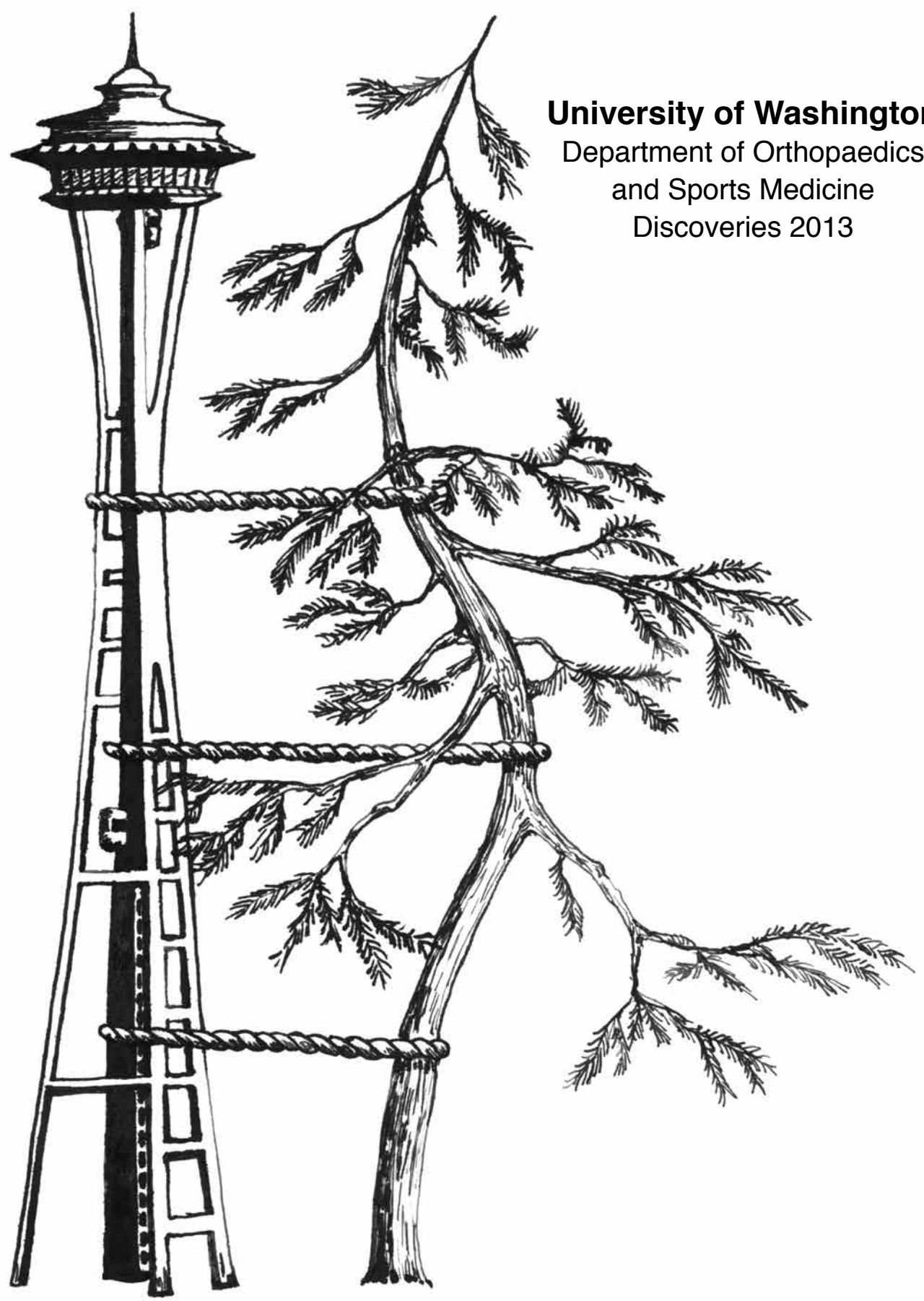
REVISED EDITION

Discoveries 2013

University of Washington
Orthopaedics and Sports Medicine



University of Washington

Department of Orthopaedics
and Sports Medicine
Discoveries 2013

UW Medicine
SCHOOL OF MEDICINE

Department of Orthopaedics and Sports Medicine
University of Washington
Seattle, WA 98195

EDITOR-IN-CHIEF:
Jens R. Chapman, MD
jenschap@uw.edu

MANAGING EDITOR:
Fred Westerberg
fwesterb@uw.edu

Front Cover Illustration: Sounds in a Chamber 3 by Ann Vandervelde. Photographer: Peter Cavanagh. Ms. Vandervelde (pictured below) is a Seattle-based abstract painter. She has exhibited her work locally and nationally at the IMA Gallery in Seattle, Petley-Jones Gallery in Vancouver, BC, and Chicago Art Source in Chicago, IL. She describes her cover painting:

> "The painting was a visual attempt to create a sense of the sounds emanating from inside an MRI Chamber. I was stuck there for 2 hours (in and out) for a series of tests last year. The way I survived was to create a visual image in my head of what these sounds looked like."

Her husband is Peter R. Cavanagh, PhD, DSc, Professor, Orthopaedics & Sports Medicine. For more of her artwork, please see her website: www.annvandervelde.com.

A pdf of this publication is available at our website: www.orthop.washington.edu. Or, connect using the QR code below:

Note: Please note this edition is a revision of the published version. Two articles were removed at the author's request. While the validity of the articles still stands, the author wishes to publish this research in other publications.

Contents

1
Foreword

4
Anne and Rick Matsen, 2013 Grateful Alumni

5
Ed Farrar, MD, 2013 Distinguished Alumnus

6
New Faculty

9
Department of Orthopaedics and Sports Medicine Faculty

13
Visiting Lecturers

15
A Very Successful Year in Orthopaedics

Demographics of Disclosure of Conflicts of Interest at the 2011 Annual Meeting of the American Academy of Orthopaedic Surgeons **19** Frederick A. Matsen III, MD, Jocelyn L. Jette, BS, and Moni Blazej Neradilek, MS

Unloader Knee Braces for Osteoarthritis: Do Patients Actually Wear Them? **23** Emily Squyer, MD, Daniel L. Stamper, PA-C, Deven T. Hamilton, PhD, Janice A. Sabin, PhD, and Seth S. Leopold, MD

Processing of Type IIA Procollagen in Cartilage Formed By the Disruption of Type II Procollagen Alternative Splicing Switch in a Transgenic Knock-In Mouse **27** Audrey McAlinden, PhD and Russell J. Fernandes, PhD

New Insights on Genetic Causes of Brittle Bone Disease: Clues to the Collagen Mineralization Mechanism **30** David R. Eyre, PhD and Mary Ann Weis, BS

Arthroscopic Surgery Skill Acquisition In the Era of Restricted Work Hours **34** Winston J. Warme, MD, Brian Gilmer, MD, Aaron Chamberlain, MD, Nathan Coleman, MD, Bryan Comstock, BS, Seth S. Leopold, MD, and Frederick A. Matsen III, MD

New Insight Into the Pathogenesis of Osteoarthritis **36** Kevin A. Lawson, BA, Colin J. Teteak, BA, Michael J. Chen, BA, John McCornack, Howard A. Chansky, MD, and Liu Yang, PhD

Immediate Weightbearing After Ankle Fracture Fixation **39** Reza Firoozabadi, MD, MA, Emily Harnden, MD, and James C. Krieg, MD

Lessons Regarding the Safety of Orthopaedic Patient Care: An Analysis of Four Hundred and Sixty-Four Closed Malpractice Claims **43** Frederick A. Matsen III, MD, Linda Stephens, PhD, Jocelyn L. Jette, BS, Winston J. Warme, MD, and Karen L. Posner, PhD

Monitoring Tibial Shock During Parabolic Flight **46** Peter R. Cavanagh, PhD, DSc, Molly D. Glauberman, MS, Alexandre E. Bykov, Andrea J. Rice, MS, Grier L. Wilt, Andrea M. Hanson, PhD, Kelly G. Gilkey, MS, and Justin H. Funk, PE

Trehalose Decreases Apoptosis in Osteochondral Grafts Stored at 4ºC **49** Kevin A. Lawson, BA, Colin J. Teteak, BA, John McCornack, Liu Yang, PhD, Howard A. Chansky, MD, and Albert O. Gee, MD

Cell Saver Use in Acetabular Surgery: Does Approach Matter? **52** Reza Firoozabadi, MD, MA, Alan Swenson, BS, and Milton L. Routt, Jr., MD

Evolutionary Origins of 3-Hydroxyproline In Fibrillar Collagens: Implications for Normal Bone and Tendon Development **55** David M. Hudson, PhD and David R. Eyre, PhD

Joint Development in the Hand: A Look at the Molecular Mechanisms **58** Colin J. Teteak, BA, Kevin A. Lawson, BA, Michael J. Chen, BA, Liu Yang, PhD, and Howard A. Chansky, MD

Entrapped Posteromedial Structures in Tibial Pilon Fractures **62** Jonathan G. Eastman, MD, Reza Firoozabadi, MD, MA, David P. Barei, MD, Stephen K. Benirschke, MD, and Robert P. Dunbar, MD

Musculoskeletal Functional Outcomes in Children with Osteogenesis Imperfecta: Associations with Disease Severity and Pamidronate Therapy **64** Ted Sousa, MD, Viviana Bompadre, PhD, and Klane K. White, MD, MSc

A Validated Predictive Model for the Safety of Spine Surgery: SpineSage.com **68** Michael J. Lee, MD, Amy M. Cizik, MPH, and Jens R. Chapman, MD

Differential Expression of Osteoactive Myokines and Muscle-enriched MicroRNAs Following Transient Muscle Paralysis **70** Leah M. Downey, Ted S. Gross, PhD, and Ronald Y. Kwon, PhD

Platelet Rich Plasma for Flexor Tendon Repair **72** Katie Kollitz Jegapragasan, BS, Erin M. Parsons, MS, and Jerry I. Huang, MD

Case Report: Salvage of a Collapsed Avascular Talus with a Fresh Talar Body Allograft **74** Michael E. Brage, MD

76
Harborview Medical Center Orthopaedics

77
Seattle Children's Hospital Orthopaedics

78
University of Washington Medical Center Orthopaedics

81
VA Puget Sound Orthopaedics

A Brief Overview of the Educational Mission of UW Orthopaedics **82** Jens R. Chapman, MD

Increasing Medical Student Exposure to Orthopaedics: Developing an Orthopaedic Surgery and Sports Medicine Interest Group **87** Dayne Mickelson, MD, Philip Louie, BS, Alex Farnand, BA, Lauren Meyer, BS, Brian Gilmer, MD, and Jens R. Chapman, MD

Digital Curriculum Initiative - Developing an iPad-based Platform for Orthopaedic Surgery Resident Education and Collaboration **90** Dayne Mickelson, MD, Kenneth R. Gundle, MD, and Douglas P. Hanel, MD

Reconciling Resident Education with MOC in the New Practice Environment **94** Gregory A. Schmale, MD and Michael J. Goldberg, MD

Musculoskeletal Medicine and Medical Student Education **96** Gregory A. Schmale, MD and Barry Goldstein, MD, PhD

Who is Responsible for Shaping the Future of Orthopaedics in the Pacific Northwest? **97** Brian Gilmer, MD, Sarah E. Timmons, BA, and Theodore A. Wagner, MD

The University of Washington Sports Medicine Clinic: Back to the Future **99** Carol C. Teitz, MD

106
Thomas M. Green, MD Retirement

107
Graduating Residents

109
Incoming Residents

111
ACEs and Fellows

113
Research Grants

115
Department Publications 2012-2013

120
Alumni

123
Endowments

124
Index

Foreword

Dear UW Orthopaedics community, this past academic year has been one of major transformations within our Department affecting all areas of our academic troika of patient care, research and education. In this Discoveries 2013 issue we are proud to present to you our customary general departmental overview with highlights of some of our currently ongoing research and clinical activities. In keeping with our new tradition of featuring a special focus segment within the journal (the Discoveries 2012 edition provided an outlook on clinical volunteer activities of our faculty abroad), this year's emphasis is on our teaching efforts to recognize a number of substantial advancements and to present the plethora of educational activities members of our Department are regularly engaged in. Before delving into the educational arena allow me to briefly reflect on our past year in patient care and research.

In our clinical domain we are excited to provide new access opportunities in partnership with our hospitals for patients with sports related injuries and joint reconstruction needs. The opening of the UW Medicine Stadium Clinic at Husky Stadium this fall and the stepwise implementation of our new Adult Reconstruction Surgery Center at Northwest Hospital will provide state-of-the-art care facilities for our patients and allow for new growth opportunities for these important subspecialties. This also includes a number of initiatives to meet the rising orthopaedically related health care needs of our aging population. Within our service lines we are actively incorporating changes to meet the demands of our evolving health care system by emphasizing patient satisfaction and utilizing care maps for common conditions. We are also starting to put into place the elements for a unique Hip and Pelvis Center based at a variety of facilities in the UW Medicine system. With this center we will be able to offer an integrative therapeutic and educational approach towards hip and pelvic disorder fracture repair, joint reconstruction and joint preserving surgeries offered by respective leaders in their field who will collaborate in consultations, decision making, teaching, and setting new standards for patient outcomes.

Along with these program expansions we have successfully recruited key new faculty over the past year with an eye towards program enhancements. While presenting these faculty developments I would like to pause to thank several colleagues who are pursuing new career opportunities elsewhere for their past work here. On behalf of our Department we wish Drs. Cordelia Carter, Jim Krieg, M.L. 'Chip' Routt and Chris Wahl well in their new endeavors.

Despite these departures we have maintained robust case volumes and in some service lines actual growth. I invite you to look through our updated faculty roster and get to know our newly added faculty colleagues in their bios in our New Faculty section. With our new faculty additions, significant programmatic enhancements and new infrastructural upgrades, such as our office suite remodel at UWMC and our new centralized departmental office space at Harborview Medical Center, we are confidently looking forward to meeting the challenges of the future.

In our Research section our

Figure 1: Group picture of Faculty and Alumni during Summit in Seattle 2012: Focus on Hip and Pelvis Disorders. The Summit continues to grow and features headline speakers such as Professor Norbert Haas from Berlin and Thomas Ruedi from Basel, Switzerland.

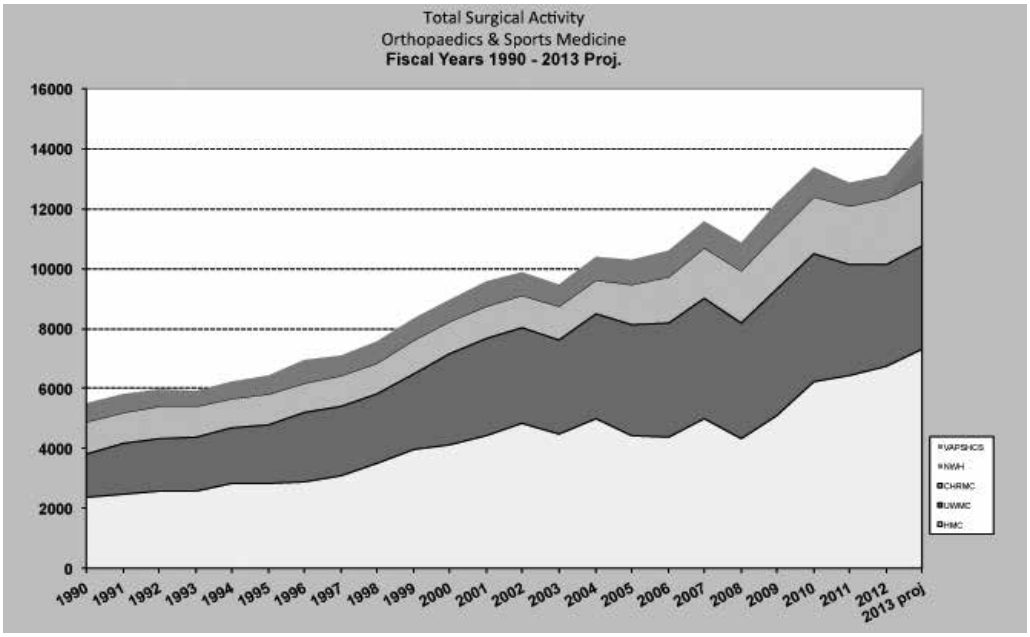


Figure 2: Surgical volumes in UW Orthopaedics and Sports Medicine continue to climb steadily with Northwest Hospital having become a practice site for several of our faculty surgeons.

Collagen, Osteobiologics and Applied Biomechanics labs as well as many other investigator groups continue to perform admirably well despite an increasingly adverse federal funding environment. The core mission of all of our researchers remains focused on enabling our patients to lead longer and happier lives through repair or reconstruction of damages or defects of their musculoskeletal system. This quest is particularly timely as one of the key insights in the landmark Lancet publication from 2012 titled 'Global Burden of Diseases, injuries and risk factors study' by Dr. Chris Murray and collaborators from the UW Department of Global Health identified that as humans around the globe we are leading increasingly longer lives with disproportionately more disabilities.[1] Of the top eight disabilities, four were found to be of musculoskeletal nature. As a Department and particularly in our Research Section we remain dedicated to finding practical solutions for the eminent musculoskeletal ailments

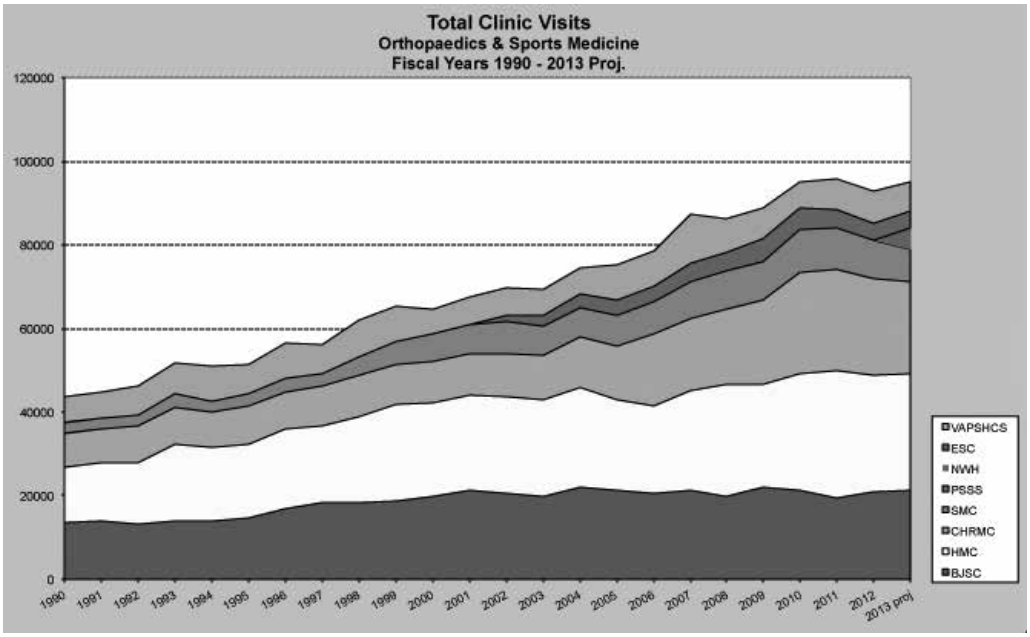


Figure 3: Our clinic visits experienced a slight decline at some sites due to faculty departures while holding steady or slightly growing at others, such as Harborview Medical Center. With the addition of our NWH outpatient clinics our total patient visits have shown continued increase as well.

Figure 4: Resident research education: the benefits of early exposure to research can be seen in Dr. Jacques Hacquebord, PGY-4 (right), whose father Heero Hacquebord (center) is a noted statistician and provided several educational sessions to our Department over the last year. Dr. Jacques Hacquebord has a number of well-received publications to his credit. (They are pictured here with Jens R. Chapman, MD.)

of our times exemplified by impaired joint function, bone loss and functional neuromuscular decline. For us to succeed in our mission we will require support beyond what our usual Federal funding sources are supplying in this era of steady cutbacks. Exciting researchers like Dr. Ron Kwon, who recently received the ASBMR Harold M Frost Young Investigator Award for his work on 'Biophysical control of bone regeneration', would tremendously benefit from a secure funding outlook to be able to turn their vision into clinical realities for our patients in the not too distant future. I hope you take a look at some of the important insights gained by our scientists and investigators over the last year in this edition of Discoveries 2013 and share our excitement, as we are eager to enter a new era of transforming the lives of our patients through translational research.

As this issue of our annual report focuses on education I wanted to highlight a number of areas where our research endeavor crosses over into our educational efforts as part of the 'scholarship' element of our resident and fellow education. Our residents now receive a more formal training approach in what it takes to do formal research through a concerted program consisting of a 'roadmap to research', which accompanies them through their 5 years with us along with instructions and reiterative mentorship guidance along the way. Under leadership of our Vice Chair of Research, Dr. Peter Cavanagh, and under active participation of many of our faculty we now provide a structured pathway to research for our residents, which starts with a formal exposure to available research projects and mentors while using milestones to assure regular progress along the way. Important seed funding for projects for residents and junior faculty has now become available through internal faculty funded grants along with an efficient and expedient grants review process. In December 2012, a Research Advisory Committee was formed to provide the Chairman with advice on research issues in the Department. This group has already initiated a program of Young Faculty Research Grants that has provided funds to four faculty members. The expectation is that these seed funds will facilitate applications to external funding organizations. The Committee is also considering recruitment strategy for new basic science faculty members. The members of the committee are Ted Gross, Mike Lee, Chris Allan, Theresa Bergholz, and Peter Cavanagh. The major breakthrough in our residents' keystone research undertaking is supposed to occur during their third year. Thanks to the fourth year clinicians, the rotations have been adapted to facilitate a dedicated and protected weekly research day. This schedule change pioneered by Drs. Doug Hanel, Michael Goldberg, as well as Greg Schmale has been a very positively received and has enhanced the 'scholarly' development phase of our residents considerably. Finally, our 'Roadmap to Research' is designed to culminate in presentable and then publishable data by the fourth and fifth years of residency and hopefully will have permanently engrained in our graduates the values and pride inherent to contributing to research discoveries. Following an initiative by Dr. Cavanagh and Amy Cizik we now also have regular formal learning opportunities and communication opportunities in our monthly Research Grand Rounds, which are organized with contributions of many, if not most, of our faculty, researchers and graduate students. This program, which consists of a number of regular features, such as review of Orthopaedic Core knowledge questions, invited brief project updates and brief lectures or round table discussions centers on pairing a basic researcher with a clinician to highlight translational opportunities in research and hopefully spark new project ideas in the process of these interdisciplinary presentations. We have received very positive responses to this regular educational feature, which you can personally witness on our website.

As shown in this brief overview, we are excited to present to you highlights of our clinical, research and highlighted educational activities over the past year in this edition of Discoveries 2013. Let me take this moment to thank all of you for your interest, continued support and active partnership in our Department's further evolution in support of our mission to help our patients lead longer and happier lives by allowing us to seek and deliver cures for their musculoskeletal problems.

Jens R. Chapman, M.D.
Professor and Chairman
HansJörg Wyss Endowed Chair
Department of Orthopaedics and Sports Medicine
Joint Professor of Neurological Surgery

References

1: Vos T et al: Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;389:2163-96

Anne and Rick Matsen
2013 Grateful Alumni
University of Washington School of Medicine

We are pleased to honor Anne and Rick Matsen by naming them our 2013 Grateful Alumni. The Matsens have generously given their time, energy, and years of experience to the University of Washington. Frederick A. Matsen III, MD, Douglas T. Harryman II/DePuy Endowed Chair for Shoulder Research, Professor, University of Washington Department of Orthopaedics and Sports Medicine, ranked as a "Top Doctor" in the category of "Orthopaedics" according to Seattle Magazine, has dedicated his entire professional life to developing excellence in Orthopaedics and Sports Medicine at the University of Washington. Starting with his residency here in 1971 he developed an interest in shoulder and elbow reconstruction. A fellowship with the father of modern shoulder surgery, Dr. Charles S. Neer II initiated his lifetime commitment to improving the art of care of patients with simple and complex problems involving the shoulder and elbow. He has partnered with Charles Rockwood, a fellow Texan, in editing the definitive text in shoulder surgery *The Shoulder*, now in its fourth edition from Saunders. He has also written *Practical Evaluation and Management of the Shoulder* and most recently, along with a former shoulder fellow Steve Lippitt, has published *Shoulder Surgery, Principles and Procedures*, also published by Saunders.

He is the former chair of the Department of Orthopaedics and Sports Medicine, a position he held from 1986 to 2009, making him amongst the longest tenured chairs among clinical departments at the University of Washington. During his tenure the Department has risen to being one of the top Departments according to rankings by U.S. News and World Report and by the National Institutes of Health. Throughout Rick's tenure as chair, Anne was a vital member of the Orthopaedics team, hosting countless memorable dinners and other events for residents, fellows, and faculty.

Besides years of service in the medical field, the Matsens have gone above and beyond voluntarily donating their time and money to our university. The Matsens have given annually to the Department of Orthopaedics and Sports Medicine for over 32 years. This includes an impressive 163 gifts to the University of Washington – totaling over $10,000, including contributions to support our residency, our research programs, Harborview Medical Center, and the Burke Museum. Their most recent gifts have made them UW Benefactors, members of the group of families who have given a total of $100,000 or more to UW.

Ed Farrar, MD
2013 Distinguished Alumnus
University of Washington School of Medicine

Dr. Edward L. Farrar III is a member of the alumni class graduating from the UW Department of Orthopaedics in 1983, which by all accounts fostered an impressive number of leaders in our field.

Originally from Florida, Ed Farrar graduated Magna Cum Laude from Emory University School of Medicine in 1978, where he also served as President of the Alpha Omega Alpha honorary medical society. While at Emory, he did elective work studying healthcare in Ladakh, northern India, a surgery rotation at Massachusetts General Hospital in Boston, and an elective in Infectious Disease at Fred Hutchinson Institute in Seattle. While there he fell in love with the Pacific Northwest and subsequently matched for Internship and Residency in our Department. Following his Chief Resident year at Harborview, Dr. Farrar went on to complete his training as a Senior Registrar in Orthopaedics at Middlemore Hospital in Auckland, New Zealand. He returned home the long way, with three months of mountain climbing in Nepal, India, and Europe.

After returning to the US, Dr. Farrar moved to Wenatchee, Washington and became a founding member of Wenatchee Orthopaedics. He and his wife Cindy raised two sons there. Tyler Farrar is an internationally recognized professional cyclist with The Garmin Cycling team, and his son Fletcher is now a commercial realtor and accomplished speed climber in Tacoma.

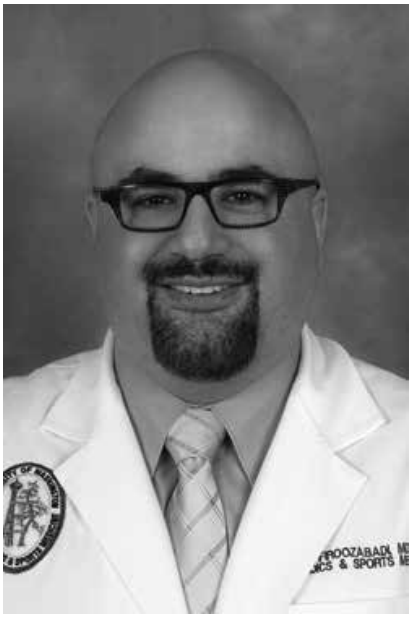
Ed's fascination with the Himalayas led him back to Kathmandu, Nepal in 1985, and 1988 as an Orthopaedics Overseas volunteer. He became a regular visitor to Nepal and has been a climbing member of 5 Himalayan Expeditions in the Khumbu region of that country. He demonstrated the value of our profession by doing two of those expeditions after undergoing a total knee arthroplasty.

In 1990, he returned to Swedish Hospital to do a Spine Fellowship with Dr. Ted Wagner. In 1994 he joined the UW clinical faculty and volunteered with monthly call at Harborview through 2001. During this time, he also served as president of the Puget Sound Spine Interest Group, and became active in the Washington State Orthopaedic Association. He served on the WSOA Board of Directors for several years, and was President of the WSOA from 2004-2006.

In 2008, while riding his bicycle to work, Dr. Farrar was hit head on and run over by a distracted driver. His chest was crushed, and he sustained a T4-5 fracture/dislocation, along with a C2 fracture. After 6 weeks of being in critical condition in the ICU in Wenatchee, he returned to Harborview, this time as a patient for rehabilitation. He has been putting life back together as a high thoracic paraplegic since that time, and now sees life as both a patient and a physician. His strict exercise discipline has allowed him to return to an independent life style, he enjoys regular 20-mile rides with a handcycle and cross-country skies in a sitski.

The choice of 2013 Distinguished Alumnus celebrates the tremendous come back spirit of Dr. Ed Farrar, which he has abundantly demonstrated against all odds. He personifies the virtues of dedication and perseverance and is an inspiration for all those around him.

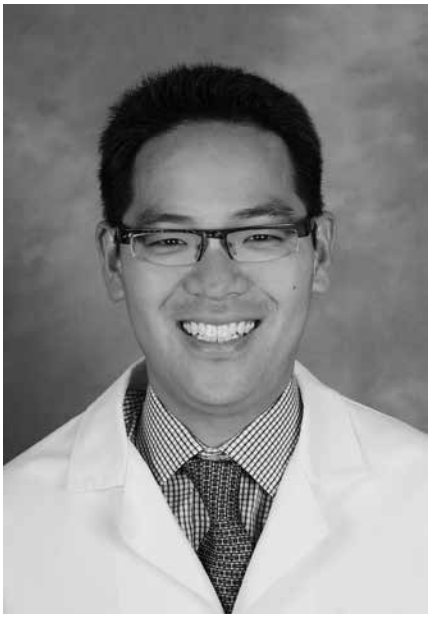
New Faculty



Reza Firoozabadi, MD, MA

Assistant Professor
Harborview Medical Center
Trauma
rezaf2@uw.edu

Dr. Firoozabadi received his bachelor's degree in Molecular Cell Biology from University of California, Berkeley. He received his M.D. from Boston University School of Medicine and graduated Magna Cum Laude. He also obtained a Masters Degree in Medical Sciences at Boston University. He subsequently completed his orthopaedic surgery residency at the University of California, San Francisco. Following his residency, he completed an Orthopaedic Trauma Fellowship at the University of Washington / Harborview Medical Center. Dr. Firoozabadi is devoted to teaching orthopaedic residents/fellows and improving the quality of patient care through research. His research interests focus on both the clinical and technical aspects of orthopaedic trauma surgery and the injured patient. Dr. Firoozabadi is an attending orthopaedic trauma surgeon at Harborview Medical Center and sees patients for trauma related injuries.



Albert O. Gee, MD

Assistant Professor
University of Washington Medical Center
Sports Medicine
ag112@uw.edu

Albert Gee is a sports medicine and shoulder surgeon with specialty training in the care of the injured athlete. He attended medical school at Washington University in St. Louis and completed his training in orthopaedic surgery at the University of Pennsylvania - the oldest training program in the United States. Dr. Gee completed a fellowship in sports and shoulder surgery at the prestigious Hospital for Special Surgery in New York City where he served as assistant team physician for the NBA's New Jersey Nets. He served as a member of the medical staff of the U.S. Open Tennis Tournament and served as an assistant team physician for the Iona College Gaels.

His clinical interests include treating shoulder injuries, knee ligament injuries (ACL, PCL, MCL, LCL) and athletic ankle problems. His research interests include ligament and tendon biomechanics and mechanobiology, meniscus and cartilage tissue engineering, and shoulder instability and reconstruction.

Dr. Gee is a member of the American Academy of Orthopaedic Surgeons, American Orthopaedic Society for Sports Medicine, and the Arthroscopy Association of North America.

He has been published in multiple peer reviewed periodicals including the Journal of Hand Surgery, American Journal of Orthopaedics, Biomaterials, Journal of Orthopaedic Trauma, Techniques in Knee Surgery, and The American Journal of Sports Medicine among others.

New Faculty

Stephen Kennedy, MD
Assistant Professor
University of Washington Medical Center
Hand and Wrist
sajk@uw.edu

Stephen Kennedy is a Hand and Upper Extremity Surgeon with subspecialty training in both Hand & Microvascular Surgery and Upper Extremity Sports Medicine and Reconstruction. He provides treatment options for problems of the entire upper extremity, including all forms of instability, trauma, deformity, and arthritis. He has a particular interest in problems of the wrist and elbow. Surgically, he performs open, arthroscopic, and microsurgical techniques for the entire upper extremity from fingertips to clavicle.

Dr. Kennedy outlines his patient care philosophy as follows: "Each person who comes into clinic has a different set of circumstances and goals. Giving the best care means listening, providing current and accurate information, and developing a plan together that best meets each person's needs. When surgery is chosen, it should be undertaken with the greatest possible precision and care. Therapy and multidisciplinary providers are often pivotal to success."

His research interests include studies of clinical outcomes in hand and upper extremity trauma, advances in joint reconstruction surgery, particularly of the wrist and elbow, and technology for the improvement of patient care.

Ronald Y. Kwon, PhD
Assistant Professor
Harborview Medical Center
Research
ronkwon@uw.edu

Joining our faculty, and PI of the Musculoskeletal Systems Biology Lab, is Ronald Y. Kwon, PhD. Dr. Kwon graduated from the University of California Berkeley in 2002 with a BS in Mechanical Engineering. He went on to get a Masters of Science in the same subject at Stanford in 2005 and completed his PhD at Stanford in 2008.

