than AF tissue. It is unclear if the formation of fibrocartilage in the disc is beneficial. Newly formed fibrocartilage may provide a structure for regenerating tissue and prevent the disc from further degeneration. Increasing the IVD height alone may reduce pressure on sensitive tissues, which would in turn reduce some low back pain.

In our gene expression studies, the data showed that injury to the disc may cause an upregulation of collagen type II, collagen type I, and matrix metallopeptidase 13 (Figures 5A-5C). When treating with nHDFs, the resident cells and nHDFs themselves may receive signals to generate fibrocartilage to fill in the injured areas. Fibrocartilage contains a mixture of fibrous and cartilaginous tissues. The ratio of collagen II to collagen I in the nHDF-treated IVDs was close to the ratio seen in the saline-treated IVDs at the 2-week time point (Figure 5D). At the 8-week time point, ratio of collagen II to collagen I was higher in the nHDF-treated samples than in the saline-treated samples, suggesting that more cartilage and less fibrous tissue was beginning to form in these IVDs. In this study, we did not determine if the nHDFs themselves had differentiated into disc-like cells after injection into the IVD or if the nHDFs sent growth signals to the resident disc cells to increase collagen type II expression. Using immunohistochemical double staining, Sakai et al determined that when mesenchymal stem cells that expressed green fluorescent protein (GFP) were transplanted into rabbit IVD, they differentiated into cells expressing NP phenotypic markers such as keratin sulfate, chondroitin-4-sulfate, chondroitin-6-sulfate, and collagen type II.[11] This study suggests transplanted cells can differentiate to express collagen type II after transplantation in the IVD environment. More detailed studies of tracking the nHDFs with a retrovirus expressing GFP and double staining for GFP and NP phenotypic markers will help elucidate if nHDFs can transdifferentiate into chondrocyte-like cells after injection into the IVD.

In conclusion, these studies have shown that nHDFs injected into the rabbit degenerating disc can remain in the disc for at least 8 weeks and help increase both disc height and expression of collagen type II. Human dermal fibroblasts can easily be obtained from patients themselves or from human foreskin donors. Since the IVD is relatively immunoprivileged, donor fibroblasts should not elicit an immune response. More studies will be needed to determine minimum effective dosage of nHDFs and biomechanical outcome measures after nHDF therapy. Also, a longer time frame may be needed to see more significant results in cell therapy studies when compared to growth factor studies. Cells may need a period of time to adjust to the new environment and differentiate before showing therapeutic results, while growth factors stimulate resident cells to get a direct therapeutic response. These data would help design future studies of cell therapies to restore the biological function and reduce symptoms of degenerative discs. ■

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

"GLENOID RECONSTRUCTION WITH FRESH DISTAL TIBIA ALLOGRAFT OFFERS AN ALTERNATIVE, VIABLE SURGICAL OPTION THAT POTENTIALLY RESTORES BOTH MECHANICS AND BIOLOGY FOR WHAT HISTORICALLY HAS BEEN AN EXTREMELY DIFFICULT PROBLEM."

A Biomechanical Perspective on Distal Tibia Osteochondral Allograft for Reconstruction of Glenoid Bone Defects

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Bone defects in the setting of glenohumeral instability can be extremely difficult to treat. An intact glenoid articular arc is crucial for maintenance of the concavity-compression mechanism and for a stable articulation with the humeral head. And loss of articular congruency can prove detrimental in cases of both primary and recurrent shoulder instability.[1,2] Despite recent advances in both surgical techniques and implant design, recurrent instability remains a concern, with rates as high 30% following both open and arthroscopic approaches. Many risk factors have been identified as potential etiologies of an unsuccessful repair, and both glenoid and humeral head bone loss have been implicated. The treatment of large glenoid bone defects in the setting of recurrent instability is difficult, mainly because of the nonanatomic, and thus incongruous, joint resulting from most bony augmentation procedures.

In cases of anterior glenoid bony deficiency, defects that represent less than 15% of the glenoid width can be treated with soft-tissue stabilization alone, while defects great than 30% often require autograft or allograft glenoid augmentation. Defects between 15% and 30% are in an indeterminate zone. And pathology- and patient-specific factors drive the decision making for surgical repair. On the other hand, posterior glenohumeral instability is rare, accounting for approximately 5% of all shoulder dislocations.[1-5] The most common underlying pathology is posterior capsule and/or labral damage. Posterior glenoid bone defects are uncommon, especially when compared to anterior glenoid bone defects, and therefore, there is no algorithm for treating posterior glenoid bony deficiency.[2,6,7]

Bony reconstruction procedures for anterior glenoid bone loss include the iliac crest bone-graft (ICBG) procedures and coracoid transfer procedures (Latarjet-Bristow). While good to excellent results with regard to maintenance of shoulder stability have been reported following the Latarjet procedure, the early development of symptomatic glenohumeral arthritis remains a concern. Similarly, the most often reported reconstructive technique for posterior glenoid bone loss is the placement of iliac crest autograft as a posterior bone block. This procedure has disappointing long-term clinical results, with high rates of patient dissatisfaction, inability to return to desired level of activity, recurrence of instability, and glenohumeral arthritis. One possible explanation is that a nonanatomic repair of the glenoid arc associated with a lack of an articular surface may, at least in part, contribute to the high incidence of arthritis following these popular bone-grafting procedures.

Studies have described the use of fresh osteochondral distal tibia allograft (DTA) as an alternative for the treatment of large glenoid bone defects in the setting of anterior, and more recently posterior, glenohumeral instability. Initial laboratory work has demonstrated a nearly identical radius of curvature between the distal tibia and the glenoid, even among nonmatched cadaveric specimens (Figure 1). Thus, similar to the glenoid surface, the distal tibia articular surface has excellent conformity to the humeral head throughout a full arc of motion. In addition to having a radius of curvature that allows for unimpeded motion due to its congruency with the humeral head, fresh distal tibia allograft contains dense, weight-bearing corticocancellous bone, making it ideal for screw fixation, and, further, contains a

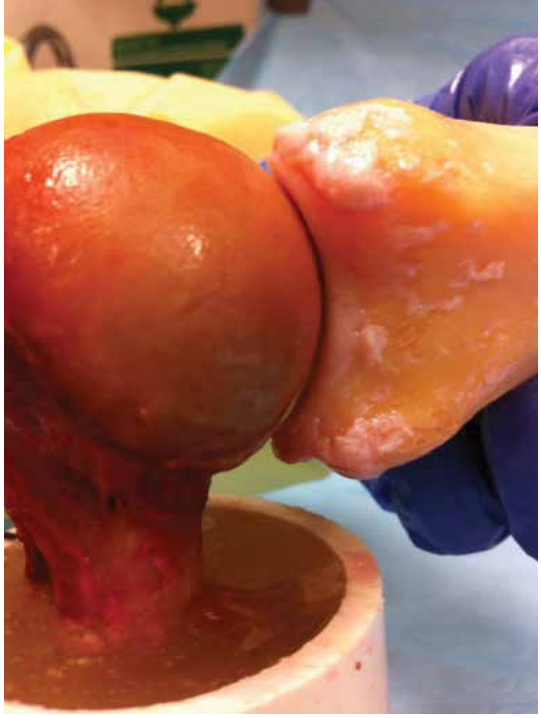

FIGURE 1. Near-identical radius of curvature between distal tibia and humeral head.

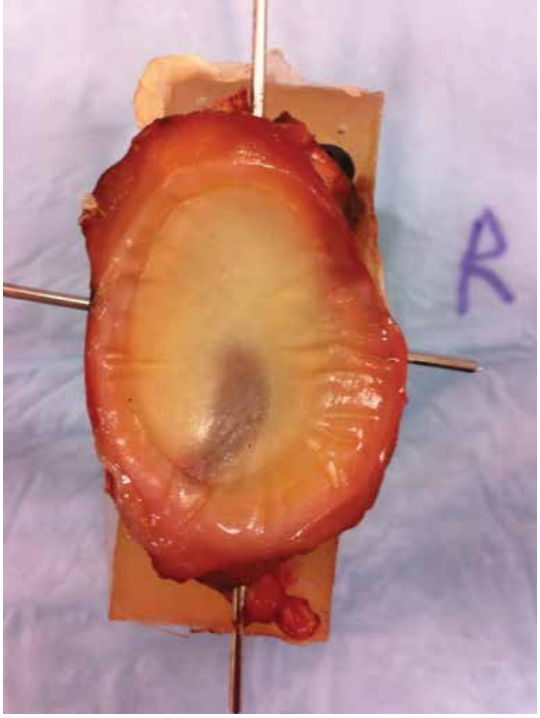

FIGURE 2. Intact glenoid specimen prior to creation of the 30% anterior–inferior glenoid bone defect (right shoulder).

robust cartilaginous surface that allows for an anatomic, osteoarticular glenoid surface reconstruction. Graft fixation, independent of the graft source, remains a critical aspect of the surgical procedure and is provided at a minimum by 3.5-mm bicortical fully threaded interference screws using lag technique.

Our laboratory has recently evaluated and published on the biomechanical properties of various bone-grafting procedures for both anterior and posterior glenoid bone loss, including the novel technique of distal tibia allograft reconstruction.[8-10] This manuscript discusses the biomechanical methodology used in these studies as well as the clinical relevance of the biomechanical findings. We anticipate that the findings from this area of research will facilitate improved understanding of the surgical options available for shoulder instability associated with large glenoid bone defects.

METHODS

This laboratory has conducted multiple studies evaluating the biomechanical properties of glenoid bone-grafting techniques.[8-10] The initial study of Ghodadra et al[8] evaluated both the Latarjet procedure as well as iliac crest bone-grafting and provided a model for future work. Bhatia and colleagues[9] performed a subsequent study, comparing reconstruction of anterior glenoid bone defects with DTA versus Latarjet. Our third study[10] evaluated the biomechanical properties of posterior glenoid bony reconstruction with DTA compared to iliac crest bone graft. It has been presented at multiple orthopedic meetings and is due for publication. The methodology for each of the studies was similar, based on the original work performed by Ghodadra et al.[8] All studies were classified as exempt from review by our university's Institutional Review Board.

For each study, we dissected fresh-frozen human cadaveric shoulders free of all soft tissue, exposing the glenohumeral joint. We then disarticulated the proximal humerus from the glenoid. We potted the glenoid specimens (Figure 2) and their corresponding humeral shafts in polyvinyl chloride (PVC) piping using dental acrylic (Isocryl, Lang Dental, Wheeling, Illinois). To conduct the biomechanical testing, we mounted the humeral shaft to the crosshead of a Materials Testing System (MTS) materials testing machine (Insight 5, MTS systems, Eden Prairie, Minnesota). We prepared the glenoid, based upon its designated testing group (as indicated in the following methodologies), and loaded each into a custom-made jig on the baseplate of the MTS machine. For each specimen, we precalibrated a dynamic, pressure-sensitive pad with a thickness of 0.1 mm (sensor model 5051, TekScan, Boston, Massachusetts) with a 56- × 56-mm matrix and a density of 62 sensels/cm^2 with the MTS machine and placed it between the humeral head and glenoid articular surfaces.

In the index study,[8] the research team tested specimens in static positions of humeral abduction (30°, 60°, and 60° abduction with 90° of external rotation ([ABER]). We evaluated biomechanical properties for multiple specimen conditions, including 1) the intact glenoid; 2) the glenoid with an anterior bone defect involving 15% or 30% of the glenoid surface area; 3) a 30% glenoid defect treated with a Latarjet or iliac crest bone graft placed 2 mm proud, placed flush, or recessed 2 mm relative to the level of the glenoid; and 4) a Latarjet bone block placed flush and oriented with either the lateral (Latarjet-LAT) or the inferior (Latarjet-INF) surface of the coracoid as the glenoid face. In the subsequent study,[9] the team tested a new set of specimens in the same 3 static positions of humeral abductions, with the testing conditions consisting of 1) the intact glenoid; 2) the glenoid with a 30% anterior bone defect; and 3) the glenoid after reconstruction with a DTA (Figures 3-4) or a Latarjet bone block. The most recent study[10] analyzed the

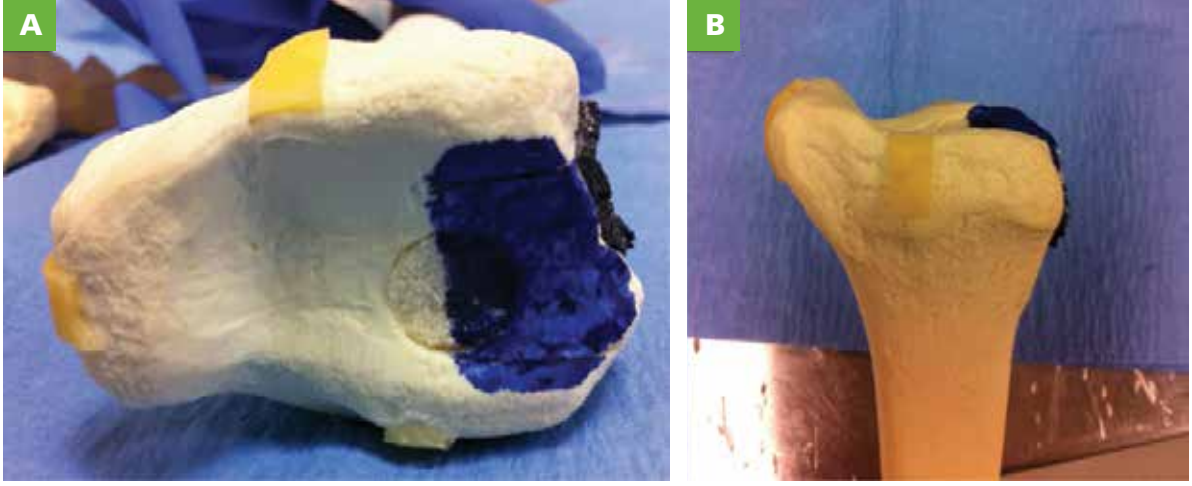

FIGURE 3. Sawbone model of the anatomy of the distal tibia; the marked portion represents the lateral one-third of the tibia used to create the DTA.

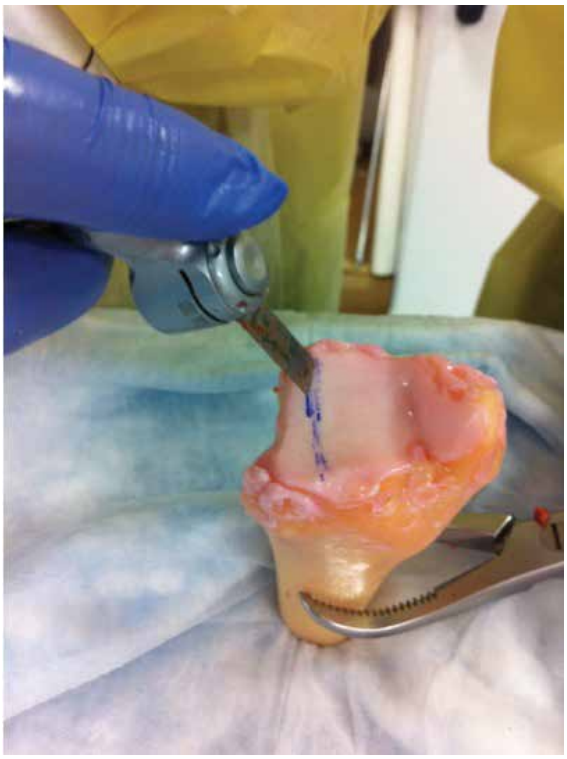

FIGURE 4. Creation of DTA from lateral one-third of the tibial graft using a sagittal saw.

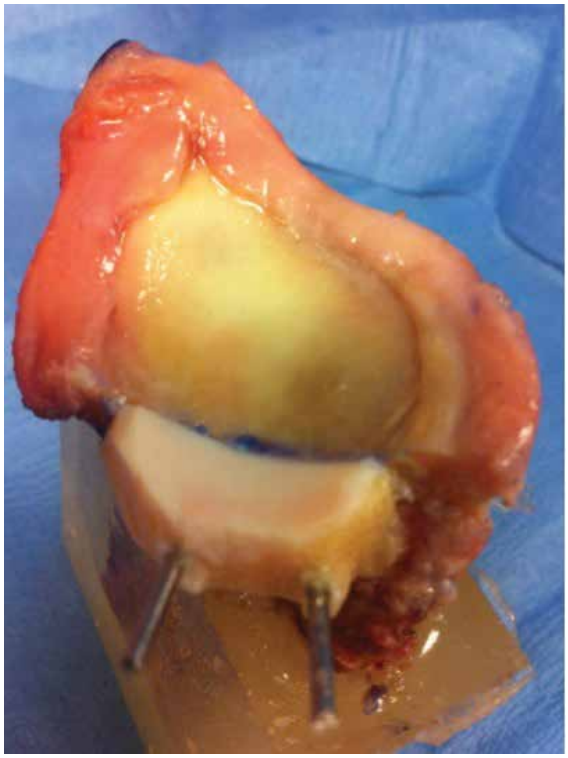

FIGURE 5. Posterior glenoid osteoarticular reconstruction of 20% posterior–inferior glenoid bone defect with DTA (right shoulder).

reconstruction of the posterior glenoid bone defect. As opposed to the first 2 studies, this study tested specimens in 3 static positions relevant to posterior shoulder instability: 1) neutral (scapular plane), 2) 60° humeral abduction, and 3) 90° of flexion with 45° internal rotation (FIR). Similar to the second study, testing conditions consisted of 1) intact glenoid; 2) 20% posterior–inferior glenoid surface area defect; 3) 20% defect reconstructed with flush ICBG; and 4) 20% defect reconstructed with fresh DTA (Figure 5).

For each specimen in all studies, the authors applied a compressive load of 440N across the glenohumeral joint,[8-10] simulating in vivo glenohumeral loading conditions throughout the course of normal shoulder motion during activities of daily living.[10] Following MTS testing of the intact glenoid, researchers performed osteotomies simulating the desired amount of glenoid bone loss (as determined by the specimen's group), using established methodology.[3,9,11] We also performed appropriate reconstruction procedures, including iliac crest grafting, Latarjet reconstruction (coracoid transfer), and DTA reconstruction. We then conducted MTS testing for each of the subsequent testing groups, in each of the clinically relevant arm positions (Figure 6). The specific methodology for each of these studies is described in detail in the respective publications.

We collected both raw and normalized data from I-scan pressure measurement software (TekScan) and utilized it for statistical analysis. The outcomes

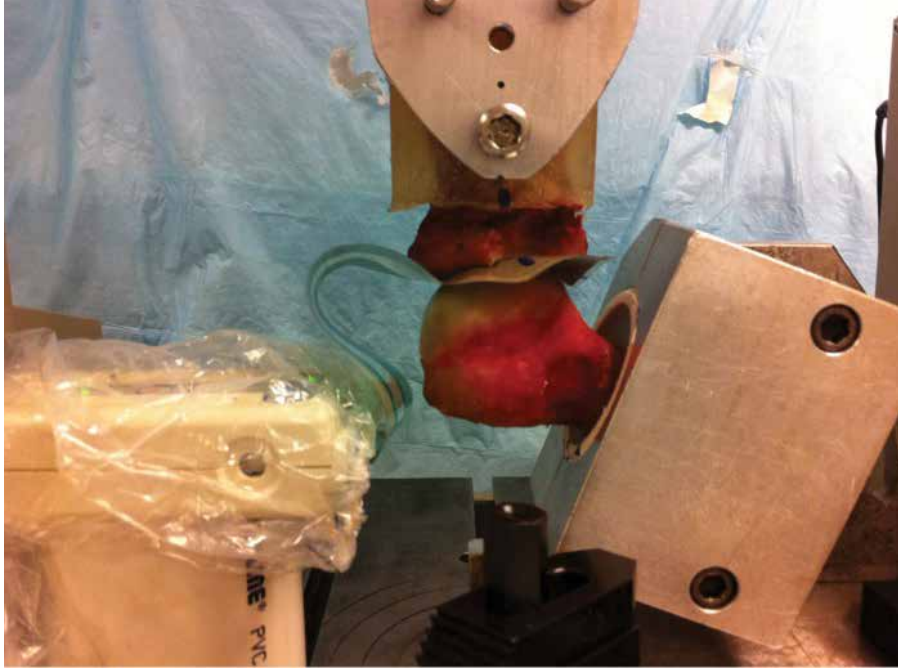

FIGURE 6. MTS setup with TekScan sensor in place of left shoulder (30° abduction) with 20% posterior–inferior glenoid bone defect.

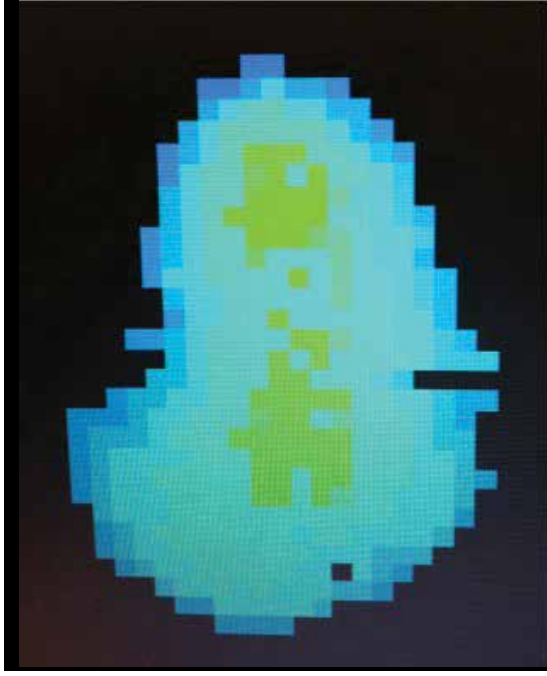

FIGURE 7. Representatitive TekScan pressure map of the intact glenoid with the arm in neutral. Higher pressures are signified by green/light blue and lower pressures by dark blue (left shoulder).

we assessed and recorded included glenohumeral contact pressure (CP) (kg/cm^2), contact area (CA) (cm^2), and joint peak force (PF) (N). We compared the data across groups, using a repeated measures 1-way analysis of variance (ANOVA) with the Tukey test when indicated. For normalization calculations, we normalized specimen data with respect to each specimen's intact state in order to account for anatomic specimen variability.

RESULTS

The respective publications describe in detail the results of all 3 studies.[8-10] In brief, the initial work by Ghodadra and colleagues[8] established a model for evaluating the biomechanical properties, including CP, CA, and PF, of both an intact glenoid (Figure 7) and a glenoid with an anterior bone defect involving up to 30% of the glenoid surface area. The authors found that when they placed the graft flush to the glenoid surface, mean peak contact pressure was restored to 116% of normal with iliac crest, 120% of normal with Latarjet-INF, and 137% of normal with Latarjet-LAT, all of which were significantly greater ($P < .05$) when compared with the peak pressure of the 30% defect model. When we placed the grafts in a proud position, mean peak contact pressures increased to 250% of normal in the anteroinferior quadrant, with a concomitant increase in pressure to 200% of normal in the posterosuperior quadrant.

Further, when comparing between the grafts, the authors reported significantly higher ($P < .05$) mean peak glenoid contact pressures with the Latarjet-LAT graft compared to the iliac crest and the Latarjet-INF graft at both 60° of abduction and at ABER. Finally, the authors noted that augmentation with the Latarjet-INF produced complete restoration of the glenoid articular contact surface (from the 30% defect state), while reconstruction with the Latarjet-LAT resulted in restoration of the surface to a 5% defect state.

Bhatia and colleagues[9] expanded on the work performed by Ghodadra[8] in an effort to compare the contact mechanics of the Latarjet procedure to those of the DTA reconstruction. Using a 30% anterior glenoid defect model, the authors reported that reconstruction with DTA resulted in significantly higher contact areas compared to Latarjet bone blocks, both in 60° of abduction and in the ABER position. In addition, we found that DTA produced significantly lower peak forces than Latarjet reconstruction in the ABER technique ($P < .05$). The authors noted that in all 3 positions, glenoid reconstruction with both DTA and Latarjet resulted in higher contact areas, lower contact pressures, and lower peak forces when compared with the 30% defect model; however, only the differences between the Latarjet reconstruction and the defect model in the ABER position were not statistically significant.

The final study[10] was the first to evaluate these same outcomes for defects and reconstructions in the posterior glenoid. The authors effectively established a model for evaluating the contact mechanics of both the intact glenoid and glenoids with a 20% posterior–inferior surface area defect. Overall, the authors found that Tekscan mapping of glenohumeral contact areas and mean contact pressures demonstrated equal or higher pressures and smaller contact areas in the defect group compared to intact. Specifically, glenoid reconstruction with DTA resulted in significantly higher CA compared to the 20% defect model at 60° ($P < .01$) and in FIR ($P < .01$). The intact state exhibited significantly higher CA than the defect ($P < .01$) in all positions ($P < .01$), and significantly higher CA than the ICBG at 60° ($P < .05$) and in FIR ($P < .05$).

DISCUSSION

The findings and interpretation from all 3 studies are described in detail in the respective publications.[8-10] The principal findings from the 3 studies are as follows: 1) glenohumeral contact pressure is optimally restored with a flush bone-graft placement as compared to proud graft placement; 2) when employing the Latarjet procedure, the inferior aspect of the coracoid better restores glenoid contact mechanics as compared to the lateral aspect of the coracoid; 3) reconstruction of

anterior glenoid bone defects with a DTA may allow for improved joint congruity and lower peak forces within the glenohumeral joint than Latarjet reconstruction at 60° of abduction and the ABER position; and 4) reconstruction of posterior glenoid bone defects with DTA conferred similar contact mechanics compared to reconstruction with iliac crest bone graft.

Studies have implicated both glenoid and humeral head bone loss as potential etiologies of surgical failure in cases of recurrent anterior glenohumeral instability.[1,2] The importance of a congruent glenoid articular arc is not only for maintaining stability, but also for maintaining the overall integrity of the entire glenoid chondral surface.[2,12] Specifically, Greis et al[13] reported an increase in glenoid anteroinferior contact pressures of 300%-400% with the creation of a 30% glenoid bone defect. Further, the work in the index study by Ghodadra et al[8] noted that bone grafts placed proud, and therefore not restoring a congruent glenoid arc, increase anteroinferior peak pressure and also shift the contact pressure to the posterosuperior quadrant. Thus, it is crucial to both recognize and appropriately address the osseous defects often found in cases of glenohumeral instability.

Anterior glenohumeral instability is by far the most common form of shoulder instability, while cases of posterior glenohumeral instability are rare, accounting for up to 5% of all cases of shoulder instability.[2,6,7] Given the overall low occurrence of posterior instability in general, the true incidence of bone deficiency in the setting of recurrent posterior instability is unknown.[6,7,14] Shoulder joint stability is produced by 3 major mechanisms including 1) concavity-compression mechanism of the joint, 2) coordinated contraction of the rotator cuff to permit smooth range of motion of the humeral head onto the glenoid surface, and 3) contribution of the glenohumeral ligaments via their direct attachment onto the rotator cuff.[15] When considering stability or lack thereof, the most important anatomy includes the dynamic and static stabilizers of the shoulder joint. The static stabilizers include the bony anatomy, rotator interval, and capsulolabral structures, while the dynamic stabilizers include the rotator cuff and scapular rotator musculature.

The bony anatomy of the shoulder joint, specifically the glenoid, plays a major role in anterior shoulder stability, and injury leads to high rates of recurrent instability. Due to the relatively small size of the glenoid compared to the humeral head, any loss of bone, such as a glenoid rim fracture, can compromise stability by decreasing the surface area for glenohumeral articulation.[16] Several clinical studies have shown that bone loss of either the humeral head[17] or glenoid surface[1,3,18,19] is the most common cause of failed arthroscopic stabilization procedures and that recurrence of glenohumeral instability increases when there is at least a 20% glenoid bone loss.[5,20]

It can be very difficult to treat patients with glenohumeral instability associated with significant anterior and posterior glenoid bone defects. Obtaining a proper history, performing a thorough physical examination, and obtaining imaging studies including magnetic resonance imaging (MRI) and computed tomography (CT) with 3-dimensional reconstructions can delineate several patient-specific factors. The activity level and postoperative expectations of the patient, as well as ability to comply with strict postoperative rehabilitation protocols including possibly permanent activity restrictions, are important considerations in this challenging patient population. Detailed discussions with the patient, including the salvage nature of allograft reconstruction procedures, are critical prior to embarking on any of these possible surgical interventions.

When bony glenoid reconstruction is indicated, such as in cases of primary or recurrent instability associated with a large (>15%-20%) glenoid bone defect, a number of surgical options are available. The options for anterior glenoid reconstruction include coracoid transfer (Latarjet), iliac crest autograft transfer, and allograft reconstruction.[2,21-26] While coracoid is clearly a viable option, concerns include the extra-articular, nonanatomic nature of this repair, and thus its poor reconstitution of the glenoid arc. Reconstruction options for posterior glenoid bone loss include augmentation with iliac crest bone block[27-35]; however long-term outcomes have been inconsistent, often with discouraging results, which include the development of glenohumeral arthritis. For obvious anatomic reasons, coracoid autograft transfer is not a viable option for the posterior glenoid.

Given the association of extra-articular, nonanatomic autograft, or allograft reconstruction techniques with the early development of symptomatic glenohumeral arthritis, an alternative treatment of large glenoid bone defects in the setting of glenohumeral instability is the use of fresh osteochondral DTA. In theory, reconstruction with fresh DTA offers the same benefits of bone block (coracoid and iliac crest) reconstruction in preventing recurrent instability, while offering the advantages of biologically restoring the glenoid articular surface and providing congruency to the glenoid-humeral head articulation throughout an entire range of motion.[36,37]

While certainly an evolving technique, recently published studies address the clinical applications of fresh DTA.[36-40] Some studies address an effective surgical technique for treatment of anterior shoulder instability associated with anterior glenoid bone loss that includes anterior glenoid augmentation with DTA, which both reduces the rate of dislocation as well as improves pain and function.[36,37] Other studies describe posterior glenoid augmentation with fresh DTA, which shows encouraging early outcomes. Millet et al[38] described their 2-year results in 2 patients following open posterior shoulder stabilization with fresh DTA and noted successful clinical and imaging (via CT) outcomes. More recently, Romeo and colleagues[40] described the surgical technique for an arthroscopic approach to posterior glenoid augmentation with fresh DTA; however, no clinical outcomes are yet available.

Initial research has demonstrated a nearly identical radius of curvature between the distal tibia and the glenoid,[41] even among nonmatched cadaveric specimens, allowing for unimpeded motion due to the congruency of the DTA with the humeral head.[36,37] Additionally, DTA contains dense, weight-bearing corticocancellous bone, which makes it ideal for screw fixation and, further, contains a robust cartilaginous surface that allows for an anatomic, osteoarticular glenoid surface reconstruction. Overall, our initial laboratory studies represent the first biomechanical research to determine glenohumeral loading mechanics in clinically relevant glenoid bone-loss models, with clinically relevant arm positions, and the first to compare those to glenohumeral loading mechanics following allograft reconstruction. Anterior

glenoid reconstruction with flush Latarjet (using the inferior aspect of the coracoid as the glenoid surface) and flush ICBG best restored contact mechanics in the index study,[8] while these same grafts placed proud, or using the lateral aspect of the coracoid, resulted in worse outcomes. Further, anterior glenoid reconstruction with DTA resulted in improved contact mechanics when compared to Latarjet reconstruction, especially in the position of abduction with external rotation,[9] while posterior glenoid reconstruction with DTA resulted in equivalent contact mechanics when compared to ICBG.[10]

These discussed studies are not without some inherent limitations. As with any cadaveric study, each of these studies are dependent on "time zero" ex vivo models, without any opportunity for bone healing to occur. Given that the reconstruction techniques in these studies depend on bone-to-bone healing, the results from this study may differ from those in an in vivo setting. Finally, these studies utilize static models of the glenohumeral joint, devoid of any dynamic soft-tissue restraints, including the capsule and rotator cuff, which may alter the normal mechanics of the glenohumeral joint, affecting at the very least, peak force and contact pressures.

Current reconstructive techniques, including the use of coracoid transfers and ICBG, aim at decreasing shoulder instability, decreasing pain, and improving function. However, the concern for the early development of symptomatic glenohumeral arthritis following these nonarticular, nonanatomic procedures remains. At a minimum, glenoid reconstruction with fresh DTA offers an alternative, viable surgical option that potentially restores both mechanics and biology for what historically has been an extremely difficult problem. Certainly, these techniques are novel, and further clinical studies can aid in describing the effects that these mechanical properties may have on postoperative outcomes after glenoid reconstruction. Of utmost importance is gaining a better appreciation of the potential for graft resorption by means of further clinical work, including follow-up imaging studies.

SUMMARY

Reconstruction of anterior glenoid bone defects with DTA demonstrates improved biomechanical properties compared to Latarjet reconstruction, especially in the provocative position of abduction and external rotation, while reconstruction of posterior glenoid bone defects with DTA demonstrated at least equivalent biomechanical properties compared to reconstruction with ICBG. Given the concern over the association of the extra-articular nonanatomic ICBG reconstruction technique with the early development of symptomatic glenohumeral arthritis, this review of studies and methods from our laboratory suggests that glenoid reconstruction with fresh DTA is a viable alternative solution, with the potential advantage of improving joint congruity via an anatomic reconstruction that includes a cartilaginous, congruent articulation with the humeral head. ■

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

"ROTATOR CUFF DYSFUNCTION FOLLOWING TOTAL SHOULDER ARTHROPLASTY MAY BE MORE COMMON THAN PREVIOUSLY REPORTED."