Dr. Kwon has received numerous honors in his career, his most recent being the NIH National Research Service Award Postdoctoral Fellowship (2008-2010), the American Society for Bone and Mineral Research Young Investigator Award (2010), the International Bone Fluid Flow Workshop Young Investigator Award (2010), and the Pfizer Inc. Endowed Scholarship to attend the Zebrafish Genetics and Development Course at the Marine Biological Laboratory (2010).

His research interests are in systems biology, mechanobiology, neuromuscular control of bone health, and bone regeneration. His work has been published in a number of peer reviewed journals including the FASEB Journal, Journal of Bone and Mineral Research, and Proceedings of the National Academy of Sciences. He has also recently published a textbook called "Introduction to Cell Mechanics and Mechanobiology".

New Faculty

Bruce C. Twaddle, BHB, FRACS
Professor
University of Washington Medical Center
Sports Medicine
btwaddle@uw.edu

In Bruce Charles Twaddle, BHB, FRACS, UW Orthopaedics has been able to attract a senior voice in international sports medicine in time for the opening of the new stadium clinic in September 2013. Dr. Twaddle comes to us from New Zealand, where he served as Director of Orthopaedic Trauma at Auckland Hospital and was also a senior partner in the UniSports Centre, which serves as FIFA Centre of Excellence for the Asia-Pacific region.

He has a unique background with residency training in Auckland, New Zealand, Oxford, London and Edinburgh, United Kingdom and is one of the few orthopaedic surgeons to have completed a formal triple fellowship (Knee Surgery in Auckland 1993-94, Traumatology at Harborview, Seattle 1994-95, Sports Medicine at Fowler-Kennedy Sports Medicine 1995-96). After his return to New Zealand he served as a consultant in orthopaedic traumatology and was instrumental in making the UniSports clinic, a joint private practice and academic enterprise, the major success it has become. This facility became the first branded Adidas clinic in the world and later was chosen as the regional FIFA Centre of Excellence, a distinction shared in the USA only by Duke University and the Santa Monica UCLA affiliated Sports Medicine practice. Dr. Twaddle was instrumental in creating a modern-day practice based database now used as the national standard for orthopaedic surgery in New Zealand and has created modern patient outcomes assessment systems for several orthopaedic specialties including sports medicine and traumatology.

Dr. Twaddle is a member of many international organizations and has received multiple honors, among them being elected to serve on the Board of Councillors of the Australasian College of Surgeons, he was chosen as the youngest ever member of the Board of Trustees of the AO Foundation and selected as the Education Chairman of the AO Asia-Pacific region. He has also served as team doctor for many professional and amateur teams in rugby, basketball, netball and other team and individual sports. Dr. Twaddle is particularly well known for his work on the severest forms of knee injuries – knee dislocations, and is also well known for his work on athletic foot and ankle injuries. He is a dedicated teacher and program builder and in his past time enjoys barbeque and swimming. We are delighted to have this experienced and highly engaging colleague on our faculty.

Department of Orthopaedics and Sports Medicine Faculty

Jens R. Chapman, MD
Professor and Chairman
Harborview Medical Center
Spine
jenschap@uw.edu

Michael E. Brage, MD
Associate Professor
Harborview Medical Center
Foot and Ankle
bragem@uw.edu

Christopher H. Allan, MD
Associate Professor
Harborview Medical Center
Hand and Wrist
callan@uw.edu

Richard J. Bransford, MD
Associate Professor
Harborview Medical Center
Spine
rbransfo@uw.edu

Steven D. Bain, PhD
Research Associate Professor
Harborview Medical Center
Research
sdbain@uw.edu

Peter R. Cavanagh, PhD, DSc
Professor
University of Washington Medical Center
Research
cavanagh@uw.edu

David P. Barei, MD
Associate Professor
Harborview Medical Center
Trauma
barei@uw.edu

Howard A. Chansky, MD
Professor
VA Puget Sound Health Care System
Adult Reconstructive Surgery
chansky@uw.edu

Daphne M. Beingessner, MD
Associate Professor
Harborview Medical Center
Trauma
daphneb@uw.edu

Ernest U. Conrad III, MD
Professor
Seattle Children's Hospital
Orthopaedic Oncology
chappie.conrad@seattlechildrens.org

Carlo Bellabarba, MD
Professor
Harborview Medical Center
Spine and Trauma
cbella@uw.edu

Mark C. Dales, MD
Clinical Associate Professor
Seattle Children's Hospital
Spine
mark.dales@seattlechildrens.org

Stephen K. Benirschke, MD
Professor
Harborview Medical Center
Foot and Ankle
beniskb@uw.edu

Darin J. Davidson, MD, MHSc
Assistant Professor
University of Washington Medical Center
Orthopaedic Oncology
djdavi@uw.edu

Department of Orthopaedics and Sports Medicine Faculty

Robert P. Dunbar, MD
Associate Professor
Harborview Medical Center
Trauma
dunbar@uw.edu

David R. Eyre, PhD
Professor
University of Washington Medical Center
Research
deyre@uw.edu

Russell J. Fernandes, PhD
Research Associate Professor
University of Washington Medical Center
Research
rjf@uw.edu

Reza Firoozabadi, MD, MA
Assistant Professor
Harborview Medical Center
Trauma
rezaf2@uw.edu

Albert O. Gee, MD
Assistant Professor
University of Washington Medical Center
Sports Medicine
ag112@uw.edu

Michael J. Goldberg, MD
Clinical Professor
Seattle Children's Hospital
Pediatric Orthopaedics
michael.goldberg@seattlechildrens.org

John R. Green III, MD
Associate Professor
University of Washington Medical Center
Sports Medicine
jgreen3@uw.edu

Ted S. Gross, PhD
Professor
Harborview Medical Center
Research
tgross@uw.edu

Douglas P. Hanel, MD
Professor
Harborview Medical Center
Hand and Wrist
dhanel@uw.edu

Sigvard T. Hansen, Jr., MD
Professor Emeritus
Harborview Medical Center
Foot and Ankle
hansetmd@uw.edu

M. Bradford Henley, MD
Professor
Harborview Medical Center
Trauma
bhenley@uw.edu

Jerry I. Huang, MD
Assistant Professor
University of Washington Medical Center
Hand and Wrist
jihuang@uw.edu

Stephen Kennedy, MD
Assistant Professor
University of Washington Medical Center
Hand and Wrist
sajk@uw.edu

Walter F. Krengel III, MD
Clinical Associate Professor
Seattle Children's Hospital
Spine
wally.krengel@seattlechildrens.org

Department of Orthopaedics and Sports Medicine Faculty

Ronald Y. Kwon, PhD
Assistant Professor
Harborview Medical Center
Research
ronkwon@uw.edu

Roger V. Larson, MD
Associate Professor
University of Washington Medical Center
Sports Medicine
drlarson@uw.edu

Michael J. Lee, MD
Associate Professor
University of Washington Medical Center
Spine
mjl3000@uw.edu

Seth S. Leopold, MD
Professor
University of Washington Medical Center
Adult Reconstructive Surgery
leopold@uw.edu

Paul A. Manner, MD
Associate Professor
University of Washington Medical Center
Adult Reconstructive Surgery
pmanner@uw.edu

Frederick A. Matsen III, MD
Professor
University of Washington Medical Center
Shoulder and Elbow
matsen@uw.edu

Vincent S. Mosca, MD
Professor
Seattle Children's Hospital
Pediatric Orthopaedics
vincent.mosca@seattlechildrens.org

Carol A. Mowery, MD
Clinical Associate Professor
Seattle Children's Hospital
Pediatric Orthopaedics
carol.mowery@seattlechildrens.org

Sean E. Nork, MD
Associate Professor
Harborview Medical Center
Trauma
nork@u.washington.edu

John W. O'Kane, MD
Associate Professor
University of Washington Medical Center
Sports Medicine
jokane@uw.edu

Bruce J. Sangeorzan, MD
Professor
Harborview Medical Center
Foot and Ankle
bsangeor@uw.edu

Gregory A. Schmale, MD
Associate Professor
Seattle Children's Hospital
Pediatric Orthopaedics
gregory.schmale@seattlechildrens.org

John A. Sidles, PhD
Professor
University of Washington Medical Center
Research
sidles@uw.edu

Douglas G. Smith, MD
Professor
Harborview Medical Center
Foot and Ankle
dgsmith@uw.edu

Department of Orthopaedics and Sports Medicine Faculty

Sundar Srinivasan, PhD
Research Associate Professor
Harborview Medical Center
Research
sundars@u.washington.edu

Suzanne E. Steinman, MD
Clinical Assistant Professor
Seattle Children's Hospital
Pediatric Orthopaedics
suzanne.steinman@seattlechildrens.org

Lisa A. Taitsman, MD, MPH
Associate Professor
Harborview Medical Center
Trauma
taitsman@uw.edu

Carol C. Teitz, MD
Professor
University of Washington Medical Center
Sports Medicine
teitz@uw.edu

Allan F. Tencer, PhD
Professor
Harborview Medical Center
Research
atencer@uw.edu

Bruce C. Twaddle, BHB, FRACS
Professor
University of Washington Medical Center
Sports Medicine
btwaddle@uw.edu

Theodore A. Wagner, MD
Clinical Professor
University of Washington Medical Center
Spine
wagner@uw.edu

Winston J. Warme, MD
Associate Professor
University of Washington Medical Center
Shoulder and Elbow
warmewj@uw.edu

Klane K. White, MD, MSc
Associate Professor
Seattle Children's Hospital
Pediatric Orthopaedics
klane.white@seattlechildrens.org

Jiann-Jiu Wu, PhD
Research Professor
University of Washington Medical Center
Research
wujj@uw.edu

Emeritus Faculty

Stanley J. Bigos, MD
Professor Emeritus

Theodore K. Greenlee, Jr., MD
Associate Professor Emeritus

Lynn T. Staheli, MD
Professor Emeritus

Visiting Lecturers

2013 LeCocq Lectureship

Thursday & Friday, January 24- 25, 2013

This year we were happy to have Dr. Harry E. Rubash (left) visit our department as the guest lecturer for the 2013 LeCocq Lectureship. On January 29th, he gave a presentation on "Risk Factors for Early Revision Following THA & TKA in Medicare Patients." That evening, at our 49th Annual John F. LeCocq Dinner, he spoke on "Highly Cross-Linked Polyethylene for Total Hip Arthroplasty – The Debate is Over!"

Dr. Harry E. Rubash is the Chief of Orthopaedic Surgery at the Massachusetts General Hospital (MGH). He received his Medical Degree (Cum Laude) and Orthopaedic training from the University of Pittsburgh. He then completed a Fellowship in the Hip and Implant Surgery Unit at the MGH, Department of Orthopaedic Surgery.

After a successful, 13-year tenure at the University of Pittsburgh where he was Professor and Clinical Vice-Chairman of the Orthopaedic Department, Dr. Rubash returned to the MGH in 1998 as Professor of Orthopaedic Surgery at Harvard Medical School (HMS) and the Chief of the Department. Under his leadership, the department has grown to include 65 surgeons and 200 staff. In 2000 he was named the Edith M. Ashley Professor of Orthopaedic Surgery at HMS.

Dr. Rubash has served in leadership roles in many professional societies including, the Orthopaedic Research Society (ORS), The American Orthopaedic Association (AOA), The Knee Society, The Hip Society, and the International Hip Society. Dr. Rubash is proud to be an honorary member of the Ruth Jackson Orthopaedic Society to support leadership training and mentor the growing number of women orthopaedic surgeons.

Dr. Rubash is a noted lecturer and a highly sought after speaker in the field of orthopaedic surgery on both a national and international level. He has had over 200 significant contributions to the literature in the area of hip and knee replacement and has served on the Editorial Boards for notable medical journals including the Journal of Bone and Joint Surgery, Clinical Orthopaedics and Related Research and The Journal of Arthroplasty. The American Academy of Orthopaedic Surgeons (AAOS) has honored Dr. Rubash with the presentation of five Hip Society Awards and one Knee Society award for outstanding contributions to the field of hip and knee replacement surgery. He has authored 9 textbooks including the highly regarded The Adult Hip and The Adult Knee volumes.

Dr. Rubash's current research interests are: investigating implant failure patterns, the development of new long-lasting implant surfaces, and novel implant designs. Dr. Rubash and his colleagues recently developed a robotic total knee system to evaluate the native knee and the knee, post-surgical intervention. This powerful testing system is used to quantify the kinematics of total knee arthroplasty and has led to improved surgical techniques and the development of new prosthetic designs that preserve the native ligaments of the knee. Through this work, Dr. Rubash and his co-investigators hope to provide new systems for implanting joint replacements, and a variety of other joint implant designs.

Visiting Lecturers

2013 Resident Research Day

Friday, June 28, 2013

We were happy to host Kristy Weber, MD (left) as the guest lecturer for our Resident Research Day on June 28, 2013.

Kristy Weber, MD is a Professor of Orthopaedic Surgery at the University of Pennsylvania. She is originally from St. Louis, Missouri and attended college at the U. Missouri-Columbia and medical school at Johns Hopkins. Kristy completed her orthopaedic residency training at the University of Iowa and followed that with a 2 year research/clinical fellowship in orthopaedic oncology at the Mayo Clinic.

She was on the faculty at University of Texas M. D. Anderson Cancer Center for 5 years where she developed a large clinical practice in orthopaedic oncology and developed a basic science research program related to osteosarcoma metastasis to lung and renal cell carcinoma metastasis to bone. Kristy was recruited to Johns Hopkins in 2003 as chief of the Division of Orthopaedic Oncology and Director of the Sarcoma Program. She also became an Associate Faculty member of the Johns Hopkins Armstrong Institute for Safety and Quality. Kristy and her team received the Kappa Delta national orthopaedic research award for work in bone metastasis in 2006.

Recently she was recruited to the University of Pennsylvania as Vice-Chair of Faculty Affairs and Chief of Orthopaedic Oncology. She also serves as the Director of the Sarcoma Program in the Abramson Cancer Center. Kristy has been a leader in many national orthopaedic and cancer organizations including the American Orthopaedic Association (AOA), the Connective Tissue Oncology Society, and the Orthopaedic Research Society.

She recently completed 4 years as Chair of the AAOS Council on Research and Quality where she oversaw initiatives related to clinical practice guidelines, evidence-based medicine, appropriate use criteria, patient safety, biomedical engineering, biological implants and the development of orthopaedic clinician-scientists. In addition, she is currently in the presidential lines of the Musculoskeletal Tumor Society and the Ruth Jackson Orthopaedic Society (RJOS). She enjoys backpacking, canoeing, and reading about environmental/conservation issues.

A Very Successful Year in Orthopaedics

Osteoporosis education at the University of Washington: Dr. Steven Kates (center) and Carrie Bradt, PA-C (laft) from University of Rochester with Dr. Lisa Taitsman (right).

The James Garrick Sports Medicine 2012 Lecturer Dr. Ned Amendola from U of Iowa (2nd from right) with Drs. Jim Garrick (left) and Carol Teitz, and Colleen Johnson (right).

Boston sports fans in Seattle: Drs. Michael Goldberg, Bob Dunbar, Visiting Professor Harry Rubash, Jens R. Chapman, Howard Chansky, Paul Manner from left to right. Conspicuously absent: Drs. Carlo Bellabarba and Lisa Taitsman.

Drs. Sam Agnew and Bruce Sangeorzan during Summit in Seattle 3. Dr. Agnew has been co-chairing the Fellowship alumni meeting at Harborview since 1990.

Drs. Frederick Matsen, Winston Warme, Albert Gee, and Nate Coleman, PGY4 after concluding Grand Rounds on labral tears.

Interdisciplinary education at Spine Grand Rounds March 2013 from left to right: Mathias Daniels, Spine ACE, Mr. Alexander Montgomery, Consultant at the Royal London Hospital (Speaker), Stan Herring, Director Musculoskeletal Medicine, John Loeser, Professor Emeritus, Neurological Surgery.

Drs. Bruce Twaddle (left) and Howard Chansky (right) with Maureen Johnson, Manager, Faculty Affairs (center).

Professor Norbert Haas, from Berlin, Germany, Honored Faculty for the Summit in Seattle 3, enjoying his visit to Seattle.

Demographics of Disclosure of Conflicts of Interest at the 2011 Annual Meeting of the American Academy of Orthopaedic Surgeons

Frederick A. Matsen III, MD, Jocelyn L. Jette, BS, and Moni Blazej Neradilek, MS

It is recognized that financial conflicts of interest affect the methodology, results and conclusions of research. This study documented the voluntary disclosures at the 2011 Annual Meeting of the American Academy of Orthopaedic Surgeons. Commonly, program committees had members who disclosed conflicts of interest. Commonly, presentations had authors who disclosed conflicts of interest. A small number of orthopaedic companies had disclosed conflicts of interest with the majority of the presentations.

Introduction

The Annual Meeting of the AAOS has a major influence on orthopaedic practice; thus, conflicts of interest among participants in the educational programs of the American Academy of Orthopaedic Surgeons are of interest.

Material/Methods

- Using the Final Program for the 2011 AAOS annual meeting, we analyzed the voluntarily disclosed conflicts of interest for presentations in the disciplines of spine, sports medicine/arthroscopy, and pediatric orthopaedic surgery as well as for the relevant program committees.

Results (Figures 1, 2, 3 and 4)

- Conflicts of interest were disclosed by participants for each of the program committees and for over 3/4ths of the presentations.
- Conflicts of interest were disclosed for all of the featured symposia and for over 3/4ths of the scientific exhibits, the podium presentations, and the posters.
- Over half of the disclosures were for paid consultancy and for research support for the principal investigator. Over 1/3 were for paid presentations, royalties, and stock.
- The highest number of

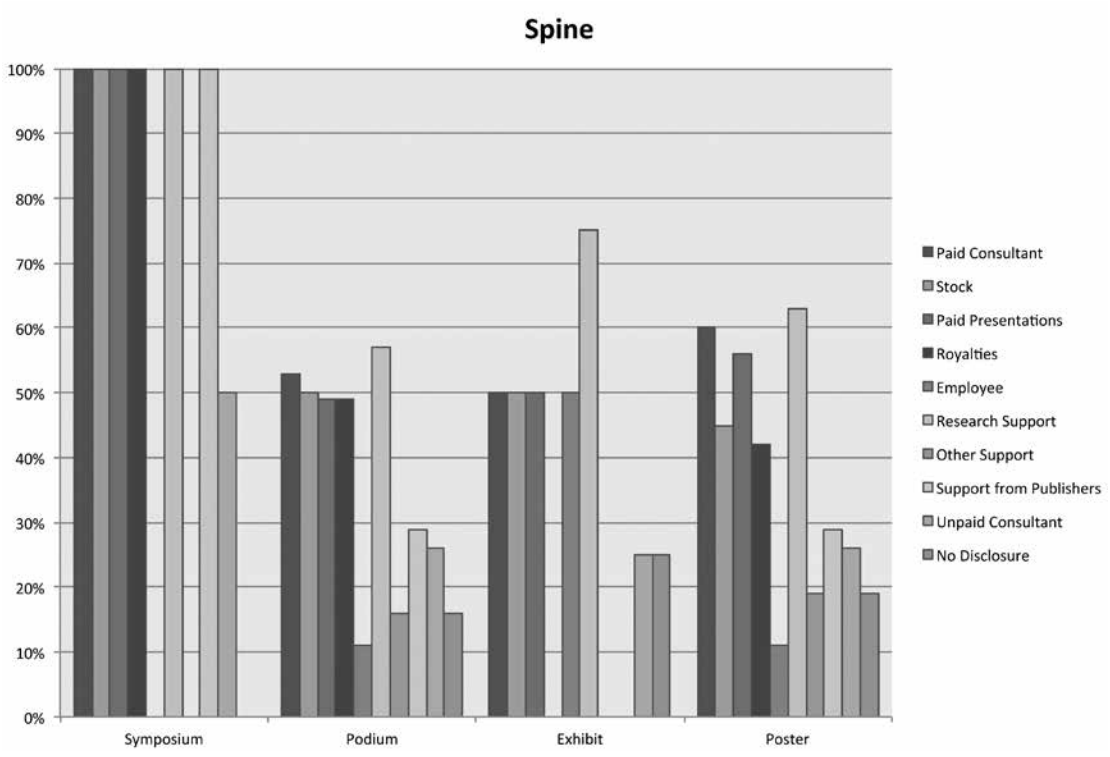


Figure 1: Disclosures of Conflicts of Interest at the 2011 Meeting of the AAOS for Spine Specialty by Type of Presentation and by Type of Disclosure. Data are expressed as the percent of the presentations of each type having authors with the different types of disclosure.

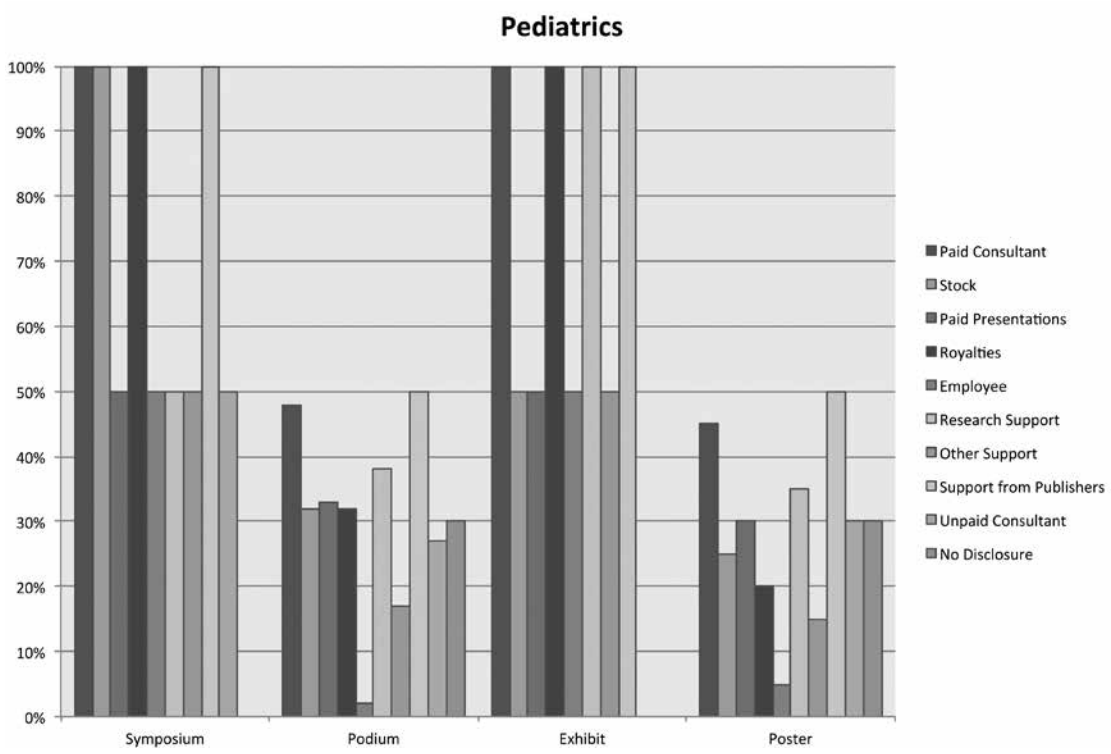


Figure 2: Disclosures of Conflicts of Interest at the 2011 Meeting of the AAOS for Pediatrics Specialty by Type of Presentation and by Type of Disclosure. Data are expressed as the percent of the presentations of each type having authors with the different types of disclosure.

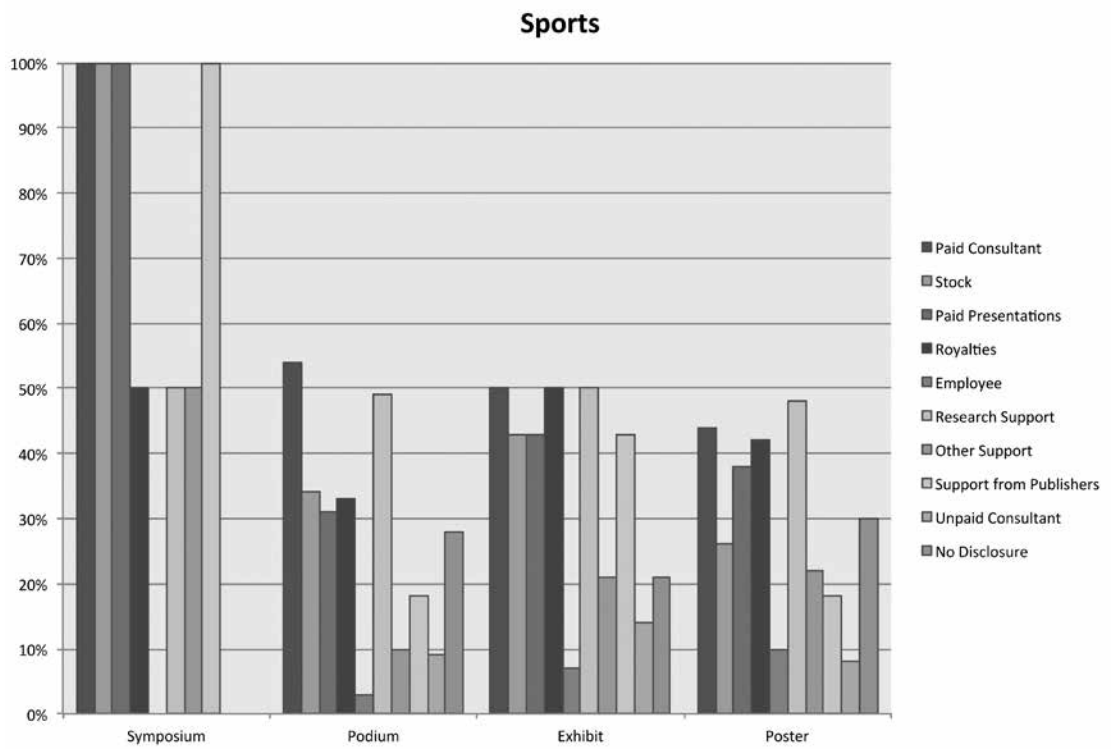


Figure 3: Disclosures of Conflicts of Interest at the 2011 Meeting of the AAOS for Sports Medicine Specialty by Type of Presentation and by Type of Disclosure. Data are expressed as the percent of the presentations of each type having authors with the different types of disclosure.

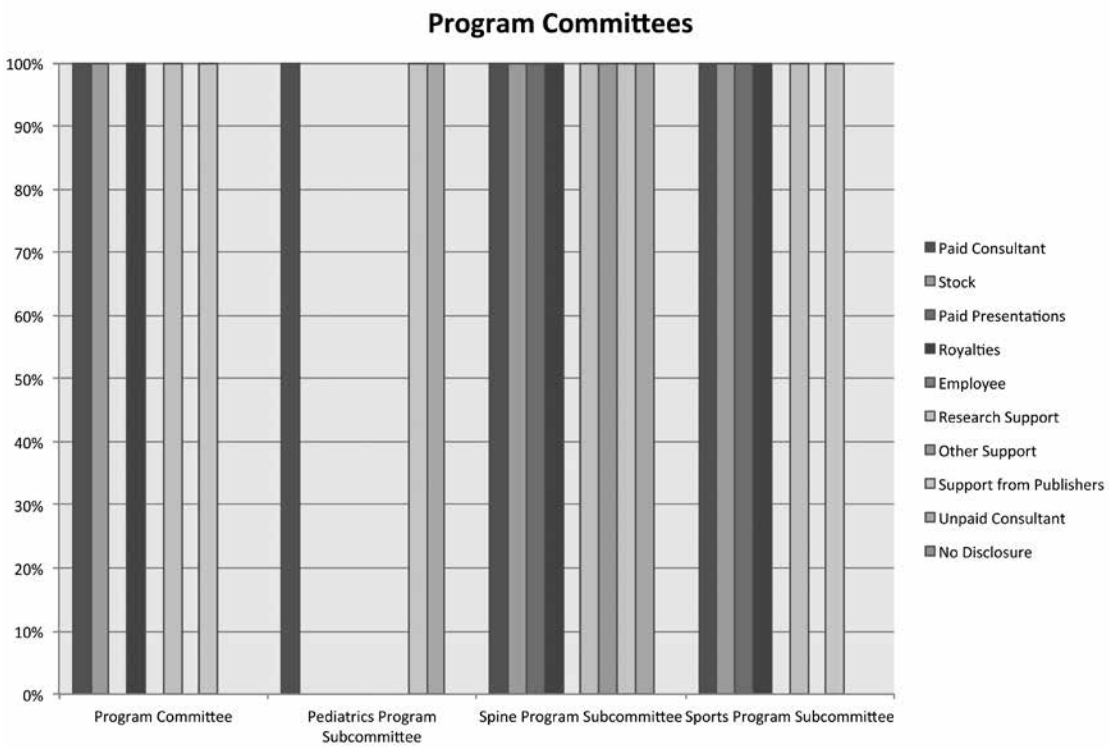


Figure 4: Disclosures of Program Committees by Type of Disclosure. Data are expressed as the percent of each committee having members with the different types of disclosure.

disclosures for an individual author was 37.

- The number of disclosures per author was positively correlated with the number of presentations per author.
- While disclosures identified 379 different companies, relationships with only 26 of these companies were listed in disclosures for 2/3rds of the presentations.

Discussion

Our results are similar to the prior work of Zuckerman et al and Okike et al, although the rates of disclosure were greater for the 2011 meeting than for the earlier meetings studied by these authors. It is of note that their work and ours are based on the voluntary self-reporting of conflicts of interest. Recently some authors have pointed to the risk of incompleteness and inaccuracy in voluntary disclosure. Okike et al found that over 1/5th of the directly related payments and half of the indirectly related payments were not disclosed at the 2008 AAOS meeting.

In light of the commonality of conflicts of interest in orthopaedic presentations, it is important to recognize that these conflicts have been documented to have a biasing effect on the design, conduct and interpretation of research, even in randomized clinical trials. Ezzet found that 3/4ths of a group of hip arthroplasty studies had commercial sponsorship; over 90% of these had positive outcomes for the implant, while independently funded studies had positive outcomes in less than 40%. Leopold et al, Okike et al and Carragee et al each found that commercially funded studies and authors receiving personal payments reported more positive outcomes and fewer complications than non commercially funded studies.

Conclusion

We conclude that voluntarily disclosed conflicts of interest were common at the 2011 AAOS meeting, especially for the featured symposia that are selected by program committees to represent the state of the art on important topics. In light of the documented frequency of undisclosed conflicts of interest as well as the documented effects of conflicts of interest on research, it may be time to improve strategies for assuring the accuracy and completeness of disclosures and for managing the biasing effects of conflicts of interest.

References

1. Als-Nielsen B, Chen W, Gluud C, Kjaergard LL. Association of funding and conclusions in randomized drug trials: a reflection of treatment effect or adverse events? JAMA 2003; 290:921-8.

2. Bhandari M, Busse JW, Jackowski D, Montori VM, Schunemann H, Sprague S, et al. Association between industry funding and statistically significant pro-industry findings in medical and surgical randomized trials. CMAJ 2004; 170:477-80.

3. Carragee EJ, Hurwitz EL, Weiner BK. A critical review of recombinant human bone morphogenetic protein-2 trials in spinal surgery: emerging safety concerns and lessons learned. Spine J 2011; 11:471-91.

4. Chimonas S, Frosch Z, Rothman DJ. From disclosure to transparency: the use of company payment data. Arch Intern Med 2011; 171:81-6.

5. Ezzet KA. The prevalence of corporate funding in adult lower

extremity research and its correlation with reported results. J Arthroplasty 2003; 18:138-45.

6. Friedman LS, Richter ED. Relationship between conflicts of interest and research results. J Gen Intern Med 2004; 19:51-6.

7. Leopold SS, Warme WJ, Fritz Braunlich E, Shott S. Association between funding source and study outcome in orthopaedic research. Clin Orthop Relat Res 2003:293-301.

8. Okike K, Kocher MS, Mehlman CT, Bhandari M. Conflict of interest in orthopaedic research. An association between findings and funding in scientific presentations. J Bone Joint Surg Am 2007; 89:608-13.

9. Okike K, Kocher MS, Wei EX, Mehlman CT, Bhandari M. Accuracy of conflict of- interest disclosures reported by physicians. N Engl J Med 2009; 361:1466-74.

10. Zuckerman JD, Prasarn M, Kubiak EN, Koval KJ. Conflict of interest in orthopaedic research. J Bone Joint Surg Am 2004; 86-A:423-8.

Please note this research has now been published in the Journal of Bone and Joint Surgery (J Bone Joint Surg Am, 2013 Mar 06;95(5):e29 1-8). The pdf is available at the following link: http://jbjs.org/data/Journals/JBJS/926435/e29.pdf

Unloader Knee Braces for Osteoarthritis: Do Patients Actually Wear Them?

Emily Squyer, MD, Daniel L. Stamper, PA-C, Deven T. Hamilton, PhD,
Janice A. Sabin, PhD, and Seth S. Leopold, MD

Unloader braces are a nonsurgical approach for predominantly unicompartmental knee arthritis. Although noninvasive, braces are expensive and it is unclear whether clinical factors, if any, will predict regular brace use. We asked the following: (1) Do patients continue to use the unloader brace more than 1 year after it is prescribed? (2) Are there clinical or radiographic factors that predict continued use of the unloader brace after the first year? (3) What are the most common subjective reasons that patients give for discontinuing the brace?

We administered 110 surveys to all patients who were fitted for unloader knee braces for predominantly unicompartmental osteoarthritis 12-40 months prior to administration of the survey. The survey response rate was 81% (89 of 110). We found 28% reported regular brace use (twice per week, an hour at a time, or more); at 2 years, 25% used the brace regularly. No clinical or radiographic factors considered were associated with ongoing brace utilization.

Level of Evidence Level III, prognostic study.

Introduction

Knee arthritis is common, underreported, and increasing in prevalence. In 2009, surgeons performed 686,000 knee arthroplasties; projections going forward predict 1.52 million procedures in 2020 and 3.48 million procedures in 2030. Arthritis unloader braces provide a low-risk intervention for selected patients. Several studies on brace compliance differ widely in terms of whether patients continue to use the unloader brace over time [1-5].

Although unloader braces do not carry much risk, these braces are expensive. The patient charge for one kind of brace is nearly $1800 at our own institution. By comparison, the professional fee for a knee arthroplasty for a Medicare patient is approximately $1450.

In light of the high cost of bracing for arthritis, and the variable estimates of whether patients continue to wear the braces after they are prescribed, we sought to look specifically at the question of brace usage. We asked three study questions: (1) Do patients continue to use an unloader brace more than 1 year after it is prescribed? (2) Are there clinical or radiographic factors that predict continued use of an unloader brace after the first year? (3) What are the most common subjective reasons that patients give for discontinuing the brace?

Patients and Methods

- 110 Surveys were administered; these were sent to all patients who were were fitted for an unloader brace (Figure 1), (Össur Americas, Foothill Ranch, CA, USA) as part of a comprehensive nonoperative approach to the management of predominantly

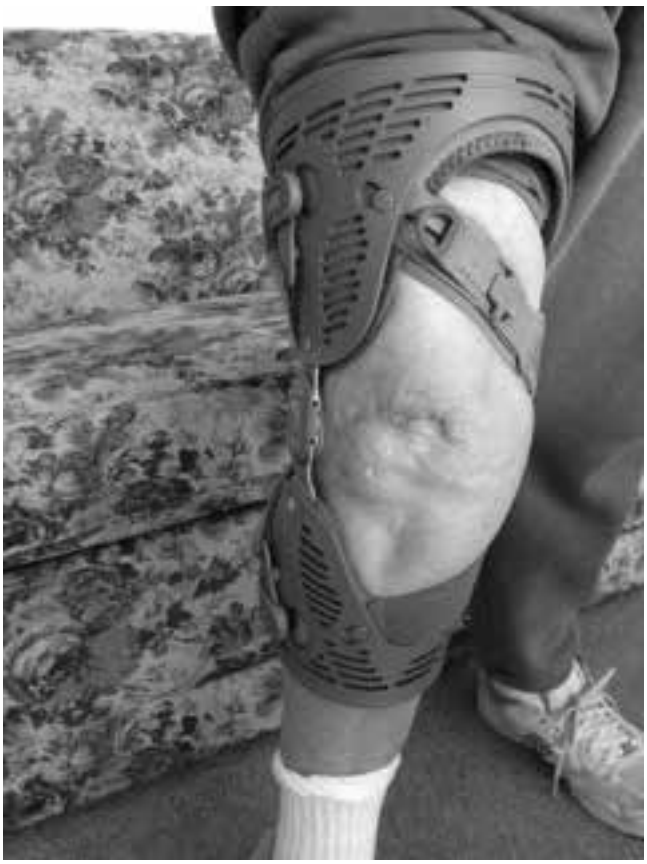


Figure 1: Unloader knee brace.

Variable	Value
Age (years)*	63 ± 9.4 (43–83)
BMI	28 ± 5.6 (20–50)
Weight (kg) *	86 ± 20 (50–145)
Sex	
Male	52%
Female	48%
Radiographic Alignment	
Varus	59%
Valgus	26%
Neutral	15%
Most Involved Compartment	
Medial	68%
Lateral	32%
Arthritis Severity (1-3)**	
Medial Compartment	2.39 ± 0.86
Lateral Compartment	1.66 ± 0.88
Patellofemoral Compartment	1.26 ± 0.49

Table 1: Demographics of Survey Responders
*Values are expressed as mean ± SD, with range in parentheses.
**Modified Kellgren-Lawrence scale as follows: Grade 1, minimal to no osteophytes or joint space narrowing; Grade 2, osteophytes and/or moderate joint space narrowing (Kellgren-Lawrence 1 or 2); and Grade 3, severe joint space narrowing (Kellgren-Lawrence 3 or 4).

Months since fitting	Number of surveys completed	Number of braces in use
> 12	89	25 (28%)
> 24	40	10 (25%)
> 36	14	3 (21%)

Table 2: Brace use through time (months since brace fitting).

Walking range improved greater than twice the range without the brace?	Number of patients		p value
	Brace used at least 1 year?		
	No	Yes	
No	23 (68%)	7 (24%)	< 0.001
Yes	2 (5.8%)	22 (76%)	

Table 3: Association of use longer than 1 year with improved walking range
Nine patients reported not having used their brace enough to comment.
Remaining surveys (26) submitted with this question omitted

Brace aspect	Number of patients	
	Brace used at least 1 year?	
	No (N=42)	Yes (N=20)
The brace causes skin irritation or swelling	17 (40%)	4 (20%)
The brace does not fit well enough or was too uncomfortable	25 (60%)	2 (10%)
The brace does not help my symptoms enough to make it worth wearing	21 (50%)	2 (10%)
The brace is hard to put on/take off	7 (17%)	0
The brace is too hard to wear with the clothes that I wanted to wear for the activities I wanted to do	13 (31%)	6 (30%)
The brace is too heavy or too bulky	14 (33%)	4 (20%)

Table 4: Self-reported aspects of brace that prevented greater use
Remaining surveys (27) submitted with this question omitted

unicompartmental osteoarthritis (either medial- or lateral-sided). Weight, BMI, radiographic severity, and limb alignment were not used as contraindications to bracing.

- The survey response rate was 81% (89 of 110). The mean age for survey responders was 63 years (SD, 9.4 years), and the mean BMI was 28 (SD, 5.6), 59% had predominately varus alignment and 68% had predominately medial compartment involvement (Table 1).
- The same surgeon (SSL) wrote all the brace prescriptions; braces were fitted by the same orthotist and fit for neutral alignment or the smallest amount of unloading that generated symptomatic relief.
- The survey asked about current and past brace utilization, knee pain, function, satisfaction with the brace, and problems related to use of the brace. We defined regular brace use as at least an hour a day, at least 2 days a week.
- Patient and radiographic parameters were obtained by chart and direct review of weight-bearing radiographs, respectively. Arthritis was graded using a modification of the Kellgren-Lawrence grading system as follows: Grade 1, minimal to no osteophytes or joint space narrowing; Grade 2, osteophytes and/or moderate joint space narrowing (Kellgren-Lawrence 1 or 2); and Grade 3, severe joint space narrowing (Kellgren-Lawrence 3 or 4).
- Descriptive statistics of available responses and data were used for primary data explorations and comparisons. Logistic regression was used to determine whether respondent attributes (BMI, weight, age, sex, radiographic severity, limb alignment) were associated with the likelihood of using a brace for 3, 6, and 12 months.

Results

- 25 of 89 (28%) patients continued to use the brace more than one year after brace

	Sample Size	Age	BMI	Arthritis Severity	Brace Compliance	Follow-Up (years)
Giori [3]	46	57	32	2.96	76%; 68%; & 61%	1; 2; & 3
Kirkley et al [4]	41	59	≤35*	3.39	NA	0.5
Brouwer et al [2]	60	59	28	NA	58%	1
Barnes et al [1]	30	57	29	2.84	41%	2.7
Wilson et al [5]	29	66	29	3.06	0%	11.2
Current Study	89	63	28	2.88**	28%; 24%; & 21%	1; 2; & 3

* Average BMI not listed; however, BMI > 35 was an exclusion criteria for this study
** Arthritis Severity of current data was obtained using a modified Kellgren-Lawrence as described and normalized here to a 4-point scale for comparison

Table 5: Comparison of published populations undergoing unloader bracing.

fitting (Table 2); mean followup was 24 months (range, 13–40 months). Most patients did not use their braces beyond the 1-month trial period: 26% reported never having used the brace regularly, and 39% did so for less than 3 months. In aggregate, 65% of the patients fitted for braces did not use them even for 3 months.

- None of the potential predictor variables analyzed (BMI, weight, age, sex, radiographic severity, limb alignment) were associated with brace use or discontinuation. Patients who described considerable improvement in walking range (defined as more than twice the walking range than without the brace) were more likely ($p < 0.001$) to continue to use the brace beyond 1 year (Table 3).
- Patients cited a number of subjective factors that led to brace discontinuation including skin irritation/swelling, poor fit, lack of symptomatic relief, difficulty donning/doffing brace, difficulty wearing with clothing, and heaviness/bulkiness of brace (Table 4).

Discussion

Our finding of only about one in four patients continuing to use the brace for more than a year may represent a best-case trial for the brace, in that all patients were given a no-charge 1-month trial with the brace, were invited to have free brace refittings as needed, and were offered cotreatments (including joint injections and nonnarcotic analgesics).

Other studies have varied in terms of reported compliance rates over time. One followup survey of patients previously braced and studied found that 41% of 30 patients were using the brace at 2.7 years, where use was defined as an average of 5 hours/day for work or weightbearing activity [1]; when those patients (n=29) were resurveyed at an average of 11.2 years follow-up, none were using the brace [5]. Another study, in a population of military veterans, found 76% of patients were still using the brace at least once a week after a year [3]. Our study employed a stricter definition of brace usage (twice a week, compared to once a week) than the others.

No patient or radiographic factors, such as sex, age, BMI, limb alignment, or arthritis severity predicted use or discontinuation of the brace. Our patient population is in many ways similar to other populations studied in the brace literature in terms of age, body-mass index, and radiographic arthritis severity (Table 5). Our sample size is larger than the other published studies on the subject. The study of Giori et al [3], like ours, concluded that continued brace use was not associated with weight, BMI, or the primary compartment affected by osteoarthritis, though Giori's study did find that age younger than 50 had better brace compliance than age over 65. Brouwer [2] claimed a non-statistically significant trend towards better patient function and pain relief with these braces in younger patients.

Our results may serve as hypothesis-generating pilot data for a prospective trial to evaluate clinical- and/or cost-efficacy of unloader braces. Insofar as these braces are expensive ($849 to $1780 here), we believe that such a study is warranted. We continue to offer the unloader brace as part of a comprehensive approach to nonsurgical management of patients whose arthritis pattern is predominantly unicompartmental; however, our findings make us less sanguine that the brace will serve as a durable intervention, and we are candid with our patients about the relatively low likelihood that a brace, once fitted, will remain in service a year later.

Acknowledgement

The authors gratefully acknowledge Leslie Meyer, BA, for her considerable assistance in manuscript preparation and submission.

Permission

Material in this article is made available with kind permission from Springer Science+Business Media: Clin Orthop Relat Res. 2013 Feb 2. Unloader Knee Braces for Osteoarthritis: Do Patients Actually Wear Them? Squyer E, Stamper DL, Hamilton DT, Sabin JA, Leopold SS. Published online 2013 Feb 2.

References

1. Barnes CL, Cawley PW, Hederman B. Effect of CounterForce brace on symptomatic relief in a group of patients with symptomatic uni-compartmental osteoarthritis: a prospective 2-year investigation. Am J Orthop. 2002;31:396-401.

2. Brouwer RW, Raaij TM, Verhaar JAN, Coene LNJEM, Bierma-Zeinstra SMA. Brace treatment for osteoarthritis of the knee: a prospective randomized multi-center trial. Osteoarthritis and Cartilage 2006;14(8):777–83.

3. Giori N. Load-shifting brace treatment for osteoarthritis of the knee: a minimum 2½ year follow-up study. J Rehabil Res Dev. 2004;41:187-194.

4. Kirkley A, Webster-Bogaert S, Litchfield R, Amendola A, MacDonald S, McCalden R, Fowler P. The effect of bracing on varus gonarthrosis. J Bone Joint Surg Am. 1999;81:539-548.

5. Wilson B, Rankin H, Barnes CL. Long-term results of an unloader brace in patients with unicompartmental knee osteoarthritis. Orthopedics 2011;34:e334-e337.

Authors' note: A full-length version of this paper was published in Clinical Orthopaedics and Related Research as: Squyer E, Stamper DL, Hamilton DT, Sabin JA, Leopold SS. Unloader braces for knee osteoarthritis: Do patients actually wear them? Clin Orthop. Published online, 02 February 2013, DOI: 10.1007/s1199-013-2814-0.

Processing of Type IIA Procollagen in Cartilage Formed By the Disruption of Type II Procollagen Alternative Splicing Switch in a Transgenic Knock-In Mouse

Audrey McAlinden, PhD[1] and Russell J. Fernandes, PhD

The biological significance of the developmentally-regulated alternative splicing of exon 2 in *Col2a1* during skeletal development has remained a mystery. The present study describes the generation of a knock-in mouse model to address this. Normally, chondroprogenitor cells synthesize predominantly exon 2-containing mRNA isoforms (type IIA) while *Col2a1* mRNA devoid of exon 2 (type IIB) is the major isoform produced by differentiated chondrocytes. Utilizing a splice site targeting knock-in approach, a 4 nucleotide mutation was created to convert the 5' splice site of *Col2a1* exon 2 from a weak, non-consensus sequence to a strong, consensus splice site. To test this knock-in approach *in vivo*, homozygote mice engineered to retain IIA exon 2 (*Col2a1+ex2*) were generated. Chondrocytes from hindlimb epiphyseal cartilage of homozygote mice were shown to express only procollagen type IIA mRNA. Protein analysis of the homozygote rib cartilage show native α1(II) collagen chains deposited in the matrix, implying procollagen type IIA molecules were processed to mature type II collagen molecules as observed normally for type IIB procollagen. As *Col2a1+ex2* homozygote mice are viable and appear healthy, the results challenge the consensus that this splicing event is as critical a switch in chondrogenesis and endochondral bone formation during fetal development.

Introduction

The *Col2a1* gene encodes α1(II) chains that form type II procollagen homotrimers. *Col2a1* also encodes α3(XI) chains that form type XI procollagen heterotrimers with α1(XI) and α2(XI) chains. Type II collagen is the major collagenous component of cartilage, providing tensile strength to the ECM. Type XI collagen is a minor component of cartilage and functions in controlling fibrillogenesis. During chondrogenesis, exon 2-containing *Col2a1* mRNA isoforms (IIA) are generated by chondroprogenitor cells while differentiated chondrocytes synthesize mRNA isoforms devoid of exon 2 (IIB). Exon 2 encodes a von Willebrand factor C-like cysteine-rich domain in the amino (NH2) propeptide of type II/XI procollagen. To understand why IIA and IIB isoforms are differentially expressed during chondrogenesis, transgenic mice engineered to express only the IIA mRNA isoform of *Col2a1* have been generated.

Methods

A splice site targeting approach was utilized to induce production of only the IIA mRNA isoform of *Col2a1* in transgenic knock-in mice (1). RNA was extracted from epiphyseal cartilage of WT, heterozygous knock-in (+/-) and homozygous knock-in (-/-) mice, post partum day 7, 14, 28 (P7, P14, P28) and analyzed by RT-PCR for expression of *Col2a1* isoforms. Immunohistochemistry of hindlimb paraffin sections was done with an antibody recognizing the IIA exon 2-encoded domain. Procollagen processing was analyzed by Western blotting of 4M guanidine extracts of P7 epiphyseal cartilage using 1C10 Ab (recognizing processed collagen II) and the anti-IIA Ab (recognizing exon 2).

Results

Only IIA mRNA isoforms were detected at all pre- and post-natal

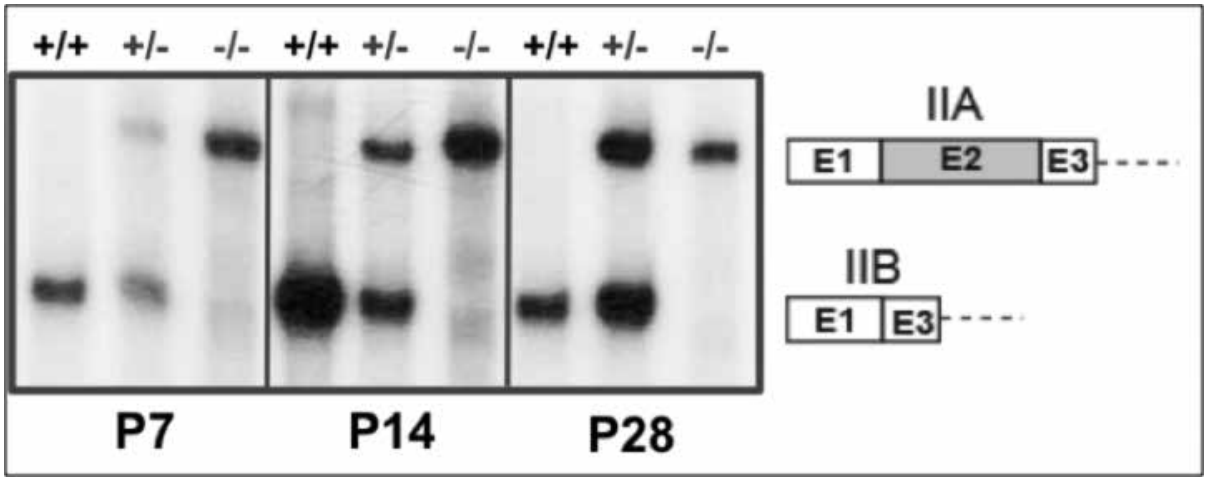


Figure 1: Expression patterns of *Col2a1* mRNA isoforms in epiphyseal cartilage from post-natal (P) day 7, 14 or 28 wild type (+/+), heterozygote (+/-) and homozygote (-/-) cartilage. IIA band = 490bp; IIB band = 283bp. IIA mRNA is the major splice form produced in homozygote post-natal cartilage tissue.

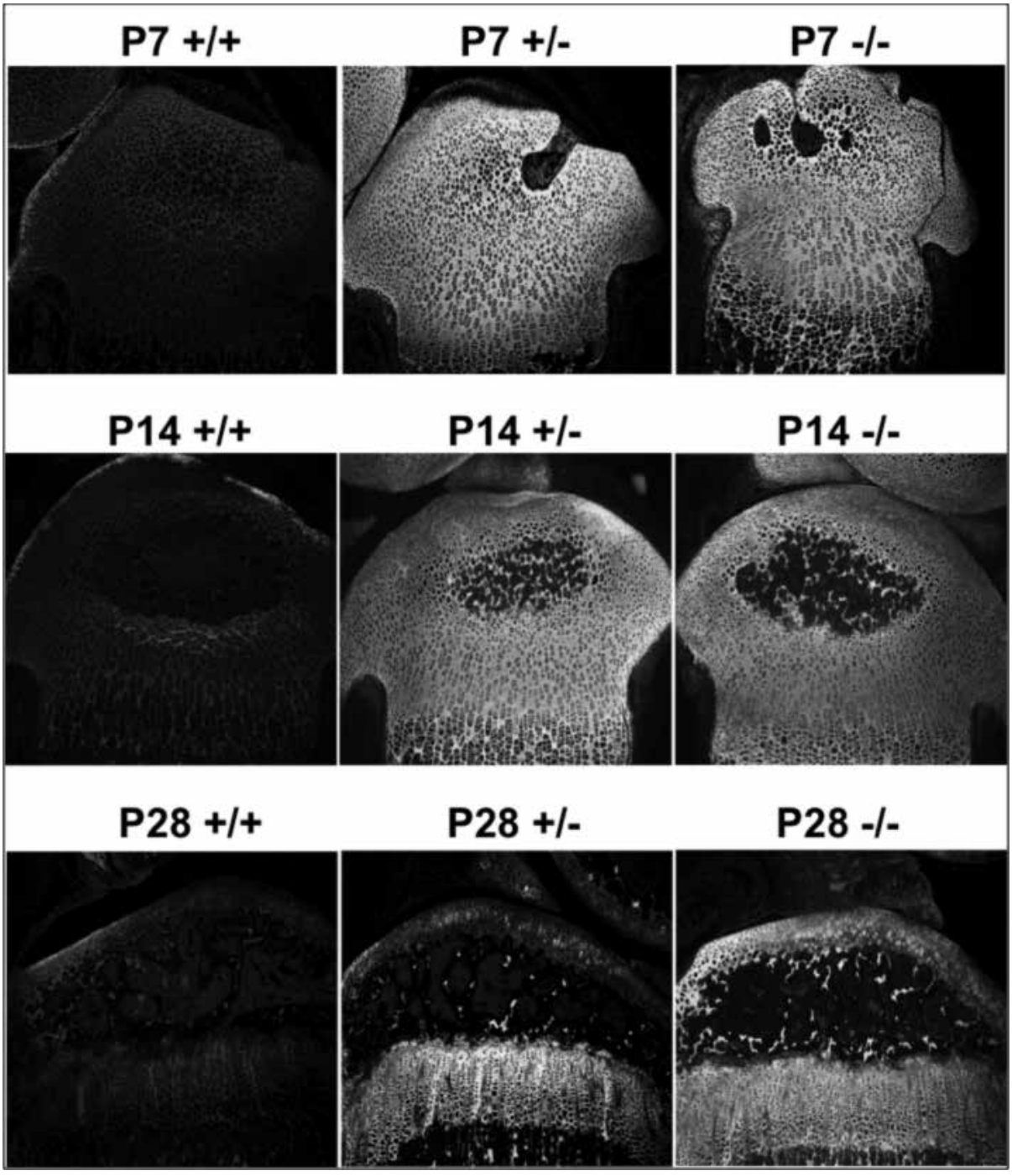


Figure 2: Immunolocalization of type II procollagen exon 2-encoded domain in proximal tibiae of P7, P14 and P28 limbs. Intense type IIA specific immunostaining was observed in the heterozygote (+/-) and homozygote (-/-) epiphyseal cartilage (P7, P14) and articular and metaphyseal bone (P28) compared to wildtype (+/+) cartilage and bone.

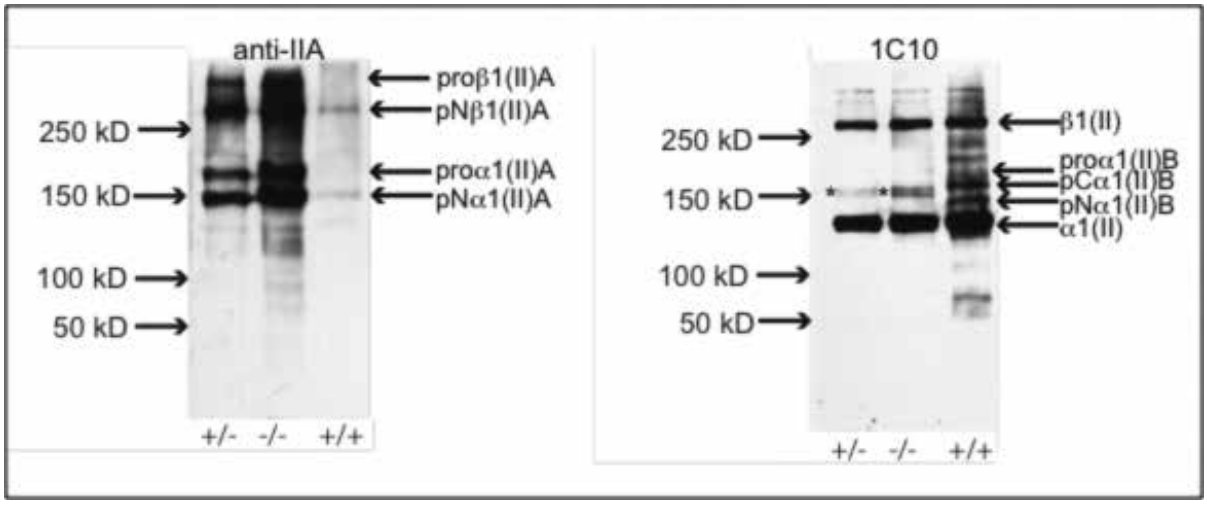


Figure 3: Western blot of the exon 2-encoded domain (anti-type IIA blot) and triple helical domain of type II procollagen (1C10 blot) from P7 epiphyseal cartilage. The anti-type IIA blot shows increased levels of IIA- containing pro-forms of type II collagen in addition to IIA-containing beta forms of type II procollagen chains in heterozygote and homozygote cartilage compared to WT. The 1C10 blot shows that processing to remove the NH2- and COOH- propeptides occurs regardless of whether IIA or IIB procollagen is the substrate. Bands marked by an asterisk (*) shows unprocessed type IIA N-procollagen in homozygote and heterozygote cartilage.

time points analyzed in -/- cartilage; no IIB mRNA was found (Figure 1) Persistent expression of type IIA collagen protein was found in the ECM of +/- and -/- cartilage and trabecular bone from P7 onwards but little to no IIA staining was detected in WT cartilage matrix at all time points (Figure 2). Type IIA procollagen, the only isoform expressed in post-natal -/- cartilage, was found to be processed similar to type IIB procollagen (Figure 3). Mis-expression of the IIA collagen isoform is apparently well-tolerated since both +/- and -/- mice are viable with no overt phenotype. However, preliminary ultrastructural studies reveal an abnormal fibrillar matrix in +/- and -/- cartilage and trabecular bone by electron microscopy (unpublished results) indicating changes in collagen fibril assembly.

Discussion

Observing no overt phenotype in +/- and -/- mice was surprising since it was assumed that the *Col2a1* IIA-to-IIB splicing switch was necessary for cartilage development. With the generation of viable type IIA knock-in mice the consensus that the type IIA to type IIB splicing switch is critical in early chondrogenesis has to be reconsidered. The data has also shown that propeptide of type IIA procollagen can be processed in the cartilage of homozygous mice and it is likely that resident cartilage type II collagen N-proteinases ADAMTS-2 or ADAMTS-3 (2) are responsible for this cleavage.

The homozygous mice that express only the collagen type IIA isoform in cartilage will now enable us to address the question: Can the type IIA splice isoform solely contribute as a structural component in cartilage? Having already shown that procollagen type IIA can be processed in the cartilage of -/- mice, research is now ongoing to determine if type IIA collagen can form cartilage-typic cross-linked heteropolymeric fibrils in cartilage.

Significance

Deciphering the biological significance of *Col2a1* alternative splicing will provide us with new concepts related to the functional role of type II/XI collagen isoforms during skeletal development. By understanding how cartilage and bone tissue properly form and are maintained, we will gain better insights into mechanisms leading to bone and cartilage regeneration. This discovery-based research is also valuable in the field of cartilage/intervertebral disc/bone tissue engineering where the translatable goal is to generate tissue that is in quality and function identical to the original.

References

1. Lewis R, Ravindran S, Wirthlin L, Traeger G, Fernandes RJ, McAlinden A. Disruption of the developmentally-regulated *Col2a1* pre-mRNA alternative splicing switch in a transgenic knock-in mouse model. Matrix Biol. 31(3):214-26. 2012.

2. Fernandes, R.J., Hirohata, S., Engle, J.M., Colige, A., Cohn, D.H., Eyre, D.R., Apte, S.S. Procollagen II amino propeptide processing by ADAMTS-3: Insights on dermatosparaxis. J. Biol. Chem. 276(34), 31502-31509, 2001.

Acknowledgements

Funding: NIH RO1 AR057025 (RJF), NIH R21 AR053513 (AM), Pilot & Feasibility Grant (AM) from P30 Core Grant AR057235.

[1]Department of Orthopaedic Surgery, Washington University School of Medicine, St Louis, MO.

New Insights on Genetic Causes of Brittle Bone Disease: Clues to the Collagen Mineralization Mechanism

David R. Eyre, PhD and Mary Ann Weis, BS

Despite intensive study over 50 years the molecular mechanism of mineralization of bone collagen to form an intimate ordered biocomposite remains elusive. The question is still contested whether bone collagen fibrils have evolved special features or are essentially generic and passive in a process largely driven by extrafibrillar inhibitors of solid-phase calcium phosphate nucleation, and so act primarily as a structural constraint on nanocrystal growth not as a nucleator (Figure 1). Here we summarize recent genetic discoveries on osteogenesis imperfecta (OI) that we believe reinforces a long-held view that highly evolved post-translational features of bone collagen itself are intimately involved in regulating the ordered composite of polymeric collagen embedded with oriented nanocrystal plates of hydroxyapatite [1]. It turns out that defects in many of the steps regulating the unique post-translational chemistry and assembly of bone collagen can cause severe OI [2].

Introduction

Most (90%) OI cases result from dominant mutations in either of the two genes encoding type I collagen. Though a systemic defect, fragile bone is the most pronounced tissue consequence. Until 7 years ago, gene defects causing recessive OI were unknown beyond a few cases of parental mosaicism for *COL1* mutations. A key advance was the discovery that mutations in *CRTAP* (encodes cartilage-associated protein) caused severe recessive OI [3]. This protein forms a complex with prolyl 3-hydroxylase 1 (P3H1) and peptidyl prolyl isomerase B (PPIB or cyclophilin B, a peptidyl prolyl isomerase), which is responsible for 3-hydroxylating a single proline residue in collagen α1(I) and α2(I) chains at α1(I) Pro986 and α2(I) Pro707. Further OI-causing mutations in *CRTAP*, *LEPRE1* (encodes P3H1), *PPIB* (encodes cyclophilin B) and a growing list of genes were quickly found [3,4; Table 1]. Bruck syndrome, which exhibits both the bone fragility of OI and joint contractures, results from

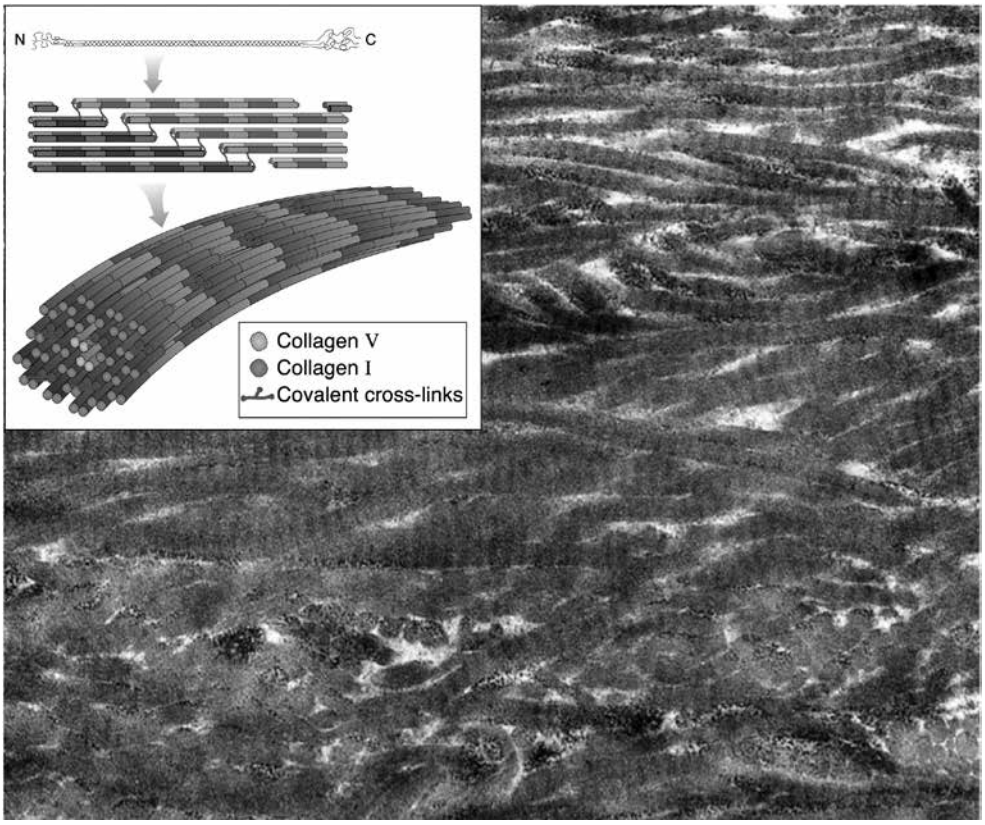


Figure 1: Transmission electron micrograph of decalcified bone matrix showing the dense, woven packing arrangement of collagen fibrils. Inset: individual fibrils are a covalently cross-linked composite of collagen type I polymerized on a template of type V collagen.