Rotator Cuff Tears after Total Shoulder Arthroplasty in Primary Osteoarthritis: A Systematic Review

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The number of total shoulder arthroplasties (TSAs) performed has increased over 3-fold from 2000 to 2008.[1] Prolonged life expectancy and increased physical activity in older populations are among many reasons we have seen a greater prevalence of shoulder osteoarthritis (OA). Results following TSA have demonstrated a low complication rate, excellent pain relief, and return of function in a majority of patients.[2,3]

When complications do arise postoperatively, component loosening, nerve injuries, instability, periprosthetic fractures, and rotator cuff tears have been reported.[4,5] Glenoid loosening has typically been the primary concern with regard to TSA longevity, but recent reports have indicated that rotator cuff tears may be more prevalent.[6,7] Bohsali et al reported a 1.3% incidence of rotator cuff tear following TSA, with a majority of these tears occurring in the subscapularis tendon.[5] Others have reported a 2%-4% incidence of rotator cuff tear following the procedure.[4,8] Young et al reported a much higher incidence (16.8%) of rotator cuff dysfunction following TSA, with "dysfunction" defined as greater than 25% superior migration of the humeral component on a true anterior-posterior radiograph of the glenohumeral joint.[6] Post-TSA rotator cuff tearing or dysfunction is associated with proximal migration of the humeral component, which can accelerate polyethylene wear and loosening of the glenoid component through the rocking-horse phenomenon.[9]

The literature on rotator cuff dysfunction after TSA remains limited. Furthermore, most studies do not differentiate patients with OA from those with other forms of arthritis. Posttraumatic and inflammatory arthritis predispose patients to cuff tears, thus confounding any post-TSA cuff complications. We are not aware of any systematic reviews examining the incidence of rotator cuff tears following total shoulder replacement for primary OA. The purpose of this investigation is to determine the incidence of rotator cuff tears after TSA for primary glenohumeral arthritis. We hypothesized that the rate of overall rotator cuff pathology would be significantly higher than the 2%-4% rate of rotator cuff tears reported in earlier investigations.

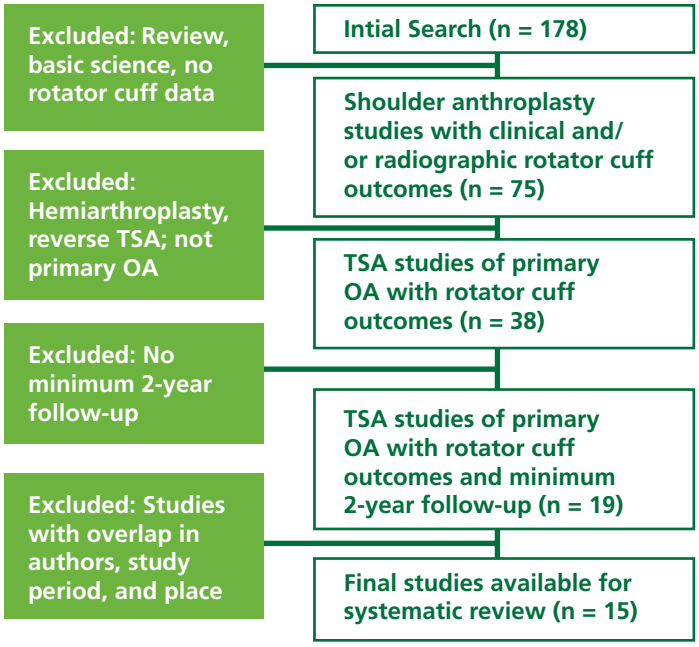
MATERIALS AND METHODS

SEARCH STRATEGY

We conducted a systematic review of the available literature according to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines using the PRISMA checklist.[10] Searches were completed in February 2013 using the PubMed Medline database and the Cochrane Central Register of Clinical Trials. The keyword selection was designed to capture all level I-IV evidence (according to the Oxford Centre for Evidence-Based Medicine) English-language studies that reported clinical and/or radiographic outcomes. This was accomplished using the keywords "shoulder" and "arthroplasty" and a series of "NOT" phrases designed to match our exclusion criteria. Study exclusion criteria consisted of cadaveric, biomechanical, histological, and kinematic results, as well as any analyses of non-operative management, hemiarthroplasty, or reverse total shoulder arthroplasty. Studies were excluded if they did not report any clinical and/or radiographic data relating to rotator cuff pathology. Patient populations were further excluded if their diagnosis was not primary OA; patients with inflammatory arthritis, posttraumatic arthritis, and postcapsulorrhaphy arthritis were excluded. Conversions from hemiarthroplasty to TSA were also excluded. Inclusion criteria consisted of a minimum of 2 years follow-up. This search yielded 178 initial results.

STUDY SELECTION

Upon encountering studies with the previously stated exclusion criteria, we did



Abbreviations: TSA, total shoulder arthroplasty; OA, osteoarthritis

FIGURE 1. Diagram demonstrating study-selection criteria.

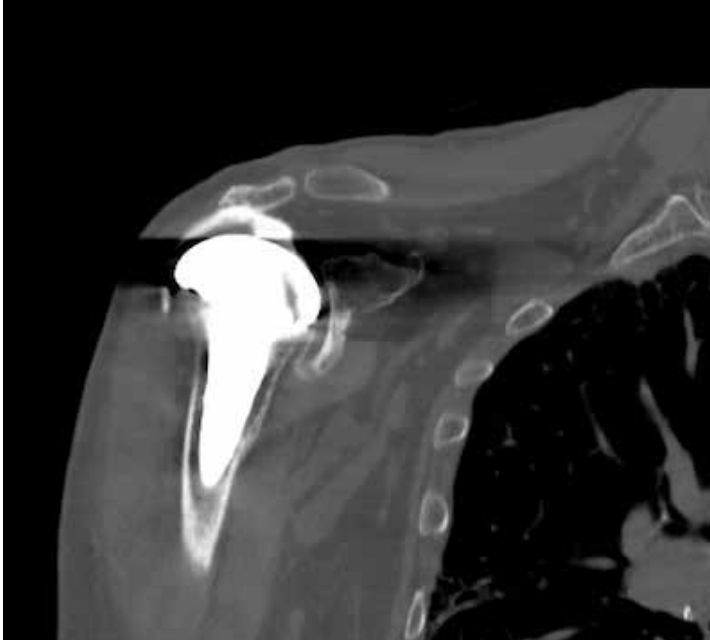


FIGURE 2. Computed tomography (CT) arthrogram of the right shoulder in a 67-year-old male demonstrating superior migration of the humeral component as well as contrast extravasation in the subacromial space indicative of a full-thickness tear of the superior rotator cuff.

not immediately exclude them, but rather reviewed them for any differentiation of patient populations. For instance, if outcomes from a TSA population were compared to or isolated from those of hemiarthroplasty patients, the clinical outcomes from the TSA population were included in our review. If a study separated outcomes by diagnosis, only those for patients with primary OA were included. If a study could not be deconstructed as such or was entirely devoted to 1 of our exclusion criteria, that study was excluded from our review. To ensure that no patients were counted twice, each study's authors, data collection period, and ethnic population were reviewed and compared to those of the other studies. If there was any overlap in authorship, period, and place, only the study with the most relevant (ie, rotator cuff outcomes) or comprehensive data was included. After accounting for all inclusion and exclusion criteria, 15 studies with 1259 patients (1338 shoulders) were selected for inclusion (Figure 1).[2,6,7,11-22]

DATA EXTRACTION

We extracted data from studies that satisfied the eligibility criteria. Details of study design, sample size, and patient demographics, including age, sex, hand dominance, and primary diagnosis were recorded. Surgical factors such as the approach, presence of preoperative rotator cuff tears, biceps treatment, use of cement, and prosthesis design were abstracted. Clinical outcomes included physical examination findings, functional assessment scores (patient satisfaction, Western Ontario Osteoarthritis Score [WOOS], Constant-Murley Shoulder Outcome Score [Constant], American Shoulder and Elbow Surgeons [ASES] score, the visual analog score [VAS], and Neer patient satisfaction), the number of revisions, changes in range of motion, and subscapularis-specific tests, such as belly press and lift-off. Radiographic outcomes focused on anterior or superior migration of the humeral head, which suggested subscapularis or supraspinatus/infraspinatus pathology, respectively. Those studies reporting superior migration of the humeral head were classified according to Torchia et al[12]: mild subluxation involves superior migration of less than or equal to 25% of the prosthetic humeral head diameter; moderate subluxation involves migration of 25% to 50% of the diameter; severe subluxation involves proximal migration greater than 50% of the humeral head diameter (Figure 2).

STATISTICAL ANALYSIS

We reported our data as weighted means with standard deviations. A mean was calculated for each study reporting on a respective data point, and each mean was then weighed according to its study sample size. This calculation was performed by multiplying 1 study's individual mean by the number of patients enrolled in that study and dividing the sum of these weighted data points by the number of eligible patients in all relevant studies. In this way, the nonweighted means from studies with a smaller sample size did not carry as much weight as those from larger studies. Comparisons were made with student *t*-test (SPSS v.18, IBM Inc., Armonk, NY), and an alpha value of 0.05 was set as statistically significant.

RESULTS

In the final dataset we included a total of 15 studies representing 1259 patients.[2,6,7,11-22] Level of evidence, conflicts of interest, study location, demographic characteristics, and clinical diagnoses of the included patients are presented in Tables 1 and 2. Of the 15 studies, 9 consist of level IV evidence. Only 1 study reported level I evidence.[20]

We present the incidence of preoperative rotator cuff tears as well as details of the surgical procedure, including type of components utilized, in Table 3. Seven studies commented on the preoperative condition of the rotator cuff and whether or not a concomitant repair was performed. Of these 7 studies, 59 shoulders (6.4%) had a complete supraspinatus tear, 39 of which (4.0%) underwent a supraspinatus repair at the time of arthroplasty. The technique of repair was not specified in any study.

TABLE 1. Levels of Evidence, Conflicts of Interest, and Study Location for Included Investigations

PARAMETER	TOTAL NUMBER
Final number of studies[2,6,7,11-22]	15
Level of evidence	
Level I[20]	1
Level II[18]	1
Level III[13,16,17,21]	4
Level IV[2,6,7,11,12,14,15,19,22]	9
Financial conflicts of interest	
Reported[2,6,7,11-20,22]	14
Present[21]	1
Study location	
United States[2,11-13,16,17,22]	7
Europe[6,7,14,15,19]	5[λ]
Canada[18,20]	2
New Zealand[21]	1

[λ] = Italy, France, Germany, United Kingdom, 1 multicenter study involving multiple European nations

TABLE 2. Demographics and Clinical Diagnoses for Final Cohort of Included Patients

PARAMETER	NUMBER
Sample size[2,6,7,11-22]	
Total number of patients	1259
Average number of patients	57.2 (range, 10-542)
Total number of shoulders	1338
Average number of shoulders	60.8 (range, 10-596)
Gender[2,6,7,11-22]	
Males	543 (43.1%)
Females	716 (56.9%)
Age[2,6,7,11-22]	67.2 ± 3.7 (range, 18-90)
Shoulder side[16,18]	
Right	95 (54.6%)
Left	79 (45.4%)
Shoulder dominance[6,12,14,19]	
Dominant	428 (59.9%)
Nondominant	286 (40.1%)
Primary diagnosis[2,6,7,11-22]	
Osteoarthritis	1335
Avascular necrosis	3
Number of shoulders with prior surgery[6,12,17]	4 (0.6%)

TABLE 3. Operative Findings, Techniques, and Implants for Final Cohort of Included Patients

PARAMETER	TOTAL NUMBER
Preoperative rotator cuff tears[2,6,7,12,13,16,19]	
Partial-thickness supraspinatus	51
Complete-thickness supraspinatus	59
Concomitant supraspinatus repair	12
Surgical approach[2,6,7,11-22]	
Deltopectoral	1321
Superior deltoid split	17
Anterior exposure[6,12-15,17-22]	
Subscapularis tenotomy	847
Lesser tuberosity osteotomy	86
Subscapularis peel	116
Biceps tendon handling[6,15,17,19-22]	
Tenodesis	372
Tenotomy	286
Glenoid component: material, fixation[2,6,7,12-20]	
All-polyethylene, cemented	1253
Metal-backed, bone in-growth	0
Glenoid component: design[6,7,12-16,18-20,22]	
Keeled	860
Pegged	185
Humeral component: fixation[2,6,7,12-22]	
Cemented	884
Uncemented	425
Humeral component: design[2,6,7,11-22]	
Stemmed	1292
Stemless	17
Prosthetic system[2,6,7,11-13,15,17-22]	
Total Evolutive (Biomet; Warsaw, Indiana)	17
Bio-Modular (Biomet; Warsaw, Indiana)	40
Comprehensive (Biomet; Warsaw, Indiana)	9
Global (DePuy; Warsaw, Indiana)	17
Global Advantage (DePuy; Warsaw, Indiana)	204
Neer I (Smith & Nephew; London, United Kingdom)	29
Neer II (3M; Saint Paul, Minnesota)	60
Cofield I (Smith & Nephew; London, United Kingdom)	34
Cofield II (Smith & Nephew; London, United Kingdom)	34
Aequalis (Tornier; Amsterdam, The Netherlands)	725
Bigliani-Flatow (Zimmer; Warsaw, Indiana)	39

TABLE 4. Pre- and Postoperative Clinical Outcome Data and Postoperative Radiological Outcomes for All Patients Included in Final Analysis

PARAMETER	WEIGHTED MEAN	*P* VALUE
Average physical exam follow-up	**6.8 ± 3.2 years**	
Positive belly-press test, incidence[15,17,21]	20.4 ± 27.7%	
Forward elevation[6,7,12-16,18,19,22]		
Preoperative	89.2 ± 15.8°	*P* < .001
Postoperative	139.7 ± 7.2°	
External rotation[6,7,12-16,18,19,22]		
Preoperative	13.5 ± 6.8°	*P* < .001
Postoperative	42.6 ± 9.3°	
Abduction[7,18,19,22]		
Preoperative	59.3 ± 15.9°	*P* < .001
Postoperative	126.8 ± 5.5°	
Average clinical survey follow-up	**6.5 ± 3.3 years**	
Neer criteria patient satisfaction, percentage[6,13,15]	90.4 ± 5.6%	
Constant[6,7,14,15,19]		
Preoperative	29.2 ± 4.7	*P* < .001
Postoperative	67.0 ± 11.5	
WOOS[18,20]		
Preoperative	26.9 ± 5.8	*P* < .001
Postoperative	86.0 ± 1.6	
ASES[2,7,18,20]		
Preoperative	31.6 ± 5.6	*P* < .001
Postoperative	83.2 ± 3.8	
VAS pain[2,13,15]		
Preoperative	8.1 ± 0.8	*P* < .001
Postoperative	2.4 ± 1.1	
DASH[18]		
Preoperative	57.0 ± 4.4	*P* = 0.013
Postoperative	19.3 ± 3.1	
Average radiological follow-up	**6.6 ± 3.1 years**	
Shoulders with superior migration of humeral head, incidence[6,7,11-16,18,19]	29.9 ± 20.7%	
Shoulders with superior migration of humeral head > 25% of humeral head diameter, incidence[6,12,16,19]	17.9 ± 14.3%	
Shoulders with anterior migration of humeral head, incidence[13,15,18,20]	11.9 ± 15.9%	

Abbreviations: ASES, American Shoulder and Elbow Surgeons score; Constant, Constant-Murley Shoulder Outcome Score; DASH, Disabilities of the Arm, Shoulder, and Hand score; VAS, visual analog score; WOOS, Western Ontario Osteoarthritis Score

Table 4 reports the clinical and radiographic outcomes from the included studies. The average length of follow-up to final physical exam was 6.8 ± 3.2 years. Eleven studies documented changes in forward elevation and external rotation pre- and postoperatively, and those 11 studies also demonstrated statistically significant improvements at final follow-up (*P* < .001). The belly-press test was found to be abnormal in 20% of patients postoperatively. Each clinical outcome score was utilized in no more than 5 studies. When reported, clinical outcome scores improved significantly after more than 6 years of follow-up (*P* < .001).

Radiographic data were reported in a majority of investigations (Table 4). Ten studies reported on proximal migration of the humeral head prosthesis. In these 10 studies, the percentage of shoulders with superior migration was nearly 30% after 6.6 ± 3.1 years. Among the 4 studies using the Torchia et al classification scheme,[12] 18% of shoulders demonstrated moderate or severe migration. These 10 studies were further subdivided into those reporting an average of greater than or less than 15% of shoulders with superior migration. When comparing these 2 groups of studies, there was no significant difference in preoperative Constant scores (*P* = .74), but there was a significant difference in postoperative scores, with lower scores in those studies reporting more than 15% of patients with superior migration (*P* = .049). Anterior humeral head migration was not specifically defined in any of the 4 studies in which it was reported. As such, the percentage of shoulders with anterior migration ranged widely from 0% to 36% for a mean of 12%.

Complications data were reported after a weighted mean of 7.0 ± 3.5 years of follow-up (Table 5). Overall revision rate for any reason was 6.8 ± 6.0%, while the independent reoperation rate for rotator cuff injury was 1.2 ± 4.5%. Among the 8 studies with documented rotator cuff tears, the weighted mean of superior cuff tears (supraspinatus or infraspinatus) was 11.3 ± 7.9%, and that of subscapularis tears was 3.0 ± 13.6%.

DISCUSSION

The goal of this systematic review was to report the incidence of rotator cuff tears following TSA for primary OA. Nearly all of the 15 analyzed studies (over 1,300 shoulders) reported indirect markers of rotator cuff dysfunction, such as radiographic humeral head migration and/or positive exam findings. Only 1 study utilized non-roentgenographic imaging (ultrasonography).[17] We found that nearly 30% of shoulders demonstrated radiographic superior migration and 12% showed anterior migration of the humeral head at a final mean follow-up of 6.6 ± 3.1 years.

Weiner and Macnab first described superior migration of the humeral head in native shoulders.[23] They found that 50% of patients with rotator cuff tears had proximal humeral migration, and subsequent clinical and cadaveric studies confirmed this finding.[24-26] Deutsch et al later described the mechanism by which this

TABLE 5. Incidence of Postoperative Rotator Cuff Tears and Reoperations

PARAMETER	WEIGHTED MEAN
Average complications follow-up	**7.0 ± 3.5 years**
Superior cuff tears[6,7,11,13,14,19,20,22]	11.3 ± 7.9%
Subscapularis cuff tears[2,6,7,13,17,19,20,22]	3.0 ± 13.6%
Revisions (reoperations)[2,6,7,11-14,17,19,20]	6.8 ± 6.0%
For rotator cuff injury	1.2 ± 4.5%
Superior cuff repair	0.5 ± 4.6%
Subscapularis cuff repair or graft	0.6 ± 1.1%
Revision to reverse total shoulder arthroplasty	0.2 ± 0.7%
For component loosening	3.5 ± 3.6%
For periprosthetic fracture	0.3 ± 0.9%
For infection	0.7 ± 1.7%
For stiffness	0.5 ± 0.2%
Revision to hemiarthroplasty	0.2 ± 0.6%

migration occurs.[27] The rotator cuff functions as a dynamic stabilizer of the glenohumeral joint and counteracts the upward pull of the deltoid muscle by depressing the humeral head during abduction. When the rotator cuff is torn or deficient, the deltoid is unopposed, leading to chronic superior subluxation of the humeral head. In a native shoulder, this can progress to impingement syndrome or cuff tear arthropathy. In a prosthetic shoulder, proximal migration may accelerate arthroplasty failure through 1 of 2 mechanisms. Greater humeral head translation diminishes the relative contact area of the glenohumeral joint and thus transmits higher contact stress to the glenoid component, leading to polyethylene deformation and wear.[28-30] In addition, humeral head migration can lead to eccentric loading and glenoid component loosening via the rocking-horse phenomenon.[9,29-33] Anterior migration theoretically could engender the same mechanisms of polyethylene wear and eccentric loading. Just as superior humeral head migration is associated with supraspinatus and infraspinatus tears, anterior subluxation may indicate a tear of the subscapularis tendon.[5] Inadequate subscapularis repair, humeral component malrotation, the use of oversized components, anterior glenoid and/or capsular deficiency, and deltoid dysfunction all have been associated with anterior instability after TSA.[8,34,35]

The clinical consequences of superior or anterior humeral migration remain controversial. The rocking-horse phenomenon of glenoid component loosening has been clearly described in patients with severe preoperative rotator cuff tears,[9] but it has not been proven in patients without preexisting tears. Young et al found that superior migration was significantly associated with glenoid radiolucencies,[6] and Miller et al reported lower ASES and patient satisfaction scores in patients with subscapularis tears.[36] However, the former also showed no difference in glenoid revisions between patients with and without superior migration. Moreover, Wirth et al found no association between rotator cuff tears and activity-altering pain,[8] and Khan et al showed that rotator cuff pathology did not adversely impact mean shoulder survey scores.[7]

Of the studies that reported postoperative rotator cuff tears, there was an 11% rate of superior cuff tears and 3% rate of subscapularis tears, confirming this investigation's hypothesis. Unfortunately, only a few authors specified their method of diagnosis.[6,7,14,17] Khan et al clinically diagnosed rotator cuff failure if the patient was pseudoparalytic or had a positive Jobe test, elevation of the humeral head on resisted cuff action, and serial radiographs with increasing proximal migration of at least 5 mm.[7] Scalise et al defined an abnormal subscapularis tendon through ultrasonography.[17] An attenuated tendon had a focal decrease of greater than or equal to 50% of the normal tendon thickness, and a full-thickness tear was defined as a gap in the tendon substance with retraction. The lack of objective data and the nature of aggregate data in a systematic review precluded direct correlation of the presence of rotator cuff tears with clinical outcomes. However, while nearly 7% of patients underwent reoperation, only 1.2% of patients did so for a rotator cuff injury.

Proximal migration of the humeral head was the most consistently reported data point that was used as an approximation of rotator cuff dysfunction. Ten studies (1,012 shoulders) commented on proximal migration. Four of these studies employed the proximal migration grading system of Torchia et al.[12] One study defined proximal migration as an acromiohumeral distance less than or equal to 7 mm,[14] and another study defined it as greater than 5 mm of migration over the follow-up period.[7] The other 4 studies did not define their parameters for superior migration of the humeral head. Only 3 studies in this review were dedicated specifically to post-TSA rotator cuff function, and all 3 used radiographic humeral head migration as their primary outcomes instrument.[6,11,12] This review's reported rate of 17.9% moderate or severe superior subluxation after 6.6 years was similar to the 16.8% rate Young et al reported after 8.6 years.

Limitations of this systematic review are imposed by the studies analyzed. There was a relative paucity of clinical and radiographic data relating to rotator cuff pathology. Magnetic resonance imaging (MRI) serves as the current gold standard for diagnosis of rotator cuff tears, but its postoperative use is limited by metal artifact. Therefore, most outcomes studies rely on indirect measures of rotator cuff quality. Proximal humeral migration may not be a reliable indicator of rotator cuff pathology, because Boyd et al showed that the incidence of cuff tears did not differ significantly between groups with and without proximal migration.[11] In addition, the belly-press test has demonstrated low sensitivity, low specificity, and low positive predictive value for diagnosis of subscapularis tears after TSA and so may be an unreliable indicator of subscapularis dysfunction.[37] Finally, because only a handful of studies described their actual clinical method of diagnosing rotator cuff tears, we accepted radiographic measurements of humeral head migration as a proxy of rotator cuff dysfunction. Selection bias was minimized in this review due to the inclusive nature of studies with levels of evidence I-IV, but this created a study design bias in that most studies consisted of level IV evidence and only 1 study cited level I evidence.

CONCLUSION

Rotator cuff dysfunction following total shoulder arthroplasty may be more common than previously reported. It remains unknown, however, whether rotator cuff dysfunction, as defined by clinical or radiographic examination of humeral head migration, leads to inferior outcomes. We hope that this review encourages researchers to design clinical and basic science studies that assess the impact of shoulder arthroplasty on the rotator cuff. ■

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

> NOT EVERY SPINAL DEFORMITY REQUIRES SURGICAL TREATMENT…. CAREFUL INDIVIDUALIZED ANALYSIS OF THE PATIENT'S PAIN AND DISABILITY IS NECESSARY TO FACILITATE CORRELATION WITH THE ANATOMIC FINDINGS.

Individualized Surgical Treatment of Degenerative Scoliosis

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An increasing number of people are seeking care for symptoms of degenerative scoliosis. Degenerative, or de novo, scoliosis, is defined by a Cobb angle of over 10° in the coronal plane in an adult who did not have scoliosis prior to skeletal maturity.[1,2,3] The condition is distinct from scoliosis that begins in childhood or adolescence. The number of people entering orthopedic clinics in search of care for this malady is increasing due to an aging population, increasing vigor and expectations of older people, and public knowledge of tremendous advancements in treatment.

Pathologic changes leading to degenerative scoliosis include loss of intervertebral disc height, facet degeneration, and wedging of the vertebral bodies secondary to osteoporosis.[4] The prevalence of degenerative scoliosis is reported as 68% in men and women aged 60 years or older.[5] As the deformity evolves, it may include anterolisthesis,[6] lateral olisthesis,[7] and/or rotatory deformity, along with sagittal and/or coronal imbalance, and central canal, lateral recess, and/or foraminal stenosis.[8]

Pain and disability, rather than imaging, generally drive decisions regarding whether to offer surgical treatment.[9] Though scoliosis can be associated with pain, some prevalence studies have noted poor correlations between scoliosis and pain scores[5] and poor correlation between symptoms and magnitude of the curve,[10] just as the degree of narrowing in lumbar spinal stenosis has shown poor correspondence with clinical affliction.[11] Not every spinal deformity requires surgical treatment, and selection for surgery cannot be made from images alone. Careful individualized analysis of the patient's pain and disability is necessary to facilitate correlation with the anatomic findings.

Unlike adolescent idiopathic scoliosis, cosmesis is often not the older patient's chief concern. Presenting complaints typically resemble a mix of mechanical back pain, flat-back syndrome, neurogenic claudication, and radiculopathy.[2] Not uncommonly, lumbar degenerative scoliosis patients will have predominantly axial back pain manifest by intolerance of standing a certain time or walking a certain distance. Symptoms are from a combination of central stenosis, foraminal stenosis, facet arthritis, deterioration of the ligaments and discs, and overall global imbalance. Because multiple pain generators act with varying intensity, it is often difficult to identify a single target. It is important to rule out mimickers of symptomatic degenerative scoliosis, such as vascular claudication and hip arthritis. Clinical observation of coronal and sagittal balance, pelvic obliquity, and leg-length discrepancy is essential. Incriminating global imbalance as the cause of symptoms could lead to recommendation for a long fusion with substantial risk; whereas, the failure to recognize those patients whose symptoms are from multilevel global imbalance could result in having them go through an operation that does not adequately address their symptoms; therefore the importance of accurate analysis is very high.

Imaging studies needed for assessment of degenerative scoliosis reflect the condition's complexity. From standing, full-length X-rays of the spine from the occiput to the hips, we measured sagittal and coronal Cobb angles (Figure 1), sagittal balance (Figure 2), maximal antero-posterior olisthesis x2, pelvic incidence (PI) (Figure 3), lateral olisthesis x2, lumbar lordosis (LL), thoracic kyphosis (TK), and upper endplate obliquities at L3 and L4[10]. Surgical planning of complex cases requires a supine, recumbent, bolstered view of kyphosis; right and left bend films of scoliosis; and flexion/extension laterals of sagittal plane segmental instability. Cross-sectional imaging, such as computed tomography (CT) or magnetic resonance imaging (MRI), evaluates central or foraminal stenosis. Areas highly susceptible to foraminal stenosis include the concavity of both the structural lumbar curve and the distal fractional curve.[12] Lumbar radiculopathy due to foraminal stenosis in the distal fractional curve is a more common source of symptoms than is the radiographically more dramatic upper lumbar curve.

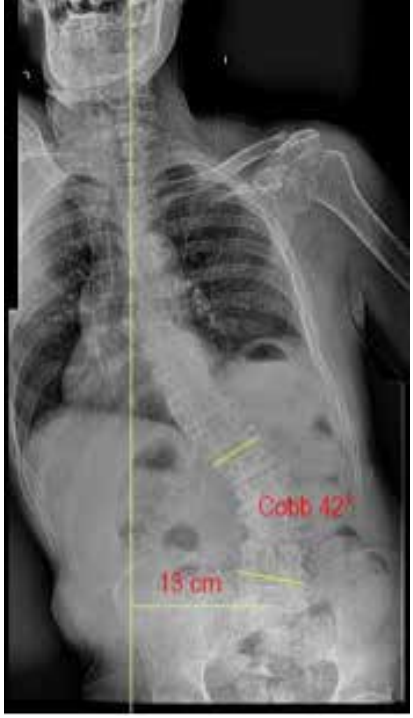

FIGURE 1. Cobb angles measure coronal plane deformity. Decompensation in the coronal plane is measured as the distance between a plumb line drawn from the center of C7 and a vertical line drawn through the center of the sacrum.

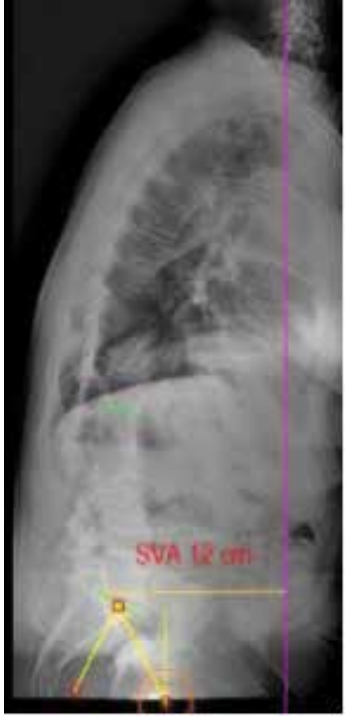

FIGURE 2. The sagittal vertical axis is the distance from the posterior superior corner of S1 to a vertical plumb line dropped from the center of the C7 body. When that distance is positive, the spine is said to be in positive sagittal balance, measured here to be 12 cm.

Assessment of sagittal imbalance plays an important role in surgical planning.[13,14] The Scoliosis Research Society's classification system for degenerative scoliosis addresses parameters affecting sagittal balance[15] and can be used to predict the impact of operative management on health-related quality of life[16] and likelihood of failing nonoperative management.[17]

For patients who are frail and have medical comorbidities, conservative options may be the best choice or at least should be explored before considering surgery. Nonsteroidal anti-inflammatory drugs, physiotherapy, activity modification, oral steroids, neuropathic pain medications, bracing, assistive devices, and facet and epidural injections all have their places,[2] though each has its own set of potential complications. Surgical indications include failure of conservative treatment, progressive spinal deformity, disabling neurological deficits, and pain sufficiently severe to warrant risks of surgery. A recent meta-analysis of 16 studies evaluating surgery for degenerative scoliosis found a mean decrease of 23 points in the Oswestry Disability Index (ODI), with an overall surgical complication rate of 49%.[18] Surgeons may counsel patients that surgery has been shown to be an effective and reasonable treatment for many patients with degenerative scoliosis, albeit with a high complication rate.

The surgical plan must reconcile the goals of decompression, stability, and balance with consideration of the patient's medical comorbidities. The patient's clinical symptoms are far more important in the surgical plan than imaging findings. Symptoms resulting from spinal deformity must be differentiated from those resulting from stenosis and neural compression. Radicular symptoms must be differentiated as originating from the primary curve versus the fractional curve.

Once the surgeon has collected the full constellation of relevant clinical, radiographic, and medical findings, the next step is to devise an appropriately tailored surgical plan, using 1 of 3 approaches: 1) decompression alone, 2) decompression with local fusion, or 3) decompression with long fusion.

DECOMPRESSION ALONE

In the spectrum of surgical interventions, the least invasive is decompression of neural elements without fusion. Strictly defined guidelines for the procedure are not available; however, there are some instructive general principles based on expert opinion. Surgical candidates for decompression without fusion should have a clinical picture of neurogenic claudication and radiculopathy.[19] They should also have smaller curves, ideally less than 20.[20] They should be well balanced globally in the coronal and sagittal planes because decompression alone will not correct imbalance. The symptomatic areas requiring decompression should not show radiographic signs of instability, such as lateral olisthesis or anterolisthesis. The site of decompression should not be at the apex of the deformity because iatrogenic instability could result. Careful surgical technique that does not violate the integrity of the pars or the stability of the facet joints is critical to avoiding instability complications. Surgeons should inform patients that decompression alone carries a risk of worsening of symptoms due to reduced stability and progression of the scoliosis after surgery.[21]

In appropriately selected patients, decompression can produce good results. Kelleher et al reported their results of decompression alone for lumbar stenosis in a series of 75 patients with and without degenerative scoliosis.[22] Both groups demonstrated clinically significant improvements in the ODI. Transfeldt and colleagues compared their results of decompression alone, local fusion, and long fusion in a series of 85 patients.[23] They noted low complication rates and significant improvements in the ODI in the decompression-alone groups, especially when compared to the fusion groups.