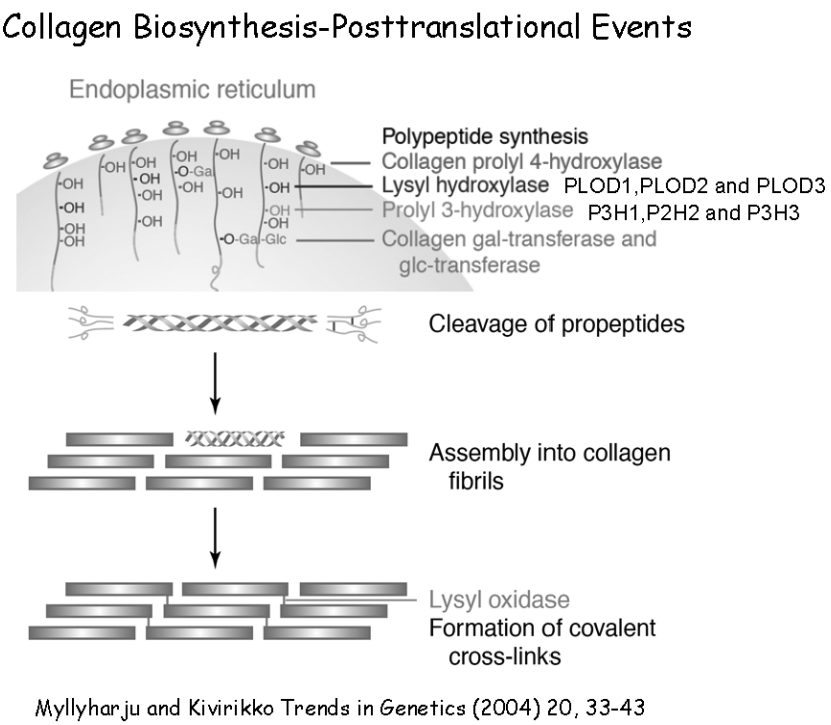


Figure 2: Collagen Biosynthesis-Posttranslational Events

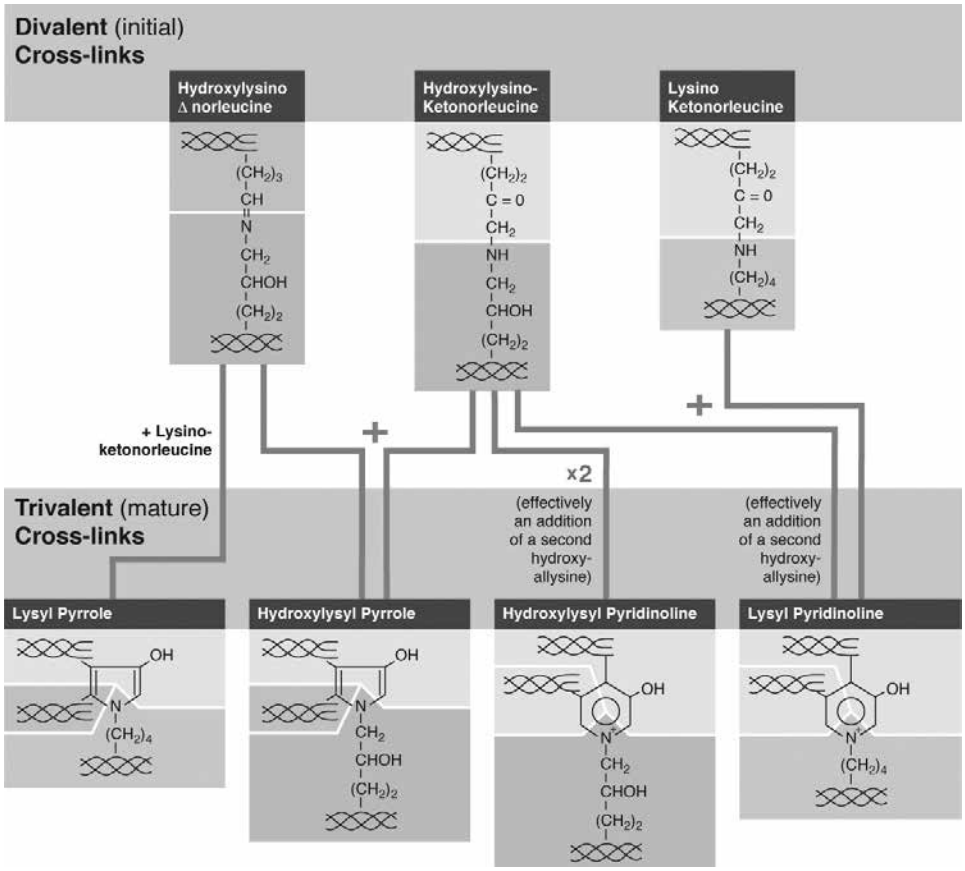


Figure 3: Location of 3Hyp residues in type I and V collagen molecules (A) with their resulting aligned positions in a fibril relative to the D-periodic stagger and band pattern of fibrils as seen by transmission EM (B). A single fully 3-hydroxylated proline occurs at Pro986 (A1 site) in α1(I) and at Pro707 (A3 site) in α2(I). All four sites (A1 – A4) are fully or partially 3-hydroxylated in α2(V), and the spacing between A2, A3 and A4 is D-periodic (234 + 3 residues). Multiple sites of 3Hyp occur in the α1(V) chain (19,41) with a high occupancy and D-periodic spacing of B1 and B3 (19). It is notable that fibril surface binding sites for one class of small leucine-rich proteoglycans (e.g., fibromodulin, lumican) are close to 3Hyp sites.

Table 1

Non-Collagen Genes in which Mutations Cause Osteogenesis Imperfecta Variants

Gene	Protein		Phenotype	Bone Collagen Abnormalities
CRTAP *LEPRE1* *PPIB*	CRTAP P3H1, prolyl hydroxylase CYPB, cyclophilin B	P3H1 Complex	AR, bone fragility, reduced mineral density	α1(I)P986 and α2(I)P707 under prolyl-3-hydroxylation, high HP/LP (CRTAP & LEPRE1), low HP/LP (PPIB)
FKPB10 *PLOD2*	FKBP65 LH2, lysyl hydroxylase 2		AR, Bruck Syndrome: bone fragility, joint contractures	Lack of telopeptide hydroxylysines produces skin-like cross-links
SERPINH1	HSP47, heat shock protein 47		AR, bone fragility (type III OI)	High HP/LP, and abnormal arrangement of cross-linking bonds
SERPINF1	PEDF pigment epithelium-derived factor		AR, bone fragility and low mineral density, osteoid seams	Failed mineralization, no other collagen abnormalities
BMP1	Procollagen type I C-propeptidase		AR, bone fragility, high mineral density	Defective C-propeptide removal, potential cross-linking defects
IFITM5	Bril, osteoblast-specific small transmembrane protein		AD, bone fragility, hyperplastic callus (type V OI)	None reported

Table 1: Non-Collagen genes in which mutations cause osteogenesis imperfecta variants.

defective lysyl hydroxylase 2 activity caused by mutations in either *PLOD2*, which encodes the enzyme, or *FKBP10*, which encodes another peptidyl prolyl isomerase required to fold the enzyme. Null mutations in either gene produce close phenocopies of Bruck syndrome both clinically and in terms of abnormal collagen cross-linking.

Cross-linking of Bone Collagen

Intermolecular cross-links between collagen telopeptide and helical domains are essential for fibril strength. Bone collagen presents a unique profile of cross-linking since the telopeptide lysines, which lysyl oxidase converts to reactive aldehydes to form the cross-links, are only 50% hydroxylated compared with 0% in skin type I collagen and 100% in type I and II collagens of cartilages. The mechanism regulating this is unclear, though three hydroxylase enzymes encoded by three genes (*PLOD 1, 2 and 3*; Figure 2) are involved. Consequently the initial divalent and mature trivalent cross-links of bone collagen give a distinctive pattern on peptide analysis with about equal amounts of mature pyrroles and pyridinolines in human bone collagen (Figure 3; 5). The ratio of pyrroles to pyridinolines has been shown to vary between bone types, for example between osteoporotic and control human bone, with a high pyrrole content associated with a finely meshed trabecular architecture.

Collagen Prolyl 3-hydroxylation

3-hydroxyproline is a highly regulated, rare modification found in collagens throughout multicellular animals. A single fully modified 3Hyp in α1(I) and α1(II) chains at Pro986 appears in ancestral type I collagen of early chordates, coinciding with the appearance of the *Crtap* gene in the genomic record just before vertebrates and bone tissue emerged. The Pro986 3Hyp site appears to be hydroxylated exclusively by a P3H1/CRTAP/PPIB protein complex (see Table 1), suggesting it became a new collagen substrate for prolyl 3-hydroxylase when CRTAP added to the enzyme in a complex [4]. To speculate, this acquired Pro986 modification may have helped equip collagen molecules for a polymeric architecture that was better suited to enable ordered hydroxyapatite nanocrystal growth within fibrils. An effect on molecular assembly and the cross-linking arrangement is one possibility we are exploring.

In summary, despite a growing list of causative genes, most OI cases whether recessive or dominant are associated with defective molecular assembly of bone collagen. The new findings reveal and highlight the importance of understanding in detail the mechanism controlling the unique post-translational quality of collagen fibrils in bone. Can these new genetic insights also aid in understanding whether certain post-translational properties unique to bone collagen are essential for its characteristic pattern of nanocrystal mineralization?

Implications for bone fragility

With this growing knowledge a repeated theme is emerging of abnormal cross-linking when bone collagen from OI patients is biochemically analyzed. This supports a hypothesis that abnormalities in cross-linking that can result from a range of defects in collagen assembly are a common causative feature of brittle bone. Clearly collagen strength depends heavily on its cross-linking but other properties of bone including ductility and resistance to micro-damage and crack propagation may depend on the unique pattern of lysyl oxidase mediated cross-links seen in normal bone collagen of higher vertebrates. The placement of cross-links and the chemically labile nature of half of them may be required to produce a malleable framework in which mineral nanocrystals can grow optimally within the fibrils. Related differences in collagen cross-linking are seen in comparison of adult osteoporotic and normal bone, implying a more general pathological mechanism associated with aging.

Future Challenges

To define how tissue-specificity in the chemistry and placement of cross-linking bonds is regulated in bone collagen and whether cross-linking irregularities are a common mechanism underlying bone fragility in OI. Are pyrrole cross-links important for the unique material properties of bone and is their loss a determining factor in bone fragility (in OI and acquired osteoporosis)?

The biggest challenge will be demonstrating cause and effect relationships between the altered post-translational features of bone collagen and material properties as a basis for predicting increased fragility. Multidisciplinary collaborations and genetic approaches will continue to be vital for progress in this rapidly advancing field.

References

1. Glimcher MJ (1960) Specificity of the molecular structure of organic matrices in mineralization. In: R.F. Sogannes, editor. Calcification in Biological Systems. American Association for the Advancement of Science, Washington, DC.

2. Cundy T (2012) Recent advances in osteogenesis imperfect. Calcif Tissue Int 90:439-449.

3. Morello R, Bertin TK, Chen Y, Hicks J, Tonachini L, Monticone M, Castagnola P, Rauch F, Glorieux FH, Vranka J, Bächinger HP, Pace JM, Schwarze U, Byers PH, Weis MA, Fernandes RJ, Eyre DR, Yao Z, Boyce BF, Lee B (2006) CRTAP is required for prolyl 3-hydroxylation and mutations cause recessive osteogenesis imperfecta. Cell 127(2):291-304.

4. Weis MA, Hudson DM, Kim L, Scott M, Wu JJ, Eyre DR (2010) Location of 3-hydroxyproline residues in collagen types I, II, III and V/XI implies a role in fibril supramolecular assembly. J Biol Chem 285(4):2580-2590. PMC2807315

5. Eyre DR, Weis MA, Wu JJ (2008) Advances in Collagen Cross-link Analysis. Methods 45:65-74.

Arthroscopic Surgery Skill Acquisition In the Era of Restricted Work Hours

Winston J. Warme, MD, Brian Gilmer, MD, Aaron Chamberlain, MD, Nathan Coleman, MD, Bryan Comstock, BS, Seth S. Leopold, MD, and Frederick A. Matsen III, MD

Arthroscopic surgical skill acquisition is difficult to acquire even in a well-balanced residency program, given the broad exposure to the various orthopaedic subspecialties and current work hour restrictions. The use of simulators to develop psychomotor skills is in vogue in many of the surgical disciplines, but has its limitations. Laboratory cadaveric courses provide more realistic training, and can be valuable adjuncts towards achieving arthroscopic competency.

Introduction

The University of Washington Orthopaedic Surgery Residency Program has a long and distinguished history of training surgeons who are capable of independently caring well for patients after graduation. With the time spent alongside renowned traumatologists at Harborview Medical Center, they have been particularly skilled in the management of fractures, but their ability to perform arthroscopic surgery has been more limited. Work hour restrictions further curtail their exposure to surgical cases. We hypothesized that an intensive one-week course for the third and fourth year residents, operating on cadaver knees and shoulders would improve the residents' competence and confidence in performing arthroscopic surgery.

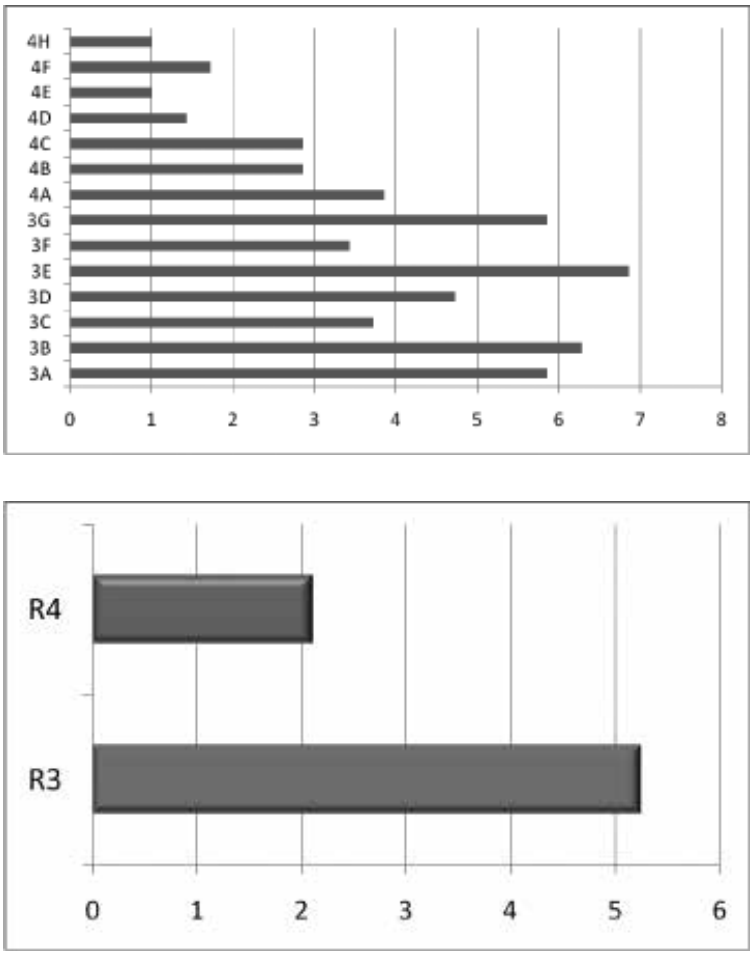


Figure 1: (Top) Improvement in confidence by resident (average of all tasks). (Bottom) Mean increase in confidence by year.

Methods

The research protocol was examined by the IRB and given an exemption. Nonetheless, each resident was issued a unique identifier, for subsequent analysis purposes, to which neither the research team nor the instructors were privy to. This was done to allay any fears that poor performance might impact their residency and to decrease any stress inherent to the testing process.

With an unrestricted educational grant, we arranged for a one-week course where the residents would have 44 hours of operative time on cadavers, and at least 4 additional hours of dry lab exercise. 16 residents (8 PGY-3 and 8 PGY- 4) participated in a five-day comprehensive intensive arthroscopic skills course, which included teaching of arthroscopic knot tying technique. The same 8 residents who completed the course as R3s, repeated the course the following year. Features of the course beyond arthroscopic knot tying included diagnostic arthroscopy of knee and shoulder, triangulation exercises, arthroscopic meniscectomy, meniscal repair, chondroplasty, microfracture, ACL/PCL reconstruction, arthroscopic Bankart and labral repair, arthroscopic distal clavicle resection, subacromial decompression and arthroscopic rotator cuff repair. At the end of the arthroscopic exercises residents were encouraged to dissect each cadaver to inspect their arthroscopic repairs directly, and review pertinent anatomy. In addition to 8 hours each day of scheduled cadaver wet lab activities, residents were provided

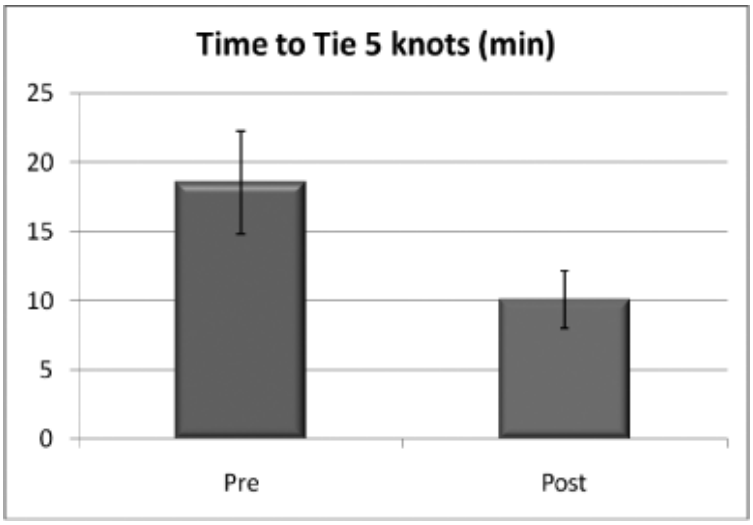


Figure 2: Results of knot tying effeciency.

unlimited access to a dry lab including arthroscopic knot tying stations, standard arthroscopic cannulas, a standard ring type arthroscopic knot pusher, and high strength polyblend arthroscopic suture material.

Their confidence in performing arthroscopic skills was assessed pre and post intervention with a questionnaire using a 10-point Likert scale. We tested their progress in tying arthroscopic knots through cannulas on knot tying stations, where they tied 5 knots for time, before and after formal knot tying instruction, and five days of practice. Skill and efficiency in arthroscopy was assessed via a timed arthroscopic triangulation exercise in cadaver knees. Five suture anchors were placed in various locations in the knee and the trainees were tasked to grasp them with a grasper, as if they were loose bodies that needed to be retrieved. The trainees performed the triangulation test on the first and last day of the course, on different cadavers. Course evaluations were obtained at the conclusion of the course.

Results

Overall arthroscopic surgical confidence improved 5.2 Likert points in the R3s and 2.1 points in the R4s, (Figure 1). Time to tie arthroscopic knots was roughly cut in half and approached the expert (attending) tying time of 9 minutes, (Figure 2). Triangulation time was also significantly improved, (Figure 3). Residents were extremely pleased with the training and articulated the course's educational value. Comments such as these were representative: "This experience accounts for my 1st and 2nd best weeks of residency." "This course was again an amazing learning experience and it has greatly enhanced my arthroscopy skills." "After day three, I could reliably get suture anchors where I wanted them and knew more portals and their uses than I'd previously ever heard of." "Those two weeks of scoping have had the single greatest impact on my technical skills and my ultimate decision to do a fellowship in sports medicine."

Discussion and Clinical Relevance

Arthroscopic skills are required for surgeons to function in a modern general orthopaedic practice. In the constrained contact hours available to current residents, educators are challenged to test old methods and develop new teaching paradigms that are both efficacious and time efficient in post-graduate medical training. Residency programs must ensure that trainees are provided with the requisite "stick time" to gain the necessary psychomotor skills to properly care for patients, and to gain surgical confidence. In some cases, an arthroscopic training wet lab exercise may be an effective adjunct to their formal operative experience. This course, along with increased exposure to arthroscopic surgeries, has significantly improved our residents' competence and confidence in performing arthroscopic surgery, and better prepared them for the arthroscopic part of their ensuing clinical practices.

References

1. Atesok K, Mabrey JD, Jazrawi LM, Egol KA. Surgical simulation in orthopaedic skills training. J Am Acad Orthop Surg. 2012;20(7):410-422.

2. Alderson D. Developing expertise in surgery. Med Teach. 2010;32(10):830-836.

3. Memon MA, Brigden D, Subramanya MS, Memon B. Assessing the surgeon's technical skills: analysis of the available tools. Acad Med. 2010;85(5):869-880.

	Pre (min)	Post (adjusted min)
Mean	5.58	3.09
SD	1.94	0.89
SEM	0.54	0.24

Figure 3: Speed in triangulation. (P Value 0.0028)

New Insight Into the Pathogenesis of Osteoarthritis

Kevin A. Lawson, BA, Colin J. Teteak, BA, Michael J. Chen, BA, John McCornack, Howard A. Chansky, MD, and Liu Yang, PhD

Articular chondrocytes from patients with osteoarthritis show 'phenotypic plasticity', comparable to mesenchymal stem cells undergoing chondrogenesis, by recapitulating aspects of chondrocyte hypertrophy. Here we demonstrate that knockout of the protein ESET histone methyltransferase in murine mesenchymal stem cells leads to ectopic hypertrophy and terminal differentiation of chondrocytes in articular tissue. This premature and abnormal hypertrophy predisposes the knockout mice to early articular cartilage degeneration.

Introduction

During skeletogenesis and joint formation, chondrocytes invariably follow two separate developmental paths. Growth plate chondrocytes display transient behavior, characterized by rapid proliferation and terminal differentiation, whereas articular chondrocytes are slow in turnover and phenotypically stable.[1] In osteoarthritis, however, articular chondrocytes fail to maintain their latent phenotype and become mitotically active, ultimately proliferating and expressing proteins such as type-X collagen, alkaline phosphatase and MMP-13 that are indicative of chondrocyte terminal differentiation.[2] This is a process reminiscent to the growth of long bones through endochondral ossification. We recently demonstrated that the protein ESET histone methyltransferase is transiently upregulated in growth plate chondrocytes to function as a 'gate keeper' in preventing entry into terminal differentiation, thus ESET knockout results in premature hypertrophy of growth plate chondrocytes and early physeal closure during skeletal development.[3] To investigate whether ESET is also essential in the regulation of articular chondrocyte differentiation, we histochemically analyzed articular cartilage of wild-type and ESET-deficient mice at different time points after birth. Here we report that deletion of the ESET gene is associated with ectopic hypertrophy of articular chondrocytes, overt signs of chondrogenic terminal differentiation, and early onset of articular cartilage degeneration.

Methods

- Generation of conditional mesenchymal specific ESET-null mice utilizing Cre-LoxP system
- Wild-type and ESET-null mice were sacked at 14 days, 1 month and 3 months after birth
- Hindlimbs were harvested, fixed overnight (4% paraformaldehyde), decalcified in EDTA and then fresh frozen tissue was sectioned for analysis
- Sections were stained utilizing immunohistochemistry for Type II collagen and Type X collagen
- Sections were also stained with hematoxylin and eosin (H and E) as well as alkaline phosphatase

Results

Type II collagen staining within the knockout (KO) articular cartilage displays large lacunae within the extracellullar matrix. These large lacunae are absent in the wild-type cartilage. Analysis of the tibial plateau stained with H and E demonstrates

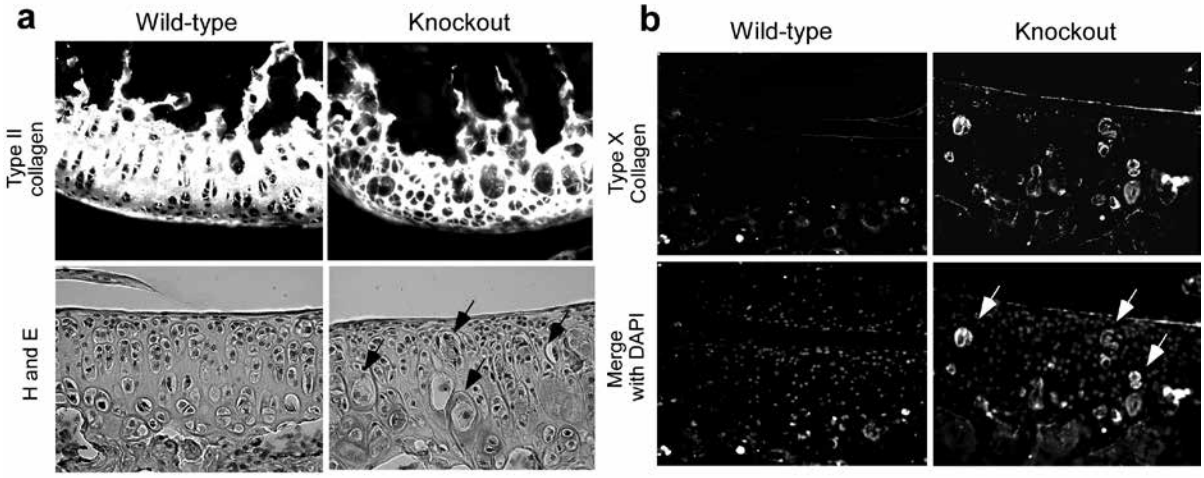


Figure 1: Ectopic hypertrophy of articular chondrocytes in one month-old ESET-null mice. a, shows type II collagen staining of medial femoral condyle and hematoxylin and eosin stain of the tibial plateau. b, shows type X collagen staining of the tibial plateau and merged image with DAPI.

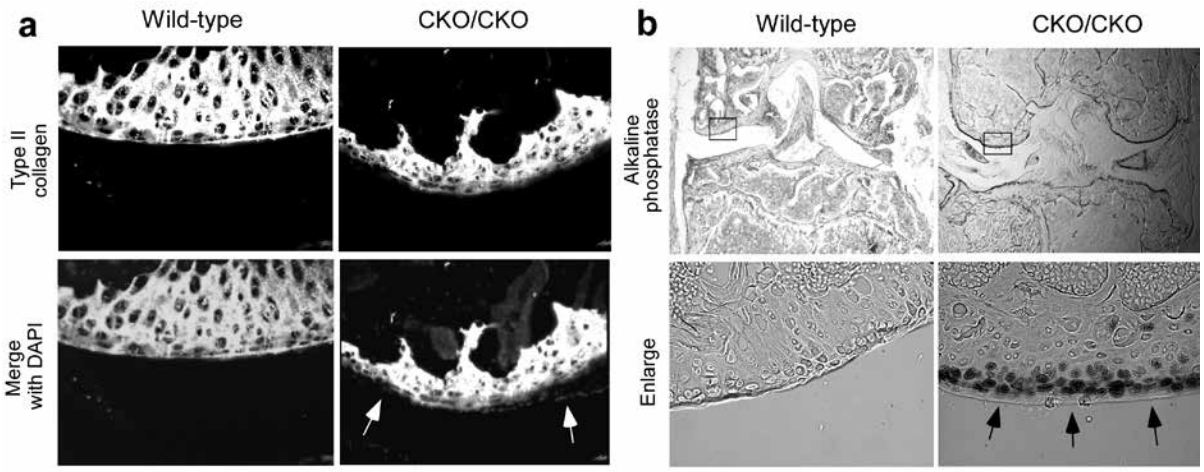


Figure 2: Degradation of articular cartilage and terminal differentiation of articular chondrocytes in three month-old ESET-null mice. a, demonstrates type II collagen staining of the femoral condyle. b, demonstrates alkaline phosphatase staining of the femoral condyle, with enlarged image.

large hypertrophic cells (black arrows) within the cartilage of the KO animals (Figure 1a). Type X cartilage is seen within the articular cartilage of knockout mice, with minimal staining viewed within control animals (Figure 1b). A merged image with DAPI staining (for nuclei) allows for the localization of the articular chondrocytes in comparison to the type X collagen. Type X staining in the KO is seen within all levels of the cartilage.

When compared to the control mice, there is a marked decrease in articular cartilage thickness in the KO model at 3 months (Figure 2a). Additionally a merged image with DAPI in Figure 2a shows Type II collagen staining in relationship to articular chondrocytes. In the control animals, all chondrocytes are contained within the type II collagen articular matrix. Within the ESET-knockout mice, in contrast, articular chondrcoytes are seen outside of the type II collagen stained tissue (white arrows). Figure 2b demonstrates a coronal image of knees from the ESET knockout mice and control control mice stained for alkaline phosphatase (ALP). An enlarged image of the distal femoral articular tissue is seen. There is positive ALP staining within the articular chondrocytes of the ESET knockout animals, specifically in the superficial zone (black arrows). Minimal ALP staining is seen within the control animal cartilage.

Discussion

Our lab has demonstrated that ESET histone methyltransferase deficiency leads to increased and premature chondrocyte hypertrophy at the growth plate.[3] In this study we investigated whether premature chondrocyte hypertrophy also occurs within the articular cartilage of ESET-knockout mice. Previous studies have shown that chondrocytes within osteoarthritic cartilage demonstrate phenotypic plasticity.[4] These cells take on morphological and histochemical characteristics of growth plate chondrocytes and subsequently undergo proliferation and hypertrophy during their participation in endochondral ossification and bone growth.[5] These changes are not seen in normal "resting" articular chondrocytes which are post-mitotic and minimally metabolic active.

Signs of chondrocyte differentiation within the articular cartilage begin to appear around 1 month after birth in the ESET knockout mice. This is demonstrated by the large, hypertrophic cells seen within the articular cartilage of the KO mice as well as the large lacunae, presumably containing these hypertrophic cells, seen in the type II collagen stained articular cartilage, (Figure 1a). Hypertrophy is a normal step in growth plate chondrocyte differentiation, but is not seen in healthy articular cartilage. Additionally we found a large increase in type X collagen staining within the articular cartilage of the ESET knockout mice as compared to control mice. Type X collagen is not only a marker of chondrocyte hypertrophy but is also found within the physis and is usually absent from normal articular cartilage. This histologic data indicates that articular chondrcoytes in ESET deficient mice fail to maintain their phenotype and demonstrate signs of terminal differentiation.

At 3 months, staining of type II collagen demonstrates a large decrease in thickness as compared to the control (Figure 2a). The decrease in type II collagen progresses with age, showing a marked decrease at 11 months in knockout mice (date not shown). Alkaline phosphatase is another marker of terminally differentiated chondrocytes. There is a large increase in the number of ALP-stained cells in 3 month-old knockout mice. Taken together this data indicates that the early hypertrophy seen at 1 month within the knockout mice predisposes their articular cartilage to wear and degradation that is seen at 3 months of age.

Conclusion

Here we demonstrate that ESET histone methyltransferase is a potent regulator of articular chondrocyte differentiation. Additionally, we show that aberrant articular chondrocyte differentiation may be involved in the pathogenesis of articular degeneration. Since similar changes are seen in osteoarthritis these findings may have clinical relevance.

References

1. Pfander, D et al. Expression of early and late differentiation markers (proliferating cell nuclear antigen, syndecan-3, annexin VI, and alkaline phosphatase) by human osteoarthritic chondrocytes. The American journal of pathology. 2001;159(5): 1777–83.

2. Dreier, R et al. Hypertrophic differentiation of chondrocytes in osteoarthritis: the developmental aspect of degenerative joint disorders. Arthritis Research & Therapy. 2010;12 (5):216

3. Yang L, Lawson KA, Teteak CJ, Hacquebord J, Zou J, Patterson DP, Ghatan AC, Mei Q, Chansky HA. ESET histone methyltransferase is essential to the control of chondrocyte hypertrophy and epiphyseal plate. Developmental Biology. Accepted March 2013.

4. Tchetina, E et al. Increased type II collagen degradation and very early focal cartilage degeneration is associated with upregulation of chondrocyte differentiation related genes in early human articular cartilage lesions. The Journal of Rheumatology, 2005;32(5): 876–86.

5. Goldring MB. Chondrogenesis, chondrocyte differentiation, and articular cartilage metabolism in health and osteoarthritis. Therapeutic Advances in Musculoskeletal Disease 2012;4(4):269-85.

Immediate Weightbearing After Ankle Fracture Fixation

Reza Firoozabadi, MD, MA, Emily Harnden, MD, and James C. Krieg, MD

Most physicians advocate a period of protected weightbearing post ankle fixation surgery. The objective of this study was to determine if a certain subset of ankle fracture surgical patients can be weightbearing as tolerated immediately following surgery. Retrospective study of prospectively gathered orthopaedic trauma data at a Level 1 trauma center was used to identify a total of 136 skeletally mature patients who underwent ORIF of an ankle fracture by the senior author; 33 were allowed immediate weightbearing as tolerated (IWBAT). Twenty six patients were included, having at least six weeks of follow up. Only 1/26 patients was noted to have loss of fixation. This was found at the 6 week follow up and was attributed to a missed syndesmotic injury. IWBAT in a certain subset of patients with stable osteosynthesis following an ankle fracture is a safe alternative to a period of protected weight bearing.

Introduction

Ankle fractures are among the most common injuries treated by orthopaedic surgeons. Open anatomic reduction and internal fixation is routinely advocated for displaced, unstable ankle fractures. Recently, emphasis has been placed on functional outcome and recovery. Faster return of function and return to work are related to rehabilitation strategy. Following operative treatment of ankle fractures, most physicians advocate a period of non-weightbearing followed by partial progressive weightbearing.

The primary aim of this project was to determine if a certain subset of ankle fracture surgical patients can be weightbearing as tolerated immediately following surgery. We assume that earlier weightbearing will allow patients to return to their activities of daily living quicker, with an overall easier time during convalescence.

Methods

This study was approved by our institutional review board. A prospectively-gathered orthopaedic trauma database at a Level 1 trauma center was reviewed retrospectively to identify patients who had sustained unstable ankle injuries treated by the senior author between January 2007 and December 2011. A total of 136 skeletally mature patients underwent ankle surgery, 33 of which were allowed IWBAT in the acute post-operative period. Patients were not made weightbearing as tolerated for a number reasons (Figure 1).

Patient demographic data and fracture characteristics (AO/OTA classification) were identified. Bimalleolar, trimalleolar, fracture dislocation, and fibular fractures with more than 4mm medial clear space widening on stress radiographs or positive gravity stress views were deemed unstable.

Surgical protocol included open anatomic reduction and internal fixation of the fibula by resident/fellow

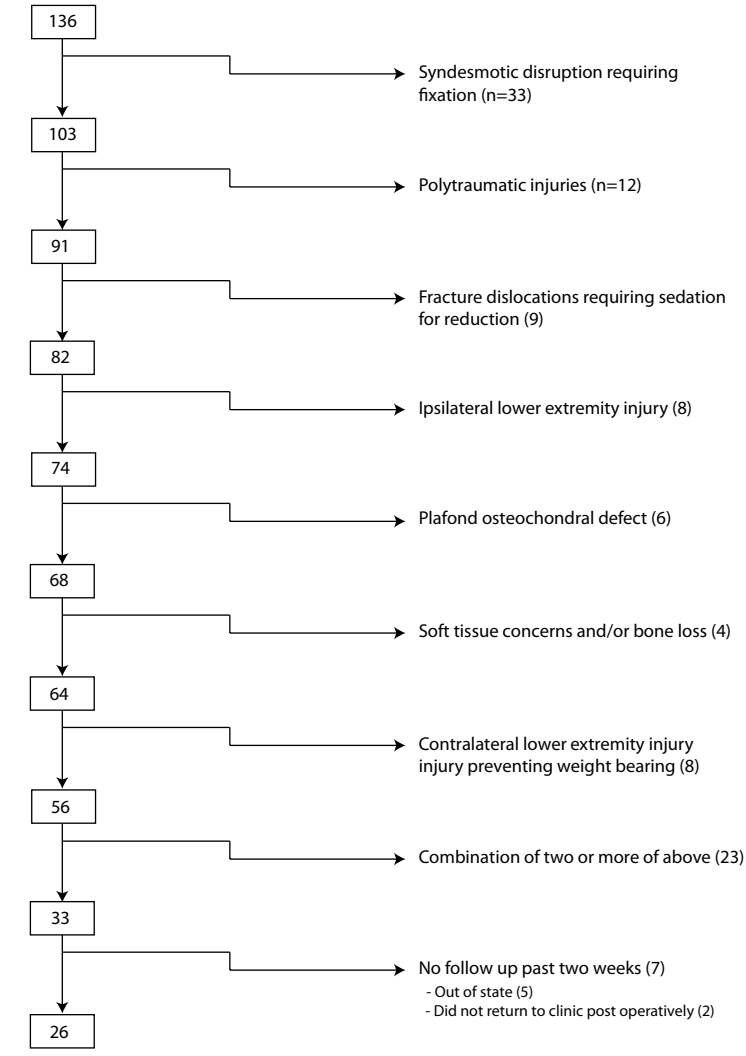
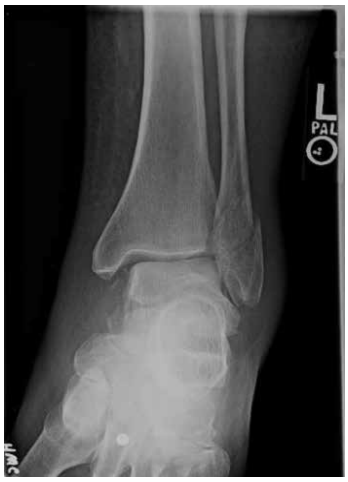
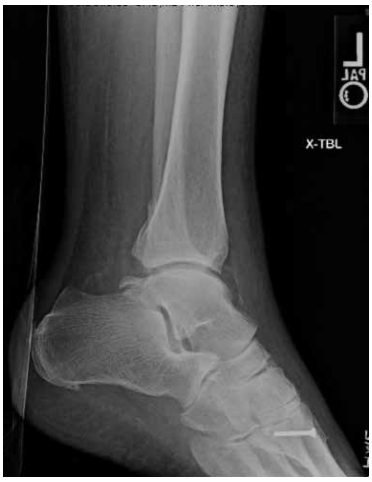


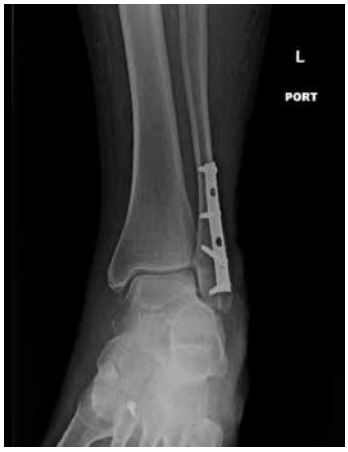
Figure 1: Ankle surgery patients between 1/07 - 11/11.



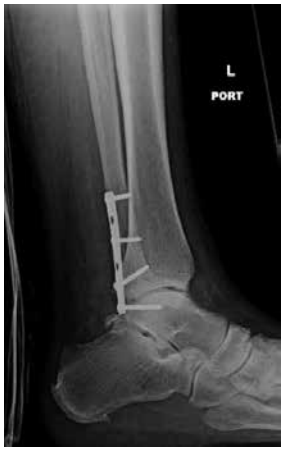
Pre-operative injury mortise view



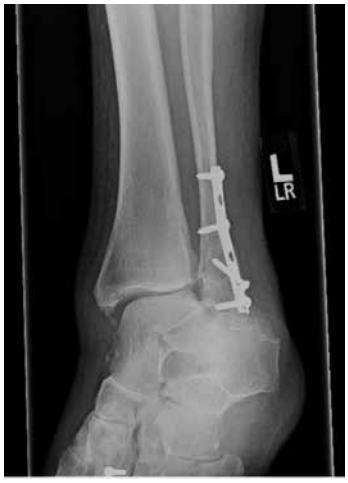
Pre-operative injury lateral view



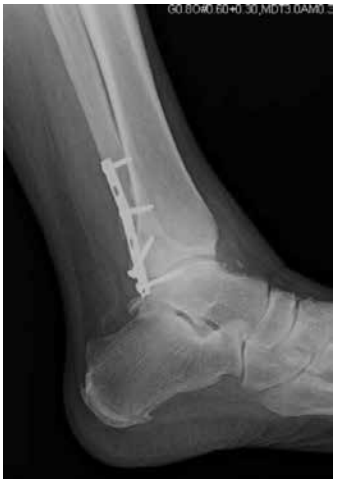
Post-operative mortise view



Post-operative lateral view

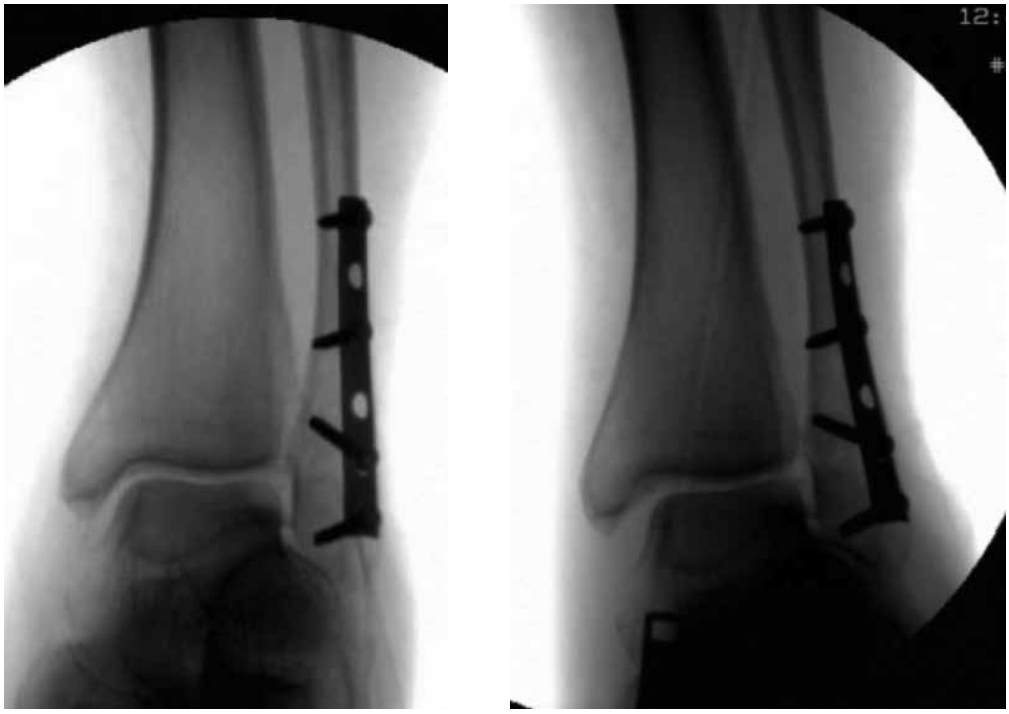


Six week mortise view



Six week lateral view

Figure 2: Single case of failure.



Intraoperative pre-stress mortise view

Intraoperative evidence of syndesmotic injury, post-stress

Figure 3: Intraoperative stress test in single case of failure.

supervised by the trauma fellowship trained senior author. In cases in which the medial malleolus was fractured, screws or small fragment plates were used for fixation. Posterior malleolus fractures were fixed on a case-by-case basis. Although no clear indications exist for fixation of small posterior malleolus fractures, many of the small fractures and all of the larger fractures were treated operatively. The syndesmosis was reduced and held in place with screw fixation if stress testing displayed widening after the malleoli were fixed.

Postoperative protocols were similar for all patients. Patients who were allowed IWBAT were protected in a Controlled Ankle Motion (CAM) Walker Boot. The boot was kept on at all times for the first two weeks. At two weeks the wound was assessed and sutures were removed. The patients were then instructed to continue wearing the CAM Walker Boot for an additional 2-4 weeks, coming out for hygiene only. Three view radiographs of the ankle were obtained at the 6, 12, 24, and 52 week time points. At 6 weeks post operation, the boot was discontinued if the patient had not already converted over to a shoe. Attempts were made to follow up with patients until clinical healing had occurred.

Results

Of the 26 patients who had at least six weeks of follow up, 20 were male and 6 were female, and their average age was 48 years (range 20-95 years). Mechanism of injury included 17 low-energy falls, three motor vehicle accidents, two pedestrians struck by motor vehicles, two twisting injuries while playing sports, one fall off bicycle, and one assault. According to AO/OTA fracture classification, there were four type-44A (4%), 21 type-44B (81%), and one type-44C(4%). Fifteen patients (58%) were cigarette smokers, and two patients (8%) had diabetes.

Lateral malleolus fixation included: 20 1/3rd tubular plates (77%), four precontoured posterolateral plates (15%), and one intramedullary nail (4%). Medial malleolus fixation was required in eleven patients (42%), screws were used in twenty two cases (85%) and a plate in four cases (15%). Posterior malleolus required fixation in five cases (19%). Twenty five patients had intraoperative post fixation radiographs that displayed symmetric joint space around the talus. One patient had 1.7mm increased lateral joint space compared to medial and superior clear space.

Average follow up time was 140 days (range 40-478 days). Clinical evaluation at two weeks was significant for two patients having peri-incisional erythema that resolved with a short course of oral antibiotics (8%). At six weeks, no wound issues were noted. Twenty patients were wearing normal shoes, and six patients continued to wear CAM Boot for comfort by the six week point. At the last clinic visit, three patients had persistent ankle stiffness, one patient had symptoms consistent with peroneal subluxation, which resolved with physical therapy, and one patient required removal of medial malleolar fixation secondary to symptomatic hardware.

Radiographic evaluation at six weeks displayed no loss of reduction in 25 patients (96%) and one loss of reduction (4%). This was the same patient that was noted to have 1.7mm of increased lateral joint space compared to medial and superior clear space. At the six week interval, the lateral joint space was 4.8mm greater than the

medial and superior clear space (Figure 2). Intraoperative fluoroscopy images were reviewed, and it was noted that the patient had a missed syndesmotic injury (Figure 3).

Discussion

This study was designed to analyze whether immediate weightbearing after stabilization of unstable ankle fractures would result in early loss of fixation. Ahl et al. prospectively compared immediate and late weight bearing post ankle fixation in a below knee cast. Radiographic and clinical analysis at three and six months did not display a difference between the two groups. More recently, Simanski et al. performed a prospective study comparing functional early weight bearing (3 weeks) versus 6 weeks without weightbearing in a below knee cast. The early weightbearing group was allowed partial weightbearing (10-15kg) in an Aircast Air-Stirrup Brace immediately after surgery. The patients were then allowed full weight bearing at 3 weeks if no problems were identified. Early weightbearing patients were able to obtain full weightbearing in advance of the delayed group (7.7 vs. 13.5 weeks, p=0.01). No disadvantage was noted in regards to the early weight bearing group both clinically and radiographically.

Our findings show that patients can fully weight bear as tolerated during the immediate post-operative period similar to patients with stable ankle fractures. Our patient group had one case of loss of reduction and fixation failure. This occurred as a result of a missed syndesmotic injury. This reaffirms the importance of identifying syndesmotic disruptions.

This study has a number of limitations inherent in any retrospective case series. The major limitation being that only a subset of patients with unstable ankle fractures was allowed immediate full weightbearing. This discretion was set by the senior author's practice guideline. Although this study does support immediate weight bearing post-operatively for a certain subset of patients with ankle fractures, we feel that a controlled, prospective trial is warranted to look further at the influence of delayed versus immediate weight bearing post ankle fixation surgery.

IWBAT in a certain subset of patients with stable osteosynthesis following an ankle fracture is a safe alternative to a period of protected weight bearing. Potential candidates for IWBAT are patients with closed ankle fractures, without syndesmotic disruption, no involvement of the tibial plafond, in whom stable fixation has been achieved.

References

1. Michelson JD. Fractures about the ankle. J Bone Joint Surg Am 1995;77:142-52.

2. Bauer M, Jonsson K, Nilsson B. Thirty-year follow-up of ankle fractures. Acta Orthop Scand 1985;56:103-6.

3. Simanski CJ, Maegele MG, Lefering R, et al. Functional treatment and early weightbearing after an ankle fracture: a prospective study. J Orthop Trauma 2006;20:108-14.

4. Ahl T, Dalen N, Holmberg S, et al. Early weight bearing of displaced ankle fractures. Acta Orthop Scand 1987;58:535-8.

5. Egol KA, Dolan R, Koval KJ. Functional outcome of surgery for fractures of the ankle. A prospective, randomised comparison of management in a cast or a functional brace. J Bone Joint Surg Br 2000;82:246-9.

Lessons Regarding the Safety of Orthopaedic Patient Care: An Analysis of Four Hundred and Sixty-Four Closed Malpractice Claims

Frederick A. Matsen III, MD, Linda Stephens, PhD, Jocelyn L. Jette, BS,
Winston J. Warme, MD, and Karen L. Posner, PhD

Background: An orthopaedic malpractice claim alleges that the patient sustained a preventable iatrogenic injury. The analysis of a representative series of malpractice claims provides a unique view of alleged orthopaedic adverse events, revealing what can potentially go wrong across a spectrum of practice settings and anatomic locations. The goal of this study was to identify high-impact targets in order to institute measures to reduce claims through efforts focused on patient safety.

Methods: The authors investigated 464 consecutive closed malpractice claims from the nation's largest insurer of medical liability. We analyzed the claims by anatomical site, type of care rendered, type of allegation, and payment. We calculated an "impact factor" for each claim type by dividing the percentage of total payments for each type by the percentage of total claims for that type.

Results: Our analysis revealed major concerns regarding patient safety within this series of malpractice claims. One-third of the claims alleged permanent disabling injuries, including amputations, brain damage, and major nerve damage. The highest impact allegations were failure to protect structures in the surgical field (41% of total payments to plaintiffs, 15% of all claims, impact factor of 2.7) and failure to prevent, diagnose, and/or treat complications of treatment (16% of total payments, 7% of all claims, impact factor of 2.3). Spine procedures had high impact (1.9), representing 28% of dollars paid and 15% of claims, with 45% of spine claims involving death or severe permanent injury. Failure of implant positioning was commonly alleged in hip and knee arthroplasty. In claims related to fracture care, the most common allegations were related to malunions, nonunions, dislocations, failure to protect structures in the surgical field, infection, and treatment complications. Total payment for the eighty-eight claims paid was $17,917,614 (U.S. dollars adjusted to 2009).

Conclusions: Regarding clinical relevance, this analysis suggests risk areas for targeted efforts to improve patient safety and reduce malpractice claims.

Introduction

Prior articles about orthopaedic malpractice have focused on negligence, surgeon demographics, informed consent, defensive imaging, risk management and tort reform, these papers have not called out the possibility of lessening the risk of malpractice claims by improving patient safety. We reviewed a large number of closed malpractice claims from a major national insurance company in search of lessons that might guide future efforts to make orthopaedic surgery safer for the patient and for the surgeon.

Material / Methods

- We reviewed 464 orthopaedic malpractice claims that were closed (that is settled, adjudicated or dropped) between January 2008 and April 2010
- We grouped the claims by type of allegation (e.g. failure to protect structures in the surgical field, complications of treatment, fracture management error, infection, implant malposition, etc).
- We also determined what we termed an impact factor to characterize the relative importance of different types of claim types in orthopaedic liability. The impact factor for each claim type is the ratio of the percent of total payment dollars divided by the percent of the total number of claims for that claim type. For example, medication-related errors accounted for 5% ($890,000) of the total payment dollars ($17,917,614) and 4% (nineteen) of the 464 claims; thus the impact factor for medication errors was 1.25 (5/4) (all payments and costs are expressed in U.S. dollars and adjusted to 2009). Impact factors greater than 1 indicated that the particular claim type was associated with above-average payment dollars per claim.

Results

- The most common claims were in cases involving, in order, fracture fixation, spine, shoulder arthroscopy, hip arthroplasty, knee arthroplasty and knee arthroscopy.
- Spine procedure claims accounted for 15% of all orthopaedic claims (sixty-nine of 457) and for 28% ($4,942,560) of all dollars paid to plaintiffs (impact factor of 1.9), with a median payment of $250,000 ($p = 0.016$). In contrast, hip and pelvis claims were 10% of all claims (forty-six of 457) but only 1% ($199,999) of dollars paid to plaintiffs (impact factor of 0.1), with a median payment of $40,000.

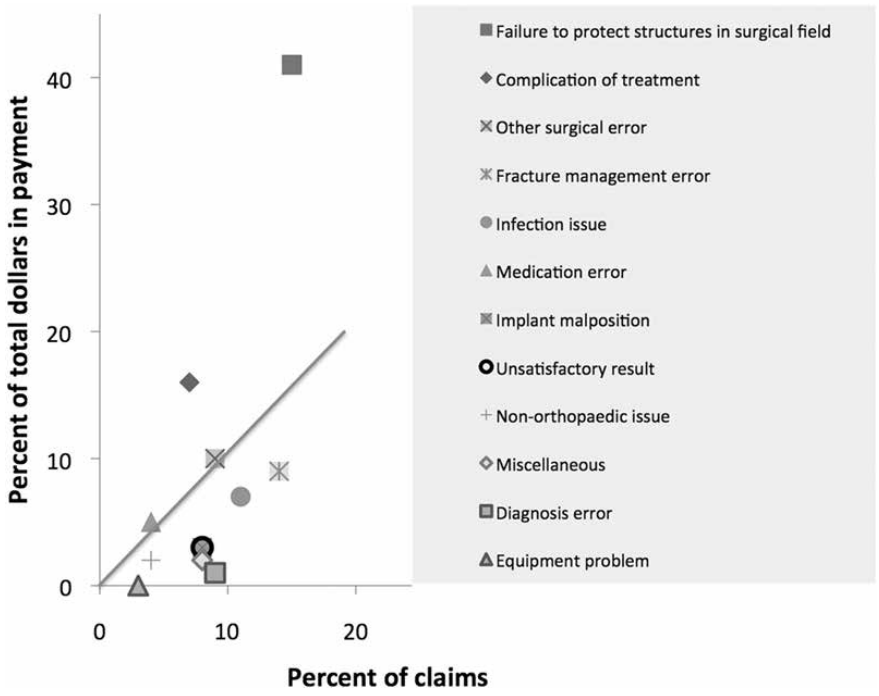


Figure 1: The relationship of relative claim payment to claim frequency for twelve claim types. The horizontal axis shows the number of claims of the indicated type divided by the total number of claims (464) expressed as a percentage. The vertical axis shows the total payment dollars for claims of the indicated type divided by the total payment for all claim types ($17,917,614) expressed as a percentage. Points above the line of identity indicate impact factors greater than 1 (i.e., those claim types where the payment is disproportionally high with respect to the claim frequency).

- Especially costly claims were those involving nerve injury, vascular injury, failure to prevent, diagnose or treat complications, and failure to diagnose a neoplasm.

Discussion

Most orthopaedic surgeons will face a malpractice claim in their career. Regardless of the outcome of a claim, it is costly in time and stress for the orthopaedic surgeon and raises concern about the safety of the care rendered to the patient. Our analysis strongly suggests that preventative efforts should be directed at the safety of intraoperative care, especially with respect to increasing awareness of high-impact errors and teaching surgical approaches that protect structures in the surgical field; this type of claim was both frequent and costly in this study. Some intraoperative issues (e.g., the safety of nerves, vessels, and other structures during spine and extremity surgery) might best be addressed by educational programs to increase surgeon awareness, knowledge, skill, and experience, with use of technique videos, cadaver laboratories, and surgical simulations aimed at safe surgical approaches. Other intraoperative patient safety issues (e.g., the timely administration of preoperative antibiotics and avoidance of wrong-site surgery) may be better addressed by systems-based strategies than by education alone.

The relative frequency of claims alleging failure to prevent, diagnose, or treat complications, especially after joint replacement and fracture fixation, indicates a need for a systematic approach to surveillance for the early diagnosis and treatment of complications (e.g., compartment syndrome, skin ulcers, pulmonary embolism, and infection). While infection was a relatively lower-impact allegation in the series of claims we investigated, it was the third most prevalent, with 11% of total claims, emphasizing the importance of systems-based approaches to ensure the timely administration of appropriate prophylactic antibiotics as well as the timely diagnosis and treatment of infections when they occur.

Many solutions are being considered to reduce costs related to orthopaedic malpractice claims, including tort reform. However, because many iatrogenic complications do not result in malpractice claims and many claims do not result in verdicts, the tort system does not appear to be an ideal target for optimizing patient safety. It seems more likely that education and systems-based approaches directed at patient safety may be helpful in reducing malpractice claims. In the absence of a national system for reporting and investigating adverse orthopaedic events, the analysis of allegations that led to malpractice claims provides an important means for identifying quality of care concerns in different practice settings and developing approaches for their avoidance. A malpractice claim is an indication of a deviation from the anticipated outcome; as such, each claim merits analysis as to the factors contributing to it and to the strategies for prevention of recurrence as a part of structured continuous learning and continuous quality improvement. Malpractice claims reveal problems, and their analysis can motivate problem solving and new knowledge that can be shared across the specialty as educational programs put in place by the national orthopaedic leadership and as systems-based strategies that can be implemented by medical centers.

References

1. Kellsey DC.The anatomy of orthopaedic malpractice. J Bone Joint Surg Am. 1975 Oct;57(7):1013-8.

2. Committee on Professional Liability. Managing orthopaedic malpractice risk. 2nd ed. Rosemont, IL: American Academy of Orthopaedic Surgeons; 2000.

3. American Academy of Orthopaedic Surgeons, Committee on Professional Responsibility. Medical malpractice: a primer for orthopaedic residents and fellows. Rosemont, IL: American Academy of Orthopaedic Surgeons; 1993

4. Greenberg MD, Haviland AM, Ashwood JS, Main R. Is better patient safety associated with less malpractice activity? Santa Monica: RAND Corporation; 2010.

5. Pegalis SE, Bal BS. Closed medical negligence claims can drive patient safety and reduce litigation. Clin Orthop Relat Res. 2012 May;470(5):1398-404.

Please note this research has now been published in the Journal of Bone and Joint Surgery (J Bone Joint Surg Am, 2013 Feb 20;95(4):e20 1-8). The article is available at the following link: http://jbjs.org/article.aspx?articleid=1653101

Monitoring Tibial Shock During Parabolic Flight

Peter R. Cavanagh, PhD, DSc, Molly D. Glauberman, MS, Alexandre E. Bykov,
Andrea J. Rice, MS, Grier L. Wilt, Andrea M. Hanson, PhD[1],
Kelly G. Gilkey, MS[2], and Justin H. Funk, PE[3]

Future space exploration missions will be of longer duration than past expeditions to the moon or to the International Space Station. In order to maintain bone health during these missions, it is essential that optimal methods for the prescription and monitoring of exercise programs that promote bone health and maintenance are identified. We have developed a system that could wirelessly monitor and classify activity in space. Such a system would enhance the quality and comprehensive nature of our knowledge regarding the daily loads experienced by astronauts. The effectiveness of the proposed system was tested in parabolic flight, during which there are brief periods of reduced gravity. Peak axial tibial acceleration scaled approximately linearly with the gravity level during the same activity when performed in simulated lunar, Martian, and 1g loads. Analysis of tibial accelerations and ground reaction forces showed large reductions in tibial shock during lunar activities compared to values for the same activities in 1g. The proposed system could easily be implemented on the International Space Station where it would be useful in monitoring astronaut-loading history to assist in the planning of individualized exercise prescriptions.

Introduction

The degree to which living and exercising in reduced-gravity environments will provide an osteo-protective stimulus remains unknown. Parabolic flight is an accessible analog of reduced gravity environments during which activity in zero G as well as lunar and Martian gravities can be studied. We have developed an activity monitoring system to recognize activities and to monitor the mechanical stimulus delivered to the lower extremity during daily activity. Remote monitoring of these quantities during spaceflight could aid in prescribing and monitoring effective exercise programs.

Methods

- A Kistler Gaitway treadmill was mounted to the floor of a Zero G Corporation modified 727-200 aircraft. The plane completed 163 parabolas in four flights over three days. At the apex of each parabola, brief periods (~10-20 seconds) of reduced gravity were available including 0g, lunar gravity (1/6g), and Martian gravity (3/8g).
- A compact subject load device (C-SLD) connected the subject to the treadmill via a pair of pressure actuators that could generate a range of gravity-replacement forces. The C-SLD, designed at NASA Glenn Research Center, was used during 0g parabolas to simulate 1/6g, 3/8g and 1g loading.
- Four female subjects (mean age 27.3 years, mean height 163.2 cm, mean weight 61.8 kg), were instrumented with 2 acceleration measurement units (Figure 1) that incorporated a tri-axial accelerometer (±12 g) and a tri-axial rate gyro (±2000°/sec). A Velcro strap tightened with 5 pounds of tension retained a tibial accelerometer 5 cm superior to the medial malleolus along the medial tibial border. The second AMU was securely placed in a running belt and tightened to the subject's waist at L5-S1 level. Bluetooth radios in the AMUs enabled real-time data streaming to laptop computers. The subjects performed walking, running, loping, hopping, squats, heel raises, and hip ab/adduction exercises on the treadmill (Figure 2).
- Parabolic flight is a challenging environment for data collection. The annotated experimental record of the resultant tibial acceleration (Figure 3) shows that often only a relatively few "steady state" cycles of locomotor exercise are available once the gravity condition is established, the treadmill is started and the subject is in equilibrium. In general, patterns of movement are much more variable than during similar exercise in 1g.

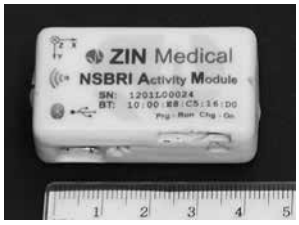


Figure 1: AMU which was attached to the tibia and waist belt.

Results

- Gravity-Replacement: The C-SLDs provided accurate gravity-replacement loads that enabled the simulation of locomotion under any gravitational condition during the 0g parabolas. There was approximately 3.5% variation in the applied load during a single running trial.
- Tibial Acceleration: Peak axial tibial acceleration scaled approximately linearly with the gravity level during the same activity when performed in simulated lunar, Martian, and 1g C-SLD loads (Figure 4). Whereas peak values during 1g ground-based running are

Figure 2: Subject running on the treadmill during a zero-gravity parabola.

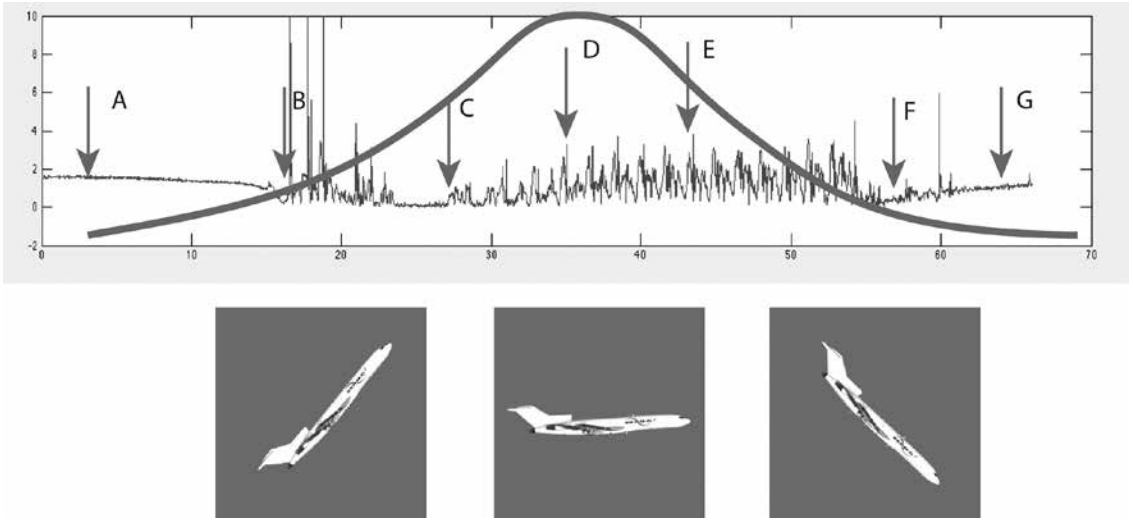


Figure 3: Resultant tibial acceleration vs. time during a single parabola during running.
A. 1.8g pull during the climb with maximum nose-up angle of 47 degrees
B. Gravity begins to "bleed off" as the apex of the parabola is approached
C. Subject mounts treadmill as 0g phase starts
D. Tibial acceleration increases as subject gains speed
E. Approximately 10 cycles of running
F. Treadmill stops as 0g phase ends
G. 1.8g pullout preparation for next parabola

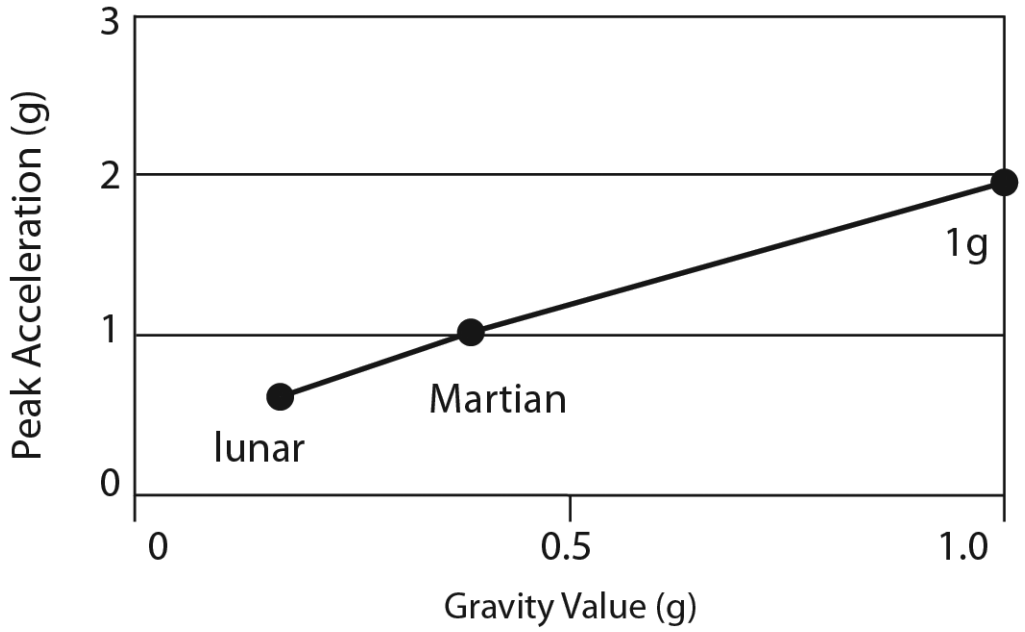


Figure 4: Mean peak tibial acceleration (n=3) during walking at 3 different gravity levels.

typically 7.5g, similar values in Martian and lunar gravity were 3.5g and 1.4g respectively.

Discussion

These results imply that locomotor exercise on the moon is unlikely to provide sufficient mechanical stimulus for bone maintenance since we know that running on the International Space Station treadmill with less than body weight loading was not sufficient to maintain bone mass (Genc et al. 2010). An examination of the components of the acceleration-time curves that have been shown to be osteogenic on Earth (Jämsä et al. 2011) is currently underway.

The insights from tibial acceleration measurements are useful in the evaluation of loading from exercise in reduced gravity. The proposed system could easily be implemented on the International Space Station where it would be useful in monitoring astronaut-loading history to assist in the planning of individualized exercise prescriptions.

References

1. Genc KO, Gopalakrishnan R, Kuklis MM, Maender CC, Rice AJ, Bowersox KD, Cavanagh PR. Foot forces during exercise on the International Space Station. J Biomech. 2010 Nov 16;43(15):3020-7

2. Jämsä T, Ahola R, Korpelainen R. Frontiers in Physiology. Measurement of osteogenic exercise–how to interpret accelerometric data? Clinical and Translational Physiology October 2011 (2). 2-4.

[1]Wyle Science, Technology & Engineering Group, Houston, Texas, USA, [2]NASA Glenn Research Center, Cleveland, Ohio, USA, and [3]ZIN Technologies, Inc., Cleveland, Ohio, USA.