While decompression alone can yield good results, potential complications must be kept in mind. Tsutsui et al, in a retrospective review of 75 patients with lumbar degenerative scoliosis and stenosis who underwent decompression alone, found 59% had good relief of axial back pain, with poor results in patients with high apical rotation of the lumbar curve who underwent decompression alone.[24] Logistic regression analysis revealed a significant association between increasing degrees of apical rotation and residual postoperative back pain. The authors highlighted the importance of mild deformity for a good

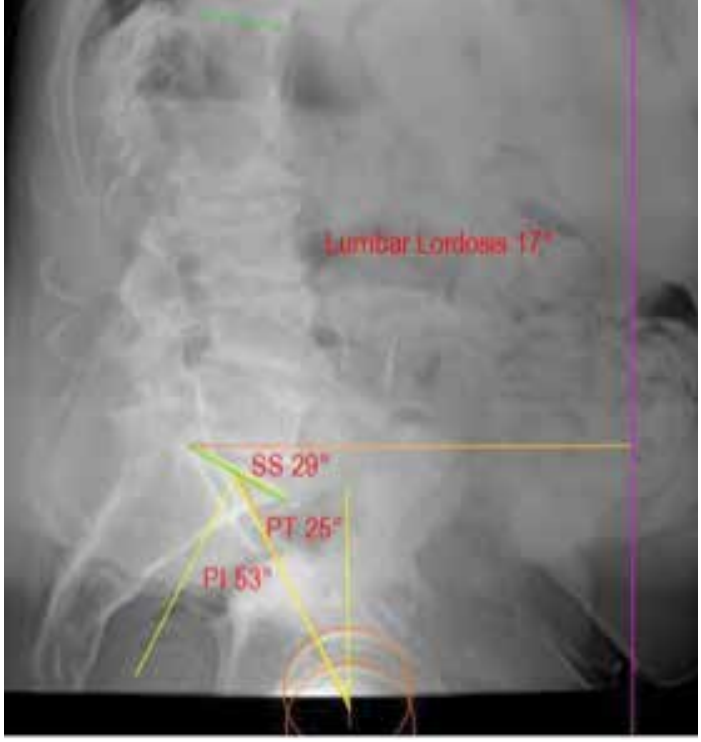

FIGURE 3. The pelvic incidence (PI) is a fixed anatomic parameter of the pelvis that is independent of pelvic orientation. It is the sum of the sacral slope (SS) and the pelvic tilt (PT). The SS is the angle between the sacral end plate and the horizontal axis. The PT is the angle between a line connecting the center of the femoral heads to the center of the sacral end plate and the vertical axis. As the anteversion/retroversion of the pelvis changes, the SS and PT change in opposite directions. Well-balanced spines generally have a lumbar lordosis (measured from superior L1 end plate to the S1 end plate) approximately equal to the PI.

outcome when performing decompression alone. Yamada and colleagues studied the progression of degenerative scoliosis following decompression without fusion.[25] Fifty consecutive patients underwent decompression without fusion for degenerative lumbar foraminal stenosis. Clinical results were good and complication rates were low; however, at 2-year follow-up, 18% of patients showed progression of scoliosis.

DECOMPRESSION WITH LOCAL FUSION

The surgeon must decide whether to offer the patient who needs arthrodesis a local fusion with some residual deformity or a longer fusion with less deformity. Transfeldt et al, in a retrospective review of 85 patients with degenerative scoliosis and radiculopathy, found that the full-fusion group had both higher complication rates and higher patient satisfaction rates.[23] The local-fusion group had significant improvements in the ODI and lower complication rates. Patients with moderate deformity without significant global imbalance and pain and radicular symptoms that can be localized to the apex of the scoliosis are good candidates for decompression with a local fusion.

Surgeons have accomplished decompression with fusion with a variety of surgical techniques. Li et al published results of selective segmental transforaminal lumbar interbody fusion (TLIF) for 46 patients with a mean age of 66 years.[26] They achieved partial correction of deformity with good clinical results and low complication rates, consistent with short-segment fusion.

Studies have investigated short-segment fusion with asymmetric pedicle subtraction osteotomy in the setting of rigid lumbar degenerative kyphoscoliosis. Employing this technique in a series of 14 patients with rigid, short kyphoscoliotic curves, Toyone achieved an average of 40° improvement in lumbar lordosis and significant improvements in sagittal balance with a postoperative satisfaction rate of 100% for 14 patients.[27]

Defining the symptomatic portions of degenerative curves can be a challenge. Pugely and colleagues divided degenerative scoliotic curves into primary (structural) and fractional segments.[12] They stratified patients into groups according to whether they had femoral nerve pain or sciatic nerve pain. By correlating pain type according to upper lumbar root versus lower lumbar root, they were able to identify whether the pain generator was from the primary versus the fractional curve. In this manner, they limited decompression and fusion to symptomatic segments with excellent results. The surgeon must keep in mind that symptoms are often due to foraminal compression on the concavity of the symptomatic curve. On the concavity, the cephalad-caudad dimensions of the foramina are reduced, and this can compress the exiting nerve roots. The surgical plan for local fusion should include distraction across the concavity typically through the pedicle screw and rod construct with or without TLIF.

O'Shaughnessy et al retrospectively compared, by matched cohort analysis, upper-thoracic-to-sacrum versus lower-thoracic-to-sacrum fusions for degenerative scoliosis.[28] Upper-thoracic-to-sacrum fusions had increased rates of pseudoarthrosis and perioperative complications. The lower-thoracic-to-sacrum fusion group had increased rates of proximal junctional kyphosis. The 2 groups had similar improvements in Scoliosis Research Society (SRS) and ODI scores.

In a large retrospective review of patients with degenerative scoliosis older than 50, Charosky and colleagues examined risk factors for neurologic and mechanical complications.[29] The study cohort included anterior only, anterior-posterior, and posterior only groups. There were a total of 175 complications in 119 patients. Risk factors for mechanical and neurological complications included numbers of levels instrumented, fusion to the sacrum, and osteotomy. Number of levels fused was associated with increased risk of reoperation.

Patients who undergo short-segment fusions risk later requiring more extensive surgery. Kasliwal and colleagues performed a retrospective cohort analysis of 30 matched patients in which they investigated whether prior short-segment surgery was a risk for poor outcomes or complications when

revised to a long fusion and found that prior short-segment surgery was not associated with poorer outcomes.[30] Concern for future extension of fusion should not be a barrier to a short-segment fusion in appropriately selected patients.

Decompression with a local fusion can achieve good results for patients who do not have excessive spinal imbalance and who can localize pain and radiculopathy to specific segments of the spine.

DECOMPRESSION WITH LONG FUSION

When more conservative surgical options cannot be pursued, decompression with long fusion (also known as full-curve fusion) is sometimes necessary. In these instances, a significant component of the patient's symptoms consists of back pain originating from imbalance. Prospective studies have shown that if high-grade sagittal imbalance is not addressed, decompression with only a local fusion is likely to lead to a poor result Once decompression with long fusion is decided upon, additional augmentative options can include interbody fusion and corrective osteotomies.[31] Interbody fusion simultaneously provides both lumbar-curve correction and foraminal distraction. Corrective osteotomies become necessary when stiff deformities impede restoration of sagittal balance.[17]

Extensive exposures, increased instrumentation, and use of osteotomies lead to increased rate of complications for decompression with long fusion. In a retrospective, single-center study evaluating 85 patients, Transfeldt and colleagues identified a complication rate of 56% in patients who received long fusion, compared to 40% in patients who received local fusion and 10% in patients who had decompression alone.[23] Prospective analysis of 58 patients at a single institution receiving fusion from either a low or high thoracic vertebra revealed that short fusions were more prone to proximal junctional kyphosis while long fusions had a significantly higher rate of perioperative complications and pseudoarthrosis.[28] Despite this complication profile, patients who receive decompression with long fusion have been shown to score highly on postoperative satisfaction questionnaires.[23,32]

To minimize risk of complication associated with larger exposures and extensive instrumentation, surgeons try to minimize the extent of fusion whenever possible. On the other hand, it is crucial that the fusion be of sufficient length to minimize the risk of postoperative fixation failure or adjacent segment disease. Cho et al reviewed 51 people who had received long fusion for degenerative scoliosis and followed them for over 2 years. The study found that proximal adjacent segment disease occurred most frequently in cases where the fusion ended at the upper-end vertebra (UEV), and least frequently when the fusion ended at the proximal horizontal vertebra (HV) or higher.[33] In their literature review and case series, Silva and Lenke recommend that, in general, one must avoid ending instrumentation at either the curve apex or at any level where rotatory subluxation is present. They went on to also endorse that the upper- and lower-most instrumented vertebrae are ideally both neutral and stable.[31] As an extrapolation of the anatomic study by Bernhardt and Bridwell, as the natural thoracic kyphotic apex occurs between T5 and T8, placement of the upper instrumented vertebrae (UIV) should be avoided for this area.[34] While sacral or pelvic fixation is not essential, it should be strongly considered whenever the fusion has been extended to T12 or above, or when the L5-S1 level demonstrates a spondylolysis, spondylolisthesis, previous decompression, or oblique (>15) take-off.[31]

CONCLUSION

Degenerative, or de novo, scoliosis, a unique disease, is causing increasing numbers of people to seek care in order to preserve quality of life. Physicians can tailor current surgical techniques to preserve function and improve prognosis for patients with degenerative scoliosis. Procedures include decompression alone, decompression with local fusion, and decompression with long fusion. Due to the highly individualized nature of care, formulaic decisions regarding surgery are unsatisfying. Surgeons must analyze symptoms, physical findings, and imaging when considering operations with both potential for success and a high rate of complications. ■

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"FOR SELECTED PATIENTS WITH DEGENERATIVE SCOLIOSIS, EXTREME LATERAL INTERBODY FUSION PROVIDES INDIRECT DECOMPRESSION AND MILD CORONAL AND SAGITTAL BALANCE CORRECTIONS, WITH LOWER COMPLICATION RATES AND SHORTER OPERATION TIMES."

Minimally Invasive Surgery for Degenerative Scoliosis

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Scoliosis in the adult population may represent childhood/adolescent scoliosis that becomes progressive in adulthood or scoliosis that appears de novo in adult life. This latter type is termed degenerative scoliosis. Adult patients receive a diagnosis of degenerative scoliosis when it occurs or becomes relevant after skeletal maturity with a Cobb angle of more than 10 degrees in the coronal plain.[1] In adult degenerative scoliosis, the deformity is thought to develop as a result of asymmetric disc collapse, vertebral body wedging, and facet arthropathy. Asymmetric loading of the spinal segment may contribute to progressive deformity.[2]

Patients with degenerative scoliosis commonly present with back pain, which occurs in more than 90% of patients seeking treatment.[3,4] Back pain may be the result of muscle spasm and fatigue (often on the convex side), spondylotic changes secondary to asymmetric facet/disc degeneration (often on the concave side), or some combination of both. The pathologic features of degenerative scoliosis may lead to spinal stenosis, manifesting with neurogenic claudication and/or radiculopathy.

Disability and treatment as a consequence of degenerative scoliosis in the adult population have significant clinical and societal impacts. Radiographic evidence of degenerative scoliosis may be present in as much as 68% of patients over 60 years old.[5] With a high prevalence of degenerative scoliosis, treatment becomes difficult to address because many of these patients are older and have significant medical comorbidities. Surgical treatment should focus primarily on dealing with the patient's symptoms, which may require fusion of the affected spinal segments, correction of deformity, and/or decompression of neural elements.

Traditional surgical treatment for degenerative scoliosis includes both posterior and posterior-anterior approaches, which typically accomplish direct decompression of neural elements as well as instrumentation with fusion for correction of deformity. These surgeries are associated with significant morbidity, and studies have shown complications rates ranging from 25% to 80% with open surgical fusion for the treatment of degenerative scoliosis.[6,7]

ALTERNATIVE TREATMENT

In recent years, less-invasive approaches, such as the lateral approach, have been utilized for indirect neural decompression, fusion, and, recently, spinal deformity. In 2005 Phillips et al first reported the use of extreme lateral interbody fusion (XLIF) technique in the treatment of adult scoliosis.[8] Ultimately, the lateral approach may avoid the complications of traditional open anterior approaches while minimizing the extent of soft-tissue dissection and blood loss with open posterior correction. The lateral approach may be utilized as an anterior-only procedure, or combined with percutaneous posterior pedicle screws or traditional open posterior techniques to obtain further coronal and sagittal balance correction.

During lateral lumbar interbody fusion, the surgeon accesses the spine via a direct lateral transpsoas approach. This procedure offers certain advantages over direct anterior and posterior interbody fusion. The procedure is typically performed without the help of an access surgeon and does not require mobilization of blood vessels or extensive paraspinal muscle stripping, thereby reducing morbidity and complications associated with traditional approaches to the spine. The transpsoas approach must avoid the neural elements located in the psoas muscle.[9,10] The procedure involves accessing the retroperitoneal space through single or dual incisions and then, using fluoroscopic guidance, advancing sequential dilators through the psoas muscle onto the lateral aspect of the intervertebral disc. This is followed by the placement of an expandable retractor onto the lateral disc. In order to navigate through the psoas muscle and avoid injury to the neural elements, accurate and reliable real-time neural monitoring is required. Following disc access and conventional discectomy, the surgeon places an interbody cage to promote arthrodesis and effect correction of both the coronal and sagittal plane deformities through ligamentotaxis. In addition, the disc-space

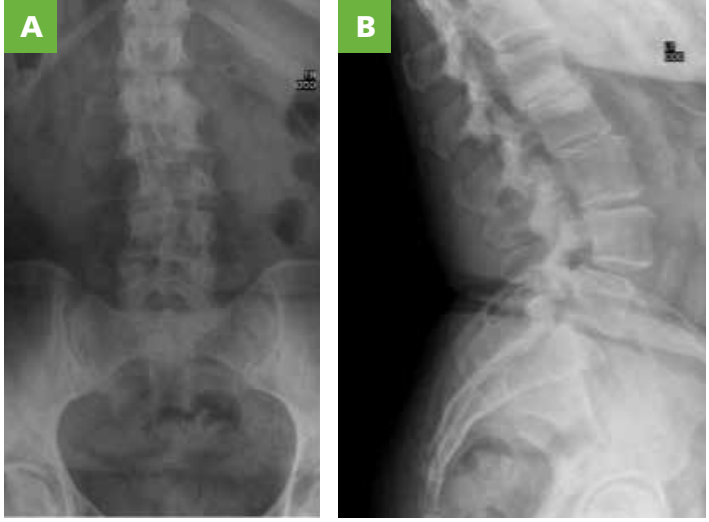


FIGURE 1. X-rays showing **A,** anterioposterior (AP) and **B,** lateral views demonstrate degenerative scoliosis with mild coronal deformity measuring approximately 19 degrees with preservation of lumbar lordosis of 55°. The lateral x-rays demonstrate relative preservation of L5-S1 disc space. Also note a L4-L5 grade 1 spondylolisthesis with retrolisthesis of L1-L2 and L2-L3.

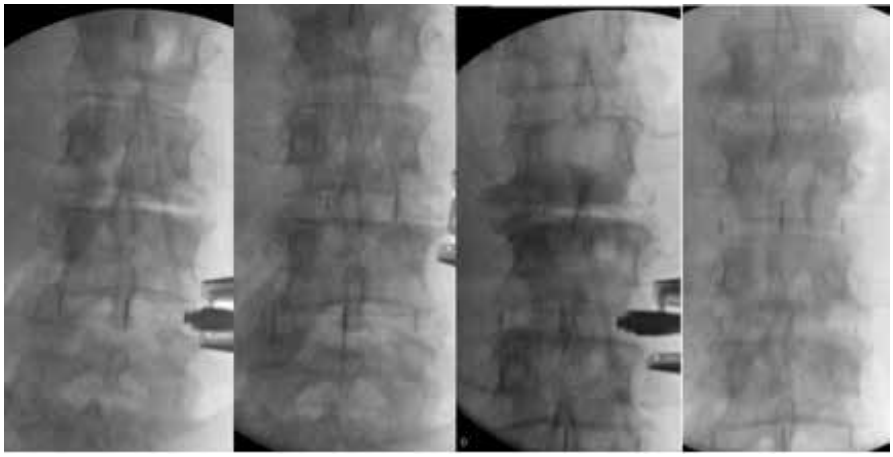


FIGURE 2. From left to right, anterioposterior (AP) intraoperative fluoroscopy demonstrating the amount of coronal correction with each subsequent level starting from L4-L5.

distraction achieved can provide indirect decompression of the neural foramen and spinal canal. Oliveira and colleagues demonstrated increases in disc height (41%), central canal diameter (33%), foraminal area (25%), and foraminal height (14%) after placement of a lateral interbody device, as measured by magnetic resonance imaging (MRI).[11]

With XLIF, surgical time has been reported at approximately 58 min per level, with average blood loss between 50-100 mL. The average hospital stay for this same group was 2.9 days for stand-alone technique.[12] Phillips et al, in a prospective study investigating XLIF for degenerative scoliosis in 107 patients, found that, at 2-year-follow-up, Oswestry Disability Index (ODI), Visual Analog Score (VAS) back, VAS leg, Short Form-36 Mental Component Summary (SF-36 MCS), and Short Form-36 Physical Component Summary (SF-36 PCS) scores were significantly improved from preoperative level. They also reported that 85% of patients were satisfied with their outcome and that 86% would repeat the surgery. Radiographs at 24 months showed improvement from the preoperative coronal Cobb angle, from an average of 20.9° to 15.2°, as well as improvement in lumbar lordosis in hypolordotic patients at 24 months.[13]

While XLIF is a minimally disruptive technique for achieving fusion, the procedure is not without the risk of both medical and surgical complications. Medical complications, with a reported incidence between 1.9% and 3.7%, are similar but less frequent than those found with other major spinal surgery procedures.[14] Transient hip-flexor weakness, likely related to the passage of retractors through the psoas muscle, has been reported in up to 23% of patients after single-level XLIF and up to 33% of patients after multilevel procedures. Because this weakness is typically due to muscle trauma and not nerve injury, near-complete recovery can be expected in most cases. Neurologic injury resulting in lower extremity weakness has been reported in 0.7% to 3.4% of patients.[12,14]

CASE REPORT

A 57-year-old female who works as a special-education nurse complained of chronic back pain with radiation to her buttocks for almost 10 years. She rated her pain 9 out of 10 and noted severe limitations to her daily activities secondary to her pain. She has tried nonsteroidal anti-inflammatories (NSAIDs), physical therapy, and epidural injections without any lasting relief.

Her past medical history is significant for asthma, hypertension, and an arrhythmia. Her past surgical history includes cholecystectomy, carpal tunnel release, tonsillectomy, and hysterectomy without any prior spine surgeries. On exam, she walked with a normal gait and stood with normal posture. She exhibited pain with lumbar extension. Her motor and sensory exams were normal. Her x-rays demonstrated degenerative scoliosis with a grade-1 spondylolisthesis at L4-L5 with lumbar kyphotic deformity (Figure 1).

She underwent an XLIF from L1-L5 with percutaneous posterior instrumentation from L1-L5 (Figure 2). She had no immediate postoperative anterior thigh pain or hip-flexor weakness. Her preoperative symptoms have resolved (Figures 3-4).

SURGICAL TECHNIQUE

The first step to the lateral surgical approach is proper positioning on a standard operative table (Figure 5). The senior author prefers positioning with the concave surface up, allowing greater distraction of the neural foramina, greater coronal correction, and easier access to the

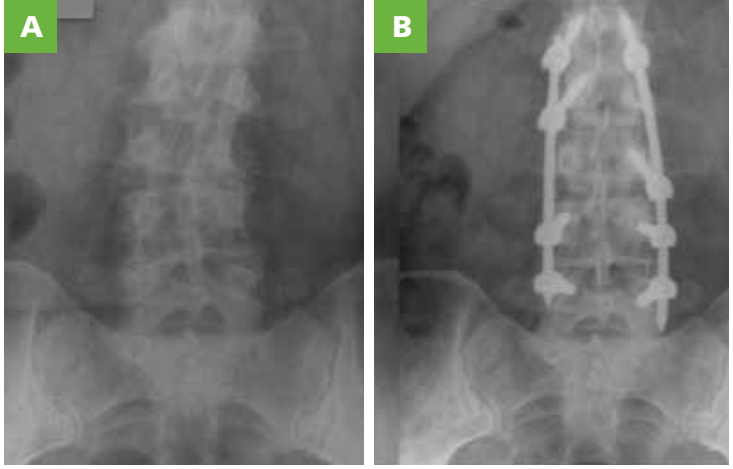


FIGURE 3. Comparison of **A,** anterioposterior (AP) preoperative x-ray to **B,** 2.5-month postoperative x-ray. The mild coronal deformity was corrected and maintained correction at follow-up.

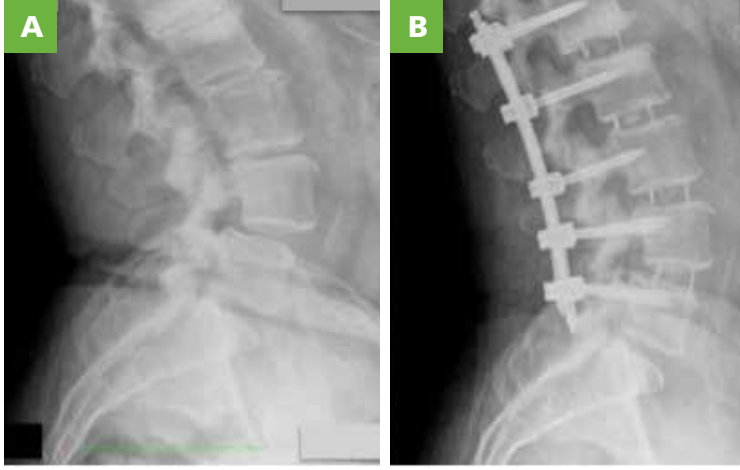


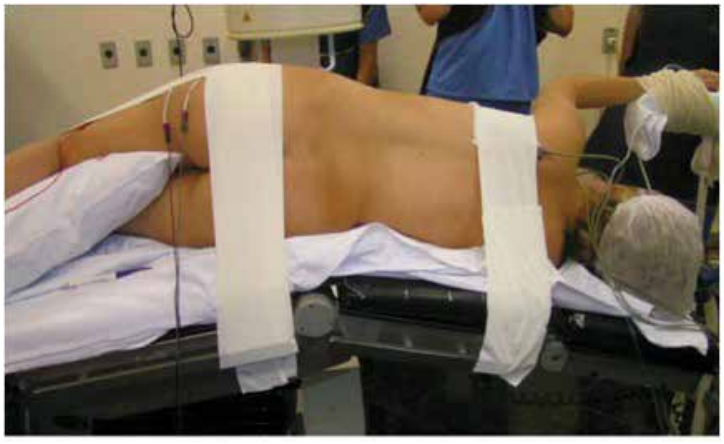
FIGURE 4. Comparison of **A,** lateral preoperative x-ray to **B,** 2.5-month postoperative x-ray. The lumbar lordosis is maintained. Disc space and foraminal height are increased postoperatively.

L4-L5 disc space, which may be blocked by the top of the iliac crest. An axillary gel roll is used to avoid brachial plexus injuries. The hips are placed in a flexed position, decreasing tension of the psoas muscle and the lumbar plexus to avoid neuropraxic injury. The break in the table is centered about the level of the iliac crest to optimally increase the distance from iliac crest to the ribs. The fluoroscopic beam is centered at the most caudal level and will need to be adjusted at each subsequent cephalad level.

Aided with fluoroscopy, the anterior and posterior border of the vertebral body is marked on the skin. The incision should be centered about the middle one-third or the junction of the anterior two-thirds and posterior one-third of the body. A postero-lateral accessory incision can be utilized to guide the dilator through the retroperitoneal space. After skin incision, the abdominal wall muscles are bluntly dissected, and the transversalis fascia is incised. The peritoneum is swept anteriorly to allow placement of a K-wire and dilators down to the vertebral body, at the junction of the anterior two-thirds and posterior one-third of the vertebral body to avoid injury to the lumbar plexus (Figure 6).

Real-time neuromonitoring is used while passing the K-wire through the psoas to avoid injury to the lumbar plexus.

Once the K-wire is docked at the appropriate position as previously described, serial dilators are placed over the K-wire to allow placement of the retractors (Figure 7). The discectomy can be completed with a combination of curettes, pituitary rongeurs, and Cobb elevators. The contralateral annulus should be released to allow for maximal disc-space distraction and curve correction. The endplates should not be violated so as to preserve the potential of distraction through the disc space with



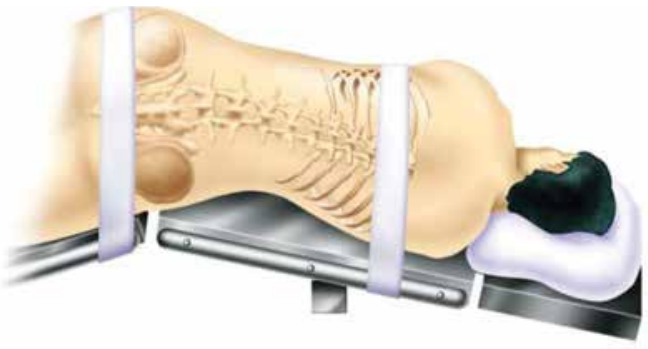


FIGURE 5. Proper positioning and taping with table break to increase the distance between the top of the iliac crest to the bottom rib.

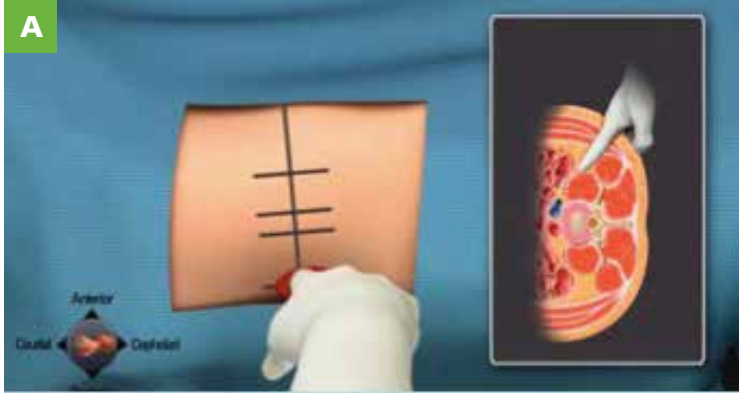


FIGURE 6. Topographical skin marking of the anterior vertebral body, posterior vertebral body, junction anterior two-thirds and posterior one-third, and accessory posterior lateral incision. The accessory incision is used to **A,** sweep the abdominal muscles anteriorly and **B,** guide the K-wire through the psoas.

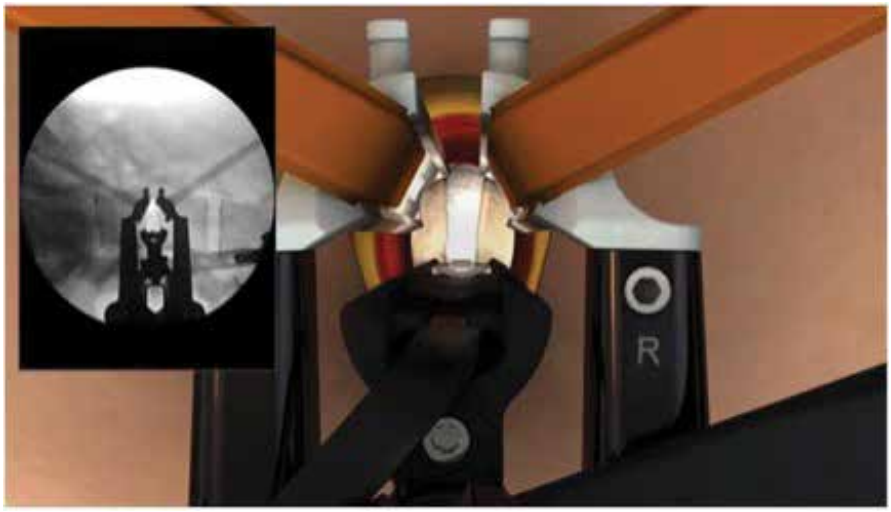


FIGURE 7. Retractors have been expanded after serial dilation over the K-wire. At this point, the discectomy is performed, using a combination of curettes, pituitary rongeurs, and Cobb elevators. The contralateral annulus is released for maximal correction while care is taken not to violate the endplate for distraction through the disc space with the interbody device.

the interbody graft, which is a key step to indirect decompression of the neural foramina and spinal canal. The widest interbody graft that is permitted by the disc space should be selected to avoid subsidence by buttressing against the stronger apophyseal ring. A lordotic interbody graft allows correction of any sagittal imbalance. Posterior instrumentation is case dependent and may be completed percutaneously to decrease blood loss.

CONCLUSION

In the past, surgeons used open decompression and posterior fusion or posterior-anterior fusion to treat patients who presented with symptomatic degenerative scoliosis. These surgical techniques have been associated with a high complication rate and long surgical times. For selected patients with degenerative scoliosis, extreme lateral interbody fusion provides indirect decompression and mild coronal and sagittal balance corrections, with lower complication rates and shorter operation times. ■

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

"WHEN DOCTORS ASSUME THAT CAM LESIONS OCCUR ONLY IN YOUNG MALES, THEY RISK OVERLOOKING CAM IMPINGEMENT AS A SOURCE OF INTRA-ARTICULAR HIP PAIN IN WOMEN."

Incidence of Cam Lesions in Female Patients with Femoroacetabular Impingement

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Abnormalities in femoral and acetabular morphology as seen in femoroacetabular impingement (FAI) can cause chondral and labral damage that potentially contributes to the development of osteoarthritis (OA).[1-3] Pincer-type FAI results from increased acetabular depth, as seen in acetabular overcoverage. Cam-type FAI is a consequence of decreased head-neck offset and/or increased radius of the femoral head. Pincer impingement leads to abnormal contact between the acetabular rim and labrum with the femoral head/neck junction. This can result in a labral tear, degeneration of the anterosuperior rim, and/or a posteroinferior contrecoup lesion.[4] Cam impingement increases shear forces, causing subsequent damage to the anterosuperior acetabular cartilage and labral avulsions from the anterosuperior acetabular rim. The etiology of both types of FAI remains unclear, but both have been associated with an increased risk of hip OA.[5-7]

Orthopedists consider cam impingement to predominate in men, while pincer impingement occurs primarily in women.[1,8-11] The morphologic configuration of the hip joint differs between males and females, and there are different hypotheses to explain the causation. Females have earlier closure of the pelvis and hip growth plates compared to males.[12] Subclinical slipped capital femoral epiphysis (SCFE) predisposes men to cam lesions because the prevalence of SCFE is much higher in adolescent males than in females.[13,14] Cam deformities have also been associated with impact sports (eg, soccer, hockey, basketball, football), which typically have higher participation among males than females.[15-17] Studies have also cited the importance of genetic influences in both types of impingement.[18]

The goal of our cross-sectional retrospective cohort study is to determine the prevalence of cam lesions in female patients who have symptomatic intra-articular hip pain without signs of arthritis. We hypothesized that the incidence of cam lesions in women presenting with FAI is higher than typically reported in the orthopedic literature.

MATERIALS AND METHODS

For our cohort, we considered new female patients presenting to the senior author's clinic with a chief complaint of "hip pain" between December 2006 and January 2013. Inclusion criteria included age under 65 years, Tönnis arthritis grade under 2, adequate anteroposterior (AP)-pelvis and lateral hip radiographs, and a clinical history and exam consistent with intra-articular hip pathology. We excluded patients who had a history of hip dysplasia or a history of hip surgery. Stiffness, groin pain, and popping or catching during hip flexion activities for greater than 3 months were considered necessary for diagnosis. We required an exam that was able to reproduce these symptoms in hip flexion.[19]

We reviewed the initial screening radiographs for each patient, documenting Tönnis grades[20] and measuring alpha angles on all available AP-pelvis, cross-table lateral, frog-leg lateral, and 90° Dunn lateral radiographs, as described by Notzli et al[21] (Figure 1). The center of the femoral head, the central axis of the femoral neck, and the resultant alpha angle were determined using the measurement tools available in the MedVIEW Picture Archive Communication System (PACS) software (Aspyra, West Lake Village, California). We used the largest alpha angle to represent the patient's cam deformity. For each patient, we collected demographic data, including age, ethnicity, and body-mass index (BMI).

In order to evaluate the prevalence of cam-type deformity, we classified all patients according to the criteria defined by Gosvig et al (pathologic > 57° and borderline 51°-56°).[22] Additionally, patients were classified as having subtle (46°-50°) and very subtle (43°-45°) lesions. We defined a normal alpha angle to be ≤ 42°.[21]

We performed a Pearson's correlation test between alpha-angle measurements and

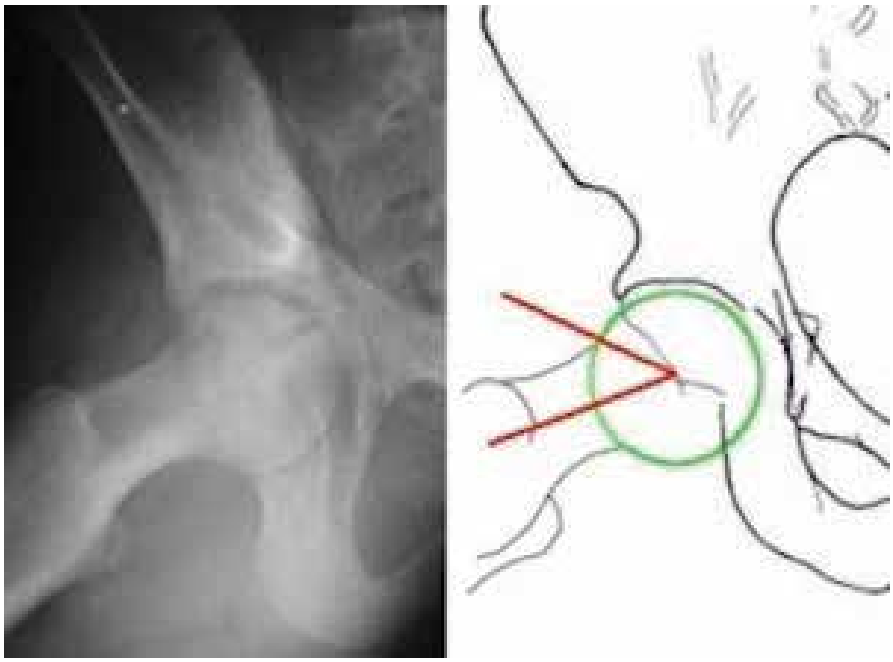

FIGURE 1. To find the alpha angle, draw a line from the center of the femoral head to the center of the femoral neck and a second line from the center of the femoral head to the point at which the femoral head loses its sphericity. The angle is found between them. Image used with the permission of Orthopaedic Studio.

age as well as alpha-angle measurements and BMI. We performed a student *t* test to assess for correlations between alpha-angle measurements and ethnicity. Measurements were performed by 2 experienced physicians. An interobserver correlation coefficient (ICC) was found between the 2 sets of measurements. *P* values of < .05 were considered significant. All statistical tests were performed using SPSS software for Windows, version 13.0 (SPSS, Chicago, Illinois).

RESULTS

A total of 969 females presented to the senior author's clinic between December 2006 and January 2013 with a chief complaint of hip pain, and we included 438 of them in the study. The mean age was 36 years old (range, 13-64). The mean alpha angle was 47.72° (SD ± 12.43°). The mean BMI was 24.02 ± 4.96 (see Table 1). Four hundred thirty-eight AP-pelvis, 169 frog-leg lateral, 40 cross-table lateral, and 276 90° Dunn lateral radiographs were reviewed. Thirteen percent of patients were categorized as pathologic; 17%, borderline; 15%, subtle; 18%, very subtle; and 37%, normal (see Table 2). Forty-two percent of patients had a lesion categorized as subtle or greater, and 68% of patients had a lesion very subtle or greater. The AP-pelvis radiograph identified a pathologic lesion in 6% of the cohort, and the lateral views identified a pathologic lesion in 12% of the cohort.

There was no correlation ($R = 0.17$) between patients' age and size of cam lesion. There was no correlation ($R = 0.05$) between patients' BMI and size of cam lesion. There was no difference between ethnicity of patient and size of cam lesion ($P = .10$).

The interobserver correlation coefficient for alpha-angle measurements was found to be 0.84.

DISCUSSION

FAI is a pathologic condition recently described by Ganz et al.[3] There is abnormal contact between the femoral head and acetabulum leading to hip pain, labral tears, chondral lesions, and early OA. We have described 2 types of FAI: cam type

TABLE 1. Demographic Information for Female Patient Cohort

Age (yr): mean (range)	36 (13-64)
Height (in): mean (SD)	65.33 (8.67)
Weight (lbs): mean (SD)	145.86 (37.09)
BMI (kg/m^2); mean (SD)	24.02 (4.96)
Race (%)	
White	431 (98%)
Nonwhite	7 (2%)

TABLE 2. Distribution of Alpha-Angle Classes for Female Patient Cohort

Classification (alpha angle)	Number (%)
Pathologic (>57°)	64 (13%)
Borderline (51°-56°)	83 (17%)
Subtle (46°-50°)	74 (15%)
Very subtle (43°-45°)	89 (18%)
Normal (≤42°)	182 (37%)

and pincer type. Cam impingement has traditionally been reported as a disease of young men, while pincer impingement has traditionally been reported as a disease of middle-aged women.[1,8-11] The notion that cam lesions occur predominantly in young males has been confirmed by recent studies[7,9-11]; however, these studies are cross-sectional evaluations of asymptomatic patients and are not focused on female patients with symptomatic impingement. The prevalence of female patients with cam lesions in these studies ranges from 0.0% to 5.4%. Our data suggest that there is a significantly higher prevalence of cam lesions found in symptomatic female patients than in asymptomatic female patients. In our cross-sectional cohort study of 438 female patients with symptomatic FAI, 13% had a pathologic cam lesion, 30% had a borderline cam lesion or greater, 45% had a subtle cam lesion or greater, and 63% had a very subtle cam lesion or greater. To our knowledge this is the first study to report the prevalence of cam deformity in female patients with symptomatic intra-articular hip pain.

When doctors assume that cam lesions occur only in young males, they risk overlooking cam impingement as a source of intra-articular hip pain in women. Femoral osteochondroplasty has been shown to be an effective treatment of cam impingement, but there is a significant learning curve associated with this technically challenging operation.[23] Furthermore, the leading cause of revision surgery is an inadequate cam resection,[24,25] underscoring the importance of recognizing and treating symptomatic cam impingement in women. Our study helps disprove the notion that cam lesions are only found in young males, and moreover, makes the case for the need to consider cam impingement in all symptomatic females, especially because lack of treatment will cause residual pain and lead to additional surgery.[24,25]

The alpha-angle cut-off of 42° chosen for normal morphology in females is based on the classification by Gosvig et al and Notzli et al.[21,22] This is a conservative threshold compared to the non-gender-specific threshold of 50.5° used in other studies.[21,26-28] To this point, the clinical relevance of subtle (46°-50°) and very subtle (43°-45°) lesions has not been established. Radiographic classifications need to be modified to reflect gender differences. Abnormal alpha-angle thresholds in females need to be lowered compared to male patients to reflect gender-specific pathomechanisms such as mixed impingement patterns, range of motion differences, and differences in hip girdle musculature.[29,30] Further studies are required to assess the extent of intra-articular pathology associated with these types of lesions and how these correlate with the risk of developing osteoarthritis.

This study is retrospective and cross-sectional, which presents limitations. Therefore, no firm causal inferences can be made. Prospectively collected data from long-term follow-up of cohorts with both genders could clarify the clinical relevance of our findings and whether different degrees of cam deformities or mixed types of FAI lesions are associated with increased risk of developing symptomatic hip OA. This study also lacks a concomitant evaluation for pincer lesions. Therefore, we cannot make an assessment of the prevalence of potential mixed lesions of pincer and cam impingement and the possible clinical relevance of mixed lesions. Our findings are based on the largest alpha angles measured from all available radiographs, which minimizes the risk of missing subtle lesions in different planes. Some radiographs were unavailable within our PACS system. If we had had access to these radiographs or radial oblique reformatted imaging, our incidence of alpha angles would have been larger.

CONCLUSION

We find that in female patients with symptomatic FAI there is a higher prevalence of cam lesions compared to the previously reported prevalence in asymptomatic females. We need to lower gender-specific radiographic alpha-angle thresholds in order to diagnose cam lesions in females. Future studies are required to assess this prospectively and establish the clinical relevance of these findings. ■

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

"PATIENTS TREATED WITH IRRIGATION AND DEBRIDEMENT WITH COMPONENT RETENTION AND A DUAL-ANTIBIOTIC REGIMEN HAD BETTER OUTCOMES THAN PATIENTS TREATED WITH A SINGLE-ANTIBIOTIC REGIMEN."

Single- vs Dual-Antibiotic Therapy for Periprosthetic Joint Infection

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Periprosthetic joint infection (PJI) is a devastating complication of total joint arthroplasty. Doctors generally treat acute infections with surgical debridement, including component retention and intravenous (IV) antibiotics, whereas for chronic infections, 2-stage exchange arthroplasty with antibiotic treatment remains the gold standard. Prior studies have shown varying success with antibiotic treatment, debridement, and component retention[1-6]. The inconsistency of results could be due to variables in diagnosis, treatment protocols, and outcome measurements. Open debridement with component retention provides a treatment option with lower morbidity and is attractive for surgeons and patients facing PJI therapy.

Optimal antibiotic treatment has typically relied on joint aspirate cultures. However, the accuracy of these cultures is unknown, and patients may receive antibiotics prior to aspirate. Additionally, the species and virulence of the infecting organism have been shown to influence outcome. This is the case in particular with infections of *Staphylococcus aureus (S aureus)*—whether methicillin sensitive (MSSA) or methicillin resistant (MRSA): treatment results of acute periprosthetic knee infection have been worse compared with those of PJI infected with other organisms[6]. The antibiotic regimen typically includes single-antibiotic therapy based on culture results; however, in situations of PJI without positive cultures to guide treatment, prolonged treatment with broad-spectrum antibiotics may give the best chance at eradication of the infection.[1,5,7]

To our knowledge, therapy with 2 antibiotics and retention of components following debridement surgery for PJI has not been studied. Therefore, our goal is to compare outcomes of patients treated with a single antibiotic to those with a dual-antibiotic regimen combined with debridement surgery and retention of components for acute, acute hematogenous, and chronic PJI.

METHODS

From a prospectively collected single-surgeon database (RAB), we identified and retrospectively studied cases of PJI diagnosed between January 2011 and December 2012. We included patients who were diagnosed with a PJI using the Musculoskeletal Infection Society guidelines, [8] and those who underwent debridement with retention of components followed by treatment with IV antibiotics as the case study group. Retention of components with debridement included exchange of the femoral head and liner in total hip arthroplasty patients and polyethylene component exchange in total knee arthroplasty patients. We excluded patients if they did not have preoperative cell count or differential data, if they underwent immediate 2-stage arthroplasty exchange, or if they did not receive operative treatment. No patients were excluded for pre-existing comorbidities or other demographic data. Patients were excluded if they did not have clinical follow-up for at least 1 year postoperatively.

The data we collected included age, type of initial surgery (unicompartmental or total knee arthroplasty, total hip arthroplasty), and presenting complaint. We calculated time between primary arthroplasty and initial debridement surgery, as well as time until further surgical procedures. Joint aspirate data, including cell count and percentage of polymorphonuclear (PMN) cells were available in each case; however, preoperative serologic values of ESR (erythrocyte sedimentation rate), CRP (C-reactive protein) and WBC (white blood cell count) were available for only 13 patients, and therefore were not analyzed. We included type of antibiotic therapy both pre- and postoperatively and noted antibiotic treatment complications if they occurred. This study was approved by the Rush University institutional review board.

The largest single-antibiotic group consisted of patients who received cefazolin (+/– rifampin), ceftriaxone, vancomycin, or ertapenem postoperatively as their primary treatment. Patients treated with a combination of 2 antibiotics postoperatively were considered the dual-antibiotic group, regardless of which antibiotics they received or if they had been treated preoperatively with a single antibiotic. All patients' antibiotics were chosen utilizing an infectious disease specialist's consultation and complications of antibiotic treatment were recorded prospectively. Culture results, when available, directed antibiotic choices.

To measure the outcome, we assessed the need for subsequent debridement surgery and therapeutic antibiotic change due to persistent clinical symptoms. We defined

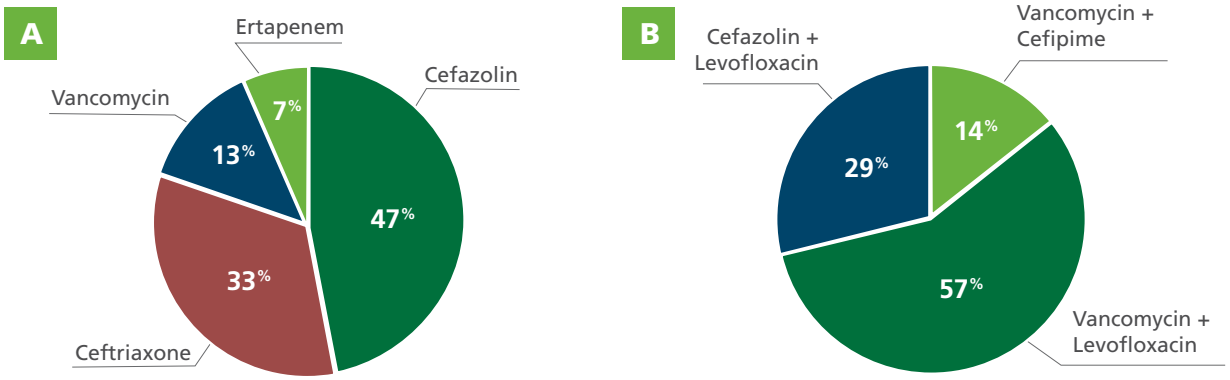

FIGURE 1. A, Single-antibiotic therapeutic agents (n = 15) and **B,** dual-antibiotic therapeutic agents (n = 7).

failure as the need for repeat debridement surgery. The need for chronic suppressive antibiotics was not considered a treatment failure, and patients with culture-positive PJI all received oral antibiotics following completion of intravenous antibiotic treatment.

We performed statistical analysis using SAS Enterprise Guide software (SAS Institute Inc., Cary, North Carolina). Due to the small sample size, we performed logistic regression and Fisher exact tests. We analyzed data at both the patient and the surgical-event level (7 patients underwent 2 or more surgical procedures). Significance was set at $P \leq .05$.

RESULTS

We identified 22 patients who met the inclusion criteria. Fourteen patients were placed on single-antibiotic courses (Figure 1A), and 7 were initially placed on combination antibiotics (Figure 1B). The median time between initial arthroplasty surgery and surgical debridement for infection was 34 days (range, 9 days-9 years). Patients greater than 21 days from surgery had a higher risk of failure of therapy, but this was not statistically significant ($P = .16$; OR, 1.079; 95% CI, 0.968-1.201). Fifteen patients (68%) were male, and the mean age of the patients was 59 years (range, 42-74) at the time of initial arthroplasty. Those patients who were older than 55 years at the time of surgery had a higher risk of failure ($P = .047$; OR, 1.176; 95% CI, 1.002-1.381). The initial arthroplasty was a total knee in 10 (45.5%), total hip in 11 (50%), and unicompartmental knee arthroplasty in 1 (4.5%).

We assigned designations of acute, acute hematogenous, and chronic based on time from initial arthroplasty to irrigation and debridement or time from presenting symptoms of acute infection to irrigation and debridement (acute hematogenous). Four weeks from the initial surgery was the cut-off level for an acute infection and from initial symptoms for acute hematogenous infection. Ten patients had acute infections (45.5%), 3 had acute hematogenous (13.6%), and 9 had chronic presentations (40.9%). Chronic infections had a higher risk of failure of therapy that was not statistically significant ($P = .17$; OR, 5.0; 95% CI, 0.499-50.068).

The most common infectious organism was MSSA (9 cases, 40.9%), and there were 0 MRSA infections (Figure 2). Other infectious organisms included *Streptococcus* species (4 cases, 18%), coagulase negative *Staphylococcus* (2 cases, 9%), and 1 case each of *Corynebacterium*, *Klebsiella*, *Haemophilus parainfluenzae*, and polymicrobial. Three cases had no growth from their cultures. The most common presenting symptoms were pain (12 cases), wound-healing issues (10 cases), erythema (7 cases), and fevers (7 cases). Five patients received antibiotics prior to surgery, 3 of whom were in the single-antibiotic group, 1 of whom went on to repeat debridement surgery. The cultures of only 1 of the 5 patients who received preoperative antibiotics had no growth.

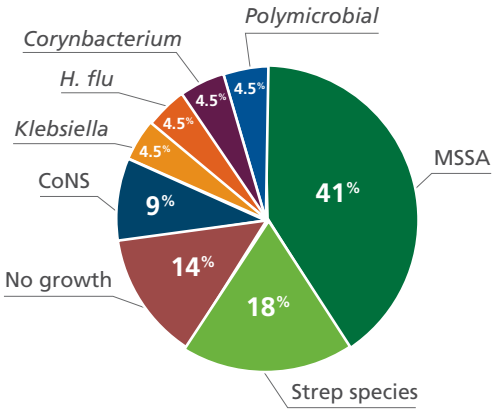

FIGURE 2. Pie graph representing infecting organisms (N = 22)

Abbreviations: MSSA, methicillin-sensitive *Staphylococcus aureus*; strep, *Streptococcus*; CoNS, coagulase-negative *Staphylococcus*; *H. flu*, *Haemophilus parainfluenzae*.

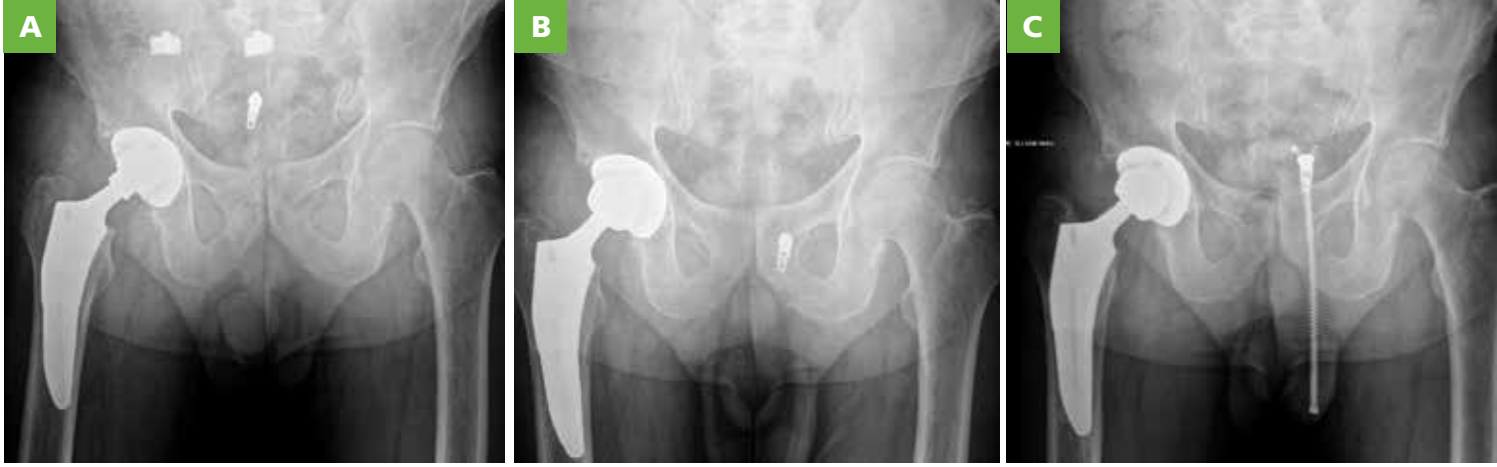

FIGURE 3. X-ray, right total hip arthroplasty (R THA) with chronic infection. **A,** 4 weeks postoperative from primary R THA, clinically diagnosed with infection. Patient returned to operating room for debridement with exchange of femoral head and acetabular liner ("retention of components"). **B,** 4 weeks postoperative from debridement with retention of components, patient now on intravenous (IV) antibiotic therapy. **C,** 18 months post-op from debridement. No evidence of component failure.

Five patients (22.7%) had failure of initial treatment, defined as need for repeat debridement surgery within the first year after initial surgical debridement and antibiotic treatment. All of these patients had a change in antibiotic therapy due to their second debridement, and 4 patients crossed over to dual-antibiotic therapy and were considered part of the dual-antibiotic group for post hoc analysis at the event level only. The average time between initial debridement and final debridement was 13 days (range, 8-18 days). Another patient had change of initial antibiotic therapy due to persistent wound drainage that resolved with change of antibiotic therapy.

Of the 7 patients initially treated in the dual-antibiotic group (31.8%), there were no failures—no repeat debridements or changes in IV antibiotic therapy. Figure 3 shows radiographic outcome of one such successful patient. Of the 4 patients whose initial therapy failed, which crossed them over to the dual-antibiotic group, there were no further failures of treatment. We could not use the traditional models of logistic regression (complete separation) because all outcomes in the dual-antibiotic group (at the patient and the event levels) were successful (complete separation), so we used Firth's penalized maximum likelihood estimation. However, there were still no statistically significant differences in the risk of failure in the single-antibiotic group (P = .13; OR, 10.9; 95% CI, 0.48-250). Eighteen patients (81.8%) required chronic oral antibiotic therapy. Six of the 22 total patients (27.2%) ultimately required removal of component and placement of static spacer and revision arthroplasty following IV antibiotic therapy (2-stage explant); however, none of the patients initially treated with a dual-antibiotic regimen required repeat surgery, therapeutic change, or had new organisms isolated. Thirteen of the 15 patients in the single-antibiotic group had complications: 5 required repeat surgery, 6 required therapeutic change, and 2 had a new organism isolated from repeat synovial fluid culture.

DISCUSSION

Although previous studies looking at outcomes of surgical debridement for PJI with retention of arthroplasty components have been encouraging,[1,3-6,9,10] the optimal treatment for PJI remains unclear, although surgical debridement and IV antibiotic therapy remain the cornerstones of treatment for acute, acute hematogenous, and chronic infections. The possibility of component retention in any of these settings is an attractive one for surgeons and patients seeking to avoid the possible morbidity and surgical complexity of 2-stage component-exchange surgery. Our goal was to evaluate the outcomes of treatment with debridement and dual-antibiotic therapy and component retention in retrospective series of cases treated by a single surgeon.

Patients treated with irrigation and debridement with component retention and a dual-antibiotic regimen had better outcomes than patients treated with a single antibiotic regimen. Although statistical significance was reached only with age at time of surgery as a variable, there were no treatment failures, defined as return to the operating room following debridement and component retention, with dual-antibiotic therapy. Patients who initially started on dual-antibiotic therapy had no failures of treatment, and patients who initially failed single-antibiotic therapy and crossed over to dual-antibiotic therapy had no further failures.

One of the primary limitations of this study is its retrospective nature. We were strict in our inclusion criteria of patients who underwent the same surgical treatment and who differed in their course of antibiotic therapy. However, without controlling the groups for other variables such as infecting organism, acute or chronic infection or even for hip vs knee arthroplasty, the results of this case series point to the need for further research in these areas.

In this case series, the small cohort of patients and single-surgeon database also raise the question of whether the results here are generalizable to other practice settings. Despite efforts to use this high-volume surgeon's clinical database to identify all reoperations in this series and confirm all laboratory values, treatment details, and identifiable demographics, a larger cohort of patients with tighter controls would certainly yield more statistically significant results. Prospective data to this effect are being collected at this time, which can hopefully guide future treatment of patients and surgeons facing periprosthetic joint infections. ■

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

"TRAINING ON ARTHROSCOPIC SIMULATORS IMPROVES PERFORMANCE ON ARTHROSCOPIC SIMULATORS... THERE IS LITTLE EVIDENCE TO CORRELATE PERFORMANCE ON SIMULATORS WITH PERFORMANCE IN THE OPERATING ROOM."

The Utility of Modern Arthroscopic Simulator Training Models: A Systematic Review

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PREVIOUS PUBLICATION

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Increased restrictions on work hours of orthopedic residents[1] may adversely affect development of advanced surgical skills, including arthroscopy. Several survey-based studies have discussed the potential decline in operative experience caused by, at least in part, work-hour restrictions.[2-7] Immerman et al[7] found from a national survey, following the 2003 changes, that both junior and senior residents, as well as program directors, felt that the new rules did not increase operative time or improve operative experience. In a different survey, Zuckerman and colleagues,[6] concluded that the majority of faculty and residents thought the work-hour changes negatively impacted their operative experience.

Concern over the impact of work hours on operative time and experience may lead to decreased confidence in surgical skills and performance in the operating room. The development of the hand-eye coordination and dexterity required to perform safe, effective, and efficient arthroscopic operations typically requires hours of experience in the operating room. Simulator-based training models have seen increasing popularity as an alternative method of obtaining surgical skills (Figure 1), as evidenced by a recent increase in publications describing the outcomes of modern arthroscopic simulator training.[8-10] Arthroscopic models exist for nearly every joint, and yet the actual clinical applicability of arthroscopic training models remains unclear for orthopedics. In contrast, studies have established the correlation between training on a simulator and improved performance in the operating room for general surgery.[11-15] In 2013, Gallagher et al[11] performed a randomized clinical trial comparing performance of both novices and experienced laparoscopic surgeons either with or without virtual-reality laparoscopic simulation. Regardless of experience level, subjects in the simulation group performed significantly better than the controls.

The purpose of this study was to systematically review the published literature on modern arthroscopic simulator training models to see if skills learned on the models are transferred to the operating room. We hypothesized that subjects who underwent arthroscopic simulator training would demonstrate objective improvement in simulator and operating room technical skills compared to those who had no training.

METHODS

We conducted a systematic review of publicly available evidence using Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines with a PRISMA checklist.[16,17] Three independent reviewers completed the search on August 5, 2013. We searched multiple databases, including Medline (Pubmed), Cumulative Index to Nursing and Allied Health Literature (CINAHL), and Cochrane Central Register of Controlled Trials. We used the search terms *arthroscopy*, *arthroscopic*, *simulation*, and *simulator*.

Our inclusion criteria were English-language studies incorporating the terms *arthroscopy* OR *arthroscopic* AND *simulation* OR *simulator*. Exclusion criteria included non-English language studies, biomechanical studies, novel technique studies, perception-based studies, scientific meeting abstracts/proceedings, and systematic reviews/meta-analyses. Levels of Evidence I, II, III, and IV were deemed inclusive (per the Oxford Centre for Evidence-Based Medicine used by the American version of the *Journal of Bone and Joint Surgery Arthroscopy*)[17] if published in the English language. Articles that were designated e-published only, e-published ahead of print, as well as print journal articles were acceptable and considered for inclusion.

FIGURE 1. Photograph of **A,** knee and **B,** shoulder arthroscopic models for simulation training.

We cross-referenced the references within included studies for potential inclusion if omitted from the initial search. Figure 2 illustrates the search methods the reviewers utilized to generate the final studies for inclusion and analysis.

We collected data including participant demographics, simulator model, type and number of tasks, method of analysis, and results of training, when available. We analyzed specific information on the participants, including level of training and history of prior experience performing arthroscopic surgeries. Other factors that we assessed included study country of origin, author conflict of interest, and single-center versus multicenter study design. We performed descriptive statistical analysis for each study and variable analyzed.

RESULTS

We identified 62 studies with the initial search. We found 1 additional study by cross-referencing the references. We excluded 44 studies, including non-English language papers (*N* = 2), abstract-only listing (*N* = 1, which was also an unrelated topic), review articles (*N* = 6), biomechanical studies (*N* = 12), studies analyzing novel techniques (*N* = 4), studies analyzing validity of simulator models (*N* = 2), studies discussing topics unrelated to orthopedic/arthroscopic simulator training (*N* = 14), and studies analyzing subject/examiner perception of simulator training (N = 3). A total of 19 studies met inclusion criteria and underwent further analysis (Figure 2). Of those, 9 studies (47%) investigated shoulder models,[18-26] 9

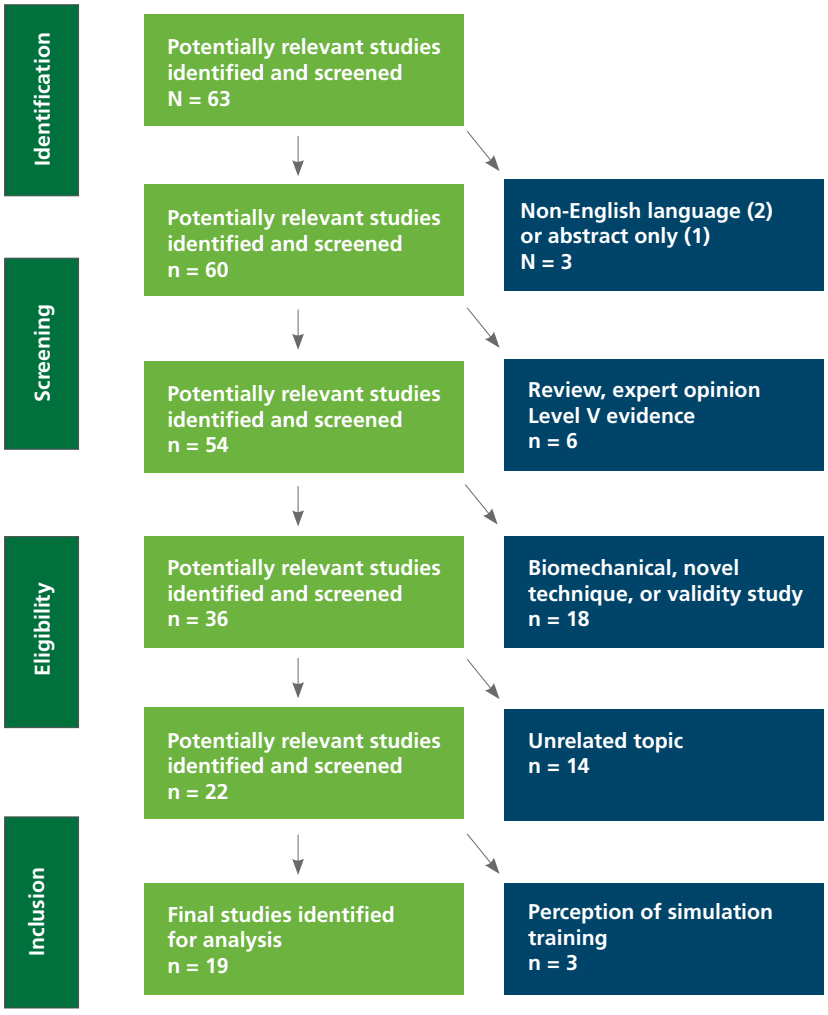

FIGURE 2. After applying exclusion criteria using PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines within Medline database, we identified 19 studies for final analysis.

TABLE 1. Summary of Shoulder Arthroscopy Simulator Studies

Author, Year, Country	LOE	No. of Subjects	Participant Details	Simulator Type	Practice Session Given?	No. of Tasks	Time Allowed
Smith 1999 UK	IV	18	• 5 OS • 6 non-OS with MIS experience • 6 MS	Procedicus arthroscopy simulator	No	4	Unlimited
Smith 1999 UK	IV	78	• 35 MS interviewing for ortho • 22 OR interviewing for sports • 21 OS	Procedicus arthroscopy simulator	Yes, 5 min	11	n/a
Srivastava 2004 USA	IV	35	• Group 1: novices • Group 2: 1-50 previous scopes • Group 3: 50+ previous scopes	Procedicus arthroscopy simulator	Yes, unlimited time	3	• Task 1: unlimited • Task 2: unlimited • Task 3: 5 min
Gomoll 2007 USA	IV	43	• 8 novices • 11 junior OR • 14 senior OR • 10 fellows/ attendings	Procedicus arthroscopy simulator	n/a	11	Unlimited
Gomoll 2008 USA	IV	10	• 10 OR	Procedicus arthroscopy simulator	n/a	10	Unlimited
Howells 2009 UK	II	6	• 6 fellowship-trained lower-limb OS	Alex Shoulder Professor benchtop simulator	Yes, 5 min	1	Unlimited
Martin 2011 USA	II	19	• 15 OR • 4 OS	Insight Arthro VR	Yes, 5 min	n/a	3 minutes
Martin 2011 USA	IV	27	• 27 OR (all years)	Insight Arthro VR	Yes, 5 min	3	Unlimited
Henn 2013 USA	I	17	• 17 MS randomized to either simulator or no simulator training	Procedicus arthroscopy simulator	n/a	5 on cadaver, 11 on simulator	n/a

Abbreviations: LOE: level of evidence; OS, orthopedic surgeon; OR, orthopedic resident; MS, medical student; MIS, minimally invasive surgery; PGY, postgraduate year; VR, virtual reality

Tasks Assessed	Attempts Given	Compared Between Different Levels of Training	Outcomes
• Identify anatomic structures • Find targets	1	Yes	OS could locate anatomic structures faster than MS, but the path they took wasn't necessarily more direct, and they made the same number of collisions as the novices
• Time • Path ratio • Collisions • Injuries	1	Yes	• Overall performance significantly better in OS • No difference in probe collisions between groups
• Hook manipulation • Anatomic identification • Scope navigation	1	Yes	• No difference in identification • Group 3 best at hook manipulation (group 2 better than group 1) • Each group improved time • Group 3 best at scope navigation (no difference between 2 and 1)
• Probing • Time • Collisions • Velocity • Distance traveled	6	Yes	• More experienced groups had better path length and time compared to less experienced groups • Number of probe collisions was significantly different between all groups except junior and senior OR • Velocity better in experienced groups compared to less experienced groups
• Probing • Time • Collisions • Velocity • Distance traveled	6	No	• 3-year follow-up of OR to evaluate simulator skills after additional residency training • Improvements in all parameters
Throw 1 Bankart suture	3x/session; 4 sessions; 1 session/ week; repeat after 6 months	No	• Time to complete tasks improved over the first set of 4 sessions and the second set of 4 sessions • No change from baseline to 6 months • Conclusions: no retentio
Probing	3	Yes	• Scoped simulator and then at least 2 weeks later scoped cadaver; compared time to complete tasks on each • Performance on simulator strongly correlated with cadaver performance • Experts were faster than novices
n/a	3	Yes	• For every 1-year increase in PGY, there was a 23-second decrease in time • For every shoulder arthroscopy case performed as a resident, there was a 0.6 second decrease in time • Total number of arthroscopies performed and the total number of surgical cases completed during residency prior to completing the simulator task correlated with shorter times
• Controlling camera • Standard series of tasks with probe	n/a	No	• All subjects completed baseline scope on cadaver, randomized to training or no training, and then all repeated cadaver testing • No difference in baseline skills • Simulator group with significantly improved scores compared to baseline (speed, subjective performance) AND compared to controls (speed) • No difference between groups with subjective scores

TABLE 2. Summary of Knee Arthroscopy Simulator Studies

Author, Year, Country	LOE	No. of Subjects	Participant Details	Simulator Type	Practice Session Given?	No. of Tasks	Time Allowed
McCarthy 1999 UK	IV	22	•10 post-grad scientists • 6 OR • 6 OS	SKATS	Yes, "brief"	1	Unlimited
Sherman 2001 UK	IV	43	43 OR	VE-KATS	Yes, unlimited time	n/a	Unlimited
Strom 2004 Sweden	I	28	28 MS randomized to either simulator or no simulator training	Procedicus VA Knee Simulator	No	6	Unlimited
Bliss 2005 USA	IV	9	9 psychology graduate students	Procedicus VA trainer	Yes, 15 minutes	11	Unlimited
McCarthy 2006 UK	IV	23	• 5 OS with 5-50 previous scopes • 7 OS with 50-100 previous scopes • 11 OS with >1000 previous scopes	SKATS	Yes, duration unknown	5	Unlimited
Howells 2008 UK	I	20	20 junior OR randomized to either simulator or no simulator training	Arthroscopy knee benchtop simulator	n/a	n/a	n/a
Tashiro 2009 Japan	II	30		Sawbones knee simulator model	Yes, 5 minutes	2	Task 1: 5 min Task 2: 6 min
Escoto 2012 Canada	IV	15		High fidelity physical knee arthroscopy simulator	No	14	3 min
Jackson 2012 UK	I	19	19 OR randomized to 3 groups	Sawbones knee simulator model	No	1	Unlimited

Abbreviations: LOE: level of evidence; OS, orthopedic surgeon; OR, orthopedic resident; MS, medical student; SKATS, Sheffield Knee Arthroscopy Training System; VA, Virtual Arthroscopy, VE-KATS, Virtual Environment Knee Arthroscopy training system

Tasks Assessed	Attempts Given	Compared Between Different Levels of Training	Outcomes
Identify 10 structures	1	Yes	• Experienced surgeons had fewer collisions and were faster at completing tasks
Identify anatomic landmarks	1	Yes	• Poor correlation between year of training and performance on simulator
Assess for probes of 6 locations: • Time • Economy • Collisions • Score	1	No	• No difference in identification • Group 3 best at hook manipulation (group 2 better than group 1) • Each group improved time • Group 3 best at scope navigation (no difference between 2 and 1)
Identify 10 anatomic landmarks	1	No	• Practice session followed by test session 1×/ day for 5 consecutive days • Tested 4 weeks later • Correctly identified 7.7 structures in the first session; 9.5 in final session • Collided 53.5 times with simulated tissues in first session; 13.2 times in final session • No significant decrease over 4 weeks
Locating loose bodies	10	Yes	• 10 separate sessions over 5 weeks • The more experienced OS significantly better and faster at locating loose bodies
Identify, Probe	n/a	No	• Simulator groups received18 sessions of training • Simulator group performed better (speed, efficiency) in operating room compared to no-simulator group
Joint inspection, probing, partial mx	1	Yes	• More experienced subjects performed better (faster, less force exerted on joint, more direct path of their instruments); • OS did better than OR who did better than the trainees
Probing, shaving, burring	n/a	Yes	• Novices applied uneven force when completing shaving and burring tasks compared to experts; • Novices slower and less accurate with probing
Meniscal repair	12	No	• All OR initially perform meniscal repair on simulator 12x over 3 weeks A: meniscal repair 1x/month for 5 months B: meniscal repair 1x total at 3 months C: no simulation for 6 months • At 6 months, all groups perform meniscal repair 12x over 3 weeks • All OR improved with each meniscal repair at initial phase • No groups with significant decrease in ability to perform meniscal repair at 6 months

TABLE 3. Summary of Hip Arthroscopy Simulator Studies

Author, Year, Country	LOE	No. of Subjects	Participant Details	Simulator Type	Practice Session Given?	No. of Tasks	Time Allowed
Pollard 2013 USA	II	20	20 OR randomized into lateral and supine groups for diagnostic arthroscopy	Hip arthroscopy benchtop simulator	No	12	Unlimited

Abbreviations: LOE, level of evidence; OR, orthopedic resident

(47%) evaluated knee models,[27-35] and 1 (6%) evaluated a hip model.[36] These studies are described in detail in Tables 1-3. (To view Tables 1-3, go to orthopedicsjournal.)