Trehalose Decreases Apoptosis in Osteochondral Grafts Stored at 4ºC

Kevin A. Lawson, BA, Colin J. Teteak, BA, John McCornack, Liu Yang, PhD,
Howard A. Chansky, MD, and Albert O. Gee, MD

Prolonged osteochondral allograft storage leads to chondrocyte death and worse clinical outcomes. Trehalose, a non-reducing disaccharide, is commonly used in tissue storage as a cryoprotectant. Here we demonstrate that prolonged storage of osteochondral grafts at 4ºC in media supplemented with trehalose decreases chondrocyte apoptosis.

Introduction

Osteochondral allograft transplantation is a common treatment option to address large articular defects, providing functional restoration of the affected joint. Traditionally "fresh" osteochondral allografts were utilized within days of harvest, resulting in good long term clinical outcomes.[1] Recently, due to increased graft demand and concerns regarding disease transmission, increased screening has prolonged the time period from tissue harvest to clinical use.[2] This prolonged storage has led to decreased graft integrity and *in vivo* performance.[3] In particular, the chondrocyte viability decreases drastically after storage, leading to a diminished population of cells capable of maintaining articular cartilage integrity within the grafted tissues.[4] Recent literature has indicated most chondrocytes die by programmed cell death or apoptosis.[5] Optimal storage of osteochondral allografts aims to preserve chondrocyte viability, which allows for maintenance of cartilage matrix, remodeling after transplantation, and the preservation of matrix biomechanical properties. Trehalose, a non-reducing disaccharide, has been shown to preserve tissue viability after storage through cell membrane and protein stabilization during freeze-thawing.[6] More recent studies have shown that these cell protectant effects are also observed at higher temperatures, such as 4°C, the current industry standard for osteochondral allografts. To assess the cell protective effects of trehalose supplementation, we analyzed fresh osteochondral graft tissue at various time points after harvest, which were preserved in storage media.

Methods

- 10 New Zealand white rabbits were sacrificed at 6 months of age.
- Tissue was harvested within 2 hours of euthanasia.
- 6 mm osteochondral plugs were harvested from each femoral condyle.
- Plugs were placed in one of two storage mediums.
- Media 1 consisted of standard chondrogenic media (-) trehalose.[7]
- Media 2 consisted of the same components of media 1, but was supplemented with 40 mM (+) trehalose.
- Plugs where analyzed at day 0 (overnight storage) and 14 days post-harvest.
- Tissue was anaylyzed with immunohistochemistry

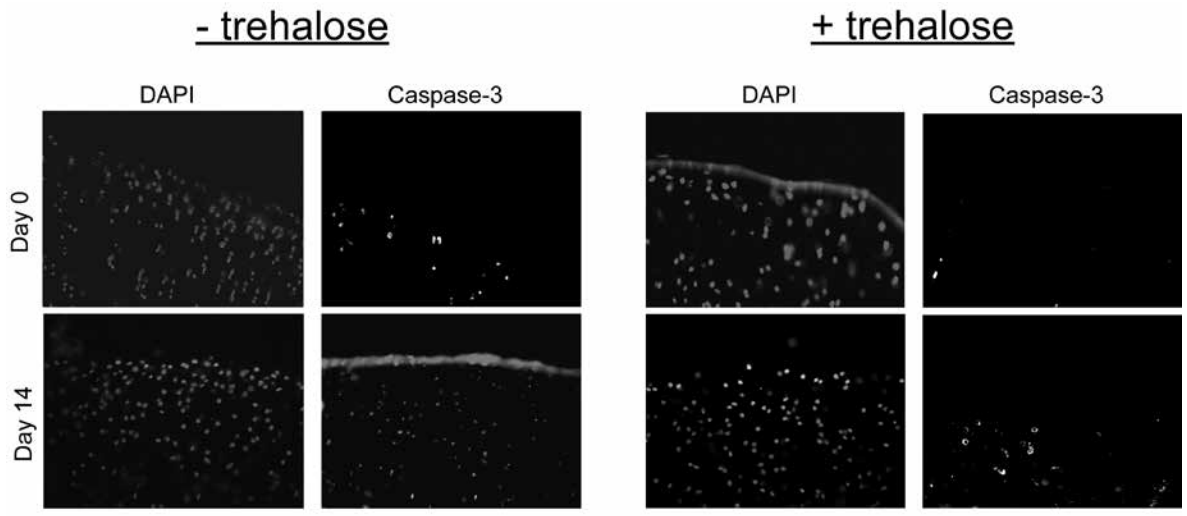


Figure 1: Increase caspase-3 staining in (-) trehalose articular chondrocytes. Figure 1 demonstrates caspase-3 staining in the articular chondrocytes of both (+) trehalose and (-) trehalose stored osteochondral grafts at days 0 and 14.

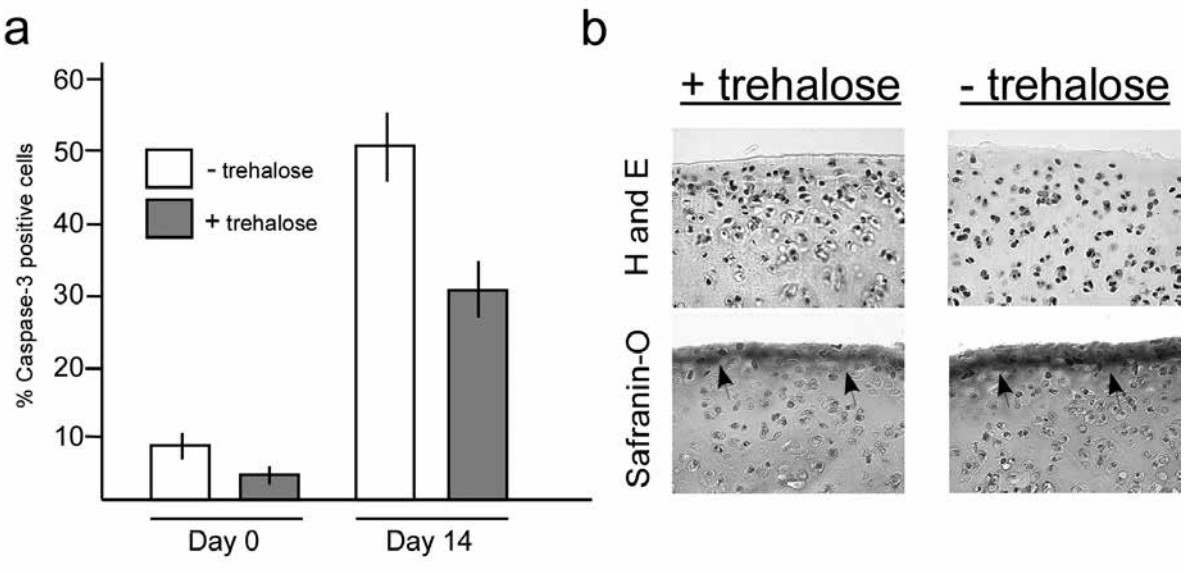


Figure 2: Quanitative analysis of caspase staining and gross morphology of articular tissue. a, demonstrates the percentage of caspase-3 positive cells in both treatment arms at day 0 and 14. b, shows a coronal section of articular cartilage stained both with H and E and as Safranin-O.

for caspase-3 (marker of apoptosis) as well as with hematoxylin and eosin staining (H and E).

- Caspase-3 positive cells were manually counted for analysis.

Results

- Caspase-3 staining was present in the articular tissue in both treatment arms and at both time points tested. There was increased caspase-3 staining in the chondrocytes of osteochondral grafts stored in (-) trehalose media as compared to those stored in (+) trehalose (Figure 1). DAPI staining (for cell nuclei) allows for the localization of the caspase-3 positive cells within the articular tissue.
- At day 0, 8.6% of the chondrocytes in (-) trehalose group stained positive for caspase-3, compared to 4.3% in the (+) trehalose group (Figure 2a) . A 50% decrease in the (+) trehalose group. At day 14, 51.3 % of the (-) trehalose group stained positive for caspase-3, compared to the (+) trehalose group, which had 32.3% of cells stained.
- No major difference in cellular morphology was seen on H and E staining (Figure 2b). Additionally, no major difference was seen with Safranin-O stain for proteoglycans. Both groups had a decrease in superficial zone staining (Figure 2b, black arrows).

Discussion

We demonstrated that chondrocytes in osteochondral grafts stored in media supplemented with 40 mM of trehalose have a decreased number of caspase-3 positive cells (Figure 1). This difference in the number of cells undergoing apoptosis was seen both after overnight storage and after 14 days. Caspase-3 positive cells at day 0 indicate cell death occurs quickly after harvest, 3-8%, subsequently slowly progressing to 32-51% caspase-3 positive cells at day 14.

We also observed no difference in cell morphology of proteoglycan content on histologic analysis. This suggests that the increased cell death seen in the stored graft tissue did not have an effect on the biochemical properties of the articular cartilage.

Long-term osteochondral graft storage has been previously shown to result in decreased tissue viability and *in vivo* performance.[3] The major mechanism of this is believed to be chondrocyte cell death via apoptosis.[5] Our results are consistent with these previous studies, as we showed increased chondrocyte apoptosis as indicated by caspase-3 staining, although we did observe a protective effect of the trehalose. Though caspase-3 is a marker of apoptosis it is more specific than sensitive, not required for all forms of programmed cell death.[8] This suggests that there may be a number of cells going through apoptosis not stained with caspase-3.

This preliminary study only investigated signs of cell death and did not screen for indicators of cell viability. Thus, future studies are needed to investigate changes in cell metabolism and viability. Additionally, osteochondral grafts in the clinical setting are commonly used after longer storage durations than the 14 days, the length of this current study. Further investigation will need to determine whether the beneficial effects of trehalose supplementation are found after 14 days of storage.

Conclusion

In this study, we demonstrated that trehalose supplementation of storage media decreases apoptosis, as measured by caspase-3, in articular chondrocytes. More research is necessary to confirm these findings and investigate the effect of trehalose on cell viability. Future studies will also

investigate the longer-term impact of trehalose storage on biomechanical and biochemical properties of fresh osteochondral grafts.

References

1. Bugbee WD. Fresh osteochondral allografts. J Knee Surg. 2002.15.3:191–5.

2. Pallante AL et al. Chondrocyte viability is higher after prolonged storage at 37 degrees C than at 4 degrees C for osteochondral grafts. Am J Sports Med. 2009.37:24S–32S

3. Malinin T et al. Transplantation of osteochondral allografts after cold storage. J Bone Joint Surg Am. 2006.88:762–770

4. Allen RT et al. Analysis of stored osteochondral allografts at the time of surgical implantation. Am J Sports Med. 2005.33:1479-1484.

5. Kim HT et al. Chondrocyte apoptosis: implications for osteochondral allograft transplantation. Clin Orthop Relat Res. 2008.466:1819–1825.

6. Elbein, A.D et al. New insights on trehalose: A multifunctional molecule. Glycobiology 3003.13:17–27

7. Wise JK et al. Chondrogenic Differentiation of Human Mesenchymal Stem Cells on Oriented Nanofibrous Scaffolds: Engineering the Superficial Zone of Articular Cartilage. Tissue Eng. 2009.15(4):913-921

8. Porter AG et al. Emerging roles of caspase-3 in apoptosis. Cell Death Differ. 1999.6:99–104

9. Kwan MK et al. Histological and biomechanical assessment of articular cartilage from stored osteochondral shell allo- grafts. J Orthop Res. 1989.7:637–644.

Cell Saver Use in Acetabular Surgery: Does Approach Matter?

Reza Firoozabadi, MD, MA, Alan Swenson, BS, and Milton L. Routt, Jr., MD

Open reduction internal fixation of acetabular fractures can lead to large volumes of intraoperative blood loss. Intraoperative autologous transfusion (IAT) by means of Cell Saver (CS) technology is routinely utilized during surgery, but at a monetary cost. The primary aim of this study was to determine if IAT rates and volumes were significantly different between anterior versus posterior approaches to the acetabulum, as well as to ascertain if blood loss was different between the two approaches. One hundred forty-five consecutive acetabular fractures treated either with an anterior or a posterior approach were included in this retrospective single-center cohort study. IAT was used in all cases. Sixty-five fractures were treated through an anterior approach and 80 fractures from a posterior approach. Mean intraoperative blood loss was 786 mL for the anterior approach versus 485 mL for posterior approach (p=0.004). CS blood was returned in 23/65 anterior cases and 6/80 posterior approach cases (p=0.04). Subgroup analysis identified male gender and anterior approach as the only risk factor for elevated blood loss and CS blood return. Potential cost saving measures can be utilized by preferentially using IAT for acetabular fractures that require an anterior approach.

Introduction

Open reduction and internal fixation (ORIF) of acetabular fractures has been associated with significant intraoperative blood losses. Intraoperative autologous transfusion (IAT) by means of Cell Saver (CS) technology is routinely utilized during a variety of surgical procedures, but at an added monetary cost. The primary aim of this study was to determine if IAT rates and volumes were significantly different between anterior and posterior acetabular surgical exposures during ORIF, as well as to ascertain if operative related blood loss was different between the two approaches. This data could potentially aid surgeons determine when CS should be used for acetabular fracture surgery.

Methods

One hundred fifty-five consecutive acetabular treated either with an anterior or a posterior approach were included in this retrospective single-center cohort study. Ten patients were excluded, resulting in 145 fractures (Figure 1). IAT was used in all cases. Demographic data, Injury Severity Score (ISS), American Society of Anesthesiologists Score (ASA), fracture classification, approach used, estimated intraoperative blood loss, CS blood returned, blood products administered during procedure, length of procedure, postoperative blood products within 48 hours and postoperative blood products administered until discharge were assessed. One-way analysis of variance (ANOVA) was performed to determine potential predictors of blood loss and CS utilization in anterior versus posterior approach cases. Fisher exact t-test and group t-test was used for subgroup analysis of cases that had EBL >700 mL and <700 mL in both the anterior and posterior approach groups.

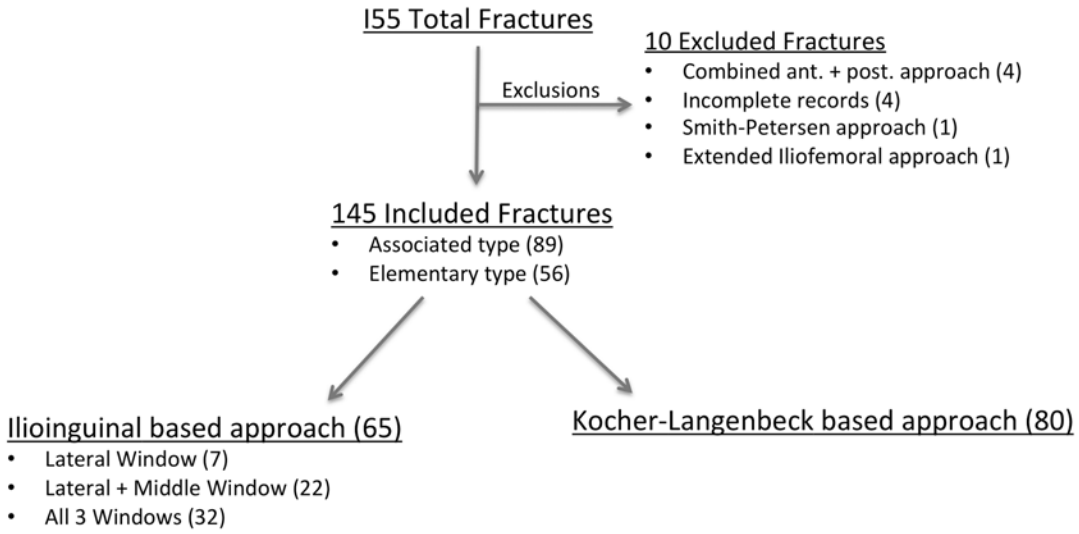


Figure 1: Fracture exclusions and surgical approach breakdown.

	Mean - Both Approaches	Anterior Approach	Posterior Approach
Intra-op. Time (p=0.732)	189 min	191 min	187 min
Mean EBL (p=0.0004)	620 ml	786 ml	485 ml
% cases with CS return (p=0.04)	20%	35% (23)	8% (6)
Mean CS return (p=0.001)	79 ml	141 ml	28 ml
Mean CS return when utilized (p=0.83)	394 (29)	379 ml (23)	398 ml (6)
Intra-op. PRBC (p=0.14)	179 ml	224 ml	143 ml
Intra-op. FFP (p=0.05)	37 ml	66 ml	23 ml
Post op PRBC 48H (p=0.33)	92 ml	116 ml	73 ml
Mean - Total post-op PRBC (p=0.73)	214 ml	230 ml	203 ml

Table 1: Comparison of operative variables between anterior and posterior approaches showing statistical difference between EBL, % cases with CS return, CS return, and Intra-op. FFP. (CS, Cell Saver; PRBC, Packed Red Blood Cell transfusion; FFP Fresh Frozen Plasma.)

Results

There were 107 men and 36 women with a mean age of 47 years old (range 15-95). The mean age in the anterior approach group was 54 years, while it was 42 years in the posterior approach group (p=0.19). The mean ISS was 19.3 (range 9-75), mean for anterior approach was 18.8 and posterior approach was 19.8 which was not statistically significant (p= 0.65). Mean BMI for the group was 28 (range 18-48), 26 for the anterior group and 29 for posterior approach group (p=0.006). Seventeen percent of the patients had an ASA of I, 58% ASA of II, 21% ASA of III, and 4% with an ASA of IV. Of the 145 fractures, 56 were elementary fractures and 89 associated fractures according to the Letournel classification scheme.

Sixty-five (45%) of the fractures were approached through an anterior ilioinguinal exposure and 80 (55%) fractures were treated surgically using a posterior Kocher-Langenbeck exposure. The ilioinguinal approach was further subdivided based on the operative windows developed: lateral window (7 cases), lateral and middle window (22 cases), and all three windows (36). Means and respective p-values are reported in Table 1 for the following measures: incision to closure time, estimated blood loss, percent of cases with CS return, CS return including cases that did not have CS return, CS return with only cases that had CS return, intraoperative PRBC transfusion, post-operative PRBC transfusion for first 48hrs, and post-operative PRBC transfusion until discharge.

Male gender and anterior surgical approach were the only significant predictors of blood loss and CS blood return (p=0.004 and p=0.001, respectively). Subgroup analysis was performed on cases that had EBL >700 mL and <700 mL in both the anterior and posterior approach. The only variable that correlated with EBL over 700 mL was male gender in the anterior approach group (Table 2).

Discussion

Autologous blood for transfusions offers a number of distinguishable advantages over allogenic blood products. Autologous blood eradicates the possibility of disease transmission, is the only source of perfectly compatible blood, does not increase

	Anterior EBL < 700 ml N=41	Anterior EBL > 700 ml N=24	p-value	Posterior EBL < 700 ml N=62	Posterior EBL > 700 ml N=18	p-value
Age	55	52	0.44	43	39	0.30
BMI	27	26	0.40	29.5	27.5	0.27
ISS	17	23	0.10	20.1	18.8	0.67
Days to surgery	3.8	3.4	0.28	3.5	6.3	0.19
Gender	9 female/ 32 male	0 female/ 24 male	0.02	20 female/ 42 male	8 female/ 10 male	0.340

Table 2: Subgroup analysis of cohort for EBL greater than/less than 700ml. Male Gender in the anterior approach group was the only factor that correlated with EBL >700 ml. (Chi-square/fisher's exact for gender calculations – Remaining values calculated via group t-test.)

the risk of wound infections, and eliminates graft versus host reaction risk. Furthermore, compatibility testing and medical identification errors are eradicated. Additionally, autologous blood periodically will be accepted by patients whose religious beliefs do not allow them to receive allogenic blood products.

A recent Cochrane review concluded that the IAT was efficacious in reducing the need for allogenic red cell transfusion in orthopaedic surgery. It should be noted that the majority of studies cited by this review were based on patients that underwent elective spine or arthroplasty procedures. To our knowledge the only other study assessing IAT in orthopaedic trauma patients was performed by Scannell et al. This study assessed if CS use in 60 selected patients with acetabular fractures would lead to reduced volume of allogeneic blood transfusions and if this resulted in a decrease in blood-related charges. The use of CS was at the surgeons own judgment. Two out of three surgeons used CS when they anticipated greater blood loss. They found no reduction in the volume of allogenic blood transfusions with use of CS.

At our institution IAT is used on patients undergoing open operative acetabular fracture repairs. In this series, one hundred forty five consecutive open operative acetabular fracture repairs were assessed. This investigation was specifically designed to determine if IAT rates and volumes were significantly different between anterior as opposed to posterior approaches to the acetabulum. CS blood return was only utilized in 35% of anterior approach cases compared to only 8% of the posterior approach cases. The estimated operative blood losses were also significantly greater in the anterior (786 mL) when compared to the posterior (485 mL) approaches for these open cases. Male gender and anterior approach were the only predictors of blood loss and CS utilization. Mean volume of autotransfused blood for the posterior and anterior cases averaged roughly over 1 unit of PRBCs in cases where CS return was utilized. These results are similar with Scannell's work where they reported an average rate of return of 345 mL. They additionally determined that CS usage led to a significant increase in blood-related charges. Our data suggests that IAT could potentially be more efficacious if used for male patients needing an anterior approach for operative repair of their displaced acetabular fractures, due to the significantly increased blood loss that is noted with the anterior approach.

To conclude, patients with acetabular fractures that require ORIF surgery have significantly increased blood loss when an anterior approach is required compared to a posterior approach. Anterior approach cases utilize CS blood return at a statistically significant higher rate compared to posterior approach cases. Potential cost saving measures can be utilized by preferentially using IAT for acetabular fractures that require an anterior approach.

References

1. Magnussen RA, Tressler MA, Obremskey WT, Kregor PJ. Predicting blood loss in isolated pelvic and acetabular high-energy trauma. Journal of orthopaedic trauma 2007;21:603-7.

2. Scannell BP, Loeffler BJ, Bosse MJ, Kellam JF, Sims SH. Efficacy of intraoperative red blood cell salvage and autotransfusion in the treatment of acetabular fractures. Journal of orthopaedic trauma 2009;23:340-5.

3. Turner RH, Capozzi JD, Kim A, Anas PP, Hardman E. Blood conservation in major orthopedic surgery. Clinical orthopaedics and related research 1990:299-305.

4. Goodnough LT. Risks of blood transfusion. Critical care medicine 2003;31:S678-86.

5. Carless PA, Henry DA, Moxey AJ, O'Connell D, Brown T, Fergusson DA. Cell salvage for minimising perioperative allogeneic blood transfusion. Cochrane Database Syst Rev 2010:CD001888.

Evolutionary Origins of 3-Hydroxyproline In Fibrillar Collagens: Implications for Normal Bone and Tendon Development

David M. Hudson, PhD and David R. Eyre, PhD

Collagen post-translational modifications have evolved to support animal life on land. Approximately half of all proline residues in fibrillar collagen are hydroxylated. The predominant form of the modification, 4-hydroxyproline (4Hyp), plays a functional role in triple helix folding and stability. A rare form of the modification, 3-hydroxyproline (3Hyp), is still without a clear function. We have recently revealed several previously unknown molecular sites of 3Hyp in fibrillar collagen. A series of biophysical analyses were carried out to characterize the physical binding properties of these 3Hyp sites in human bone and tendon. Intermolecular recognition resulting in self-association was observed in tissues with this post-translational modification. This study enhances our understanding of the unique properties of vertebrate skeletal tissues, such as the mineralization of bone collagen, and how these properties are disrupted in musculoskeletal disorders.

Introduction

Bone Development

It seems unlikely that a post-translational modification as rare but conserved as 3Hyp would not contribute basically to collagen structure and function. Perhaps nowhere is this more evident than in forms of recessive osteogenesis imperfecta (OI) in which the prolyl 3-hydroxylation modification is missing. The single 3Hyp residue per chain of type I collagen is formed by an enzyme complex composed of the proteins P3H1, CRTAP and PPIB. Gene mutations that disrupt expression of any protein in this complex have been shown to cause recessive OI [3]. Whether the resulting brittle bone phenotype is caused by the lack of the 3-hydroxyl addition or by another function of the enzyme complex is unknown. We speculate that the most efficient mechanism to explain the chemistry of collagen intermolecular cross-linking is for pairs of collagen molecules in register to be the subunit that assembles into fibrils. We propose that 3Hyp is a requirement for this collagen molecule alignment.

Tendon Development

Using peptide mass spectrometry, we have recently revealed several previously unknown molecular sites of 3Hyp in fibrillar collagen (Figure 1) [1, 2]. In fibril-forming A-clade collagens, these sites have been divided into three

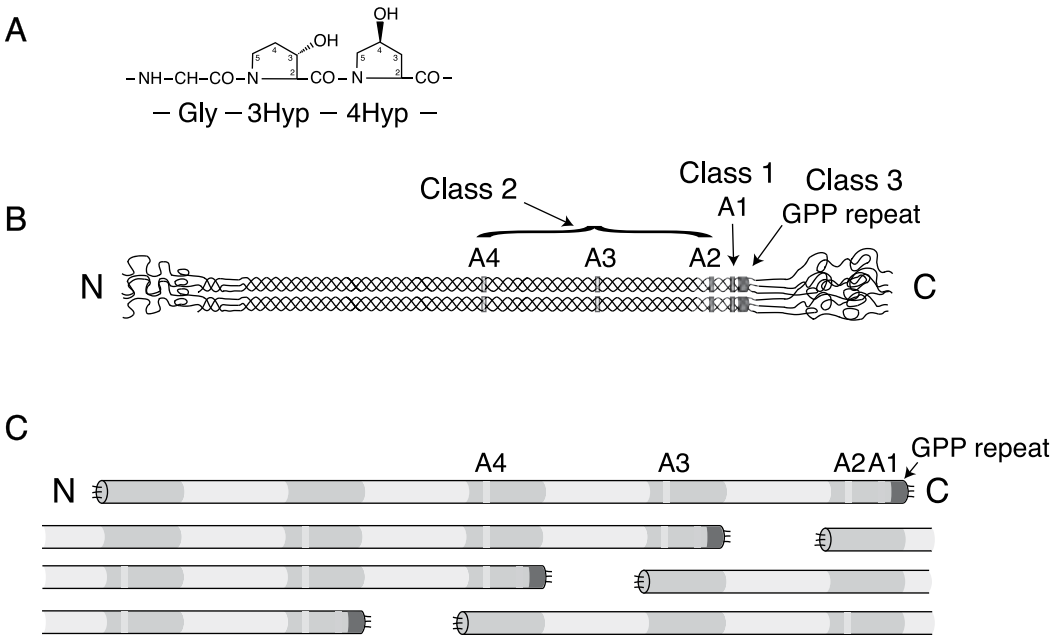


Figure 1: Model of the three classes of 3Hyp substrate sites identified in A-clade collagen. 3Hyp always occurs in the sequence Gly-3Hyp-4Hyp, A. We proposed the Class 1 3Hyp (P986) functions in collagen fibril molecular packing, in which the subunits are in-register dimers staggered axially by D-periods, B. D-periodic spacing is evident between the Class 2 3Hyp residues (P470, P707 and P944) in the triple helix, C. Occurrence of the Class 3 3Hyp residues in the $(GPP)_n$ motif exhibited clear tissue specificity, with the modification occurring almost exclusively in tendon.

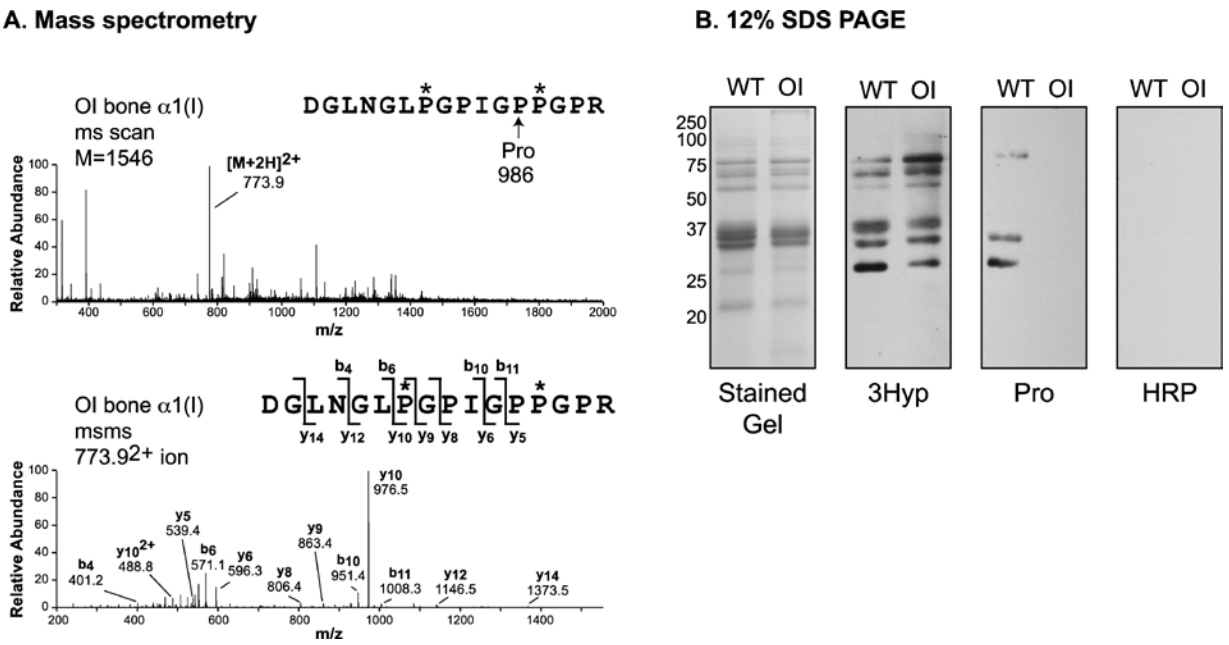


Figure 2: Self-association is not observed in OI bone where Pro986 is not 3-hydroxylated. Full scan spectra from LC-MS profile and MS/MS analysis of in-gel trypsin digests confirm Pro986 is not 3-hydroxylated in OI bone, A. Far western blots of CNBr digested OI and normal (WT) human bone were probed using 3Hyp peptide, Pro peptide, or extravidin-HRP without incubation with a primary probe (HRP), B. These data confirm that intermolecular recognition resulting in self-association is specific to collagen peptides containing 3Hyp at position 986.

classes: the original 3Hyp site at residue Pro986 termed A1 (Class 1), three new sites termed A2, A3 and A4 (Class 2) and the C-terminal $(GPP)_n$ motif (Class 3). To assess the evolutionary origins of the $(GPP)_n$ as a substrate, we surveyed the pattern of 3Hyp occupancy from early chordates through amphibians, birds and mammals.

Hypothesis

We hypothesize that 3Hyp evolved to confer fundamental and tissue-specific properties to collagens. We predict the function of prolyl 3-hydroxylation is in close-range chain registry and molecular binding. For example, 3Hyp residues may site-specifically alter interactions within the triple-helical structure, as well as interactions between triple-helices.

Methodology

Far western analysis

- Biotinylated synthetic peptides (3Hyp and Pro) were designed to mimic the Pro986 domain of collagen α1(I).
- Probe sequence: (α1(I) residues 974-99;KDGLNGLP *GPIGP$^{\#}$P*GPRGRTG). 3Hyp probe: P* is 4Hyp and P$^{\#}$ is 3Hyp; Pro probe: P* is 4Hyp and P$^{\#}$ is Pro.
- Blots were detected with extravidin-conjugated horse radish peroxidase (HRP).

Mass spectrometry

- Collagens were isolated from multiple tissue sources (type I collagen: tendon, bone and skin) and (type II collagen: cartilage and notochord).
- 3Hyp content in collagen α-chains was analyzed by electrospray mass spectrometry using an LTQ XL linear quadrapole ion-trap mass spectrometer equipped with in-line Accela 1250 liquid chromatography automated sample injection.

Results

Bone Development

The physical binding properties of 3Hyp in bone collagen chains were investigated using far western analysis [4]. Evidence of self-association was observed between 3Hyp-containing synthetic peptide and the 3Hyp-containing domain of the α1(I) chain. It is clear that the interaction is highly dependent upon the 3-hydroxylation of Pro986. Indeed, using collagen from a case of severe recessive OI with a CRTAP defect, in which Pro986 was minimally 3-hydroxylated, such binding was not observed (Figure 2). This is the first study to provide direct evidence that the brittle bone phenotype observed in OI could be specifically caused by the lack of the 3-hydroxyl modification, as opposed to an alternative function of the enzyme complex.

Tendon Development

It appeared from our mass spectral analysis of extant animal tissues that the $(GPP)_n$ as a substrate for 3-hydroxylation was peculiar to vertebrate fibrillar collagen. In higher vertebrates (mouse, bovine, human) as many as five 3Hyp residues per $(GPP)_n$ motif were detected, with a mean of about two residues per chain. Notably, in type I collagen from all species tested, the modification exhibited clear tissue specificity, with 3Hyp occurring almost exclusively in tendon. The structural significance is still unclear but the level of 3-hydroxylation seems to have increased as tendons evolved.