Simulators varied by study and included the Procedicus arthroscopy simulator (Mentice Corp, Göteborg, Sweden) in 6 of 9 shoulder studies,[18-21,25,26] the Alex Shoulder Professor benchtop simulator (Sawbones Europe, Malmö, Sweden) in 1 of 9 shoulder studies,[22] and the Insight Arthro Virtual Reality (VR) (Immersion, San Jose, California) in 2 of 9 shoulder studies.[23,24] For the knee, the Procedicus Virtual Arthroscopy (VA) trainer, (Mentice Corp, Göteborg, Sweden) was used in 2 of 9 studies,[32,34] an arthroscopy knee benchtop simulator (Sawbones Europe, Malmö, Sweden) in 3 of 9 studies,[22,29,31] the Sheffield Knee Arthroscopy Training System (SKATS, University of Sheffield, Sheffield, UK) in 2 of 9 studies,[30,35] a high-fidelity physical knee arthroscopy simulator in 1 of 9 studies,[27] and the Virtual Environment Knee Arthroscopy training system (VE-KATS) in 1 of 9 studies.[33] The hip study used a hip arthroscopy benchtop simulator (Sawbones Europe, Malmö, Sweden).[36]

The average age of the 465 subjects was 30 years (range, 21-55). They had various degrees of experience including students, orthopedic residents, fellows, and faculty. Twelve studies[19,20,23-27,30,31,33,35,36] (63%) compared task performance between participants of different experience levels with 100% reporting a positive correlation between experience level and simulator performance. A total of 8 studies[1,18,22,28-30,32,34,36] (42%) evaluated task performance before and after simulator training, with 6[18,28-30,32,36] (75%) of these studies showing improvement after training; 1 study[34] (6%) noted no difference in performance after 1 hour of training. Common arthroscopic tasks included probing identified structures, throwing a suture, hook-manipulation of identified structures, and shaving/burring. Of the shoulder studies, only 2 studies tested subjects on the simulator both before and after training, with 1[18] showing improvement in speed and the other[22] showing improvement within each training sessions, but not between training sessions. Of the knee studies, 5 tested subjects on the simulator both before and after training, and 80% showed improvement in task performance following training[28-30,32] while the study by Strom et al[34] showed no improvement in simulator task performance after 1 hour of training. The single study analyzing hip arthroscopy did evaluate performance on the simulator both before and after training and showed improvement within the training sessions.[36]

A single study (6%) commented on improved operating room performance after simulator training.[28] In their study, Howells and colleagues[28] randomized 20 junior orthopedic residents to either receive a standardized protocol of knee arthroscopy simulator training in 3 sessions of 6 simulations or no training at all. A blinded senior surgeon in the operating room then evaluated all residents on their ability to perform a diagnostic knee arthroscopy on an actual patient. The authors noted a statistically significant improvement in the simulator group compared to the control group.

No studies commented on the number of training sessions needed to translate technical skills learned on the models to the operating room, though the single study utilizing a hip model examined the learning curve of performing diagnostic hip arthroscopy in either the supine or lateral position.[36] Only 2 studies[18,23] (12%) incorporated the use of cadaveric specimens as part of their methodology. There were 4 level I studies,[18,28,29,34] 4 level II studies,[22,23,31,36] and 11 level IV studies.[19-21,24-27,30,32,33,35]

Seven studies[18,23,24,26,29,31,36] listed potential conflict-of-interest (COI) information on the manuscript. Four studies[20-22,28] listed no potential COI, while the remaining 9 studies[19,25,27,30,32-35] did not provide information on COI. Of the 7 studies reporting COI information, 5 studies [18,26,29,31,36] reported conflicts related to the topic, with all 5 receiving research grants supporting simulation studies. Of these 5 studies, only 1 demonstrated a direct benefit from simulator training. Henn[18] rreported that subjects who underwent simulator training significantly performed an arthroscopic probing task on cadavers at a faster speed than controls. Pedowitz[26] reported significantly superior shoulder simulator performance in more experienced subjects but did not compare to controls; Tashiro[31] reported similar findings in a knee arthroscopy simulator model. Pollard[36] showed improvement in hip arthroscopic task performance over the time period of a single session but did not compare to controls or analyze the results over time. Finally, Jackson[29] compared 3 randomized groups of orthopedic residents who all initially performed a simulated arthroscopic meniscal repair and then either had monthly simulator training, a single session of training, or no simulator training. Overall, the authors found no loss of skill in all 3 groups over a 6-month period, including the group of residents without any simulator training.

DISCUSSION

Our principal findings were that training on arthroscopic simulators improves performance on arthroscopic simulators; more experienced subjects perform better on arthroscopic simulators than do less experienced subjects; and there is little evidence to correlate performance on simulators with performance in the operating room. A summary of the key findings is presented in Table 4.

Since popularization of arthroscopy in the 1960s,[37] advances in technique and instrumentation have revolutionized our

Tasks Assessed	Attempts Given	Compared Between Different Levels of Training	Outcomes
Diagnostic arthroscopy	12	Yes	• Both groups improved significantly • Lateral group initially slower but caught up by attempt #9 • Junior OR initially worse but performed at level equal to senior OR by end of study

ability to diagnose and treat a wide variety of intra-articular pathology. To be performed safely and effectively, arthroscopic surgery requires unique skills, most of which require substantial hands-on training.[38] These skills include visual-spatial coordination to interpret 3-dimensional structures from 2-dimensional camera images, hand-eye coordination to triangulate and adjust the visual field, and psychomotor skills to perform the desired procedure without causing injury.[26,38,39] In open surgery, training often occurs "on the job," with residents learning skills by assisting. With increasing restrictions on work hours, acquisition of arthroscopic skill is more challenging, often consisting of unsupervised "trial and error" training that is not only inefficient, but potentially harmful for patient care.[26] Residents may ultimately be spending less time obtaining these vital skills in the actual operating room and may find their arthroscopic skill set unacceptably deficient.[40,41] With the constant evolution of complex, advanced arthroscopic techniques, practicing orthopedic surgeons must learn new skills or procedures in a safe and controlled environment. Given their already demanding time constraints, practicing surgeons are often forced to "learn" novel arthroscopic skills by simply attending a course or visiting another institution as an observer. While educational, the limited, if any, hands-on training offered in these situations is insufficient to adequately allow surgeons to develop a level of proficiency that would make them comfortable in the operating room. Simulator training provides an opportunity for surgeons to practice the new skills learned in such courses, but a standardized objective measurement scheme to evaluate performance (and improvement) based on simulator use is necessary. There is also potential for simulators to test aptitude of potential future surgeons. Similar to any surgical skill, the development of arthroscopic surgical skills is clearly dependent on a multitude of factors, including the quality and quantity of training hours. On the other hand, arthroscopic skills may also involve inherent potentials that cannot be taught, and simulators may be beneficial in identifying individuals with these inherent, natural abilities.

Overall, alternative methods for garnering these essential arthroscopic skills are imperative, and simulation-based approaches are becoming more prevalent in residency programs. In fact, in July of 2013, the Accreditation Council for Graduate Medical Education (ACGME) introduced a drastic change in requirements for postgraduate year 1 (PGY-1) orthopedic surgery residents,[42] requiring all interns to complete a formal skills curriculum, including the development of basic arthroscopy skills. This new curriculum, and specifically the requirement for surgical skills training, reflects change in educational focus within orthopedic surgery residency programs. Meanwhile, though it seems intuitive that arthroscopic simulators should play a role in development and objective evaluation of psychomotor skills, the translatability of simulator-learned skills to the operating room remains undetermined.

Responding to reports in the general surgery literature,[11-15] the American Board of Surgery recently implemented the requirement for surgeons seeking board certification to successfully complete the Fundamentals of Laparoscopic Surgery (FLS) training program.[43] The FLS is an education model that was designed for surgical trainees and practicing physicians "to learn and practice laparoscopic skills to have the opportunity to definitely measure and document those skills" whose use directly translates to improved operative performance.[43] For example, Stefanidis and colleagues[14] conducted a randomized trial comparing operating room skills in a group of inexperienced subjects randomized to either receive FLS training (experimental group) or not (control group). The authors reported that subjects who participated in the FLS suturing task module demonstrated significantly improved operative performance.

Only 1 study in our review commented on improved operating room performance after simulator training.[28] The authors evaluated the simulator-trained junior residents and untrained controls, using the Orthopaedic Competence Assessment Project score intraoperatively. This scoring system, which has been incorporated into the United Kingdom's competency-based surgical training structure, includes 14 criteria, 9 of which are relevant to arthroscopy. The authors noted a statistically significant improvement in the simulator group compared to the control group.

A perhaps more preferred approach to arthroscopic training utilizes cadaveric specimens (Figure 3), which are clearly best suited to simulate all facets of human tissue, especially with regard to appearance, texture, and quality.[44] Only 2 studies in this review, both analyzing shoulders, incorporated cadaveric models. In 2013, Henn et al[18] randomized 17 first-year medical students to either receive simulator training (experimental group) or not (control group). All students first completed a baseline arthroscopy on a cadaveric shoulder and then received either simulator training or no training. All students then repeated the cadaver arthroscopy 3 months after the initial arthroscopy. The simulator group received 6 training sessions on the model over the 3-month time period. The authors then evaluated subjects on the basis of camera-control and probing skills. There were no significant differences in baseline skills between the groups; however, while both groups improved, the simulator group was significantly faster at completing the tasks compared to the control group. Interestingly, there was no difference between the groups with subjective assessment of technical performance. Martin and colleagues[23] also evaluated arthroscopic

TABLE 4. Key Points Regarding Modern Arthroscopy Simulator Training Models

Residents are concerned about decreasing operative experience with increasing work-hour restrictions.
Simulation may be helpful for residents and practicing surgeons alike.
Training on arthroscopic simulation models improves performance on models.
More experienced subjects perform better on models than less experienced subjects.
Transferability of training on simulator models is unclear.
Author conflict of interest with simulator models does not impact study results.

FIGURE 3. Residents practicing arthroscopic skills on cadaveric specimens.

task performance in simulator and cadaveric models. In this study, 15 orthopedic residents and 4 orthopedic surgeons all underwent an orientation and 5-minute practice session with the Insight Arthro Virtual Reality (VR) (Immersion, San Jose, California) shoulder simulator, followed by testing on the model with probing as the main task. The authors then tested each subject on a cadaveric model at least 2 weeks following the simulator model test. The authors noted a strong correlation with performance time on the simulator and performance time on the cadavers and determined the time required to complete tasks on the simulator to be a significant predictor of the time required to complete the same tasks on the cadaver. By using cadaveric shoulders, this study suggests that simulator performance may correlate with actual operative performance.

The majority of these studies demonstrate that practicing arthroscopic skills via simulator training improves arthroscopic skills on the simulator. The clinical relevance of improving arthroscopic skills on a simulator remains undetermined. Interestingly, other variables, including the experience level of the trainee (student, resident, fellow, or attending), as well as the actual number of procedures performed prior to simulator training, were also shown to be correlated with simulator performance in the majority of the studies. Twelve of the 19 studies[19,20,23-27,30,31,33,35,36] compared task performance between participants of different experience levels, with all 12 studies showing a positive correlation between experience level and simulator performance, suggesting that actual operative experience, as opposed to training on the simulator, is correlated with improved simulator performance and/or the ability to get a more beneficial experience from the simulator training.

Both Smith et al[25] and Pedowitz et al[26] analyzed subjects from medical student to orthopedic surgeon experienced at arthroscopy. While both authors noted significantly superior simulator performance in the experienced groups, the number of injury collisions (number of times the probe or arthroscope contacted any tissue beyond a threshold force) was not significantly different. In contrast, Gomoll and colleagues[20] showed a significantly lower number of probe collisions in more experienced subjects compared to lesser experienced subjects (except between senior and junior residents). In this study, probe collision (in addition to average velocity) showed the largest improvement following training, suggesting that using a simulator early in training may be beneficial in development of skills to avoid collision.

In the only study of hip arthroscopy simulators available in the literature, Pollard et al[36] conducted an elegant, Level I study that evaluated performance of both junior (training years 1 and 2) and senior (training years 3 or above) residents in simulated hip arthroscopy in either the lateral or supine position. Trainees of all levels were randomized to simulation in either the lateral or supine position, and the task consisted of correctly probing multiple identified landmarks (multiple points on labrum, acetabular cartilage lesions, ligamentum teres) with the camera in both the anterolateral and anterior portals. Each subject probed all landmarks from 1 portal and then the other and repeated the process for a total of 3 times in weekly sessions for a total of 4 weeks (12 total sessions). The authors used motion analysis software (PATRIOT; Polhemus, Colchester, Vermont) to determine subject hand-path length, total number of hand movements, and time taken to complete the task. Both groups significantly improved in median time to perform the task ($P < .0001$), with the plateau for the learning curve reached after 9 training sessions in both groups, though the lateral group was slower. During weeks 1 and 2, the senior residents were superior to the junior residents in all 3 parameters; however, by the last week, there were no significant differences between the groups, with the exception of the juniors with improved distance traveled compared to the seniors. While this study identifies a learning curve for performance on the model, it makes no correlation to actual operating room performance; thus it is difficult to draw conclusions regarding the actual learning curve of hip arthroscopic simulation training.

While no studies were able to evaluate the true learning curve of simulator training as it relates to operative skills, the previously described assessment, as well as the study by Jackson et al[29], did determine a "learning curve" for mastering a specific skill on a specific model. Jackson and colleagues evaluated the ability of residents to perform meniscus repair on the arthroscopy knee benchtop simulator (Sawbones Europe, Malmö, Sweden). In this study, 19 residents initially performed a meniscal repair on the simulator 12 times over a period of 3 weeks and then were randomized to either perform a simulated meniscal repair 1 time per month for 5 months, 1 time total at 3 months, or no simulation for 6 months. At 6 months, all groups performed meniscal repair 12 times over 3 weeks, without significant differences. Some residents reached a plateau within 12 training episodes, while others continued to improve up to 21 episodes before achieving consistent performance. Interestingly, even the group who did not train at all during the 6-month time period between evaluations

TABLE 5. Limitations of Current Arthroscopy Simulator Training Models

There is significant variability among modern arthroscopic simulator models. - No current systematic approach - Not proficiency based - Not based on performance metrics
Efficiency and cost of training on current arthroscopy models are unclear.
Validation of arthroscopy models as a whole is not yet available.
Studies analyzing performance on models of the wrist and ankle are not yet available.
No clear correlation (yet) between model training and cadaveric operative performance.
No clear correlation (yet) between model training and actual operative performance.
No data available on learning curve.

demonstrated improvement and retention of skill and performance.

Modi et al performed a systematic review of 9 studies assessing the validity of computer simulation software as it relates to teaching arthroscopic skills.[45] In their study, Modi et al demonstrated that simulators with force feedback, haptic technology, and computer-generated outcome data produce high levels of internal consistency and reliability. The authors noted that the measures best able to discriminate skill level and user experience included time to task completion, distance traveled by probe, path taken by probe, and number of probe collisions, but they noted that additional work is needed to determine the ability of such training to transfer to the operating room.

When considering the potential influence of author/institution conflict of interest, only 58% of the studies listed either the presence or absence of COI. Of the 7 studies that listed potential conflict of interest (COI) information on the manuscript,[18,23,24,26,29,31,36] only 5 reported conflicts related to the topic,[18,26,29,31,36] and only 1 of these demonstrated a direct benefit in arthroscopic skill development from simulator training.[18] Given the substantial expense of the hard- and software components of arthroscopic simulators, it is essential to be aware of the potential for author bias in reporting outcomes. Nearly all sources of funding for these studies came from national or societal grants as opposed to industry, illustrating the desire of better understanding the potential role for simulators in arthroscopic skill development.

To our knowledge, this is the first study to analyze the operative translatability of arthroscopic surgical simulation training. This review, however, is not without limitations, most of which are inherent to the limitations of the studies it describes. Given the different methodology used in each of the studies, it was not possible for us to conduct quantitative statistical analysis of the studies as a whole, and instead we performed descriptive analysis. This type of analysis makes it difficult to draw statistical conclusions; however, given the variability in outcomes reported in each individual study, direct comparison was not feasible. The studies in this review vary with regard to level of evidence; however, we included multiple level I studies. The methodology of evaluating simulator task performance was extremely variable between studies, making it difficult to compare outcomes even between studies analyzing the same joint with the same simulator, thus introducing detection bias. Heterogeneity between the subjects (e, age, gender, and experience level) also contributed to bias. Finally, the lack of standardized performance measures confounds the conclusions drawn.

Overall, the study still has not answered the question of translatability of arthroscopic simulation trainers. Further, we have not analyzed the learning curve of simulation training, and specifically the number and timing (daily, weekly, monthly, etc.) of repetitions required to achieve proficiency, or, more importantly, maintain proficiency of these skills in the operating room. Similarly, methods for evaluating arthroscopic simulator performance are not standardized, making it difficult to compare one simulation system to another. Further research on arthroscopy simulations for knee, shoulder, and hip, as well as other joints, including the wrist, elbow, and ankle, is warranted. In addition, further investigation will help determine the type and number of training sessions necessary to translate technical skills learned on the models to the operating room, which will enable educators to use this training as a core component of resident education in the best way possible. We have included a summary of the limitations of current arthroscopy simulator models in Table 5.

CONCLUSION

Arthroscopic simulators have the potential to enable residents and surgeons to further develop their skills in a safe environment. This review supports the belief that practice on arthroscopic simulators improves performance on arthroscopic simulators. We cannot, however, definitively comment on whether or not simulator training correlates to an improved arthroscopic skill set in the operating room. We must conduct further work to determine the type and number of training sessions needed to translate technical skills learned on the models to the operating room. ■

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

"RECURRENCES OR WORSENING OF SYMPTOMS WERE NOT REPORTED WITH ANY TREATMENT MODALITY, BUT OPEN CURETTAGE WAS ASSOCIATED WITH A GREATER RESOLUTION OF HEEL PAIN AND RADIOGRAPHIC CONSOLIDATION THAN NON-OPERATIVE TREATMENT."

Treatment of Unicameral Bone Cysts of the Calcaneus: A Systematic Review

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Unicameral bone cysts (UBCs) are benign, fluid-filled lesions that represent 3% of all primary bone tumors.[1,2] Also known as simple bone cysts, UBCs most commonly occur in the metaphyseal regions of the proximal humerus and femur. The calcaneus is the sixth-most common UBC site, but it is the most common carpal or tarsal bone affected and, when affected, may cause significant pain, disability, and gait difficulties.[3] UBCs affect males more often than females in a 2.5:1 ratio and traditionally present in the first 2 decades of life,[1,2] although some authors suggest that UBCs localized to the calcaneus occur more frequently in middle-aged populations.[4,5] UBCs affect the proximal humerus and femur in 75% of patients under 17 years of age but are located in the ilium and calcaneus in over 50% of patients older than 17 years.[6,7]

The type of intervention for unicameral bone cysts remains controversial.[3,810] Some authors suggest that operative intervention is not indicated for calcaneal UBCs,[3,5] but others—most notably Neer in his experience with UBCs of the shoulder[11]—advise against watchful waiting, out of concern for pathologic fractures. Following pathologic fracture, UBCs have demonstrated just a 14.8% rate of complete healing without supplemental fixation or biologic enhancement.[12] Pathologic fractures of the calcaneus affect a weight-bearing bone and may present a surgical emergency with soft-tissue compromise.[13] Therefore, intervention is indicated in cases of persistent pain and/or impending pathologic fracture of the calcaneus.

Open curettage and bone grafting for UBCs has resulted in high recurrence rates if the graft is incompletely packed.[14] Methylprednisolone acetate injections were introduced by Scaglietti et al in 1979 but have been limited by the need for repeat injections and a perceived inability to achieve complete bone healing.[8,15,16] Alternative therapeutic options include autologous bone-marrow injection; various methods of cyst decompression; and use of calcium phosphate, calcium sulfate, and cannulated screws.[2,17-23]

The purpose of this study is to review all relevant literature on the treatment of unicameral bone cysts of the calcaneus in order to help guide physicians toward the best therapeutic modality. To our knowledge, no systematic review has been conducted on this topic.

MATERIALS AND METHODS

SEARCH STRATEGY

We conducted a systematic review of the available literature, according to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines using the PRISMA checklist.[24] Two reviewers independently conducted the search on October 1, 2013, using the following databases: PubMed (from 1948 to week 1 of October 2013), Medline (from 1946 to week 1 of October 2013), Embase (from 1947 to week 40 of 2013), and the Cochrane Central Register of Controlled Trials. The keyword selection was designed to capture all level I-IV evidence (according to the Oxford Centre for Evidence-Based Medicine) English-language studies that reported clinical and/or radiographic outcomes. This was accomplished using the keywords *unicameral*, cyst, and *calcaneus* and a series of *NOT* phrases designed to match our exclusion criteria. Studies were excluded if they did not report clinical and/or radiographic data relating to unicameral bone cysts of the calcaneus. Within each study, we included only data pertaining to unicameral cysts; data pertaining to aneurysmal bone cysts or other bony tumors were excluded. The bibliography from each search result was reviewed for any relevant titles. This search yielded 26 initial results.

STUDY SELECTION

Studies with the previously stated exclusion criteria were not immediately excluded but rather reviewed for any differentiation of patient populations. For instance, if a study reported on unicameral bone cysts in several anatomic locations, we included in our review only the data pertaining to calcaneal cysts. We excluded studies that could not be deconstructed or that were devoted entirely to 1 of our exclusion criteria. All case reports and studies with

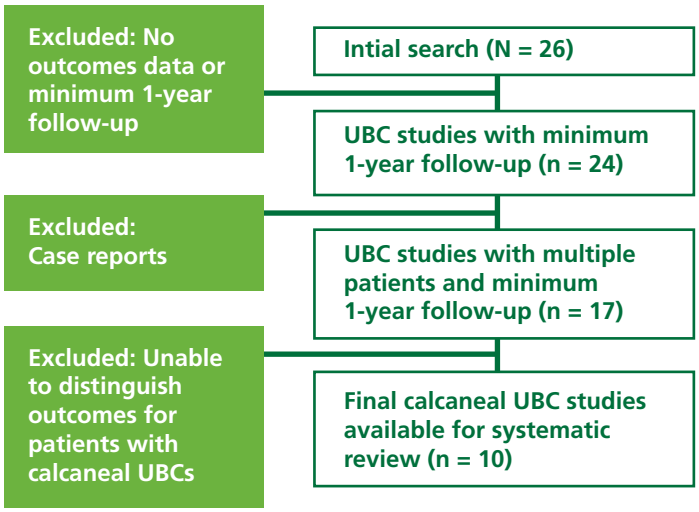

FIGURE 1. Diagram demonstrating study-selection criteria.

Abbreviation: UBC, unicameral bone cyst

a minimum follow-up of less than 1 year were also excluded. After accounting for all inclusion and exclusion criteria, 10 studies with 174 patients (181 cysts) were selected for inclusion (Figure 1).[3-5,8-10,13,23,25,26]

DATA EXTRACTION

We extracted data from studies that satisfied the eligibility criteria and recorded details of study design, sample size, and patient demographics, including age and sex. The mode of diagnosis (roentgenography, computed tomography [CT], magnetic resonance imaging [MRI], histology) (Figure 2), presence or absence of heel pain, and number of pathologic fractures were abstracted. Treatment was divided into non-operative and operative measures. Non-operative measures included observation[3-5,8,9] and intraosseous steroid injections.[8] All operative methods included curettage[3-5,12,16-18,20,28] and were further divided into 2 groups: cannulated-screw decompression[10,23] and bone augmentation with autograft,[3-5,8,9,26] allograft,[4,8,25,26] and/or cement substitutes such as calcium phosphate and calcium sulfate.[4,9] No validated clinical outcomes were utilized in more than 1 of the included studies. Therefore, posttreatment outcomes data focused on the persistence or resolution of heel pain, surgical complications or revisions, and radiographic evidence of cyst consolidation.

STATISTICAL ANALYSIS

Data are reported as weighted means with standard deviations. A mean was calculated for each study reporting on a respective data point, and each mean was then weighed according to its study sample size. This calculation was performed by multiplying 1 study's individual mean by the number of patients enrolled in that study and dividing the sum of these weighted data points by the number of eligible patients in all relevant studies. In this way, the nonweighted means from studies with a smaller sample size did not carry as much weight as those from larger studies. Using a 1-tailed paired *t* test, we compared the presence of pain before and after each treatment type. We compared 1) non-operative to operative interventions, 2) cannulated-screw decompression to bone curettage with grafting, and 3) autografting to allografting, using a 2-sample *z* test because of the difference in sample sizes between compared groups. For all statistical calculations, a *P* value of .05 was set as statistically significant.

RESULTS

A total of 10 studies with 174 patients (181 cysts) were included in this review. Nine studies consisted of level-IV evidence[3-5,8-10,13,23,26] and there was 1 level III case-control study.[25] The earliest study was published in 1974[3] and the most recent in 2011.[13] Only 1 study each reported on corticosteroid injections, calcium phosphate, and calcium sulfate as treatment for calcaneal unicameral bone cysts. In all studies combined, the weighted mean age was 25.7 ± 8.1 years, and the percentage of male patients was 67.1%.

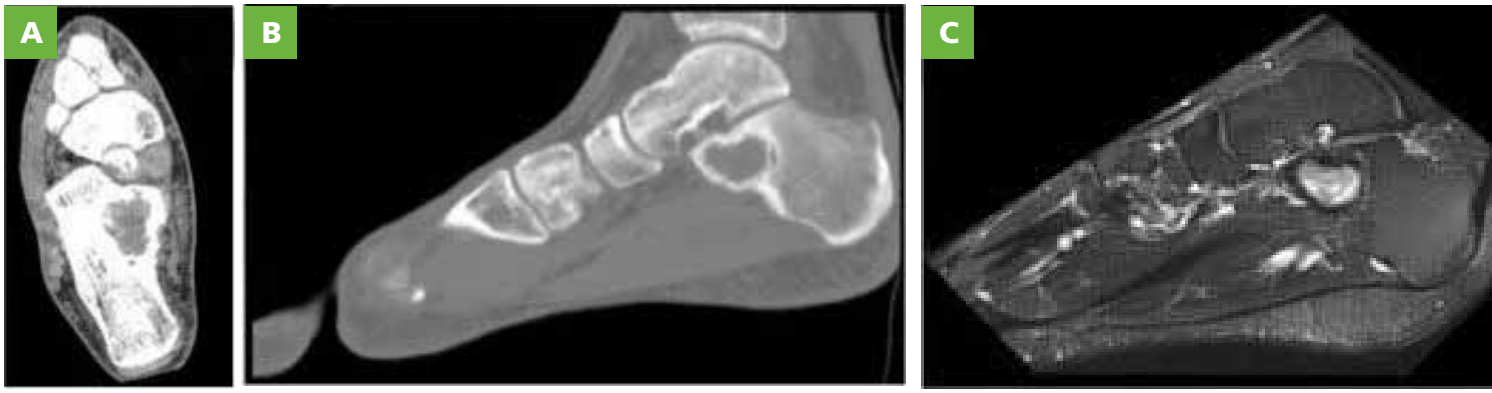

FIGURE 2. Computed tomography (CT) of the right foot in a 26-year-old male with foot pain demonstrates a large unicameral bone cyst in the less common anteromedial aspect of the calcaneus on **A,** axial and **B,** sagittal projections. **C,** A T2-weighted sagittal magnetic resonance imaging (MRI) view confirms that the bone lesion is fluid filled.

Comparative z tests revealed preoperative differences between treatment groups (Tables 1 and 2). Patients managed non-operatively were older than operative patients (32.0 ± 7.3 years vs 21.8 ± 10.2 years, $P < .001$, $z = 7.2$), more frequently male (74.7 ± 10.6% vs 59.9 ± 13.2%, $P < .001$, $z = 7.9$), and had a lower incidence of heel pain (9.2 ± 11.4% vs 76.7 ± 29.2%, $P < .001$, $z = 19.7$) than those patients who underwent surgery. In the operative group, those who had a cannulated screw placed for continuous decompression were younger than those in the non-operative group (14.1 ± 1.1 years vs 24.3 ± 10.5 years, $P < .001$, $z = 7.8$) and had a lower incidence of preoperative heel pain (47.3 ± 12.7% vs. 86.2 ± 26.7%, $P < .001$, $z = 9.0$) than patients who had bone substitute placed in their cystic defect. There was no difference between the 2 groups in the percentage of male patients (58.6 ± 10.1% vs 60.4 ± 14.1%, $P = 0.524$, $z = 0.6$). Patients who had autogenous bone graft placed were older than those who had an allograft (27.5 ± 10.1 years vs 20.2 ± 9.9 years, $P = .005$, $z = 2.8$), but there were no significant differences in male gender (62.7 ± 12.6% vs 56.9 ± 15.4%, $P = .096$, $z = 1.7$) or incidence of preoperative heel pain (87.3 ± 25.5% vs 85.1 ± 28.4, $P = .789$, $z = 0.3$).

Heel pain resolved spontaneously in 1.1 ± 1.0% of patients treated non-operatively (Table 2). The change in pain before and after treatment was not statistically significant ($P = .187$). In the cannulated-screw operative group, 47.3 ± 12.7% of patients had heel pain before surgery compared to 0 patients after surgery, but this improvement did not achieve statistical significance ($P = .085$). All

TABLE 1. Demographic Information, by Treatment Group

Treatment	Studies	Patients	Cysts	Mean age (years)	Males (%)	Incidentally Discovered Cysts (%)Ψ
Non-operative	5[3-5,8,9]	69	75	32.0 ± 7.3	74.7 ± 10.6	86.5 ± 9.1
Corticosteroid injection(s)	1[8]	6	6	12.7	83.3	66.7
Operative curettage	9[3-5,8-10,23,25,26]	83	86	21.8 ± 10.2	59.9 ± 13.2	25.2 ± 29.6
Cannulated-screw decompression	2[10,23]	20	21	14.1 ± 1.1	58.6 ± 10.1	52.7 ± 12.7
Curettage + bone augmentation	7[3-5,8,9,25,26]	63	65	24.3 ± 10.5	60.4 ± 14.1	15.4 ± 27.7
Autograft	6[3-5,8,9,26]	39	39	27.5 ± 10.1	62.7 ± 12.6	14.4 ± 26.7
Allograft	4[4,8,25,26]	25	27	20.2 ± 9.9	56.9 ± 15.4	16.1 ± 29.2
Calcium phosphate	1[9]	9	9	33.2	76.5	0.0
Calcium sulfate	1[4]	2	2	37.9	57.6	0.0

Ψ = Percentage of cysts per study that were discovered incidentally on roentography when patients were being evaluated for concomitant foot and ankle injuries

TABLE 2. Presence of Heel Pain, by Treatment Group

Treatment	Mean Follow-Up (years)	Pretreatment Heel Pain (%)	Posttreatment Heel Pain (%)	Heel Pain Resolved with Treatment (%)	*P* Value
Non-operative	2.7 ± 2.5	9.2 ± 11.5	8.1 ± 12.2	1.1 ± 1.0	.187
Corticosteroid injection(s)	2.8	33.3	0.0	33.3	N/A
Operative curettage	3.8 ± 1.9	76.7 ± 29.2	7.0 ± 9.1	69.7 ± 26.5	< .001
Cannulated-screw decompression	5.5 ± 2.4	47.3 ± 12.7	0.0 ± 0.0	47.3 ± 12.7	.085
Curettage + bone augmentation	3.3 ± 1.3	86.2 ± 26.7	9.2 ± 9.4	77.0 ± 25.7	< .001
Autograft	2.8 ± 0.8	87.3 ± 25.5	12.5 ± 8.3	74.7 ± 24.3	< .001
Allograft	3.9 ± 1.5	85.1 ± 28.4	4.9 ± 9.3	80.2 ± 27.2	.005
Calcium phosphate	2.7	100.0	11.1	88.9	N/A
Calcium sulfate	2.1	100.0	22.2	77.8	N/A

TABLE 3. Radiographic Outcomes, by Treatment Group

Treatment	Cysts with Radiographic Healing (%)
Non-operative	1.1 ± 1.0
Corticosteroid injection(s)	66.7
Operative curettage	93.0 ± 13.0
Cannulated-screw decompression	95.3 ± 5.6
Curettage + bone augmentation	92.3 ± 14.6
Autograft	97.4 ± 11.1
Allograft	85.1 ± 15.8
Calcium phosphate	100.0
Calcium sulfate	100.0

patients who underwent operative curettage with bone substitute—whether autograft or allograft—enjoyed a statistically significant improvement in heel pain ($P < .001$). Surgery was significantly more effective at alleviating heel pain than no surgery (69.7 ± 26.5% vs 1.1 ± 1.0%, $P < .001$, $z = 24.1$). At final follow-up, more patients had symptomatic heel pain in the bone substitute group than the cannulated-screw group (9.2 ± 9.4% vs 0.0 ± 0.0%, $P < .001$, $z = 7.8$), but the percentage of patients whose original heel pain had resolved following treatment was significantly greater in the former group (77.0 ± 25.7% vs 47.3 ± 12.7, $P < .001$, $z = 7.0$). Significantly more patients who received an autograft had heel pain at final follow-up compared to the allograft group (12.5 ± 8.3 vs 4.9 ± 9.3, $P = .001$, $z = 3.3$), but there was no significant difference in the percentage of patients whose original pain had resolved from treatment (74.7 ± 24.3 vs 80.2 ± 27.2, $P = .4$, $z = 0.8$).

Radiographic resolution of the cyst(s) was seen in significantly more patients who underwent surgery than not (93.0 ± 13.0% vs 1.1 ± 1.0%, $P < .001$, $z = 65.4$) (Table 3). Healing on final radiographs was also significantly more common in patients who underwent an autograft compared to allografting (97.4 ± 11.1% vs 85.1 ± 15.8%, $P < .001$, $z = 3.5$). There were no significant radiologic differences between cannulated-screw and bone-substitute placement (95.3 ± 5.6% vs 92.3 ± 14.6, $P = .165$, $z = 1.4$). No recurrences were reported in any cysts that had previously consolidated on radiography.

DISCUSSION

This systematic review evaluates the treatment options for UBCs of the calcaneus. Heel pain and radiographic cyst consolidation were the primary outcomes. Three different group comparisons were made with regard to these outcomes: 1) non-operative treatment vs surgery, 2) cannulated screws vs bone augmentation, and 3) autografting vs allografting.

Observation is reserved for the majority of calcaneal UBCs that are asymptomatic and discovered incidentally on radiographs. In this review, only 9.2 ± 11.4% of patients treated non-operatively had heel pain, a significantly lower percentage than the 76.7 ± 29.2% rate in the operative group. Only 1.1 ± 1.0% of cysts healed spontaneously with observation. No complications or pathologic fractures were noted in the non-operative group, but these patients were followed for an average of just under 3 years. UBCs typically occur in the anterolateral aspect of the calcaneus, avoiding the thick medial cortex through which most of one's body weight is transmitted.[5,12] The lateral calcaneus has a thinner cortex and fewer trabeculae, so, if a UBC is present and expanding, the weak lateral bone may be predisposed to eventual pathologic fracture.[21] This is a risk that must be weighed when managing a calcaneal cyst conservatively.

Unlike non-operative treatment, open curettage led to a significant improvement in patients' heel pain. Just under 70% of cases of heel pain resolved with surgery, and radiographs revealed that 93.0 ± 13.0% of cysts healed. Cannulated screws and bone augmentation were the 2 primary types of surgery evaluated. In 2002, Abdel-Wanis et al developed the technique of minimal curettage, multiple drilling, and continuous decompression via a cannulated screw for treatment of calcaneal UBCs.[23] This was viewed as an alternative to autografting, of which a large volume is often difficult to harvest in children, and allografting, which carries a small risk of disease transmission. Continuous decompression allows for drainage of cyst fluid and normalization of intracystic pressure, while drilling multiple small bone canals is believed to stimulate new bone formation.[27] Abdel-Wanis et al demonstrated complete radiographic healing in all 12 calcaneal UBCs in their study and complete pain relief without recurrence in the 7 symptomatic patients. Only 1 case needed revision surgery for replacement of a titanium screw, and this cyst healed 26 months after the index operation. Saraph et al demonstrated similarly positive outcomes and 1 revision operation in 9 calcaneal UBCs.[10] The sample sizes from these 2 populations, however, are too small to make definitive conclusions about the outcomes of cannulated screws. Furthermore, in this review, the percentage of patients with preoperative heel pain was significantly lower in the cannulated-screw group than in the bone-augmentation group (47.3 ± 12.7% vs 86.2 ± 26.7). As such, even though all cases of heel pain resolved with cannulated-screw placement, a significantly lower percentage of patients improved symptomatically in the cannulated-screw group than the bone-augmentation group. A fair comparison cannot be made between cannulated screws and bone augmentation if the respective patient populations differ preoperatively.

Open curettage with bone augmentation is the traditional treatment of symptomatic calcaneal UBCs, and it demonstrated the best outcomes in this review. Almost 80% of patients in the bone-augmentation group had heel pain that had completely resolved after a weighted mean of 3.3 ± 1.3 years. There was no clear distinction between autografting and allografting. The latter demonstrated a greater percentage of patients with resolved heel pain than did the former (80.2 ± 27.2% vs 74.7 ± 24.3%), but, due to a large variance, this trend did not reach statistical significance. Conversely, cysts treated with autografts consolidated on radiography at a significantly greater rate than those treated with allografts (97.4 ± 11.1% vs 85.1 ± 15.8%). There is no clear biological reasoning to explain a difference in outcomes between autogenous bone and allogenic bone. No patients in either group had recurrences, complications, or reactions suggestive of graft rejection.

Surgeons turned to corticosteroid injections as a treatment for UBCs after a chemical analysis by Scaglietti et al revealed that intracystic fluid is similar in composition to tissue transudate.[16] These injections are associated with minimal morbidity and may be done operatively or in the office setting. In the initial series by Scaglietti et al, only 24% of patients demonstrated cyst healing after a single injection: multiple injections are often required.[1,16,28] Only Glaser et al separated outcomes of steroid injections for calcaneal UBCs specifically, and all 9 injections performed in 6 patients failed to heal the cysts.[8] Resolution of pain and radiographic healing occurred only in patients who eventually underwent curettage with a grafting procedure; the 2 patients who had repeat injections without surgery never healed in the follow-up period. Curettage with bone graft substitutes such as calcium phosphate and calcium sulfate is another under-reported treatment modality for UBCs localized to the calcaneus but showed generally better outcomes than steroid injections.[4,9]

Limitations within this systematic review are primarily related to the studies analyzed. All studies with the exception of 1[25] consisted of level IV evidence, and there were few objective data reported. Heel pain was typically reported as a categorical measure rather than on a visual analog scale, and no validated clinical outcomes survey was utilized in more than 1 study. Only 2 studies utilized validated scoring systems for cystic healing on radiography,[10,25] but all other studies reported cystic healing or consolidation on a binary scale (healed or not healed). Cyst recurrence rates and the average time to heel-pain resolution or radiographic consolidation were also underreported. Other interesting parameters that were not reported consistently include prognostic factors, operative indications, and the average time to return to athletic activities. Pogoda et al suggested that larger cyst diameter may be correlated with an increased risk of pathologic fracture and clinical symptoms,[9] but this finding was not explored by the other included studies. Publication bias is present in that the authors excluded non-English-language studies and medical conference abstracts and may have omitted potentially eligible studies not discoverable in our search methodology. Performance bias is a factor in any systematic review with multiple surgeons who use a wide variety of surgical techniques. The included studies in this review suffered from information bias and a failure to control for potentially confounding effects. Finally, although all studies satisfied the inclusion criterion for a minimum mean follow-up of 1 year, the varying duration of follow-up—ranging from 2.7 ± 2.5 to 5.5 ± 2.4 years—complicates direct comparisons between treatment groups.

CONCLUSION

In this systematic review, UBCs of the calcaneus affected patients in their third decade of life compared to UBCs in other locations, which usually present in the first 2 decades of life. Recurrences or worsening of symptoms were not reported with any treatment modality, but open curettage was associated with a greater resolution of heel pain and radiographic consolidation than non-operative treatment. Curettage with bone augmentation demonstrated significant improvements over curettage with cannulated-screw placement, but differences in preoperative baseline characteristics make it difficult to compare these 2 treatment groups. Finally, autograft procedures resulted in significantly greater radiographic healing compared to allografting. The authors encourage higher-level clinical evidence to further elucidate differences between treatment types. Although randomized, controlled trials are difficult to conduct for surgical treatments, future studies of calcaneal UBCs should report objective, validated clinical and radiographic outcomes and endeavor to compare these outcomes between treatment groups. ■

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

"MODERN [TOTAL HIP ARTHROPLASTY] WAS REFINED... IN THE EARLY 1960S, BUT PRIOR ATTEMPTS TO CURE ARTHRITIS LAID THE FOUNDATION FOR... THE LOW-FRICTION TOTAL HIP ARTHROPLASTY."

Development of the Modern Total Hip Arthroplasty

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EDITOR'S NOTE: This is the first of a 2-part article that presents the history of hip arthritis and arthroplasty from the ancient to modern era. Progress from the modern era to the present, emphasizing Rush's contributions, will be presented in the 2015 *Rush Orthopedics Journal.*

Hip arthritis has affected humans throughout our existence: its occurrence has been demonstrated in hominids from Paleolithic times[1] and in skeletons exhumed from Saxon burials.[2] Despite its ubiquitous presence, satisfactory treatment with total hip arthroplasty (THA) is a relatively recent development. Modern THA was refined and popularized by John Charnley, MD, in the early 1960s, but prior attempts to cure arthritis laid the foundation for his discovery of the low-friction THA.

Excisional arthroplasty was one of the earliest surgical treatment modalities, and the first record of this procedure was of one performed by Anthony White, MD, (1782-1849) at Westminster Hospital of London in 1821. The patient was 13 years old at the time of surgery, and his medical history was described in White's obituary[3]:

> *Four years and a quarter before the excision of the bone, the patient, a boy, at that time 9 years old, was thrown down. The injury was followed by disease of the hip, which was treated with leeches, blisters, rest and other usual means. Large abscesses formed, and burst around the joint, with extreme pain, and copious discharge of pus; and the head of the femur was dislocated far on the dorsal ilii. The patient was reduced to a very debilitated state; and during the two years and a half in which the discharge continued, became exceedingly emaciated.*

After White's procedure, which included excision of "the head, neck, and trochanters of the femur," the patient showed rapid improvement, "surviving the operation 12 years, and then dying consumptive."[3] Despite this successful outcome, White's peers were initially unimpressed with his new procedure, possibly due to his less than savory reputation at the time. His colleagues considered him to be the "laziest man in his profession," "habitually unpunctual," and his mentor, Sir Anthony Carlisle, threatened to report him to the college of surgeons for performing this surgery.[4] In spite of these transgressions, he eventually went on to become a respected member of the Royal College of Surgeons.

Philadelphia surgeon, John Barton, MD, (1794-1871) performed a variation of White's procedure, and the shear fracture of the distal radius bears his name (Barton's fracture).[5] Respected for his surgical skill and modest lifestyle, Barton's means of transportation was

> *not a showy phaeton drawn by fiery steeds whose pedigree might be traced to the stalls of Solomon, no buttons, no tigers, no obsequious lackeys to herald the presence of the man, but a plain substantial horse and buggy, driven by those hands whose cunning had wrought so many marvels of surgical skills.*[6]

He performed his first osteotomy of an ankylosed hip in 1826, and the procedure lasted only 7 minutes.[6] Barton was reportedly ambidextrous and was said to never have moved once positioned for a surgery, which likely contributed to his speed.[7] Postoperatively, he would manipulate the hip in an effort to maintain motion by creation of a pseudarthrosis. This was done with "gentle and daily motion of the limb" to prevent "the formation of bony union."[6] His initial procedure, the first of many, was performed on a 21-year-old sailor who had an ankylosed hip as a result of trauma. Some patients did have success with regaining motion, but unfortunately the procedure carried a 50% mortality rate, which was unacceptably high for an elective procedure designed to increase hip motion.

A more modern version of the resection arthroplasty, which is still sometimes performed today, was popularized in the 1940s by British surgeon Gathorne Robert Girdlestone, MD (1881-1950). Girdlestone was known to be a tall, handsome man, with the "charm, the piety, and some of the haughty individualism of an Elizabethan."[8] Lesser known is the speculation that his devotion to surgery may have been his only barrier to a professional golf career: he was the Irish Amateur Golf champion.[8] At that time, the primary indications for his resection arthroplasty were tuberculosis and infection. Interestingly, his religious values may have influenced his surgical decision making, as his rationale for this procedure was based on a variation of the biblical quote from Matthew 5:29 (KJV): "if thine femoral head offend thee, pluck it out and cast it from thee."[9]

Many variations of resection arthroplasty were performed around this time period.[10] Royal Whitman, MD, a surgeon operating at the Hospital for the Ruptured and Crippled, later renamed the Hospital for Special Surgery, developed a form of resection

FIGURE 1. Vitezslav Chlumsky (1867-1941), Czech surgeon and outspoken critic of early arthroplasty techniques.

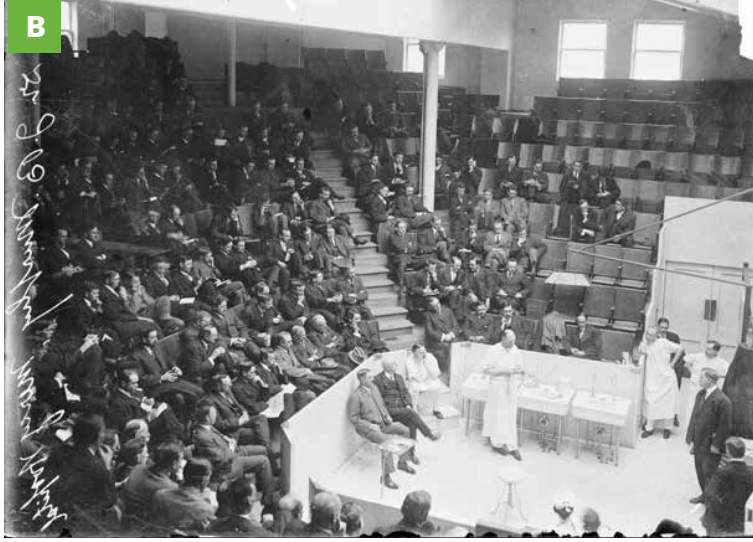

FIGURE 2. John Murphy **A,** outside Mercy hospital in 1912 after an assassination attempt on President Roosevelt where he was the treating physician. President Roosevelt expressed his sentiment regarding the event by stating, "I've hunted long enough doctor, to know that you can't kill a bull moose with a short gun."[33] and **B,** teaching at Chicago's Mercy Hospital in 1910.

arthroplasty that he felt was superior to arthrodesis. In his opinion, resection

> *is effective in removing the disease, but is defective from the functional standpoint because the trochanter is brought into contact with the rim of the acetabulum thus mechanically limiting abduction.*[11]

In his paper, "The reconstruction operation for arthritis deformans of the hip joint," he describes resection of the head and neck and then advances the greater trochanter in order to maintain muscle tension, allowing for better hip stability and improved motion.[11] His further credits include his commitment to teaching residents, and his style is mirrored by many present-day counterparts. One trainee noted that his "method of teaching was not always a placid procedure," as "he often used the difficult, and not always agreeable, method of sarcastic criticism."[12]

As an alternative to joint excision, there were many attempts at interpositional arthroplasty, the first of which was performed by American neurosurgeon John Carnochan, MD, who in 1840 implanted a wooden block in a patient's temporomandibular joint for pain relief.[13] Orthopedic surgeons eventually followed this example and attempted a similar procedure in the hip. A variety of different materials were tried, with a wide spectrum of results. Fat interposition was first attempted by Louis Ollier, MD, (1830-1900) at the Hôtel-Dieu in Lyon, France, in 1885. Ollier is considered by some to be the "father of orthopaedic surgery," and his wide interests included bone and skin grafting, and reconstructive surgery.[14] He kept meticulous records and, up to 20 years after a patient's death, would send assistants on horseback to remote villages for the purpose of retrieving specimens.[15] While many of his procedures were great successes, his adipose interpositional arthroplasties ultimately failed, likely because there was no means of securing the adipose tissue to the bones. However, his attempts inspired surgeons throughout Europe and the rest of the world to experiment with other interpositional materials. A Czech surgeon, Vitezslav Chlumsky, MD, (1867-1943) (Figure 1), was dismayed at the poor results in the treatment of ankylosed joints, stating,

> *The treatment of complicated contractures and ankyloses of the joints, as far as restitutio ad integrum is concerned, gives very unsatisfactory results. I have collected 14 cases of ankylosis, other than tuberculosis of the knee joint, with contraction, which occurred in the last 10 years in the Breslau Surgical Clinic and were there treated, and in not a single case was there improvement in the mobility of the joint.*[13]

He attempted to use a variety of interpositional materials including muscle, celluloid, zinc, glass, decalcified bones, rubber, and magnesium. The large number of different substances he used suggests he did not find one specific material that was significantly effective. However, he was diligent enough to test these materials on animals prior to insertion in humans, an uncommon practice at the time.

John Murphy, MD, (Figure 2), a Chicago surgeon who entered Rush Medical College in 1878 and 6 years later became a professor of surgery, was also interested in re-creating motion in ankylosed joints. He questioned whether interposition of biologic materials could recreate motion by inducing the formation of embryologic endothelial lined sacs. He mused,

> *What are joints? What is the embryology of joint formation? What is the pathologic histology of acquired arthroses or false joints? What is the pathology of hygromata (acquired endothelial lined sacs)? Can they be produced artificially?*[13]

Therefore, in contrast to the mostly inorganic materials used by Chlumsky, John Murphy's attempts were with fascia, skin, muscle, and periosteum.

Around 1918, in the United States, William Baer, MD, an orthopedic surgeon at Johns Hopkins who organized the first outpatient orthopedic clinic at that institution, popularized the use of chromatized pig bladder as an interpositional material. It was perhaps his memorable and larger-than-life personality that helped him convince patients to have a pig's bladder surgically inserted into their hip.

> *Frequently he came to the amphitheater from the operating room in operating clothes that were loose, baggy, and perhaps stained. He would give a graphic description and a demonstration of disease. He would limp up and down in front of the class, mimicking the limp of a congenital dislocation of one hip or both hips, the limp of a child with early tuberculosis of a hip, or the limp of a late stage of tuberculosis. He was a large man, overweight and puffing, with or without a cigar which he held in his mouth until he reached the operating table, when he would permit a nurse to remove it… No one slept during Baer's lectures.*[16]

Sir Robert Jones, MD, (1855-1933) is credited with establishing orthopedic surgery as a specialty in England by founding the British Orthopaedic Association and was known not only for his surgical skill, but also for his Victorian values and attractive personality.[17] He characterized his eponymously named fracture after he suffered that injury to his own foot while dancing.[18] His attempt at an arthritic cure was the use strips of gold to cover arthritic femoral heads.[10] He published one report of a woman who maintained satisfactory motion 21 years after her procedure. Perhaps it was his careful attention to detail in the operating room that produced these excellent results. It was stated that his assistants "often could expect a brisk and painful rap across the knuckles if any blood got on the surgical drapes."[17]

In 1923, Marius Smith-Peterson, MD, a Norwegian-born American surgeon working in Boston, implanted a glass mold into the hip of a patient. He conceived the idea after he excised of a piece of glass from a patient's back and noted that the glass had become covered with a fibrous membrane. He reasoned that he could implant the glass into a hip joint, protect motion until this membrane had formed, remove the glass, and consequently restore a cartilaginous membrane to the hip. In his words, he felt that a "mould of some inert material, interposed between the newly shaped surfaces of the head of the femur and the acetabulum, would guide nature's repair so that defects would be eliminated." However, in retrospect, he stated, "I was amazed I had the courage to use them."[19] In theory it was a good idea, but many of the glass molds broke within months. During the ensuing year Smith-Peterson attempted to use a form of celluloid, but this resulted in a vigorous foreign body reaction. In 1933, he used a more durable form of glass (Pyrex), however, some of these broke so they were used sparingly. Smith-Peterson noted that

> *"the majority of patients [for whom these were used] did well. When the moulds were removed after 15 to 25 months the joint surfaces were smooth, glistening, firm, and congruous."*

In 1938, at the suggestion of his own dentist, John Cooke, DDS, he used Vitalium, a cobalt-chrome-molybdenum alloy, as an interpositional mold. The Vitalium arthroplasties were relatively successful, and Smith-Peterson implanted over 500 in the ensuing years. In an article published in 1947, he stated that his complications included 53 revisions, 20 infections (12 new infections, 8 in patients with a prior septic hip), an unlisted number of pulmonary embolisms (none fatal), and no operative mortalities.[19] Of note, he also described his eponymously named approach to the hip, which he used for these procedures, in 1917 while still a resident. He expressed shock at the currently used hip approach, finding that "it was bloody; it was brutal." He said to his senior Roy Abbott, MD, "There must be some other way of exposing the hip," to which Abbott replied, "Why don't you figure one out"?[19]

Smith-Peterson's "cup arthroplasties" were further improved upon by 2 surgeons in Boston, Otta Aufranc, MD, a protégé of Smith-Peterson, and William Harris, MD. Cup arthroplasty was also performed by a surgeon in Iowa City, Carroll Larson, MD, who incidentally applied this technique to ankle arthroplasty in the 1960s. Successful results at 40-years follow-up were recently published.[20]

At the same time surgeons were experimenting with interpositional arthroplasty, others were attempting arthroplasty with the use of prosthetics. One of the pioneers of this field was Themistocles Gluck, MD (1853-1942) (Figure 3), a Romanian-born German surgeon, who was son of the attending physician to the royal family. His first attempt at arthroplasty was a knee replacement in a 17-year-old girl with tuberculosis. He initially experimented in animals with a variety of prosthetic materials including aluminum, wood, glass, and steel, but eventually settled on ivory. He experimented with replacement in many joints, including the hip.[21] He also tried a variety of fixation methods including nickel-plated screws, plaster of Paris, powdered pumice, and even press-fit with osseo-integration. He demonstrated excellent short-term results but had major issues of infection and loosening in the long term. Unfortunately, opposition from his colleagues ultimately forced him to abandon work on joint replacement.

Pierre Delbet, MD, (1861-1925) (Figure 4), a French surgeon working in Paris, borrowed the idea of prosthetic replacement and in 1919 was the first to use a rubber prosthesis as a hemiarthroplasty in the femur.[9] Interestingly, he was better known for his advocacy of magnesium sulfate as an antiseptic than he was for the implantation of this rubber hemiarthroplasty.[22] Phillip Wiles, MD, (1899-1966), son of a wealthy corn merchant who chose surgical medicine over a life of leisure performed at implant in 1938 at Middlesex Hospital in London, using a stainless steel device with both femoral and acetabular components that bears some similarity to modern hip designs. At that time he noted that the treatment for the arthritic hip included 3 options, as he put it: "move it, keep it still, or cut it out."[10] He stated that

> *once there is a strong possibility that it surgery will make the condition better and not worse, the choice is then between attempting to give a movable joint by some form of arthroplasty or osteotomy, keeping still with arthrodesis, and cutting out the head of the femur.*[10]

FIGURE 3. Themistocles Gluck (1853-1942) Romanian-born German surgeon who tried various arthroplasty materials in various joints.

FIGURE 4. Pierre Delbet (1861-1925) French surgeon, first to use rubber as a bearing surface in arthroplasty.

However, his initial attempt at arthroplasty was to "spray metal on the denuded joint surfaces." This fortunately was attempted only in cats. He presumed it failed because "metals that can be melted fast enough in an oxyacetylene flame are unsuitable to leave permanently in the body."[10] His further attempts at arthroplasty included utilizing a stainless-steel acetabulum and femoral head. He secured the components to bone using screws and bolts. He tried 2 generations of this prosthesis but never had great success because of significant problems with bone resorption and loosening.

Around 1940, Robert and Jean Judet, MD, brothers residing in Paris, were implanting short, straight-stemmed acrylic femoral prostheses. They felt that they needed an improvement on Royal Whitman's resection arthroplasty technique that "shows out of every 5 cases, only 1 is good, 1 is fair, and 3 are poor." Their early acrylic prosthesis had a "shape that resembles that of a mushroom," but this prosthesis had a tendency to break, which they attempted to resolve by reinforcing it with "steel core, completely surrounded and insulated by acrylic resin." They published a series of 300 cases in which 70% of patients had no or negligible pain, and "80 percent of patients are able to return to work, and some can walk several miles, ride a bicycle, and do a heavy day's work."[24] However, there were concerns of loosening with this prosthesis.

At the same time, Harold Bohlman, MD, and Austin Moore, MD, 2 surgeons from South Carolina, collaborated to insert a custom-made 12-inch long Vitalium femoral prosthesis in a patient who had a giant cell tumor. While Bohlman had previously created a short-stemmed prosthesis out of Vitalium, this collaboration resulted in the first-ever custom-designed mega prosthesis. Their implant was fabricated to include multiple protruding ring structures to allow reattachment of muscles. Nine months post-operatively, the patient ambulated well and required a cane only for long distances. Unfortunately, the patient died of heart failure soon afterwards, in 1942. Autopsy revealed a normal appearing prosthesis without signs of significant wear or corrosion.[23] Bohlman and Moore continued to develop and modify their implant and in 1952 unveiled a model that allowed for bone ingrowth due to its fenestrated stem. This prosthesis was produced in collaboration with Howmedica Inc. (Mahwah, New Jersey) and was the first hip implant to be commercially distributed.[23] This very popular implant became generally known as an Austin Moore prosthesis.

Frederick Thompson, MD, working out of St. Luke's Hospital in New York City, created a similar long-stem cemented cobalt chrome implant for hemiarthroplasty, which improved on prior designs because the stem fit further down the medullary canal, providing a more secure fit for the prosthesis.

Kenneth McKee, MD, who previously trained with Wiles in Europe, took these prostheses a step further by adding an acetabular component to the Thompson femoral model to create a joint with a metal-on-metal bearing surface. The acetabular component screwed into the roof of the acetabulum and was also composed of cobalt chrome. McKee performed 40 replacements between 1956 and 1960, with a 54% success rate. Failure occurred because of loosening.

Peter Ring, MD, working in England, continued to implant a metal-on-metal prosthesis, similar to the one designed by McKee, with the acetabular component anchored into the pelvis via a long, threaded stem. His 1968 article in the *Journal of Bone and Joint Surgery* expressed his reasoning for this method of pelvic fixation despite the development of cement fixation:

> *The Charnley and McKee-Farrar prosthesis overcomes the problem of fixing the pelvic component by embedding it in acrylic cement. This is a material which does not bond firmly with bone, and may therefore allow the acetabular component to loosen. It may carry with it a slightly increased risk of infection, and is thought occasionally to have produced at the time of insertion an intense and dangerous hypotensive reaction.*[26]

The modern era of hip arthroplasty began with John Charnley, MD, who was born in 1911 in Bury, Lancashire, United Kingdom, and graduated from medical school in 1935 at Victoria University of Manchester. At age 25 he became a fellow of the Royal College of Surgeons, the youngest physician to ever receive this honor. His interest in THA stemmed from trying to discern why so many of the previous designs produced only marginal results. In 1956 he noted, "The cart has been put before the horse; the artificial joint has been made and used, and now we are trying to find out how and why it fails."[27] He initially considered the failure mechanisms of the Judet hip, for which loosening was a problem. The hips would initially squeak, but the squeaking would cease as the prosthesis would stick more, and then motion would begin to arise at the prosthesis-bone interface rather than at the joint. He concluded that a low-friction device would solve this issue. Charnley had begun to study friction in the early 1950s and was convinced this would reduce the risk of loosening.

Charnley's use of Teflon to surface the femoral head and acetabulum was complicated by wear, avascular necrosis, and foreign body reaction. He then used acrylic cement to attach a Teflon head to a Moore prosthesis. He studied tissue reaction by getting a colleague to implant Teflon in his thigh, resulting in large nodules. (Charnley had a prior history of surgical experimentation on his own leg, resulting in osteomyelitis). In 1961, he reported 97 hip arthroplasties in *The Lancet*, followed by a 1963 letter stating his dissatisfaction with Teflon.

In the ensuing years, Charnley combined the use of ultra-high-molecular-weight-polyethylene as a bearing surface, with more effective use of bone cement as a grout, as opposed to previous application as a glue.

With an improved low-friction bearing surface and better understanding of the use of cement, the modern total hip arthroplasty was born.The results of this design have been excellent, with Callaghan et al[32] demonstrating 88% of prostheses intact at time of final follow-up or patient's death at a minimum of 30 years. Centuries of basic sciences and clinical observations and experimentation had, by 50 years ago, produced a trunk of knowledge poised to branch and bear fruit. Research institutions such as Rush embrace the challenge to deal with special problems and complications as well as to train orthopedic surgeons to effectively bring this knowledge to those patients who need it. ■

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

Publications

PUBLICATIONS (2013)*

Abrams GD, Alentorn-Geli E, Harris JD, **Cole BJ**. Treatment of a lateral tibial plateau osteochondritis dissecans lesion with subchondral injection of calcium phosphate. *Arthrosc Tech*. 2013;2(3):e271-274.

Abrams GD, **Cole BJ**, Cerza F, Carcangiu A. Hyaluronic acid and platelet-rich plasma, intra-articular infiltration in the treatment of gonarthrosis: letter to the editor. *Am J Sports Med*. 2013;41(5):NP27.

Abrams GD, Frank RM, Fortier LA, **Cole BJ**. Platelet-rich plasma for articular cartilage repair. *Sports Med Arthrosc*. 2013;21(4):213-219.

Abrams GD, Frank RM, Gupta AK, Harris JD, McCormick FM, **Cole BJ**. Trends in meniscus repair and meniscectomy in the United States, 2005-2011. *Am J Sports Med*. 2013;41(10): 2333-2339.

Amstutz HC, Campbell PA, Dorey FJ, Johnson AJ, **Skipor AK, Jacobs JJ**. Do ion concentrations after metal-on-metal hip resurfacing increase over time? A prospective study. *J Arthroplasty*. 2013;28(4):695-700.

Anderson DG, Markova D, **An HS**, Chee A, Enomoto-Iwamoto M, Markov V, Saitta B, Shi P, Gupta C, Zhang Y. Human umbilical cord blood-derived mesenchymal stem cells in the cultured rabbit intervertebral disc: a novel cell source for disc repair. *Am J Phys Med Rehabil*. 2013;92(5):420-429.

Badlani N, Foran JR, **Phillips FM**, Pelton M, **Singh K**, Garfin SR, Allen RT. Patient perceptions of physician reimbursement for spine surgery. *Spine*. 2013;38(15):1288-1293.

Barão VA, **Mathew MT**, Yuan JC, Knoernschild KL, Assunção WG, **Wimmer MA**, Sukotjo C. Influence of corrosion on lipopolysaccharide affinity for two different titanium materials. *J Prosthet Dent*. 2013;110(6):462-470.

Barrack RL, Berend KR, Cui Q, Fehring TK, **Della Valle CJ**, Gehrke T, Lombardi AV Jr, Mont MA, Parvizi J, Springer BD. Cement spacers in periprosthetic joint infection. *Clin Infect Dis*. 2013;57(2):328-329.

Barrack RL, Ruh EL, Berend ME, **Della Valle CJ**, Engh CA Jr, Parvizi J, Clohisy JC, Nunley RM. Do young, active patients perceive advantages after surface replacement compared to cementless total hip arthroplasty? *Clin Orthop Relat Res*. 2013;471(12):3803-3813.

Bell R, Li J, Gorski DJ, Bartels AK, Shewman EF, **Wysocki RW**, **Cole BJ**, **Bach BR Jr**, **Mikecz K**, Sandy JD, Plaas AH, **Wang VM**. Controlled treadmill exercise eliminates chondroid deposits and restores tensile properties in a new murine tendinopathy model. *J Biomech*. 2013;46(3): 498-505.

Bell R, Li J, Shewman EF, **Galante JO**, **Cole BJ**, **Bach BR Jr**, Troy KL, **Mikecz K**, Sandy JD, Plaas AH, **Wang VM**. ADAMTS5 is required for biomechanically-stimulated healing of murine tendinopathy. *J Orthop Res*. 2013;31(10): 1540-1548.