Discussion

The current study support a concept that 3Hyp is an ancient and widespread residue in collagen fibril biology that contributes fundamentally to collagen structure and the diversification of

	Tendon		Bone		Skin		Cartilage
Species	**α1(I)**	**α2(I)**	**α1(I)**	**α2(I)**	**α1(I)**	**α2(I)**	**α1(II)**
Human	1.1	2.0	0	0	0	0	0.3
Mouse	1.4	1.8	0	0	0	0	0.5
Bovine	0.2	1.4	0	0	0	0	0.4
Chicken	0.1	1.1	0	0.8	0	0.7	0.1
Xenopus	0.1	0.2	0.1	0.3	0.1	0.2	0.05

Table 1: Mean number of 3Hyp residues per $(GPP)_n$ motif in type I and II collagen α-chains. Molecular locations of 3Hyp were identified using mass spectrometry. 3Hyp content was determined by scrolling the full scan to include all post-translational modification variations.

connective tissues. The occurrence of 3Hyp in the $(GPP)_n$ appears to have arisen with early vertebrates, with equal distribution across type I collagen from bone, skin and tendon. Evolutionarily, 3Hyp may have emerged in $(GPP)_n$ motif to help define type I collagen in tendon, which has several distinctive qualities, including its manner of cellular assembly, cross-linking and material properties. In bone, the interaction between the 3Hyp-containing domains of two individual collagen molecules supports the hypothesis that 3Hyp could be involved in the supramolecular assembly between adjacent collagen triple helices through hydrogen bonds. The formation of in-register dimers through inter-triple helical hydrogen bonds would also ensure the alignment of collagen molecules necessary for the observed placement of mature cross-linking bonds. Post-translational modifications, particularly cross-linking, are believed to be important in modulating the unique tissue-specific properties of type I collagens.

References

1. Weis MA, Hudson DM, Kim L, Scott M, Wu JJ, Eyre DR. Location of 3-hydroxyproline residues in collagen types I, II, III, and V/XI implies a role in fibril supramolecular assembly. The Journal of biological chemistry. 2010;285(4):2580-90.

2. Eyre DR, Weis M, Hudson DM, Wu JJ, Kim L. A novel 3-hydroxyproline (3Hyp)-rich motif marks the triple-helical C terminus of tendon type I collagen. The Journal of biological chemistry. 2011;286(10):7732-6.

3. Cundy T. Recent advances in osteogenesis imperfecta. Calcified tissue international. 2012;90(6):439-49.

4. Hudson DM, Kim LS, Weis M, Cohn DH, Eyre DR. Peptidyl 3-hydroxyproline binding properties of type I collagen suggest a function in fibril supramolecular assembly. Biochemistry. 2012;51(12):2417-24.

Joint Development in the Hand: A Look at the Molecular Mechanisms

Colin J. Teteak, BA, Kevin A. Lawson, BA, Michael J. Chen, BA,
Liu Yang, PhD, and Howard A. Chansky, MD

The patterning and development of interphalangeal joints is regulated by many known and unknown factors that must all be present in order to form a functional joint. In the absence of key regulatory factors joint formation will not proceed correctly and either abnormal joints will form or joints may not form at all. Here we report a novel model for the study of epigenetic regulation of interphalangeal joint development and demonstrate that ESET is necessary for normal joint formation.

Introduction

During the process of interphalangeal joint formation, developing joints must pass through two critical stages. The first of which is the formation of a cell dense region known as the interzone at the site of the future joint. The second stage is cavitation, in which the two opposing sides of the future joint become separated at the interzone.[1] Subsequent steps of morphogenesis then mold the opposing sides of the joint into a reciprocally-shaped and interlocking structure. Increasing evidence suggests that mammalian development is tightly controlled through epigenetic mechanisms. At the present time however, little is known about epigenetic regulation of interphalangeal joint formation. The ERG-associated protein with a SET domain (ESET) is a histone methyltransferase that has been recently shown to be expressed in prehypertrophic chondrocytes and to play a critical role during chondrogenesis.[2] This study examines whether ESET protein is involved in joint formation, whether ESET exerts its effects on joint development through epigenetic regulation of genes critical to joint formation, and whether the precise timing of ESET expression is critical to joint development.

Materials/Methods

- A Cre-LoxP system was used via the Prx1-Cre deleter strain to achieve mesenchymal specific knockout of the ESET gene in mice.
- Whole mount forelimbs and hindlimbs of mice at embryonic day 18.5 (E18.5) were stained with alizarin red for calcified bone and alcian blue for cartilage.
- Fixed sections of embryonic forelimbs of mice at embryonic day 15.5 (E15.5) were stained with hematoxylin-eosin for general morphology.
- Immunohistochemical staining for type-II collagen and Indian Hedgehog (Ihh) protein was performed on E15.5 forelimb tissue sections and imaged on a fluorescence microscope.

Results

The ESET gene contains a C-terminal SET domain that catalyzes methylation of histone H3-lysine 9 residue (H3-K9). ESET also contains an N-terminal tudor domain that mediates interactions with other chromatin enzymes (Figure 1). This model utilizes two distinct knockout lines. The first knockout line contains mesenchymal-specific deletion of exon 4 (exon 4 KO) from the ESET gene which causes a complete deletion of the ESET protein via a frame-shift mutation. The second

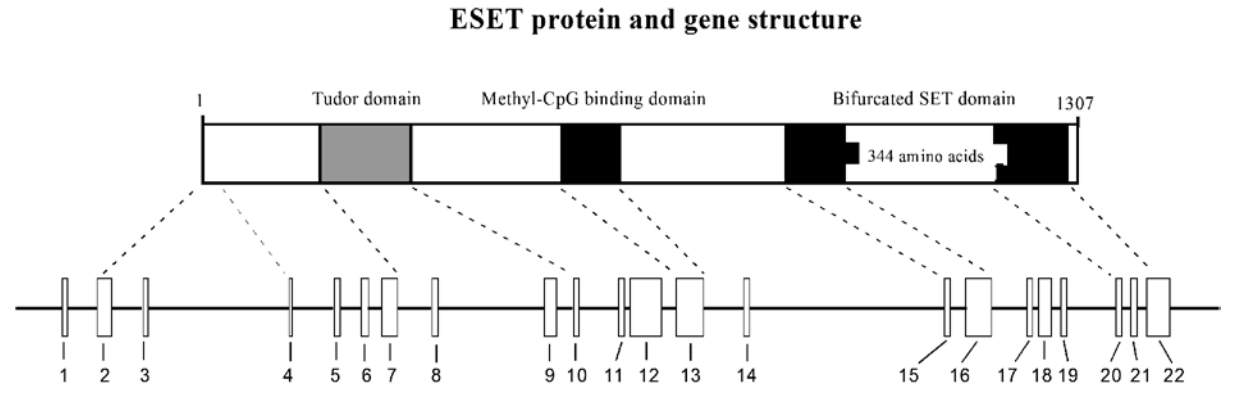


Figure 1: Diagrams of ESET protein and ESET allele. The tudor domain is involved in recruiting other chromatin enzymes, the methyl-CpG binding domain is involved in interaction with methylated DNA, and the SET domain is responsible for methylation of histone H3 at lysine 9. The numbers designate the corresponding exons within the ESET gene.

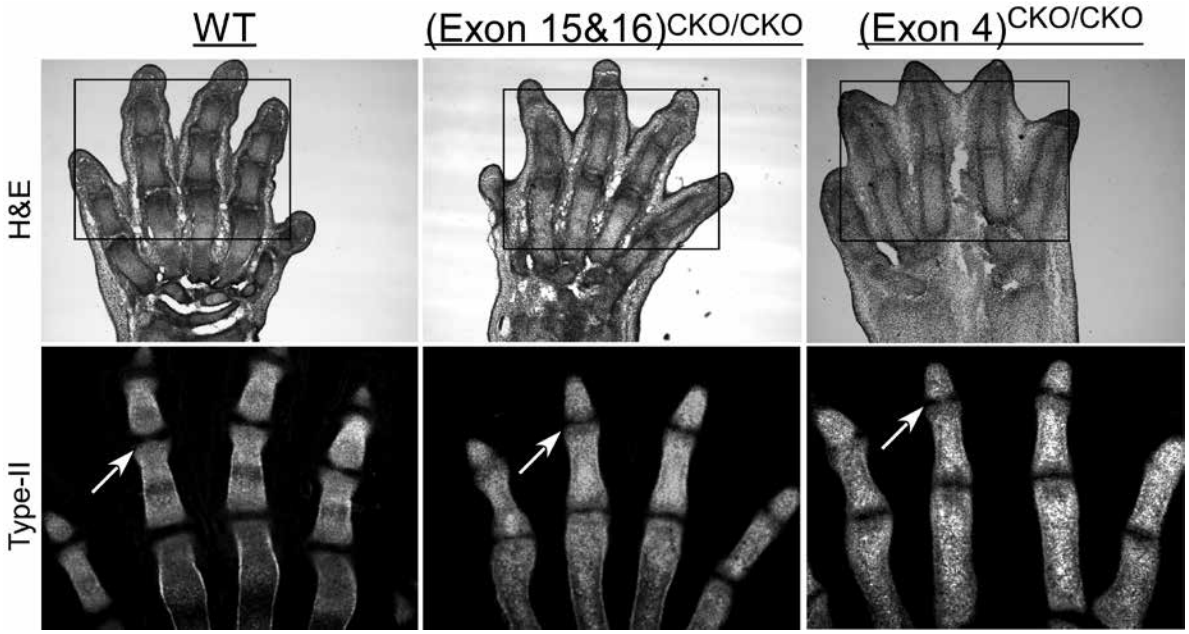


Figure 2: Formation of the interzone in both wild-type and knockout mice. Forelimb sections of E15.5 mice stained with hematoxylin-eosin. Boxed areas on H&E are enlarged below and show staining for type-II collagen. Arrows indicate examples of interzone location.

line contains deletions of exons 15 & 16 (exon 15/16 KO) from the ESET gene which only eliminates the protein's catalytic SET domain while sparing the N-terminal protein-interacting tudor domain (Figure 1).

When stained for type-II cartilage at E15.5, both the wild-type and knockout forelimbs show similar staining patterns (Figure 2) indicating the digits from each group contain interzones.

At E18.5 wild-type digits in the forelimb are separated at each joint site by a clear space and contain calcified tissue at the diaphysis (Figure 3a). In contrast, the exon 15/16 KO shows incomplete cavitation, and digits in the exon 4 knockout are uninterrupted and appear to lack joints (Figure 3a). In the hindlimb, at E18.5, both the wild-type and knockout digits appear to be unaffected. The hindlimbs all contain regularly spaced gaps as marked by the clear spaces indicating they have undergone cavitation (Figure 3b).

Sections of E15.5 forelimbs stained for Ihh show that wild-type digits stain positively in both the interzone and diaphysis (Figure 4). In contrast, exon 15/16 knockout sections show some staining in the diaphysis and very little in the interzone, while exon 4 knockout sections show globally decreased staining.

Discussion

In this study we have demonstrated that the ESET protein is essential to the normal development of interphalangeal joints. Identification of the forming interzone can be accomplished with type-II collagen staining as it should not be present in the presumptive joint region.[3] While we can see that both the wild-type and knockout forelimbs do develop interzones by E15.5 (Figure 2), only the wild-type goes on to fully cavitate at each joint site (Figure 3a). However, it appears that the N-terminal protein-interacting tudor domain of ESET plays a critical role in the cavitation of joints as evidenced by the complete lack of cavitation seen in the exon 4 KO forelimb when compared to the exon 15/16 KO (Figure 3a). Due to the unique properties of the Prx1-Cre deleter strain used in this study, initiation of ESET deletion occurs one day earlier in forelimbs than in hindlimbs (E9.5 vs E10.5).[4] Therefore, the reason the ESET-deficient hindlimbs appear to be unaffected is that the hindlimbs are exposed to the ESET protein for an additional day and that the precise timing of ESET expression is critical to joint formation.

Indian hedgehog (Ihh) protein is a member of the mammalian hedgehog family of proteins that are essential to proper embryonic patterning. Ihh is specifically involved in the differentiation, proliferation and maturation of chondrocytes. It has been previously shown that Ihh expression is necessary for the formation of interphalangeal joints, and that a lack of Ihh can produce a phenotype similar to what is shown in our ESET-null embryos.[1] Staining for Ihh protein demonstrates, for the first time, that the interzone is marked by Ihh protein in wild-type embryos. In ESET-deficient embryos, however, Ihh protein expression at the interzone is significantly decreased (Figure 4).

Conclusions

The entire ESET protein is important to the process of cavitation during development of interphalangeal joints. The precise timing of ESET expression also appears to be critical to interphalangeal joint formation. Our results indicate that ESET's effects on joint development are mediated (at least in part) through regulation of Ihh, a chondrocyte-secreted factor known to be essential for interphalangeal joint formation. These findings may have implications in human joint developmental diseases.

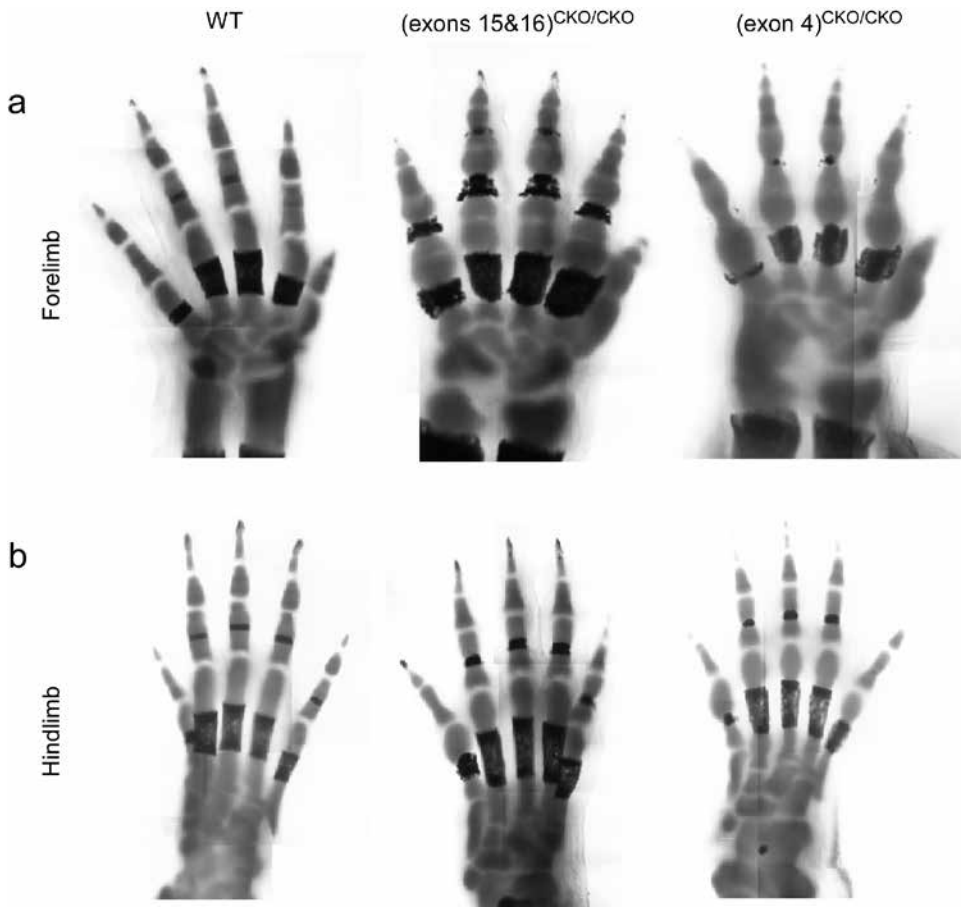


Figure 3: Comparison of whole mount forelimbs (a) and hindlimbs (b) from E18.5 wild-type and ESET-null mouse embryos. Specimens were stained with alcian blue for cartilage (grey) and alizarin red for bone (black).

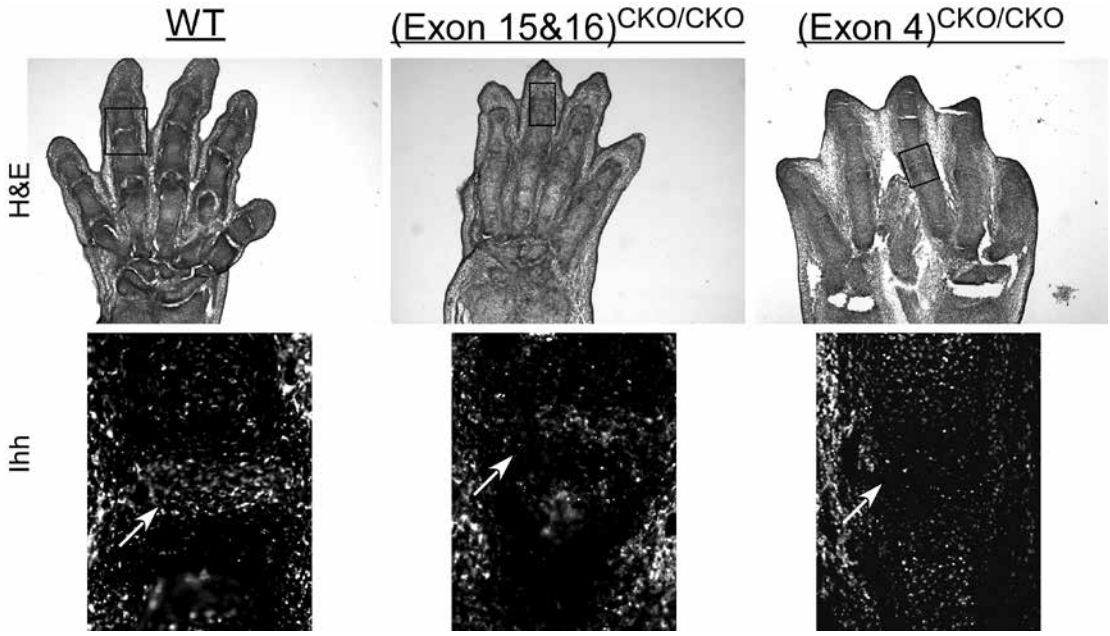


Figure 4: Ihh expression in the developing interzone of wild-type and ESET-null mice. Forelimb sections of E15.5 mice stained with hematoxylin-eosin. Boxed areas on H&E are enlarged below and show a single joint stained for Indian Hedgehog protein. Arrows indicate interzone location.

References

1. Pacifici M, Koyama E, Iwamoto M 2005 Mechanisms of synovial joint and articular cartilage formation: recent advances, but many lingering mysteries. Birth Defects Res C Embryo Today 75(3):237-48.

2. Yang L, Hacquebord J, Zou J, Patterson DP, Ghatan AC, Mei Q, Zielinska-Kiatkowska A, Lawson KA, Chansky HA 2013 ESET histone methyltransferase is essential to the control of chondrocyte hypertrophy and epiphyseal plate Proceedings of the Annual Meeting of Orthopedic Research Society, San Antonio, TX, pp 0277.

3. Craig F.M., Bentley G., Archer C.W. The spatial and temporal pattern of collagens I and II and keratan sulphate in the developing chick metatarsophalangeal joint (1987) Development, 99 (3), pp. 383-391.

4. Logan M, Martin JF, Nagy A, Lobe C, Olson EN, Tabin CJ 2002 Expression of Cre Recombinase in the developing mouse limb bud driven by a Prxl enhancer. Genesis 33(2):77-80.

Entrapped Posteromedial Structures in Tibial Pilon Fractures

Jonathan G. Eastman, MD*, Reza Firoozabadi, MD, MA, David P. Barei, MD, Stephen K. Benirschke, MD, and Robert P. Dunbar, MD

Tibial pilon fractures have historically been subject to high rates of complications, including wound breakdown and deep wound infection.[1,2] These complications have led many orthopaedic surgeons to seek ways to minimize these complications. One way has been to minimize incisions and to treat patients in a somewhat nihilistic fashion.

Tibial pilon fractures have been shown to have consistent fracture lines, usually dividing the distal tibial articular surface into a Chaput anterolateral fragment, a Volkmann posterolateral fragment, a medial malleolar fragment and various amounts of central depressed segments.[3]

The posteromedial structures of the ankle, including the posterior tibial tendon, the flexor digitorum longus tendon, the flexor hallucis longus tendon and the posterior tibial nerve, and posterior tibial artery and vein all course adjacent to the posteromedial portion of the distal tibia. In fact, these structures are slightly tented by the distal tibia and as they course behind the ankle to their respective endpoints in the plantar foot. Published reports have noted how these structures may become displaced and cause both ankle fractures and subtalar dislocations to become irreducible by closed means.[4] We have noted that when the tibial articular surface fragments, these posteromedial structures would sometimes become either trapped in the space created by the fragment (the fracture gap) or be impaled by the sharp bony edges of the fracture. In either case, these structures must be approached in an open fashion, freed from the fracture and returned to their native positions to ensure proper reduction of the fracture and for the tendons, nerve and vascular structures to function properly.

This study seeks to determine the incidence of this problem, by way of a review of the pre-operative CT scans.[5] Additionally, we seek to determine which structures are involved, whether a separate incision was required to address the problem. As well, we looked at how often an entrapped structure was noted on the official radiological report.

Introduction

Our concern is that this problem of entrapped posteromedial structures may have been missed at times in the past. While formal open approaches, either through an anterolateral or anteromedial approach, might allow for addressing this issue, closed management, or percutaneous clamp placement and screw fixation would not.

We have the luxury of a large volume of tibial pilon fractures at our institution. This volume permits us to get an idea of the incidence of this phenomenon of posteromedial ankle structures entrapped in the fracture between the medial edge of the posterolateral Volkmann fragment and the posterior edge of the medial malleolar fragment.

Material / Methods

- Retrospective review of a prospectively collected trauma database, 394 patients with 420 tibial pilon fractures who were treated between Jan 2005 & Nov 2011.
- Each patient's preoperative radiographs and computed tomography (CT) images were reviewed in detail.
- The chart of each patient with an entrapped structure was reviewed to determine:
  - ◊ The presence of a preoperative neurologic deficit
  - ◊ Whether a separate posteromedial incision was utilized
  - ◊ If the final attending radiology interpretation of the CT imaging commented on the interposed structure

Results

- Of 420 tibial pilon fractures, 40 patients with 40 fractures (9.5%) were found to have an entrapped posterior soft tissue structure.
- Of these 40 fractures, 26 fractures (65%) were AO/OTA 43-C3 injuries, 12 fractures (30%) were 43-C2 injuries, and 2 fractures (5%) were 43-C1 injuries.
- The CT scans were taken before any surgical intervention in 6/40 fractures (15%) and were performed after initial spanning external fixation with or without fibular plating in 34/40 fractures (85%).
- The tibialis posterior tendon was interposed in 38/40 fractures (95%).
- The flexor digitorum longus tendons in 9/40 fractures (22%).
- The posterior tibial neurovascular bundle in 4/40 fractures (10%).
- The flexor hallucis longus in 1/40 fractures (2.5%).
- A preoperative neurological deficit with plantar dysesthesia was present in 5/40 patients (12%).
- A separate posteromedial approach was used in 11/40 fractures (27%).
- The final attending radiology interpretation of the CT scan commented on the interposed structure in only 8/40 fractures (20%).

Discussion

- We found that the tibialis posterior tendon was most commonly incarcerated but that the posterior tibial

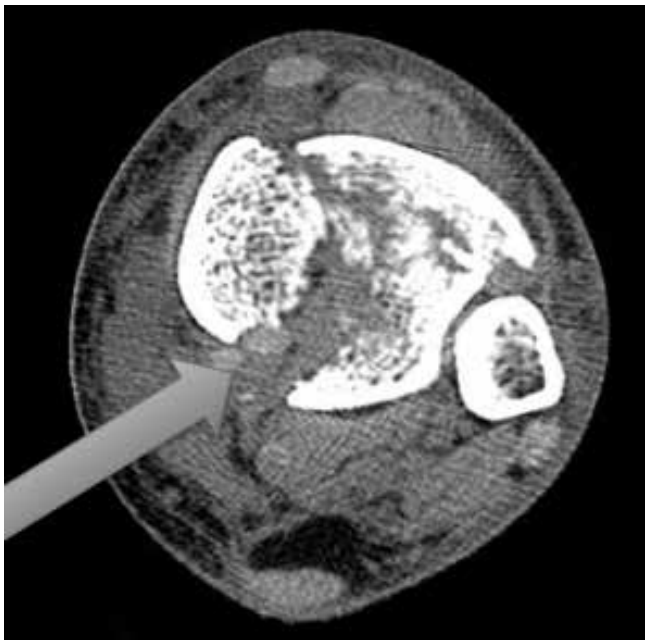


Figure 1: Posteromedial structures (arrow) within displaced tibial pilon fracture.

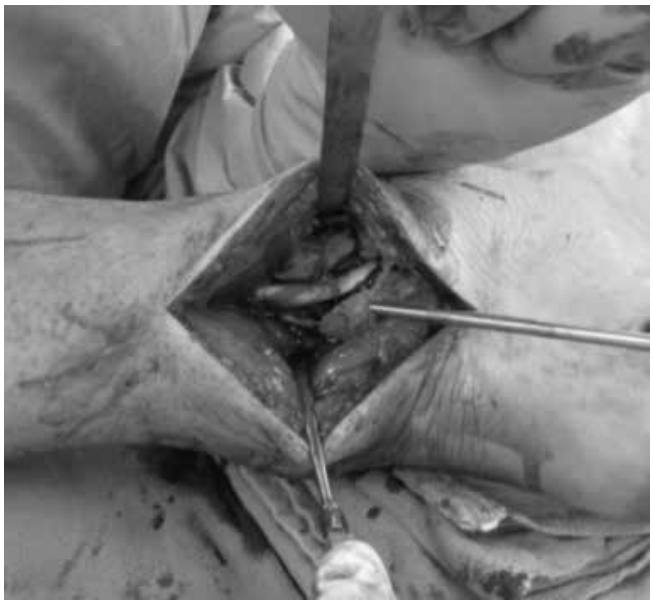


Figure 2: Clinical photo of posterior tibial tendon within fracture, between posterior edge of medial malleolar component and medial edge of Volkmann posterolateral fragment.

neurovascular bundle and long foot flexor tendons were occasionally also involved.

- Entrapment was more common in more complex articular injury patterns, but it could also occur in more simple articular injuries.
- Our study is the first to focus this problem in a large series of pilon fractures. Previous literature focused on dislocated tendons, which caused fractures or subtalar dislocations to be "irreducible", at least by closed methods.
- We found that official radiology reports cannot be relied upon to determine the existence of entrapped posteromedial structures.
- Because of our findings, we are significantly more aware of this phenomenon and are closely examining patients and their imaging studies for signs of this issue. When noted, we are prepared to perform a separate, direct posteromedial approach to address the problem if the entrapped structures cannot first be cleared from the fracture, and then the fracture reduced and clamped, by standard anterolateral or anteromedial approaches.

References

1. McFerran MA, Smith SW, Boulas HJ, Schwartz HS. Complications encountered in the treatment of pilon fractures. J Orthop Trauma. 1992; 6(2):195-200.

2. Pollack AN, McCarthy ML, Bess RS, et al. Outcomes after treatment of high-energy tibial pilon fractures. J Bone Joint Surg Am 2003;85-A(10):1893-900.

3. Topliss CJ, Jackson M, Atkins RM: Anatomy of pilon fractures of the distal tibia. J Bone Joint Surg Br 2005;87(5):692-697.

4. Anderson JG, Hansen ST. Fracture-dislocation of the ankle with posterior tibial tendon entrapment within the tibiofibular interosseous space: a case report of a late diagnosis. Foot Ankle Int. 1996 Feb;17(2):114-8.

5. Tornetta P 3rd, Gorup J. Axial computed tomography of pilon fractures. Clin Orthop. 1996;(323):273-276

*Assistant Professor, University of California at Davis, Davis, CA.

Musculoskeletal Functional Outcomes in Children with Osteogenesis Imperfecta: Associations with Disease Severity and Pamidronate Therapy

Ted Sousa, MD, Viviana Bompadre, PhD, and Klane K. White, MD, MSc

Intravenous pamidronate has been used off-label in the treatment of severe osteogenesis imperfecta for almost twenty years. Previous studies have found correlations between function and bone density in patients with osteogenesis imperfecta (OI), but have not used a validated outcome measure. The goal of this investigation is to describe the functionality and comfort of children with OI. We hypothesize that function is impaired in children with severe OI as measured using the PODCI and that improvements in the function of children with severe OI may be observed in association with intravenous bisphosphonate therapy. A total of 25 patients with OI were evaluated, of those, 15 received pamidronate therapy and evaluated using a validated instrument known as the Pediatric Outcomes Data Collection Instrument (PODCI). There was a statistically significant difference at baseline between patients with "mild" and "severe" OI in the sports/physical functioning scale (p=0.0032). Among the children who received bisphosphonanate therapy, PODCI scores in the sports/physical functioning domain were significantly improved after pamidronate therapy (p=0.0364). This study indicates that children with mild forms of OI can be differentiated from their more severe counterparts by their ability to participate in high-level play activities. Furthermore, patients with severe OI show a significant improvement in their ability to participate in high-level play after one year of IV pamidronate.

Introduction

Osteogenesis Imperfecta (OI) is a genetic disorder of bone fragility resulting from a variety of mutations affecting the structure and function of type I collagen. There is a wide spectrum of disease manifestations from fractures and death in-utero to individuals who have mild-non-deforming disease. Intravenous bisphosphonates have been used, off-label, in the treatment of severe osteogenesis imperfecta for over twenty years. Bisphosphonates in OI have shown a decrease in bone turnover, improved bone mineral density, decreased fracture rates, improved mobility and ambulation, and a substantial reduction of chronic pain and fatigue.[1] Our indications for treatment are the presence of spinal compression fractures or two or more long bone fractures per year. Children at our center are treated with intravenous pamidronate therapy requiring three consecutive days of treatment, every four months for two years.

Studies have shown functional improvements with bisphosphonate therapy but to our knowledge none has used a validated instrument. The Pediatric Outcomes Data Collection Instrument (PODCI) is a validated questionnaire to assess function and health related quality of life in patients with moderate to severe orthopaedic disease between ages 2 and 18 years of age.[2] It has not been used to assess OI.

The aim of the study is to evaluate the function and health-related quality of life in children with OI. Our hypothesis is that function is impaired in children with more severe OI as measured using the PODCI and that improvements in the function of children with severe OI may be observed in association with the administration of intravenous bisphosphonate therapy.

Methods

- Patients prospectively complete the Pediatric Outcomes Data Collection Instrument (PODCI) once a year or before and after major surgical and medical interventions.
- Age, gender, Sillence classification, height, weight, DEXA scores, number of fractures, pamidronate dose,

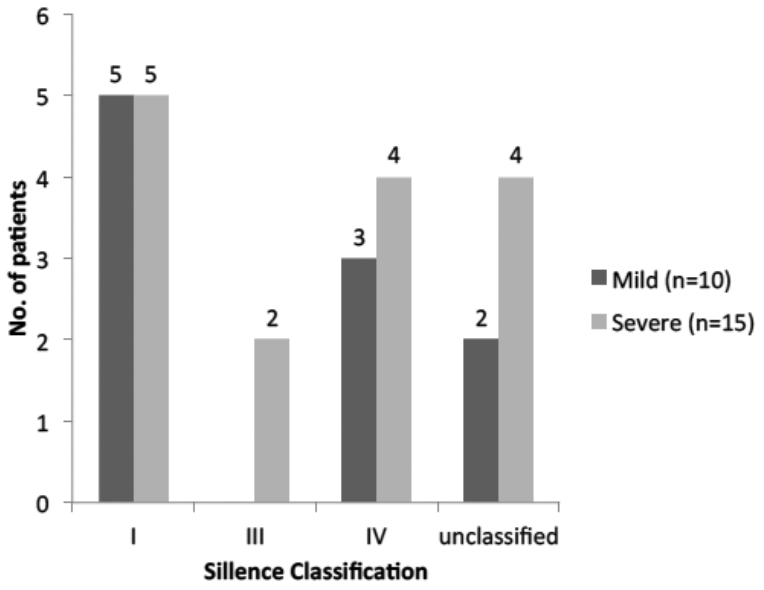


Figure 1: Description of study participants by Sillence classification.

	Mild	Severe
Number of Subjects	10	15
Age (yrs) (average±std)	9.5±5.2	10.2±5.0
Height (cm) (average±std)	106.6±37.5	107.7±40.9
Weight (kg) (average±std)	29.4±20.6	24.0±16
Surgery (no. of patients)	1	9
Gender (M/F)	5/5	9/6

Table 1: Comparison of descriptive data for study participants not receiving pamidronate therapy (Mild OI) with those that received bisphosphonate therapy (Severe OI).

Upper Extremity and Function	Difficulty encountered in performing daily personal care and student activities
Transfer and Basic Mobility	Difficulty experienced in performing routine motion and motor activities in daily activities
Sports/Physical Functioning	Difficulty or limitations encountered in participating in more active activities or sports
Pain/Comfort	Level of pain experienced during the past week
Treatment Expectations	Long term expectations of treatment
Happiness	Overall satisfaction with personal looks and sense of similarity to friends and others of own age
Global Functioning Scale	Combined scale calculated from the first four scales listed above

Table 2: PODCI domain scales

treatment duration, and surgical procedures were all recorded of 25 eligible participants (Table 1).

- 10 patients were classified with "mild disease", (less than 2 fractures per year). These patients did not receive pamidronate therapy.
- 15 patients were classified with "severe disease", (greater than 2 fractures per year), all were offered and received pamidronate therapy.
- The Sillence classification is often used when classifying OI[3] (Figure 1)
- The PODCI measures overall health, pain and ability to participate in normal daily activities, as well as in more vigorous activities associated with young people. It is divided into multiple domains (Table 2).