Berschback JC, Lynch TS, Kalainov DM, **Wysocki RW**, Merk BR, **Cohen MS**. Clinical and radiographic comparisons of two different radial head implant designs. *J Shoulder Elbow Surg*. 2013;22(8):1108-1120.

Bhatia S, Frank RM, Ghodadra NS, Hsu AR, **Romeo AA**, **Bach BR Jr**, Boileau P, Provencher MT. The outcomes and surgical techniques of the Latarjet procedure. *Arthroscopy*. 2014;30(2): 227-235.

Bhatia S, Korth K, Van Thiel GS, Gupta D, **Cole BJ**, **Bach BR Jr**, **Verma NN**. Effect of reamer design on posteriorization of the tibial tunnel during endoscopic transtibial anterior cruciate ligament reconstruction. *Am J Sports Med*. 2013;41(6):1282-1289.

Bhatia S, Mather RC III, Hsu AR, Ferry AT, **Romeo AA**, **Nicholson GP**, **Cole BJ**, **Verma NN**. Arthroscopic management of recalcitrant stiffness following rotator cuff repair: a retrospective analysis. *Indian J Orthop*. 2013;47(2):143-149.

Bhatia S, Van Thiel GS, Gupta D, Ghodadra N, **Cole BJ**, **Bach BR Jr**, Shewman E, **Wang VM**, **Romeo AA**, **Verma NN**, Provencher MT. Comparison of glenohumeral contact pressures and contact areas after glenoid reconstruction with Latarjet or distal tibial osteochondral allografts. *Am J Sports Med*. 2013;41(8): 1900-1908.

Bishop N, Witt F, Pourzal R, **Fischer A**, Rütschi M, Michel M, Morlock M. Wear patterns of taper connections in retrieved large diameter metal-on-metal bearings. *J Orthop Res*. 2013;31(7): 1116-1122.

Biswas D, Haughom B, Mayle RE Jr, **Della Valle CJ**. Case report: Failure of rotating-hinge total knee prosthesis by disengagement of the hinge-post extension. *Clin Orthop Relat Res*. 2013;471(4):1389-1392.

Biswas D, Van Thiel GS, Wetters NG, Pack BJ, **Berger RA**, **Della Valle CJ**. Medial unicompartmental knee arthroplasty in patients less than 55 years old: minimum of two years of follow-up. *J Arthroplasty*. 2014;29(1):101-105.

Biswas D, **Wysocki RW**, **Cohen MS**, **Fernandez JJ**. Radioscapholunate arthrodesis with compression screws and local autograft. *J Hand Surg Am*. 2013;38(4):788-794.

Bozic KJ, Ward DT, Lau EC, Chan V, Wetters NG, Naziri Q, Odum S, Fehring TK, Mont MA, Gioe TJ, **Della Valle CJ**. Risk factors for periprosthetic joint infection following primary total hip arthroplasty: a case control study. *J Arthroplasty*. 2014;29(1):154-6.

Bragdon CR, Doerner M, Martell J, Jarrett B, Palm H; Multicenter Study Group, Malchau H. The 2012 John Charnley Award: Clinical multicenter studies of the wear performance of highly crosslinked remelted polyethylene in THA. *Clin Orthop Relat Res*. 2013;471(2): 393-402.

Brown NM, Foran JR, **Della Valle CJ**. Hip resurfacing and conventional THA: comparison of acetabular bone stock removal, leg length, and offset. *Orthopedics*. 2013;36(5):e637-641.

Caicedo MS, Samelko L, McAllister K, **Jacobs JJ**, **Hallab NJ**. Increasing both CoCrMo-alloy particle size and surface irregularity induces increased macrophage inflammasome activation in vitro potentially through lysosomal destabilization mechanisms. *J Orthop Res*. 2013;31(10):1633-1642.

Cha TD, **An HS**. Cervical spine manifestations in patients with inflammatory arthritides. *Nat Rev Rheumatol*. 2013;9(7):423-432.

Chahal J, Gross AE, Gross C, Mall N, Dwyer T, Chahal A, Whelan DB, **Cole BJ**. Outcomes of osteochondral allograft transplantation in the knee. *Arthroscopy*. 2013;29(3):575-588.

Chahal J, Thiel GV, Hussey K, **Cole BJ**. Managing the patient with failed cartilage restoration. *Sports Med Arthrosc*. 2013;21(2): 62-68.

Chalmers PN, Frank RM, Gupta AK, **Yanke AB,** Trenhaile SW, **Romeo AA**, **Bach BR Jr**, **Verma NN**. All-arthroscopic patch augmentation of a massive rotator cuff tear: surgical technique. *Arthrosc Tech*. 2013;2(4):e447-451.

**This is a partial list of published works for the faculty members of the Department of Orthopedic Surgery at Rush in 2013. Works with electronic publication dates in 2013 and print publication dates in 2014 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.*

Chalmers PN, Hammond J, Juhan T, **Romeo AA**. Revision posterior shoulder stabilization. *J Shoulder Elbow Surg*. 2013;22(9):1209-1220.

Chalmers PN, Karas V, Sherman SL, **Cole BJ**. Return to high-level sport after meniscal allograft transplantation. *Arthroscopy*. 2013;29(3): 539-544.

Chalmers PN, Mall NA, **Cole BJ**, **Verma NN**, **Bush-Joseph CA**, **Bach BR Jr**. Anteromedial versus transtibial tunnel drilling in anterior cruciate ligament reconstructions: a systematic review. *Arthroscopy*. 2013;29(7):1235-1242.

Chalmers PN, **Sporer SM**, **Levine BR**. Correlation of aspiration results with periprosthetic sepsis in revision total hip arthroplasty. *J Arthroplasty*. 2014;29(2):438-442.

Chee AV, Ren J, Lenart BA, Chen EY, Zhang Y, **An HS**. Cytotoxicity of local anesthetics and nonionic contrast agents on bovine intervertebral disc cells cultured in a three-dimensional culture system. *Spine J*. 2013 Nov 15. pii: S1529-9430(13)01217-5.

Chen D, **Berger RA**. Outpatient minimally invasive total hip arthroplasty via a modified Watson-Jones approach: technique and results. *Instr Course Lect*. 2013;62:229-236.

Chen JL, Allen CR, Stephens TE, Haas AK, Huston LJ, Wright RW, Feeley BT; Multicenter ACL Revision Study (MARS) Group. Differences in mechanisms of failure, intraoperative findings, and surgical characteristics between single- and multiple-revision ACL reconstructions: a MARS cohort study. *Am J Sports Med*. 2013;41(7):1571-1578.

Christensen CP, Bedair H, **Della Valle CJ**, Parvizi J, Schurko B, Jacobs CA. The natural progression of synovial fluid white blood-cell counts and the percentage of polymorphonuclear cells after primary total knee arthroplasty: a multicenter study. *J Bone Joint Surg Am*. 2013;95(23):2081-2087.

Cipriano CA, Brown NM, **Della Valle CJ**, Moric M, **Sporer SM**. Intra-operative periprosthetic fractures associated with press fit stems in revision total knee arthroplasty: incidence, management, and outcomes. *J Arthroplasty*. 2013;28(8):1310-1313.

Cohen MS, Jupiter JB, Fallahi K, Shukla SK. Scaphoid waist nonunion with humpback deformity treated without structural bone graft. *J Hand Surg Am*. 2013;38(4):701-705.

Cooper HJ, **Della Valle CJ**. Advances in the diagnosis of periprosthetic joint infection. *Expert Opin Med Diagn*. 2013;7(3):257-263.

Cooper HJ, **Urban RM**, Wixson RL, Meneghini RM, **Jacobs JJ**. Adverse local tissue reaction arising from corrosion at the femoral neck-body junction in a dual-taper stem with a cobalt-chromium modular neck. *J Bone Joint Surg Am*. 2013;95(10):865-872.

Cunningham BW, **Hallab NJ**, Hu N, McAfee PC. Epidural application of spinal instrumentation particulate wear debris: a comprehensive evaluation of neurotoxicity using an in vivo animal model. *J Neurosurg Spine*. 2013;19(3):336-350.

Cvetanovich GL, McCormick F, Erickson BJ, Gupta AK, Abrams GD, Harris JD, **Romeo AA**, **Bach BR**, Provencher MT. The posterolateral portal: optimizing anchor placement and labral repair at the inferior glenoid. *Arthrosc Tech*. 2013;2(3):e201-204.

Delemarre EM, Roord ST, van den Broek T, Zonneveld-Huijssoon E, de Jager W, Rozemuller H, Martens AC, Broere F, Wulffraat NM, **Glant TT**, Prakken BJ, van Wijk F. Brief report: autologous stem cell transplantation restores immune tolerance in experimental arthritis by renewal and modulation of the teff cell compartment. *Arthritis Rheumatol*. 2014;66(2):350-356.

Della Valle CJ. CORR Insights®: no infection reduction using chlorhexidine wipes in total joint arthroplasty. *Clin Orthop Relat Res*. 2013;471(10):3126-3127.

Dhawan A, Mather RC III, Karas V, Ellman MB, Young BB, **Bach BR Jr**, **Cole BJ**. An epidemiologic analysis of clinical practice guidelines for non-arthroplasty treatment of osteoarthritis of the knee. *Arthroscopy*. 2014;30(1):65-71.

Ellman MB, Kim J, **An HS**, Chen D, Kc R, Li X, Xiao G, Yan D, Suh J, van Wjnen AJ, Wang JH, Kim SG, Im HJ. Lactoferricin enhances BMP7-stimulated anabolic pathways in intervertebral disc cells. *Gene*. 2013;524(2):282-291.

Ellman MB, **Levine BR**. Fracture of the modular femoral neck component in total hip arthroplasty. *J Arthroplasty*. 2013;28(1):196.e1-5.

Ellman MB, **Yanke A**, Juhan T, **Verma NN**, **Nicholson GP**, **Bush-Joseph C**, **Romeo AA**. Open repair of retracted latissimus dorsi tendon avulsion. *Am J Orthop* (Belle Mead NJ). 2013;42(6):280-285.

Ellman MB, **Yanke A**, Juhan T, **Verma NN**, **Nicholson GP**, **Bush-Joseph C**, **Bach BR Jr**, **Romeo AA**. Open repair of an acute latissimus tendon avulsion in a major league baseball pitcher. *J Shoulder Elbow Surg*. 2013;22(7): e19-23.

Ellman MB, **Yanke A**, Juhan T, **Verma NN**, **Nicholson GP**, **Bush-Joseph C**, **Romeo AA**. Open repair of retracted latissimus dorsi tendon avulsion. *Am J Orthop* (Belle Mead NJ). 2013;42(6):280-285.

Erickson BJ, Chalmers PN, **Yanke AB**, **Cole BJ**. Surgical management of osteochondritis dissecans of the knee. *Curr Rev Musculoskelet Med*. 2013;6(2):102-114.

Farr J, **Verma N**, **Cole BJ**. Validation study of an electronic method of condensed outcomes tools reporting in orthopaedics. *J Knee Surg*. 2013;26(6):445-451.

Fehring TK, Odum SM, Berend KR, Jiranek WA, Parvizi J, Bozic KJ, **Della Valle CJ**, Gioe TJ. Failure of irrigation and débridement for early postoperative periprosthetic infection. *Clin Orthop Relat Res*. 2013;471(1):250-257.

Foran JR, Brown NM, **Della Valle CJ**, **Berger RA**, **Galante JO**. Long-term survivorship and failure modes of unicompartmental knee arthroplasty. *Clin Orthop Relat Res*. 2013;471(1):102-108.

Foran JR, Brown NM, **Della Valle CJ**, **Levine BR**, **Sporer SM**, **Paprosky WG**. Prevalence, risk factors, and management of proximal femoral remodeling in revision hip arthroplasty. *J Arthroplasty*. 2013;28(5):877-881.

Foucher KC, **Wimmer MA**. Does hip implant positioning affect the peak external adduction moments of the healthy knees of subjects with total hip replacements? *J Orthop Res*. 2013;31(8):1187-1194.

Frank JM, Riedel MD, McCormick FM, **Nho SJ**. Isolated vastus lateralis tendon avulsion. *Am J Orthop* (Belle Mead NJ). 2013;42(10):464-465.

Frank RM, **Cole BJ**. Complex cartilage cases in the athletic patient: advances in malalignment, instability, articular defects, and meniscal insufficiency. *Phys Sportsmed*. 2013;41(4):41-52.

Frank RM, Erickson B, Frank JM, **Bush-Joseph CA**, **Bach BR Jr**, **Cole BJ**, **Romeo AA**, Provencher MT, **Verma NN**. Utility of modern arthroscopic simulator training models. *Arthroscopy*. 2014;30(1):121-133.

Frank RM, Hsu AR, Gross CE, Walton DM, **Lee S**. Open and arthroscopic surgical anatomy of the ankle. *Anat Res Int*. 2013;2182650.

Friel NA, **Wang VM**, Slabaugh MA, Wang F, Chubinskaya S, **Cole BJ**. Rotator cuff healing after continuous subacromial bupivacaine infusion: an in vivo rabbit study. *J Shoulder Elbow Surg*. 2013;22(4):489-499.

Ghanem E, Heppert V, Spangehl M, Abraham J, Azzam K, Barnes L, Burgo FJ, Ebeid W, Goyal N, Guerra E, Hitt K, Kallel S, Klein G, Kosashvili Y, **Levine B**, Matsen L, Morris MJ, Purtill JJ, Ranawat C, Sharkey PF, Sierra R, Stefansdottir A. Wound management. *J Arthroplasty*. 2014;29(suppl 2):84-92.

Ghodadra N, Mall NA, Karas V, Grumet RC, Kirk S, McNickle AG, Garrido CP, **Cole BJ**, **Bach BR Jr**. Articular and meniscal pathology associated with primary anterior cruciate ligament reconstruction. *J Knee Surg*. 2013;26(3):185-193.

Gitelis S, Bayne CO, Frank JM, Filingham Y, Kent PM. Surgery in malignant bone tumors. *Curr Probl Cancer*. 2013 Jul-Aug;37(4):192-197.

Glant TT, Besenyei T, Kádár A, Kurkó J, Tryniszewska B, Gál J, Soós G, Szekanecz Z, Hoffmann G, Block JA, Katz RS, **Mikecz K**, **Rauch TA**. Differentially expressed epigenome modifiers, including aurora kinases A and B, in immune cells in rheumatoid arthritis in humans and mouse models. *Arthritis Rheum*. 2013;65(7):1725-1735.

Green JM, **Hallab NJ**, Liao YS, Narayan V, Schwarz EM, Xie C. Anti-oxidation treatment of ultra high molecular weight polyethylene components to decrease periprosthetic osteolysis: evaluation of osteolytic and osteogenic properties of wear debris particles in a murine calvaria model. *Curr Rheumatol Rep*. 2013;15(5):325.

Gross CE, Chalmers PN, Ellman M, **Fernandez JJ**, **Verma NN**. Acute brachial plexopathy after clavicular open reduction and internal fixation. *J Shoulder Elbow Surg*. 2013;22(5):e6-9.

Gross CE, Hellman M, Freedman R, Hart M, Reddy A, Salata M, **Bush-Joseph C**, **Nho SJ**. Effect of anterior acetabular rim recession on radiographic parameters: an in vivo study. *Arthroscopy*. 2013;29(8):1292-1296.

Gross CE, Hsu AR, Chahal J, **Holmes GB Jr**. Injectable treatments for noninsertional Achilles tendinosis: a systematic review. *Foot Ankle Int*. 2013;34(5):619-628.

Gross CE, Hsu AR, **Lin J**, **Holmes GB**, **Lee S**. Revision MTP arthrodesis for failed MTP arthroplasty. Foot *Ankle Spec*. 2013;6(6):471-478.

Günther KP, Schmitt J, Campbell P, Delaunay CP, Drexler H, Ettema HB, García-Cimbrelo E, Hannemann F, Hartmann A, Huberti H, Knahr K, Kunze J, Langton DJ, Lauer W, Learmonth I, Lohmann CH, Lützner J, Morlock M, Seidler A, **Wimmer MA**, Zagra L. Consensus statement "Current evidence on the management of metal-on-metal bearings"—April 16, 2012. *Hip Int*. 2013 Jan-Feb;23(1):2-5.

Gupta AK, Bruce B, Klosterman EL, McCormick F, Harris J, **Romeo AA**. Subpectoral biceps tenodesis for failed type II SLAP repair. *Orthopedics*. 2013;36(6):e723-728.

Gupta AK, Chalmers PN, Klosterman E, Harris JD, Provencher MT, **Romeo AA**. Arthroscopic distal tibial allograft augmentation for posterior shoulder instability with glenoid bone loss. *Arthrosc Tech*. 2013;2(4):e405-411.

Gupta AK, **Forsythe B**, Lee AS, Harris JD, McCormick F, Abrams GD, **Verma NN**, **Romeo AA**, **Inoue N**, **Cole BJ**. Topographic analysis of the glenoid and proximal medial tibial articular surfaces: a search for the ideal match for glenoid resurfacing. *Am J Sports Med*. 2013;41(8):1893-1899.

Gupta AK, McCormick FM, Abrams GD, Harris JD, **Bach BR Jr**, **Romeo AA**, **Verma NN**. Arthroscopic bony bankart fixation using a modified sugaya technique. *Arthrosc Tech*. 2013;2(3):e251-255.

Hall A, Eilers M, Hansen R, Robinson BS, Maloney WJ, **Paprosky WG**, Ries MD, Saleh KJ. Advances in acetabular reconstruction in revision total hip arthroplasty: maximizing function and outcomes after treatment of periacetabular osteolysis around the well-fixed shell. *J Bone Joint Surg Am*. 2013;95(18):1709-1718.

Hallab NJ, Bao QB, Brown T. Assessment of epidural versus intradiscal biocompatibility of PEEK implant debris: an in vivo rabbit model. Eur *Spine J*. 2013;22(12):2740-2751.

Hallab NJ, Caicedo M, McAllister K, **Skipor A**, Amstutz H, **Jacobs JJ**. Asymptomatic prospective and retrospective cohorts with metal-on-metal hip arthroplasty indicate acquired lymphocyte reactivity varies with metal ion levels on a group basis. *J Orthop Res*. 2013;31(2):173-182.

Hammond LC, Lin EC, Harwood DP, Juhan TW, Gochanour E, Klosterman EL, **Cole BJ**, **Nicholson GP**, **Verma NN**, **Romeo AA**. Clinical outcomes of hemiarthroplasty and biological resurfacing in patients aged younger than 50 years. *J Shoulder Elbow Surg*. 2013;22(10):1345-1351.

Hansen E, Tetreault M, Zmistowski B, **Della Valle CJ**, Parvizi J, Haddad FS, Hozack WJ. Outcome of one-stage cementless exchange for acute postoperative periprosthetic hip infection. *Clin Orthop Relat Res*. 2013;471(10):3214-3222.

Harris JD, Erickson BJ, Abrams GD, Cvetanovich GL, McCormick FM, Gupta AK, **Bach BR Jr**, **Cole BJ**. Methodologic quality of knee articular cartilage studies. *Arthroscopy*. 2013;29(7):1243-1252.

Harris JD, Erickson BJ, **Bach BR Jr**, Abrams GD, Cvetanovich GL, **Forsythe B**, McCormick FM, Gupta AK, **Cole BJ**. Return-to-sport and performance after anterior cruciate ligament reconstruction in National Basketball Association players. *Sports Health*. 2013;5(6):562-568.

Harris JD, Erickson BJ, **Bush-Joseph CA**, **Nho SJ**. Treatment of femoroacetabular impingement: a systematic review. *Curr Rev Musculoskelet Med*. 2013;6(3):207-218.

Harris JD, Frank JM, Jordan MA, **Bush-Joseph CA**, **Romeo AA**, Gupta AK, Abrams GD, McCormick FM, **Bach BR Jr**. Return to sport following shoulder surgery in the elite pitcher: a systematic review. *Sports Health*. 2013;5(4):367-376.

Harris JD, Gupta AK, Mall NA, Abrams GD, McCormick FM, **Cole BJ**, **Bach BR Jr**, **Romeo AA**, **Verma NN**. Long-term outcomes after Bankart shoulder stabilization. *Arthroscopy*. 2013;29(5):920-933.

Harris JD, Gupta AK, Mall NA, Abrams GD, McCormick FM, **Cole BJ**, **Bach BR Jr**, **Romeo AA**, **Verma NN**. Authors' reply. *Arthroscopy*. 2013;29(10):1602-1603.

Harris JD, Gupta AK, Mall NA, Abrams GD, McCormick FM, **Cole BJ**, **Bach BR Jr**, **Romeo AA**, **Verma NN**. Long-term outcomes after Bankart shoulder stabilization. *Arthroscopy*. 2013;29(5):920-933.

Harris JD, McCormick FM, Abrams GD, Gupta AK, Ellis TJ, **Bach BR Jr**, **Bush-Joseph CA**, **Nho SJ**. Complications and reoperations during and after hip arthroscopy: a systematic review of 92 studies and more than 6,000 patients. *Arthroscopy*. 2013;29(3):589-595.

Harris JD, **Romeo AA**. Arthroscopic management of the contact athlete with instability. *Clin Sports Med*. 2013;32(4):709-730.

Harris JD, Slikker W III, Gupta AK, McCormick FM, **Nho SJ**. Routine complete capsular closure during hip *Arthroscopy*. *Arthrosc Tech*. 2013;2(2):e89-94.

Healy WL, **Della Valle CJ**, Iorio R, Berend KR, Cushner FD, Dalury DF, Lonner JH. Complications of total knee arthroplasty: standardized list and definitions of the Knee Society. *Clin Orthop Relat Res*. 2013;471(1):215-220.

Heard WM, Chahal J, **Bach BR Jr**. Recognizing and managing complications in ACL reconstruction. *Sports Med Arthrosc*. 2013;21(2):106-112.

Hellman MD, Riff AJ, Haughom BD, Patel R, Stover MD, **Nho SJ**. Operative treatment of FAI: open hip preservation surgery. *Curr Rev Musculoskelet Med*. 2013;6(3):258-263.

Holmes GB Jr, Hsu AR. Correction of intermetatarsal angle in hallux valgus using small suture button device. *Foot Ankle Int*. 2013;34(4):543-549.

Holmes GB Jr. Comment on "postoperative second metatarsal fractures associated with suture-button implant in hallux valgus surgery". *Foot Ankle Int*. 2013;34(6):917-918.

Hsu AR, Gross CE, **Lee S**, Carreira DS. Extended indications for foot and ankle *Arthroscopy*. *J Am Acad Orthop Surg*. 2014;22(1):10-19.

Hsu AR, Gross CE, **Lee S**. Intraoperative O-arm computed tomography evaluation of syndesmotic reduction: case report. *Foot Ankle Int*. 2013;34(5):753-759.

Hsu AR, Gross CE, **Lin JL**. Bilateral hallux varus deformity correction with a suture button construct. *Am J Orthop* (Belle Mead NJ). 2013;42(3):121-124.

Hsu AR, **Levine BR**, **Skipor AK**, **Hallab NJ**, **Paprosky WG**, **Jacobs JJ**. Effect of a second joint arthroplasty on metal ion levels after primary total hip arthroplasty. *Am J Orthop* (Belle Mead NJ). 2013;42(10):E84-87.

Huffman GR, **Romeo AA**. Massive rotator cuff tear. *Orthopedics*. 2013;36(8):625-7.

Hussain M, Nassr A, **Natarajan RN**, **An HS**, **Andersson GB**. Biomechanics of adjacent segments after a multilevel cervical corpectomy using anterior, posterior, and combined anterior-posterior instrumentation techniques: a finite element model study. *Spine J*. 2013;13(6):689-696.

Hussain M, Nassr A, **Natarajan RN**, **An HS**, **Andersson GB**. Relationship between biomechanical changes at adjacent segments and number of fused bone grafts in multilevel cervical fusions: a finite element investigation. *J Neurosurg Spine*. 2014;20(1):22-29.

Iorio R, **Della Valle CJ**, Healy WL, Berend KR, Cushner FD, Dalury DF, Lonner JH. Stratification of standardized TKA complications and adverse events: a brief communication. *Clin Orthop Relat Res*. 2014;472(1):194-205.

Iwata T, Miyamoto K, Hioki A, Ohashi M, **Inoue N**, Shimizu K. In vivo measurement of lumbar foramen during axial loading using a compression device and computed tomography. J Spinal Disord Tech. 2013;26(5):E177-82. *J Bone Joint Surg Am*. 2014;96(3):237-243.

Jacobs JJ, **Wimmer MA**. An important contribution to our understanding of the performance of the current generation of metal-on-metal hip replacements. *J Bone Joint Surg Am*. 2013;95(8):e53.

Kancherla VK, Del Gaizo DJ, **Paprosky WG**, **Sporer SM**. Utility of trephine reamers in revision hip arthroplasty. *J Arthroplasty*. 2014;29(1):210-213.

Kang RW, **Yanke AB**, **Espinoza Orias AA**, **Inoue N**, **Nho SJ**. Emerging ideas: novel 3-D quantification and classification of cam lesions in patients with femoroacetabular impingement. *Clin Orthop Relat Res*. 2013;471(2):358-362.

Karas V, Hussey K, **Romeo AR**, **Verma N**, **Cole BJ**, Mather RC III. Comparison of subjective and objective outcomes after rotator cuff repair. *Arthroscopy*. 2013;29(11):1755-1761.

Katz JN, Brophy RH, Chaisson CE, de Chaves L, **Cole BJ**, Dahm DL, Donnell-Fink LA, Guermazi A, Haas AK, Jones MH, Levy BA, Mandl LA, Martin SD, Marx RG, Miniaci A, Matava MJ, Palmisano J, Reinke EK, Richardson BE, Rome BN, Safran-Norton CE, Skoniecki DJ, Solomon DH, Smith MV, Spindler KP, Stuart MJ, Wright J, Wright RW, Losina E. Surgery versus physical therapy for a meniscal tear and osteoarthritis. *N Engl J Med*. 2013;368(18):1675-1684.

Kido M, Ikoma K, Imai K, Tokunaga D, **Inoue N**, Kubo T. Load response of the medial longitudinal arch in patients with flatfoot deformity: in vivo 3D study. *Clin Biomech* (Bristol, Avon). 2013;28(5):568-573.

Kim JS, Ellman MB, Yan D, **An HS**, Kc R, Li X, Chen D, Xiao G, Cs-Szabo G, Hoskin DW, Buechter DD, Van Wijnen AJ, Im HJ. Lactoferricin mediates anti-inflammatory and anti-catabolic effects via inhibition of IL-1 and LPS activity in the intervertebral disc. *J Cell Physiol*. 2013;228(9):1884-1896.

Klein GR, Levine HB, **Sporer SM**, Hartzband MA. Fracture of the proximal tibia after revision total knee arthroplasty with an extensor mechanism allograft. *J Arthroplasty*. 2013;28(2):375.e9-375.e12.

Knowlton CB, **Wimmer MA**. An autonomous mathematical reconstruction to effectively measure volume loss on retrieved polyethylene tibial inserts. *J Biomed Mater Res B Appl Biomater*. 2013;101(3):449-457.

Kornblum MB, Turner AW, Cornwall GB, Zatushevsky MA, **Phillips FM**. Biomechanical evaluation of stand-alone lumbar polyether-ether-ketone interbody cage with integrated screws. *Spine J*. 2013;13(1):77-84.

Kurkó J, Besenyei T, Laki J, **Glant TT**, **Mikecz K**, Szekanecz Z. Genetics of rheumatoid arthritis - a comprehensive review. *Clin Rev Allergy Immunol*. 2013;45(2):170-179.

Lee AS, Ellman MB, Yan D, Kroin JS, **Cole BJ**, van Wijnen AJ, Im HJ. A current review of molecular mechanisms regarding osteoarthritis and pain. *Gene*. 2013;527(2):440-447.

Lee JY, Whang PG, Lee JY, **Phillips FM**, Patel AA. Lumbar spinal stenosis. *Instr Course Lect*. 2013;383-396.

Levine B, **Rosenberg AG**. The simple unicondylar knee: extramedullary technique. *Clin Sports Med*. 2014;33(1):77-85.

Levine BR, Hsu AR, **Skipor AK**, **Hallab NJ**, **Paprosky WG**, **Galante JO**, **Jacobs JJ**. Ten-year outcome of serum metal ion levels after primary total hip arthroplasty: a concise follow-up of a previous report. *J Bone Joint Surg Am*. 2013;95(6):512-518.

Liao Y, Hoffman E, **Wimmer M**, **Fischer A**, **Jacobs J**, Marks L. CoCrMo metal-on-metal hip replacements. *Phys Chem Chem Phys*. 2013;15(3):746-756.

Lichstein P, Gehrke T, Lombardi A, Romano C, Stockley I, Babis G, Bialecki J, Bucsi L, Cai X, Cao L, de Beaubien B, Erhardt J, Goodman S, Jiranek W, Keogh P, Lewallen D, Manner P, Marczynski W, Mason JB, Mulhall K, **Paprosky W**, Patel P, Piccaluga F, Polkowski G, Pulido L, Stockley I, Suarez J, Thorey F, Tikhilov R, Velazquez JD, Winkler H. One-stage vs two-stage exchange. *J Arthroplasty*. 2014;29(suppl 2):108-111.

Lin EC, Mall NA, Dhawan A, Sherman SL, McGill KC, Provencher MT, **Nicholson GP**, **Cole BJ**, Solomon DJ, **Verma NN**, **Romeo AA**. Arthroscopic primary rotator cuff repairs in patients aged younger than 45 years. *Arthroscopy*. 2013;29(5):811-817.

Lombardi AV Jr, Nunley RM, Berend KR, Ruh EL, Clohisy JC, Hamilton WG, **Della Valle CJ**, Parvizi J, Barrack RL. Do patients return to work after total knee arthroplasty? *Clin Orthop Relat Res*. 2014;472(1):138-146.

Low K, Smith J, **Lee S**, Newbury-Ecob R. A mother and daughter with a novel phenotype of hand and foot abnormalities and severe pectus excavatum. *Am J Med Genet A*. 2013;161A(8):2056-2059.

Lu Y, Abbassi S, Li F, Ding M, Wu G, Gu J, **Zheng Q**. Distinct function of P63 isoforms during embryonic skeletal development. *Gene*. 2013;519(2):251-259.021.

Lundberg HJ, Knowlton C, **Wimmer MA**. Fine tuning total knee replacement contact force prediction algorithms using blinded model validation. *J Biomech Eng*. 2013;135(2):021015.

Ma Y, Ren Y, Han EQ, Li H, Chen D, **Jacobs JJ**, **Gitelis S**, O'Keefe RJ, Konttinen YT, Yin G, Li TF. Inhibition of the Wnt-βcatenin and Notch signaling pathways sensitizes osteosarcoma cells to chemotherapy. *Biochem Biophys Res Commun*. 2013;431(2):274-279.118.

Mall NA, Foley E, Chalmers PN, **Cole BJ**, **Romeo AA**, **Bach BR Jr**. Degenerative joint disease of the acromioclavicular joint: a review. *Am J Sports Med*. 2013;41(11):2684-2692.

Mall NA, Hammond JE, Lenart BA, Enriquez DJ, Twigg SL, **Nicholson GP**. Suprascapular nerve entrapment isolated to the spinoglenoid notch: surgical technique and results of open decompression. *J Shoulder Elbow Surg*. 2013;22(11):e1-8.

Mall NA, Lee AS, Chahal J, Sherman SL, **Romeo AA**, **Verma NN**, **Cole BJ**. An evidenced-based examination of the epidemiology and outcomes of traumatic rotator cuff tears. *Arthroscopy*. 2013;29(2):366-376.

Mall NA, Lee AS, Chahal J, Van Thiel GS, **Romeo AA**, **Verma NN**, **Cole BJ**. Transosseous-equivalent rotator cuff repair: a systematic review on the biomechanical importance of tying the medial row. *Arthroscopy*. 2013;29(2):377-386.

Mall NA, Van Thiel GS, Heard WM, Paletta GA, **Bush-Joseph C**, **Bach BR Jr**. Paget-schroetter syndrome: a review of effort thrombosis of the upper extremity from a sports medicine perspective. *Sports Health*. 2013;5(4):353-356.

MARS Group. Radiographic findings in revision anterior cruciate ligament reconstructions from the Mars cohort. *J Knee Surg*. 2013;26(4):239-247.

Martin EJ, Pourzal R, **Mathew MT**, Shull KR. Dominant role of molybdenum in the electrochemical deposition of biological macromolecules on metallic surfaces. *Langmuir*. 2013;29(15):4813-4822.

Mascarenhas R, Bonci G, Bowman KF, **Forsythe B**, Harner CD. Combined ACL-posterolateral corner injury in a skeletally immature athlete. *J Knee Surg*. 2013;26 Suppl S94-9.

Mather RC III, Koenig L, Kocher MS, Dall TM, Gallo P, Scott DJ, **Bach BR Jr**, Spindler KP; MOON Knee Group. Societal and economic impact of anterior cruciate ligament tears. *J Bone Joint Surg Am*. 2013;95(19):1751-1759.

Mazzocca AD, McCarthy MB, Ledgard FA, Chowaniec DM, McKinnon WJ Jr, Delaronde S, Rubino LJ, Apolostakos J, **Romeo AA**, Arciero RA, Beitzel K. Histomorphologic changes of the long head of the biceps tendon in common shoulder pathologies. *Arthroscopy*. 2013;29(6):972-981.

McCormick F, Bhatia S, Chalmers P, Gupta A, **Verma N**, **Romeo AA**. The management of type II superior labral anterior to posterior injuries. *Orthop Clin North Am*. 2014;45(1):121-128.

McCormick F, Cvetanovich GL, Kim JM, Harris JD, Gupta AK, Abrams GD, **Romeo AA**, Provencher MT. An assessment of the quality of rotator cuff randomized controlled trials: utilizing the Jadad score and CONSORT criteria. *J Shoulder Elbow Surg*. 2013;22(9):1180-1185.

McCormick F, Harris JD, Abrams GD, Frank R, Gupta A, Hussey K, Wilson H, **Bach B Jr, Cole B**. Trends in the surgical treatment of articular cartilage lesions in the United States: an analysis of a large private-payer database over a period of 8 years. *Arthroscopy*. 2014;30(2):222-226.

Meyer JN, **Mathew MT**, **Wimmer MA**, LeSuer RJ. Effect of tribolayer formation on corrosion of CoCrMo alloys investigated using scanning electrochemical microscopy. *Anal Chem*. 2013;85(15):7159-7166.

Misják P, Bββze S, Horváti K, Pásztói M, Pálóczi K, Holub MC, Szakács F, Aradi B, György B, Szabó TG, Nagy G, **Glant TT**, **Mikecz K**, Falus A, Buzás EI. The role of citrullination of an immunodominant proteoglycan (PG) aggrecan T cell epitope in BALB/c mice with PG-induced arthritis. *Immunol Lett*. 2013;152(1):25-31.

Moss IL, Zhang Y, Shi P, Chee A, Piel MJ, **An HS**. Retroperitoneal approach to the intervertebral disc for the annular puncture model of intervertebral disc degeneration in the rabbit. *Spine J*. 2013;13(3):229-234.

Nho SJ, Freedman RL, Federer AE, Mather RC III, **Espinoza Orias AA**, **Wang VM**, Van Thiel GS. Computed tomographic analysis of curved and straight guides for placement of suture anchors for acetabular labral refixation. *Arthroscopy*. 2013;29(10):1623-1627.

Nho SJ, Kymes SM, Callaghan JJ, Felson DT. The burden of hip osteoarthritis in the United States: epidemiologic and economic considerations. *J Am Acad Orthop Surg*. 2013;21(suppl S1-6).

Nicholson GP. CORR Insights(®): Assessing Shoulder Motion in Children: Age Limitations to Mallet and ABC Loops. *Clin Orthop Relat Res*. 2014;472(2):749.

O'Keefe JA, **Espinoza Orías AA**, Khan H, **Hall DA**, Berry-Kravis E, **Wimmer MA**. Implementation of a markerless motion analysis method to quantify hyperkinesis in males with fragile X syndrome. *Gait Posture*. 2014;39(2):827-830.

Pan TC, Zhang RZ, Markova D, Arita M, Zhang Y, Bogdanovich S, Khurana TS, Bönnemann CG, Birk DE, Chu ML. COL6A3 protein deficiency in mice leads to muscle and tendon defects similar to human collagen VI congenital muscular dystrophy. *J Biol Chem*. 2013;288(20):14320-14331.

Paprosky WG, Cross MB. CORR Insights®: Validity and reliability of the Paprosky acetabular defect classification. *Clin Orthop Relat Res*. 2013;471(7):2266.

Parvizi J, Nunley RM, Berend KR, Lombardi AV Jr, Ruh EL, Clohisy JC, Hamilton WG, **Della Valle CJ**, Barrack RL. High level of residual symptoms in young patients after total knee arthroplasty. *Clin Orthop Relat Res*. 2014;472(1):133-137.

Pascual-Garrido C, Moran CJ, Green DW, **Cole BJ**. Osteochondritis dissecans of the knee in children and adolescents. *Curr Opin Pediatr*. 2013;25(1):46-51.

Phillips FM, Isaacs RE, Rodgers WB, Khajavi K, Tohmeh AG, Deviren V, Peterson MD, Hyde J, Kurd M. Adult degenerative scoliosis treated with XLIF: clinical and radiographical results of a prospective multicenter study with 24-month follow-up. *Spine*. 2013;38(21):1853-1861.

Phillips FM, Lee JY, Geisler FH, Cappuccino A, Chaput CD, DeVine JG, Reah C, Gilder KM, Howell KM, McAfee PC. A prospective, randomized, controlled clinical investigation comparing PCM cervical disc arthroplasty with anterior cervical discectomy and fusion. 2-year results from the US FDA IDE clinical trial. *Spine*. 2013;38(15):E907-918.

Phillips FM, Slosar PJ, Youssef JA, **Andersson G**, Papatheofanis F. Lumbar spine fusion for chronic low back pain due to degenerative disc disease: a systematic review. *Spine*. 2013;38(7):E409-422.

Plummer DR, Haughom BD, **Della Valle CJ**. Dual mobility in total hip arthroplasty. *Orthop Clin North Am*. 2014;45(1):1-8.

Pui CM, Bostrom MP, Westrich GH, **Della Valle CJ**, Macaulay W, Mont MA, Padgett DE. Increased complication rate following conversion total hip arthroplasty after cephalomedullary fixation for intertrochanteric hip fractures: a multi-center study. *J Arthroplasty*. 2013;28(suppl 8):45-47.

Qasim M, Hong JT, **Natarajan RN**, **An HS**. A biomechanical comparison of intralaminar C7 screw constructs with and without offset connector used for C6-7 cervical spine immobilization : a finite element study. *J Korean Neurosurg Soc*. 2013;53(6):331-336.331.

Qasim M, **Natarajan RN**, **An HS**, **Andersson GB**. Damage accumulation location under cyclic loading in the lumbar disc shifts from inner annulus lamellae to peripheral annulus with increasing disc degeneration. *J Biomech*. 2014;47(1):24-31.

Riff AJ, Sah AP, **Della Valle CJ**. Outcomes and complications of unicondylar arthroplasty. *Clin Sports Med*. 2014;33(1):149-160.

Rihn JA, Currier BL, **Phillips FM**, Glassman SD, Albert TJ. Defining the value of spine care. *J Am Acad Orthop Surg*. 2013;21(7):419-426.

Rodeghero R, Cao Y, Olalekan SA, Iwakua Y, **Glant TT**, Finnegan A. Location of CD4+ T cell priming regulates the differentiation of Th1 and Th17 cells and their contribution to arthritis. *J Immunol*. 2013;190(11):5423-5435.

Romeo AA. CORR Insights ®: Foreign body reaction to acellular dermal matrix allograft in biologic glenoid resurfacing. *Clin Orthop Relat Res*. 2013;471(8):2459-2460.

Rosenthal BD, Hulst JB, Moric M, **Levine BR**, **Sporer SM**. The effect of payer type on clinical outcomes in total knee arthroplasty. *J Arthroplasty*. 2014;29(2):295-298.

Salata MJ, Bailey JR, Bell R, Frank RM, McGill KC, Lin EC, Kercher JS, **Wang VM**, Provencher MT, Mazzocca AD, **Verma NN**, **Romeo AA**. Effect of interference screw depth on fixation strength in biceps tenodesis. *Arthroscopy*. 2014;30(1):11-15.

Salata MJ, **Nho SJ**, Chahal J, Van Thiel G, Ghodadra N, Dwyer T, **Romeo AA**. Arthroscopic anatomy of the subdeltoid space. *Orthop Rev* (Pavia). 2013;5(3):e25.

Salata MJ, Sherman SL, Lin EC, Sershon RA, Gupta A, Shewman E, **Wang VM**, **Cole BJ**, **Romeo AA**, **Verma NN**. Biomechanical evaluation of transosseous rotator cuff repair: do anchors really matter? *Am J Sports Med*. 2013;41(2):283-290.

Samadian S, **Phillips FM**, Deeab D. Mycobacterium bovis vertebral osteomyelitis and discitis with adjacent mycotic abdominal aortic aneurysm caused by intravesical BCG therapy: a case report in an elderly gentleman. *Age Ageing*. 2013;42(1):129-131.

Samelko L, Caicedo MS, Lim SJ, **Della-Valle C**, **Jacobs J**, **Hallab NJ**. Cobalt-alloy implant debris induce HIF-1βhypoxia associated responses: a mechanism for metal-specific orthopedic implant failure. *PLoS One*. 2013;8(6):e67127.

Sershon RA, Mather RC, Sherman SL, McGill KC, **Romeo AA**, **Verma NN**. Low accuracy of interpretation of rotator cuff MRI in patients with osteoarthritis. *Acta Orthop*. 2013;84(5):479-482.

Sershon RA, Van Thiel GS, Lin EC, McGill KC, **Cole BJ**, **Verma NN**, **Romeo AA**, **Nicholson GP**. Clinical outcomes of reverse total shoulder arthroplasty in patients aged younger than 60 years. *J Shoulder Elbow Surg*. 2014;23(3):395-400.

Shakoor N, Lidtke RH, **Wimmer MA**, Mikolaitis RA, Foucher KC, Thorp LE, Fogg LF, Block JA. Improvement in knee loading after use of specialized footwear for knee osteoarthritis: results of a six-month pilot investigation. *Arthritis Rheum*. 2013;65(5):1282-1289.

Shen H, Perez RE, Davaadelger B, **Maki CG**. Two 4N cell-cycle arrests contribute to cisplatin-resistance. *PLoS One*. 2013;8(4):e59848.

Sheth NP, Brown NM, Moric M, **Berger RA**, **Della Valle CJ**. Operative treatment of early peri-prosthetic femur fractures following primary total hip arthroplasty. *J Arthroplasty*. 2013;28(2):286-291.

Sheth NP, Nelson CL, **Paprosky WG**. Femoral bone loss in revision total hip arthroplasty: evaluation and management. *J Am Acad Orthop Surg*. 2013;21(10):601-612.

Sheth NP, Nelson CL, Springer BD, Fehring TK, **Paprosky WG**. Acetabular bone loss in revision total hip arthroplasty: evaluation and management. *J Am Acad Orthop Surg*. 2013;21(3):128-139.

Sierra RJ, Kassel CA, Wetters NG, Berend KR, **Della Valle CJ**, Lombardi AV. Revision of unicompartmental arthroplasty to total knee arthroplasty: not always a slam dunk! *J Arthroplasty*. 2013;28(suppl 8):128-132.

Smyth NA, Murawski CD, Fortier LA, **Cole BJ**, Kennedy JG. Platelet-rich plasma in the pathologic processes of cartilage: review of basic science evidence. *Arthroscopy*. 2013;29(8):1399-1409.

Sporer SM. 2012 annual meeting of the American Association of Hip and Knee Surgeons (AAHKS). *J Arthroplasty*. 2013;28(suppl 8):1.

Stoyanov P, Stemmer P, Järvi TT, Merz R, Romero PA, Scherge M, Kopnarski M, Moseler M, **Fischer A**, Dienwiebel M. Friction and wear mechanisms of tungsten-carbon systems: a comparison of dry and lubricated conditions. *ACS Appl Mater Interfaces*. 2013;5(13):6123-6135.

Strauss EJ, **Verma NN**, Salata MJ, McGill KC, Klifto C, **Nicholson GP**, **Cole BJ**, **Romeo AA**. The high failure rate of biologic resurfacing of the glenoid in young patients with glenohumeral arthritis. *J Shoulder Elbow Surg*. 2014;23(3):409-419.

Sundman EA, **Cole BJ**, Karas V, **Della Valle C**, Tetreault MW, Mohammed HO, Fortier LA. The anti-inflammatory and matrix restorative mechanisms of platelet-rich plasma in osteoarthritis. *Am J Sports Med*. 2014;42(1):35-41.

Szekanecz Z, Meskó B, Poliska S, Váncsa A, Szamosi S, Végh E, Simkovics E, Laki J, Kurkó J, Besenyei T, **Mikecz K**, **Glant TT**, Nagy L.

Pharmacogenetics and pharmacogenomics in rheumatology. *Immunol Res*. 2013;56(2-3):325-333.

Tetreault MW, Shukla SK, Yi PH, **Sporer SM**, **Della Valle CJ**. Are short fully coated stems adequate for "simple" femoral revisions? *Clin Orthop Relat Res*. 2014;472(2):577-583.

Tetreault MW, Wetters NG, Aggarwal V, Mont M, Parvizi J, **Della Valle CJ**. The Chitranjan Ranawat Award: Should prophylactic antibiotics be withheld before revision surgery to obtain appropriate cultures? *Clin Orthop Relat Res*. 2014;472(1):52-56.

Tetreault MW, Wetters NG, Aggarwal VK, Moric M, Segreti J, Huddleston JI III, Parvizi J, **Della Valle CJ**. Should draining wounds and sinuses associated with hip and knee arthroplasties be cultured? *J Arthroplasty*. 2013;28(suppl 8):133-136.

Van Thiel GS, Harris JD, Kang RW, Chahal J, **Della Valle CJ**, **Bush-Joseph CA**, **Nho SJ**. Age-related differences in radiographic parameters for femoroacetabular impingement in hip arthroplasty patients. *Arthroscopy*. 2013;29(7):1182-1187.

Verma NN, Harris JD. Surgery: Preserving shoulder movement in advanced OA—yes we CAM! *Nat Rev Rheumatol*. 2013;9(7):386-388.

Wetters NG, Murray TG, Moric M, **Sporer SM**, **Paprosky WG**, **Della Valle CJ**. Risk factors for dislocation after revision total hip arthroplasty. *Clin Orthop Relat Res*. 2013;471(2):410-416.

Wimmer MA, **Laurent MP**, Dwiwedi Y, Gallardo LA, Chipps KA, Blackmon JC, Kozub RL, Bardayan DW, Gross CJ, Stracener DW, Smith MS, Nesaraja CD, Erikson L, Patel N, Rehm KE, Ahmad I, Greene JP, Greife U. Wear measurement of highly cross-linked UHMWPE using a 7Be tracer implantation technique. *J Biomed Mater Res B Appl Biomater*. 2013;101(3):423-429.

Yanke AB, Bell R, Lee A, Kang RW, Mather RC III, Shewman EF, **Wang VM**, **Bach BR Jr**. The biomechanical effects of 1.0 to 1.2 Mrad of β irradiation on human bone-patellar tendon-bone allografts. *Am J Sports Med*. 2013;41(4):835-840.

Yanke AB, Bell R, Lee AS, Shewman E, **Wang VM**, **Bach BR Jr**. Central-third bone-patellar tendon-bone allografts demonstrate superior biomechanical failure characteristics compared with hemi-patellar tendon grafts. *Am J Sports Med*. 2013;41(11):2521-2526.

Yanke AB, Hart MA, McCormick F, **Nho SJ**. Endoscopic repair of a gluteus medius tear at the musculotendinous junction. *Arthrosc Tech*. 2013;2(2):e69-72.

Yanke AB, Mall NA, Sherman SL, **Bach BR Jr**. 5 points on transtibial anterior cruciate ligament reconstruction. *Am J Orthop* (Belle Mead NJ). 2013;42(7):305-308.

Yi PH, Cross MB, Moric M, **Sporer SM**, **Berger RA**, **Della Valle CJ**. The 2013 Frank Stinchfield Award: diagnosis of infection in the early postoperative period after total hip arthroplasty. *Clin Orthop Relat Res*. 2014;472(2):424-429.

Zmistowski B, **Della Valle C**, Bauer TW, Malizos KN, Alavi A, Bedair H, Booth RE, Choong P, Deirmengian C, Ehrlich GD, Gambir A, Huang R, Kissin Y, Kobayashi H, Kobayashi N, Krenn V, Lorenzo D, Marston SB, Meermans G, Perez J, Ploegmakers JJ, **Rosenberg A**, Simpendorfer C, Thomas P, Tohtz S, Villafuerte JA, Wahl P, Wagenaar FC, Witzo E. Diagnosis of periprosthetic joint infection. *J Arthroplasty*. 2014;29(suppl 2):77-83.

Zmistowski B, Tetreault MW, Alijanipour P, Chen AF, **Della Valle CJ**, Parvizi J. Recurrent periprosthetic joint infection: persistent or new infection? *J Arthroplasty*. 2013;28(9):1486-1489.

Bernard R. Bach Jr., MD (left), and Charles A. Bush-Joseph, MD.

Team Player

An interview with renowned sports medicine surgeon, respected teacher, and dedicated family man, Bernard R. Bach Jr, MD

BY CHARLES A. BUSH-JOSEPH, MD

When it comes to the knee injury that sidelined Bernard R. Bach Jr, MD, you might say football's loss was sports medicine's gain.

Bach would not be one of the leaders in knee ligament repair today had he not blown out his own anterior cruciate ligament (ACL) and medial collateral ligament (MCL) before his senior year at Northville High School in suburban Detroit. The injury was typically career ending at that time, the Division I scholarship offers evaporated, and Bach was forced to contemplate his future—not the future he had envisioned since childhood, playing at the University of Michigan, but life after football.

That's how, at an age when "thinking ahead" usually means making weekend plans, Bach decided on his career path. With his own successfully repaired knee, he was able to play both football and baseball in college—for Harvard. An Ivy League degree and 2 additional ACL injuries later, he traded in his jersey for a white coat and never looked back.

After training under some of the earliest pioneers in the emerging field of sports medicine, Bach came to Chicago in 1986 and started the sports medicine program at Rush. Under his leadership, our program has achieved a reputation for excellence, fueled by cutting-edge research and one of the most prestigious sports medicine fellowships in the United States.

Being the consummate team player, Bach prefers to downplay his individual accomplishments, so I won't list them here. I will simply say that throughout his career, he has been a mentor, teacher, partner, friend, and doctor to more people than I can count—myself included. I'm honored to interview him and share his story as part of this year's *Rush Orthopedics Journal*.

BUSH-JOSEPH: Let's start by talking about how that knee injury in high school put you on the path to becoming a sports medicine surgeon.

BACH: For many people who go into sports medicine, there is a sentinel moment where an injury results in consulting with an orthopedic surgeon, and that contact plays a role in choosing this particular specialty. My sentinel moment was that catastrophic ACL-MCL injury.

Both the surgery and my treating surgeon, Tom Peterson, MD, had a huge impact on me—so much so that I was inspired to write a paper on knee ligament injuries as a high school senior. The *Detroit Free Press* did a student-athlete profile on me in 1970, and when the reporter asked what I wanted to do career-wise, I said I wanted to be an orthopedic surgeon who did knee surgeries and took care of athletes.

BUSH-JOSEPH: So the injury opened up an avenue that you would not otherwise have pursued.

BACH: Yes, because my whole life I wanted to play football at Michigan.

BUSH-JOSEPH: Instead, you went to Harvard.

BACH: I did. It was fortuitous, because that's where I met my first mentor, Thomas "Bart" Quigley, MD, who was the Harvard team physician. I met him my freshman year. Then I injured my other knee playing football, and I needed another surgery, and Dr Quigley did the procedure.

After that, I started hanging out in the Harvard field house training room, listening to Dr Quigley's stories and watching him evaluate student-athletes. He became my thesis advisor on Achilles tendon ruptures. I was the only undergraduate he ever sponsored for a thesis, which was a big deal for me.

I was able to come back from the injury and play sports again before re-injuring my initial reconstruction during my senior season. Tom Peterson did the repair on that one. By the time I was 21, I'd had 3 major knee operations and spent a total of 6 months in casts.

BUSH-JOSEPH: Those times when you were in casts, did you ever have a sense of frustration, or a feeling of, "Why does this keep happening to me?"

BACH: No, but the injuries definitely opened up my eyes to the realization that I wasn't—and this is something I stress today with young athletes—immortal. They also gave me an appreciation for what some disabled people go through; I developed a real sense of empathy as a result.

BUSH-JOSEPH: The shift from big incisions and lots of time in a cast—which you experienced as a patient—to arthroscopically assisted repair for ACL injuries happened right around the time you finished your training. In your experience, has any other orthopedic procedure come further over the past 25 years than ACL repair?

BACH: It's certainly come a very long way. As you know, arthroscopy evolved for the most part in the 1970s. With the advances in visualization and instrumentation, we went from being able to do diagnostic arthroscopy to being able to treat problems—take out a loose body or trim a meniscal tear; to being able to perform reconstructive procedures.

As a resident, I had the opportunity to observe ACL surgeries that were big procedures, with extensive incisions and considerable postoperative time in casts.

I wanted to train at a place where I could potentially learn a different way. When I started my fellowship at the Hospital for Special Surgery under Russell Warren, MD, they did have different approaches. It was the dawn of the era of early protected motion, although they were still doing open procedures.

Halfway through my fellowship year in 1986, Dr Warren started to do arthroscopically assisted, 2-incision ACL surgical reconstructions for chronic ACL-deficient knees. Within 6 months after I left, he was doing even acute or semi-acute ACLs arthroscopically assisted. It happened that quickly.

BUSH-JOSEPH: It seems like it was perfect timing for you.

BACH: It really was. You'll remember that when I got here in 1986, Rush still had archaic instrumentation. We had virtually no arthroscopic equipment. My first year, most of the procedures we did were open.

Once we invested in the technology, with the improvements in instrumentation and cameras, we were able to embark on the arthroscopic technique. I don't think I've done an open repair since.

I remember one time while fixing a torn meniscus, I was struggling with a loose body, and one of the residents said, "Well, Dr Bach, why don't you just open up the knee?" I told him, "You may have done that in the past, but I'm never opening up another knee again."

BUSH-JOSEPH: What made you decide you wanted to build a sports medicine program at Rush, as opposed to just being another busy orthopedic surgeon like so many other surgeons of your era?

BACH: There were 2 individuals during my training who impacted my thought process about my career. The first was Art Boland, MD, who followed Dr Quigley at Harvard as team physician. I rotated with Art as a fourth-year medical student at the University of Cincinnati, and I was impressed with what a truly down-to-earth individual he was; you felt like you knew him very well within 15 minutes. Clearly, he forgot to take the medical school course on arrogance. His humility and genuineness were qualities I decided I wanted to emulate in my career.

The other person was Dr Warren. He was the "coach" who really pushed me. He would say, "You're good at teaching, you like doing this, and you seem to have an ability to do research and write. If you go into private practice, you'll be bored." He was probably right.

As trainees we become a synthesis of our mentors, and I was very fortunate to have Drs Frank Noyes (at the University of Cincinnati), Art Boland, and Russ Warren imprint their professional DNA upon me.

BUSH-JOSEPH: What attracted you to Rush specifically?

BACH: Dr Warren was actually the one who directed me to Dr Jorge Galante, MD, DMSc, who was then the chairman of the Department of Orthopedic Surgery at Rush. I just saw great opportunities here.

Looking at the giants in sports medicine in Chicago at that time, Howard Sweeney, MD, was well known in the Evanston area, Bruce Reider, MD, was at the University of Chicago, and Gordon Nuber, MD, was at Northwestern. As for Rush, it had a world-class joint-replacement program, a spine program, and an orthopedic oncology program. There were no hand surgeons, no foot and ankle surgeons, and no sports surgeons.

Fortunately, Dr Galante and I were on the same page. I remember him saying, "I don't want you to build a practice, I want you to build a program." My whole approach over the last 28 years for the Division of Sports Medicine has been "we" rather than "me," trying to recruit partners who are as good as—if not better than—I am, and we all share in the glory.

BUSH-JOSEPH: Once the division was up and running, when did you feel you were ready to start the sports medicine fellowship program?

BACH: I don't think I was ever really ready, but we started one in 1988. We had 1 fellow doing a 6-month rotation, and the program has evolved to having 5 sports medicine fellows annually. We also added a shoulder fellow several years ago.

BUSH-JOSEPH: Where does the program stand today?

BACH: It's become one of the most sought-after sports medicine fellowships in the nation. Nearly 80 fellows have trained at Rush, and we have gone from training clinicians who went out into a private practice setting to really training the next generation of sports medicine leaders.

What's wonderful about the program is that it's so clinically diverse. Our trainees are exposed to state-of-the-art knee ligament surgery and arthroscopic rotator cuff surgery, and Rush is unique in the amount of shoulder joint replacement surgery—including complex procedures—that our fellows experience. We are also probably the premier center in the U.S. for advanced articular cartilage preservation, and we are rapidly becoming one of the busiest hip arthroscopy centers in the country.

BUSH-JOSEPH: It's also recognized as probably the most academic sports medicine fellowship program. Is that something you're especially proud of?

BACH: Absolutely. You know, we really owe that reputation to some of our fellows from about a decade ago. They showed us how much a fellow could accomplish in just 1 year. We realized that they could easily publish a dozen papers, so that's what the expectation became. It's quite competitive among our fellows these days. We've had some publish 30, 40, even 50 papers related to their 1-year fellowship experience.

We've come to expect a lot from our fellows, but in return we give a tremendous amount. For instance, we encourage them to attend as many courses as they can, based on time constraints and economics. They often go to 5, 10, 20 courses, whereas if you look at most fellowship programs, a fellow is lucky to attend 1 or 2 courses. We are setting the "professional dining table" for their careers.

The high standards set by the fellows have also changed how our residents perceive themselves and what *they* are capable of doing. Our residents are receiving world-class postgraduate training, and they now have greater expectations in terms of becoming the next generation of leaders as well. It has evolved to where each year, at least 1 of our residents is the gold standard to which I compare all of our sports fellowship applicants. That's the caliber of our residents these days.

BUSH-JOSEPH: You've been able to develop a relationship with every individual you've trained. How do you manage to balance that mentorship approach with the high demands of your practice?

BACH: I've always tried to foster a premium on education within the division. I think of our fellows as part of the family, just as I do my partners. No one is mandating that a person spend that extra year of training, and I think it's admirable when someone—especially in this economy and given how medicine is changing—is willing to do that. They're giving us a year of their lives, and it's the last year of supervision before they go out into the world on their own. I feel privileged to be able to give them a good foundation on which to build their careers, as my mentors did for me.

BUSH-JOSEPH: Our division has been quite prolific in both basic science and translational research over the years. What are some of the projects that stand out to you as really helping to improve treatment for sports injuries?

BACH: With knee ligament research, some of our basic science studies that looked at ligament fixation and ligament healing have impacted what we do clinically in the operating room. We have published numerous clinical follow-up studies on ACL surgery results, and I think we've done a great job looking at different forms of fixation and healing in rotator cuff surgery. The challenge there has always been trying to get the darn rotator cuff tendons to heal.

Our specialists in articular cartilage have done spectacular research on meniscal transplants and articular cartilage healing. We have also made some nice contributions to the literature regarding bone deficiency situations with shoulder dislocations (instability). We've learned, for instance, that you have to reinforce that deficiency with additional bone to more predictably stabilize the shoulder.

BUSH-JOSEPH: As a result of this type of research, we're doing a much better job of treating many sports injuries today than a few decades ago. Do you think we're also better at preventing injuries?

BACH: Yes and no. For example, we haven't been able to figure out how to prevent ACL injuries in girls and women. A female high school basketball, soccer, or volleyball player is 6 to 8 times more likely to tear her ACL than her male counterpart. The sports medicine community is trying to address that issue, but so far we haven't figured out how to keep those girls from sustaining ACL tears.

THE NATURAL: Bach during his playing days at Northville High School.

I think one of the biggest challenges with prevention is that sports have become so specialized. When I was in high school, we didn't use the term "cross-training," but we varied things up. When football season ended, we moved to basketball, and at the end of basketball season, it was time for baseball. At Harvard, we didn't have mandatory winter training or spring football, so I was able to play baseball, too. You could play 2 sports back then with a lot less time involvement than it takes to play 1 sport today.

Now we are dealing with kids who are being forced to commit to 1 sport at a very early age. If the kids are talented, they're told that

to make it at the next level, they have to play on the traveling team. By the time they get to junior high, they're playing on multiple teams for the same sport, and it's a year-round commitment. Many kids drop out of organized sports by age 13 because of coaches, parents, and "burnout." I wish I had a quarter for every parent who told me their kid was playing at an "elite level."

There's been some progress at the Little League baseball level in terms of regulating pitch counts and mandating not throwing curve balls. It's tough, though, because these kids often play in several different leagues at once, so they get their 60 or 80 or 90 pitches for their house league, and then 60 or 80 or 90 more over the weekend with their traveling team. That puts them at risk of developing elbow or shoulder problems very early on. Overuse injuries are epidemic in these young kids.

BUSH-JOSEPH: During your career, you've taken care of world-class and pro athletes all the way down to recreational and high school athletes. Who do you most enjoy taking care of?

BACH: You see all the highlights about Doctor X who took care of this pro athlete or that celebrity. For almost all of us in sports medicine, our practices are primarily based on taking care of recreational and high school athletes. I love taking care of high school athletes. You were also one of my favorite patients, Chuck.

BUSH-JOSEPH: I picked the best doctor I knew to fix both of my ACLs.

BACH: Well, the best one you could find whose office is literally right next door to yours!

BUSH-JOSEPH: Were you nervous operating on a friend and partner?

BACH: No, I wasn't nervous. Patients have a right to go wherever they want for their care. When I'm asked to take care of physicians and their families, I see it as the ultimate compliment.

The problem is that because you want your peers to do well, there is that potential for you to do things differently. You have to treat those individuals—whether they're high profile athletes, CEOs, or physicians—the same exact way you treat all your patients. When you cut corners or try to do "extra things," that's when you run into problems. With you, I did it the way I always do it. Fortunately, you had very good outcomes, and we're still great friends.

BUSH-JOSEPH: You've received numerous honors during your career. Which have been the most meaningful to you?

BACH: Being named president of the American Orthopaedic Society for Sports Medicine (AOSSM) was definitely a highlight of my career. There were a number of people who I feel were equally deserving, so I'm still not certain how they chose me. That was a wonderful honor.

The George D. Rovere, MD, Award for Education from the AOSSM was another special award, because teaching has always been so important to me and such an emphasis throughout my career. Induction into the Illinois Athletic Trainers Association and the National Athletic Trainers' Association was very meaningful as well.

BUSH-JOSEPH: With all the success you've achieved, how do you stay grounded?

BACH: I grew up in a middle-class household as the oldest of 4 kids. For many families, the oldest kids are the ones who are pushed the hardest, and for whom the expectations are the highest. That was definitely true for me. I've had jobs since I was in the second grade—I used to have an egg route instead of a paper route, and I mowed lawns and shoveled snow to earn money. Even as a kid, I never took anything for granted and wasn't afraid of hard work.

I was mature beyond my years in a lot of ways. Perhaps because I was always physically big—until I stopped growing in eighth grade, at 6'1"—I always had these positions of leadership. From being class president, to being captain of nearly every team I ever played on, to being class marshal at Harvard, there were always expectations about my behavior. I was so square growing up, I took it to another dimension: They called me "The Cube."

BUSH-JOSEPH: As busy as your work keeps you, how are you able to balance your professional and personal lives?

BACH: I've always made my family a priority. As I try to stress to the residents and fellows, as well as my partners, kids grow up so quickly; all of a sudden they're out of the house, and you can't get back the time you missed while you were working 60-80 hours a week.

I've been fortunate that I've able to have almost every dinner at home. Not many surgeons can say that. I have had the opportunity to coach my kids' sports teams, attend their games and recitals and really be a dad. I make time for those things because nothing is more important. I have also been incredibly fortunate to have my wife, Elizabeth, keep me grounded as well.

BUSH-JOSEPH: What else do you enjoy doing when you're not working?

BACH: I've always seemed to be a frustrated Renaissance man, a dabbler of sorts. I started collecting antique, pre-1880 patent medicine bottles when I was in my teens. For some reason they fascinated me. That hobby has evolved over many years to where I've acquired a very nice collection that my wife is probably ready to get rid of. I took up glass blowing several years ago. I also do woodworking and carpentry, home projects, and gardening. These activities are relaxing for me, and I love being creative.

BUSH-JOSEPH: Do you ever think about how your life might have been different if you hadn't gotten hurt back in high school?

BACH: I don't think I would have ended up being an orthopedic surgeon or even going into medicine. Since I've always been a "go-getter" and a hard worker, I probably would have been a wealthy businessman who retired 10 years ago.

BUSH-JOSEPH: When you do eventually step away from medicine, what do you want your colleagues to remember about you and your career?

BACH: First and foremost, that I was an educator. Second, that I had a measure of integrity: I didn't cut corners. Third, that I enjoyed "doctoring." Finally, that I valued the careers of other individuals, not just my own: I truly cared about every member of my team.

Charles A. Bush-Joseph, MD, is a sports medicine surgeon at Rush who performs more than 400 surgeries annually and is among the busiest hip arthroscopic surgeons in Illinois. He serves as head team physician for the Chicago White Sox and associate team physician for the Chicago Bulls and is associate director of Rush's orthopedic sports medicine fellowship program. ■

Principal photography for the *2014 Rush Orthopedics Journal* provided by Adam Daniels and Kevin Horan.

A Tradition of Excellence

> "MIDWEST ORTHOPAEDICS AT RUSH SPECIALISTS ARE LEADING THE WAY IN THE DEVELOPMENT AND REFINEMENT OF CUTTING-EDGE 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.

Throughout MOR's history, our surgeons and physicians have been on the cutting edge of orthopedic care, pioneering a number of the procedures and therapies used to treat patients today—from cementless implants, to minimally invasive surgery for spinal deformities and degenerative disk disease, to expandable prosthetics that help children with bone cancers avoid amputation. That spirit of innovation continues today, as MOR specialists are leading the way in the development and refinement of advanced treatments that benefit patients at Rush—and around the globe.

Physicians from MOR also hold key leadership positions in national societies and organizations. An example is Joshua J. Jacobs, MD, who completed his term as president of the American Academy of Orthopaedic Surgeons in March 2014. Our physicians also serve as the team physicians for a variety of professional, collegiate, and high school teams and clubs, such as the Chicago Bulls, Chicago White Sox, and DePaul University.

These impressive clinical, research, and administrative activities distinguish the orthopedics program at Rush as one of America's best. In 2014, *U.S. News & World Report* ranked our program No. 6 in the nation. ■

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PLEASE NOTE: All physicians featured in this publication are on the medical faculty of Rush University Medical Center. Many of the physicians featured are in the private practice Midwest Orthopaedics at Rush and, as independent practitioners, are not agents or employees of Rush University Medical Center.

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