Results

- The non-therapy group with mild disease was composed of 10 patients (5 females and 5 males. The severe disease group consisted of 15 patients (6 females and 9 males) (Table 1).
- For the PODCI questionnaires, a comparison of baseline values between the mild group and severe group demonstrated a statistically significant difference in the sports/physical function PODCI domain (means of 82.6±19.4 vs 45.1; ±31.8 respectively; p=0.0032) (Table 3).
- Of the 15-member severe cohort, 11 had PODCI data available after pamidronate therapy. They showed a significant improvement in the sports/physical function domain after therapy (p=0.0364; mean score increase from 47.2±29.9 to 57.7±32.1) (Table 4).
- Bone mineral density, whole body DEXA Z-scores were significantly improved before and after treatment (p=0.042) in the severe group, averaging -2.4 pre-therapy and -1.2 post therapy (Table 5).

	Patients with Mild OI (n = 10)					
PODCI Domain	Upper Extremity	Transfer and Mobility	Sports and Physical	Pain Comfort	Happiness	Global
Average STD	80.3 33.8	85.7 30.5	82.6* 19.4	80.1 19.0	86.3 16.6	87.1 8.3
	Patients with Severe OI (n = 15)					
Average STD	60.2 39.3	56.5 42.5	45.1* 31.8	70.1 23.3	85.6 15.8	68.8 24.3

Table 3: PODCI scores for study participants with Mild and Severe OI (averages and standard deviations) *p<0.05

Discussion

This study confirms what previous studies have found: that physical functioning is diminished in children who have more severe disease in comparison to those with milder disease. Furthermore, this study demonstrates functional improvements in association with pamidronate treatment in children with severe OI as shown by the sports/physical function domain. The sports/physical function domain measures difficulty or limitations encountered in participating in higher-level play or sports. This scale looks at participation in recreation, outdoor activities, participation in pickup games and in competitive level sports.

Previous studies on bisphosphonates have demonstrated improvements in mobility. The PEDI is a standardized exam for the evaluation of functional performance in children with a disability from 6 months to 7.5 years of age and has been used to assess functional improvements with bisphosphonates in multiple studies. Löwing et al. in 2007 found score increases within the self-care, mobility, pain, and well-being domains of the PEDI.[4] Forin et al. looked at PEDI scores in 22 children ranging in ages from 2.6-16.1 years and found increases in function.[5] In a 2-year randomized placebo controlled study of 34 Dutch children, there was no significant difference in a generalized functional score or the PEDI score.[6] The limitation of these studies is that the PEDI is not is not validated for patient's whose development exceeds 7.5 years of age. The *median* age of the patients in Forin's study was 7 years, and in the Dutch study, 10 years. Therefore the PEDI is not a validated measure for these populations.

This study also confirms that the Sillence classification does not predict which patients would be candidates to receive bisphosphonate therapy. The Sillence classification system was developed as a clinical description of 154 patients prior to the advent of consistent genotype evaluation.[3] Further modification of the Sillence classification has been rapid in the recent past with subsequent identification of non-type I collagen mutations. This has had the effect of making the classification increasingly cumbersome and less useful for the clinician. We advocate simplifying the classification of osteogenesis imperfecta to severe and mild disease to avoid confusion and help guide therapies.

In conclusion, this study represents the first to quantify functional outcomes in OI after pamidronate therapy using the PODCI. We feel that the PODCI is an appropriate instrument for this patient population, as previously utilized functional questionnaires are not validated for children over age 7. This study demonstrates that significant increases in function after pamidronate therapy occur in the exact domain (sports/physical function) that seems to be reduced in children with more severe disease. We also think that while helpful for understanding the course of the disease, the Sillence classification is not uniformly predictive of which patients with OI will qualify for pamidronate therapy, using the accepted indications of greater than two long bone fractures per year. While we do utilize the Sillence classification when classification is possible, we find that many patients are not easily classified. Consequently we advocate for the simplification of classification for

	Before Treatment					
PODCI Domain	Upper Extremity	Transfer and Mobility	Sports and Physical	Pain Comfort	Happiness	Global
Average STD	71.4 24.7	64.4 31.2	47.2* 29.9	67.5 25.4	83.8 28.6	69.8 29.4
	After Treatment					
Average STD	72.7 29.96	79.2 25.2	57.7* 32.1	72.7 24.6	83.6 28.14	70.1 26.1

Table 4: PODCI scores before and after treatment with IV pamidronate for Severe OI patients (averages and standard deviations; n=11) *p<0.05

	Pre-Therapy	Post-Therapy
Height (cm) (average±std)	107.7±40.9*	125±34.7*
DXA Lumbar Spine Z-score (average±std)	-1.8±1.7	-0.8±1.7
DXA Hip Z-score (average±std)	-2.7±1.5	-1.3±0.8
DXA Distal Femur Z-score (average±std)	-3.1±2.4	-1.6±2.8
DXA Whole Body Z-score (average±std)	-2.4±1.4*	-1.2±1.4*

Table 5: Comparison of height and z-score results from dual beam absorptiometry scans before and after one year of pamidronate therapy. *p<0.05

patients with OI based on the criteria of fracture frequency of greater than or less than two long bone fractures per year, and to simply refer to these patients as “mild” or “severe”.

References

1. Glorieux FH, Bishop NJ, Plotkin H, Chabot G, Lanoue G, Travers R. Cyclic administration of pamidronate in children with severe osteogenesis imperfecta. N. Engl. J. Med. 1998;339(14):947–952. doi:10.1056/NEJM199810013391402.

2. Daltroy LH, Liang MH, Fossel AH, Goldberg MJ. The POSNA pediatric musculoskeletal functional health questionnaire: report on reliability, validity, and sensitivity to change. Pediatric Outcomes Instrument Development Group. Pediatric Orthopaedic Society of North America. J Pediatr Orthop. 1998;18(5):561–571.

3. Sillence DO, Senn A, Danks DM. Genetic heterogeneity in osteogenesis imperfecta. Journal of Medical Genetics. 1979;16(2):101.

4. Löwing K, Aström E, Oscarsson KA, Söderhäll S, Eliasson A-C. Effect of intravenous pamidronate therapy on everyday activities in children with osteogenesis imperfecta. Acta Paediatr. 2007;96(8):1180–1183. doi:10.1111/j.1651-2227.2007.00390.x.

5. Forin V, Arabi A, Guigonis V, Filipe G, Bensman A, Roux C. Benefits of pamidronate in children with osteogenesis imperfecta: an open prospective study. Joint Bone Spine. 2005;72(4):313–318. doi:10.1016/j.jbspin.2004.08.011.

6. Sakkers R, Kok D, Engelbert R, et al. Skeletal effects and functional outcome with olpadronate in children with osteogenesis imperfecta: a 2-year randomised placebo-controlled study. The Lancet. 2004;363(9419):1427–1431. doi:10.1016/S0140-6736(04)16101-1.

A Validated Predictive Model for the Safety of Spine Surgery: SpineSage.com

Michael J. Lee, MD, Amy M. Cizik, MPH, and Jens R. Chapman, MD

Previous reports examining the safety of spine surgery have identified and quantified as relative risks or odds ratios. While these statistical tools are valuable from a research perspective, they carry little practical value when discussing risks of surgery with a patient. Patients do not ask about relative risks or odds ratios; they ask about absolute likelihood of complication. However the likelihood of medical complication depends greatly on the patient's health and the extensiveness of surgery. We have created and validated a model for predicting medical complications after spine surgery using our prospective surgical registry of more than 1700 patients. Because of the complexity of the formula in accounting for patient co-morbidity and surgical invasiveness, we have created a website (SpineSage.com) to facilitate the use of this model. This model and others like it can be beneficial for patient counseling and shared decision-making regarding surgical treatments. In addition, this can be beneficial for risk adjustment in the future as quality metrics continue to gain traction.

Study rationale

- Several risk factors for medical complication after spine surgery have been reported and quantified as Relative Risks (RR) or Odds Ratios (OR)(1-5).
- The value of an RR or OR is evident in academic circles, but is of minimal value to a patient. Furthermore, the translation of OR and RR from physician to patient is challenging.
- When counseling patients on the risks of surgery, the prediction of absolute risk (% likelihood) rather than RR and OR is more valuable to a patient.

Research Purpose

- To Create and Validate a Predictive Model for the safety of spine surgery, specifically in regard to medical complication and surgical site infection.

Materials and Methods

- The Spine End Results Registry 2003-2004 is a high quality prospectively collected registry of all surgical spine patients at University of Washington Medical Center and Harborview Medical Center.
- These data have been analyzed for the risk factors for medical complication and surgical site infection using multivariate log-binomial regression analyses (2-5).
- The population of more than 1700 patients was randomly divided into two populations A&B.
- Beta Coefficients from these analyses were used to create predictive models for 1) any medical complication, 2) Major Medical complication and, 3) Surgical Site Infection. This was done in each group (A&B). Each model was then cross validated in the other group.
- Final predictive models for any medical complication, major medical complication, and surgical site infection were created.

Results

- The area under the Receiver Operator Characteristic Curve was 0.76 for the predictive model for Any Medical complication (Figure 1).
- The area under the Receiver Operator Characteristic Curve was 0.81 for the predictive model for Major Medical complication.
- The area under the Receiver

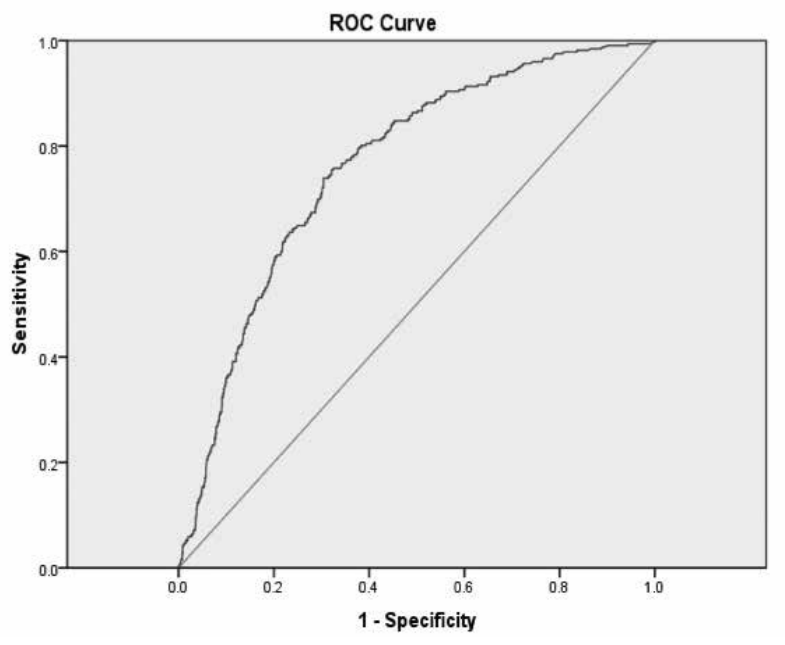


Figure 1: Area under the Receiver Operator Characteristic Curve (ROC Curve) was 0.76 for the predictive model for Any Medical complication. (Note: Diagonal segments are produced by ties.)

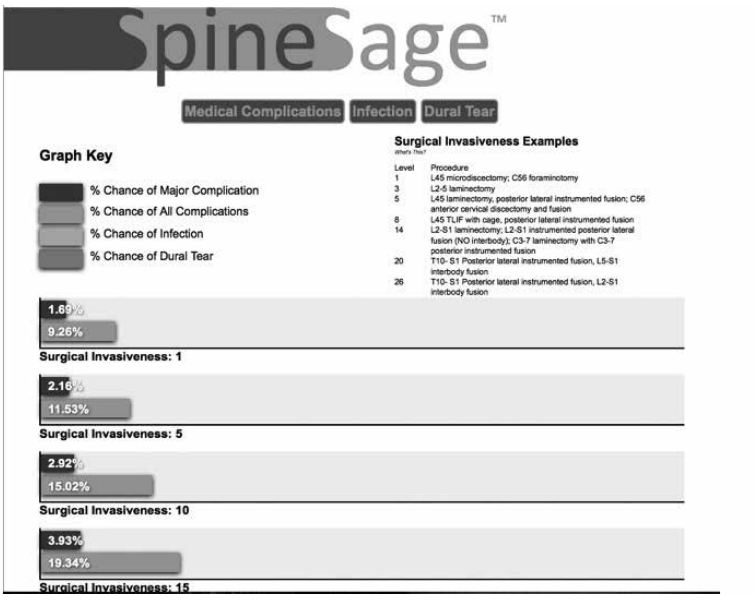


Figure 2: Using SpineSage.com, a 65 year old male with a history of rheumatoid arthritis and diabetes can be evaluated for the risk of spine surgery.

Operator Characteristic Curve was 0.72 for the predictive model for Surgical Site Infection.

- These values represent fair to good assessments of the accuracy of the predictive model. These values are also comparable to the widely utilized and highly regarded Revised Cardiac Risk Index predictive model.
- The results of this formula can be freely accessed at SpineSage.com.

Discussion

- Using SpineSage.com, a 65 year old male with a history of rheumatoid arthritis and diabetes can be evaluated for the risk of spine surgery (Figure 2). Using this tool, this particular patient would have an estimated likelihood of 1.69% for major medical complication and 9.26% for any medical complication after undergoing a surgery with an invasiveness score of 1 (such as a single level laminectomy). One can assess how this particular patient profile's risk changes based on the invasiveness of the surgery proposed.
- Predictive models such as SpineSage.com are important first and foremost for the safety of spine surgery. They can allow optimal pre-operative counseling of patients considering surgical intervention.
- A models such as this is not intended to be a substitute for decision, but an important adjunct for decision making.
- Predictive models such as SpineSage.com are also important in the consideration of risk adjustment. As quality metrics and complications are increasingly scrutinized, predictive models such as these can allow for a standardization of comparison. Currently there is insufficient risk adjustment in the assessment of quality metrics.

References

1. G. A. Baker et al., Risk factors for unintended durotomy during spine surgery: a multivariate analysis. Spine J 12, 121 (Feb, 2012).

2. A. M. Cizik et al., Using the spine surgical invasiveness index to identify risk of surgical site infection: a multivariate analysis. J Bone Joint Surg Am 94, 335 (Feb 15, 2012).

3. M. J. Lee et al., Risk factors for medical complication after lumbar spine surgery: a multivariate analysis of 767 patients. Spine (Phila Pa 1976) 36, 1801 (Oct 1, 2011).

4. M. J. Lee et al., Risk factors for medical complication after spine surgery: a multivariate analysis of 1,591 patients. Spine J 12, 197 (Mar, 2012).

5. M. J. Lee et al., Risk Factors for Medical Complication After Cervical Spine Surgery: A Multivariate Analysis of 582 Patients. Spine (Phila Pa 1976), (Jul 14, 2012).

Differential Expression of Osteoactive Myokines and Muscle-enriched MicroRNAs Following Transient Muscle Paralysis

Leah M. Downey, Ted S. Gross, PhD, and Ronald Y. Kwon, PhD

Our understanding of how muscle and bone interactively cooperate to maintain tissue health has dramatically changed and expanded in the past few years. Understanding the mechanisms underlying this relation holds potential to enable novel treatment of a variety of musculo-skeletal pathologies. This study addresses one aspect of this relation, the potential involvement of muscle-derived circulatory factors in regulating bone cell function following muscle paralysis.

Introduction

Regulation of bone homeostasis by muscle has been widely accepted to occur through the generation and sensation of mechanical forces. However, recent evidence suggests that muscle and bone are also coupled biochemically via muscle-derived circulating factors. More specifically, it has been speculated that myokines (cytokines released by muscle fibers) may regulate bone cell activity (1). In addition, it has been recently demonstrated that microRNAs (short non-coding transcripts, miRNAs) can be released into the extracellular environment and act on remote tissues including bone (2). Given the potential for myokines and circulating miRNAs to function as soluble factors that couple muscle and bone during muscle dysfunction-induced bone loss, in this study we tested the hypothesis that BTxA-induced paralysis alters muscle gene expression of a) myokines with putative bone cell interactions and b) muscle-enriched miRNAs.

Materials and Methods

- 22-week old female C57BL/6J mice were used for this study, and 2U/100g BTxA (Allergan) was administered via intramuscular injection in the calf to transiently inhibit calf function (3; Figure 1).
- RNA was isolated from calf muscles using the mirVana miRNA Isolation kit (Ambion) and fractionated by size. Large RNA fractions were reverse transcribed and analyzed by qRT-PCR. miRNA-enriched small fractions were reverse transcribed using the TaqMan MicroRNA RT kit and analyzed by qRT-PCR using TaqMan MicroRNA Assays (Applied Biosystems). The housekeeping genes for the large and small fractions were b-actin and snU6, respectively.
- Based on a literature review, we constructed a panel of myokines with putative osteotropic activity (2) and three miRNAs enriched in skeletal muscle. The panel of genes was screened for differential alteration 14 days following BTxA administration.
- To select candidate genes for secondary analysis, we computed the strictly standardized mean difference b for each gene and used a selection criterion of b=2, similar to that used in high throughput screening (4).

Results

- Of the eleven candidate genes screened, two (myokine FGF2 and microRNA mir-206) were found to exceed the selection criterion (Figure 2) and were

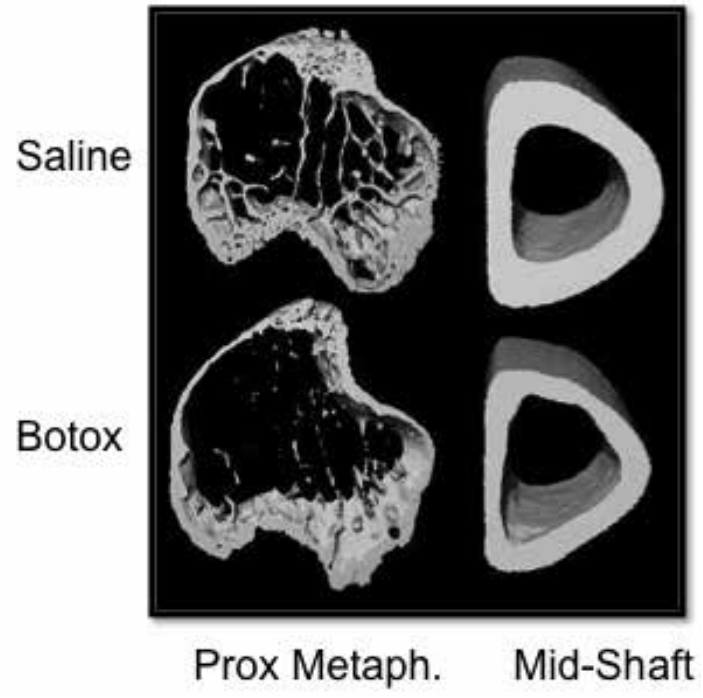


Figure 1: Micro-CT images of the proximal tibia metaphysis and tibia mid-shaft in mice exposed to saline calf injection or BTxA calf injection. The transient muscle paralysis leads to rapid and profound loss of bone trabecular and cortical bone.

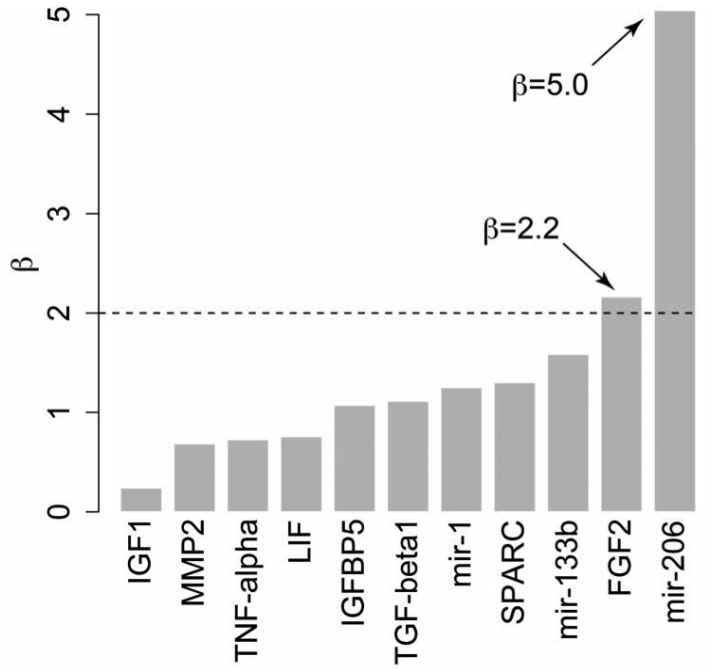


Figure 2: Gene expression screen for altered osteoactive myokines and muscle-enriched miRNAs 14 d following transient muscle paralysis. Only FGF2 and mir-206 exceed the selection criterion of β=2 when compared with saline-injected controls (n=2-3 per group).

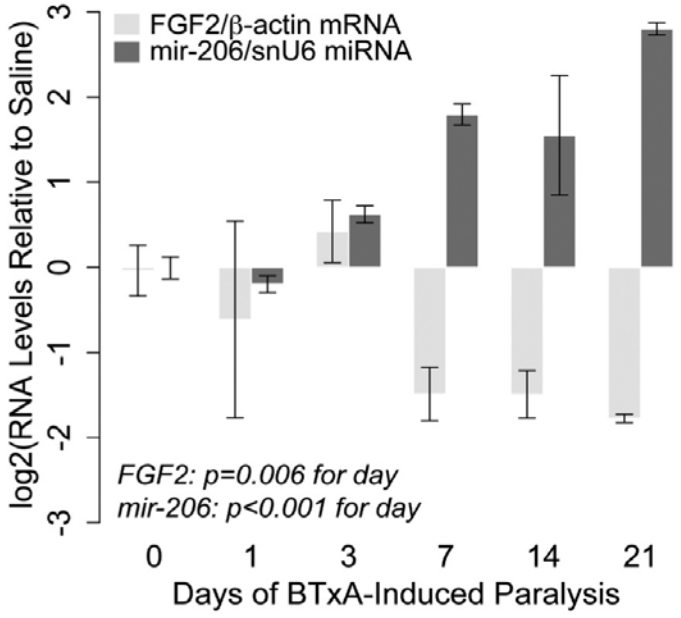


Figure 3: Time course of FGF2 and mir-206 gene expression in muscle following BTxA-induced paralysis. Both genes were substantially altered, as within seven days FGF2 demonstrated *decreased* expression and mir-206 *increased* expression (n=2-3 per group).

subjected to further analysis.

- Time course assessment revealed detectable decreases in FGF2 levels seven days following BTxA administration, with levels decreased by almost 70% by day 21 (p=0.006 for day; Figure 3). For mir-206, increases in expression were detectable seven days following paralysis, reaching five-fold elevation by day 21 (p<0.001 for day).

Discussion

We screened a small but comprehensive panel of two types of soluble factors, osteoactive myokines and muscle-specific microRNAs, for altered gene expression in paralyzed muscle. From this panel, we found that FGF2 and mir-206 demonstrated distinct temporal alterations in expression following muscle paralysis. Both FGF2 and mir-206 have diverse functions outside the skeleton (e.g., limb development, wound healing, and neuronal regeneration). Within the skeleton, FGF2 and mir-206 have potential to affect both osteoclast (FGF2) and osteoblast (FGF2 and mir-206) function (5,6). Given that muscle paralysis-induced bone loss is dominated by focal osteoclastic resorption within the first twelve days post-paralysis (3), followed by a later shift toward osteoblast function in an attempt to compensate for lost bone, the anabolic/catabolic influences of FGF2 and mir-206 in this process must be temporally related to the underlying bone cell response. If so, and if FGF2 and mir-206 are delivered to the bone microenvironment in sufficient concentration to influence bone cell function, we believe that coupling between muscle and bone during muscle dysfunction may be mediated in part by these muscle-derived circulating factors.

References

1. Hamrick, 2011, Exerc Sport Sci Rev, 39: 43-47

2. Itoh et al., 2012, J Mol Hist,

3. Aliprantis et al., 2012, Faseb J, 26:1110-1118

4. Zhang et al., 2007, J Biomol Screen, 12: 497-509

5. Downey et al., 2009, Bone, 44: 266-274

6. Inose et al., 2009, Proc Natl Acad Sci, 106:20794-20799

Acknowledgements

This work was supported by NIH grants AR060304 (TSG) and the Sigvard T. Hansen, Jr. Endowed Chair (TSG).

Platelet Rich Plasma for Flexor Tendon Repair

Katie Kollitz Jegapragasan, BS, Erin M. Parsons, MS, and Jerry I. Huang, MD

Flexor tendon repair remains a clinical challenge due to the balance between tendon healing and scar formation. Autologous Platelet Rich Plasma (PRP) has shown promise in improving tendon healing, notably in rotator cuff and Achilles tendon repairs.[1,2] The purpose of this study was to examine the effect of PRP on flexor tendon healing in a rabbit model. Thirty New Zealand White rabbits underwent complete transection of the 4th deep flexor tendon and immediate repair. Half of the toes were infiltrated with PRP prepared from autologous blood, while remaining toes served as controls. In contrast to previous findings, PRP did not have a significant effect on ultimate tensile strength of the repaired tendons or scar formation, as assessed by range of motion.

Introduction

Zone II flexor tendon repair is a surgical challenge due to the complex anatomy in the region. The tendon glides within a synovial sheath through a series of pulleys. Scar tissue or excess surgical knots can negatively impact a patient's function after repair. Early active and passive motion has been shown to improve healing and reduce adhesions, however, this activity puts the repair at risk for failure.

Given this delicate balance, a number of growth factors have been investigated with the goal of stimulating intrinsic tendon healing and tensile strength while preserving tendon gliding properties and preventing adhesion formation. Platelet-rich Plasma (PRP) contains several cytokines thought to be important in wound healing.[3]

We hypothesize that intraoperative use of PRP in zone II flexor tendon repair can improve tendon healing by improving repair strength and increase range of motion by decreasing scar formation.

Methods

- Thirty New Zealand White rabbits were divided into treatment with PRP (Arthrex, Naples, FL) or control groups. The deep flexor tendon of the 4th toe on the front paw was surgically cut and immediately repaired using a 4 strand technique with 6-0 prolene suture.
- In the treatment group, 0.5mL of PRP prepared from blood drawn intraoperatively was applied to the repair. Blood drawn from the control group rabbits was discarded.
- Rabbits were sacrificed at 2, 4, or 8 weeks postoperatively.
- Tendon glide was assessed with measurements of angular range of motion (ROM) over the metacarpophalangeal (MP) and proximal interphalangeal (PIP) joints as well as tendon excursion from the A1 pulley.
- Tendons were then dissected free and the ultimate tensile strengths were determined using a custom materials testing system. An R2000 hexapod robot (Mikrolar, Boston, MA) was used to pull the tendon apart at a constant velocity of 0.2mm/s. The force was recorded with an in-line LCFD-10 (Omegadyne, Sunbury, OH) load cell, with a reported accuracy of ±0.067 N.
- A one-way analysis of variance was carried out on excursion, ROM and ultimate strength. Categories with $p<0.05$ were further analyzed with Turkey's HSD.

Results

- The ultimate tensile strength (Figure 1) increased significantly in the control and PRP groups between 4 and 8 weeks ($p=0.002$ for each).
- There was no significant difference in ultimate tensile strength between the control and PRP groups at 2 weeks ($p=0.45$), 4 weeks ($p=0.99$), or 8 weeks ($p=0.44$).
- There was a trend towards lower tensile strength at 2 weeks and higher ROM and excursion at 8 weeks in the PRP group, but these were not statistically significant (Figure 1).
- There was no significant difference between time points or treatment groups for excursion ($p=0.32$), MP range of motion ($p=0.31$), PIP range of motion ($p=0.90$), or total range of motion ($p=0.33$, Figure 1).

Discussion

While advances in surgical technique and postoperative rehabilitation protocols have improved outcomes in flexor tendon repair over the past two decades, attention has more recently turned to biologic adjuvants to tendon repair in order to hasten healing, increase strength and minimize scarring.[4] Several growth factors including bFGF, PDGF-BB and BMP have been studied in animal models, however there are so far no effective biologics for use in human flexor tendon surgery. PRP contains many growth factors that make it theoretically appealing for use in tendon healing, both in improving repair strength and minimizing scar formation.

The use of PRP in orthopaedic applications has remained controversial, however. Many studies have been criticized for poor design and results have been difficult to replicate. A recent meta-analysis found that evidence marginally favored beneficial effects of PRP in orthopaedic applications, however the differences were not statistically significant.[3] Differing protocols for production and application of PRP, heterogeneity of study design and small sample sizes in human and animal trials have all been suggested

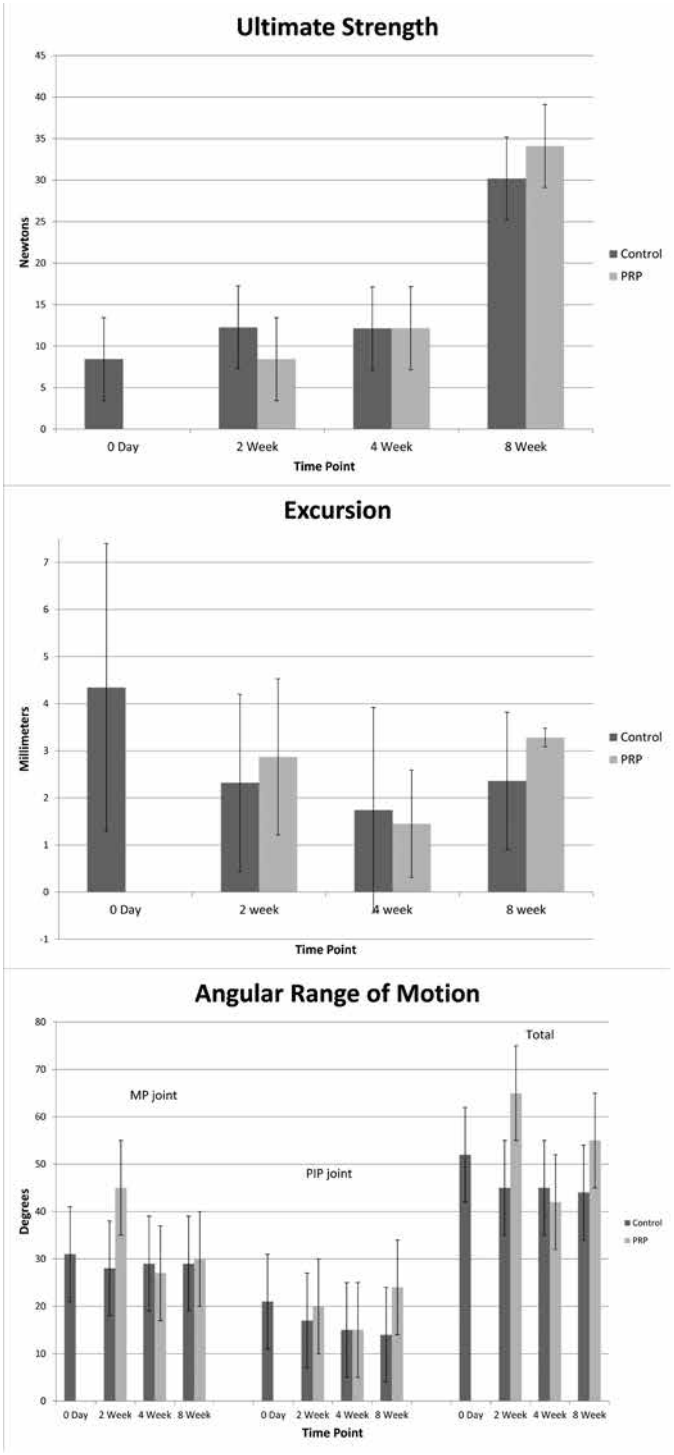


Figure 1: Results of biomechanical testing are above. Ultimate strength increased significantly between 4 weeks and 8 weeks in both control and PRP groups. There were no significant differences between control and PRP groups in ultimate strength, excursion or range of motion.

as explanations for the lack of clear evidence on the efficacy of PRP in the literature.

A recent study of PRP for flexor tendon repair in a rabbit model demonstrated statistically significant increase in tendon tensile strength with addition of PRP-impregnated fibrin-matrix at 2 weeks postoperatively as compared to controls repaired without PRP.[5] In contrast, our study found that PRP did not have a significant effect on the ultimate strength, excursion or ROM in a rabbit flexor tendon model with no difference at 2, 4, or 8 weeks. At 2 weeks, PRP trended towards having a negative effect on tendon healing with an average tensile strength of 8.42N for treatment rabbits versus 12.26N in control rabbits. PRP has been shown to play an active role in regulation of matrix metalloproteinases, which may have an effect on the inflammatory phase of tendon healing.

Our study was powered to detect a difference in ultimate strength of 5N with 80% probability assuming a standard deviation of 3N, as previously described.[5] The variability in ultimate strength was actually much higher, with a pooled standard deviation of 11.25N. A post-hoc power analysis performed assuming our experimental averages and standard deviation found that a sample size of 48 rabbits per group would have been needed to detect an effect at 2 weeks or 4 weeks.

In contrast to published studies on tendon repair, PRP did not seem to enhance intrinsic tendon healing or minimize scar formation in flexor tendon repair. Histologic and immunohistochemical analysis will be performed to further elucidate the effects of PRP on levels of cytokines involved in the tendon healing process.

References

1. Sadoghi P, Rosso C, Valderrabano V, Leithner A, Vavken P. The role of platelets in the treatment of Achilles tendon injuries. J Orthop Res 2013;31:111-8.

2. Gumina S, Campagna V, Ferrazza G, et al. Use of platelet-leukocyte membrane in arthroscopic repair of large rotator cuff tears: a prospective randomized study. J Bone Joint Surg Am 2012;94:1345-52.

3. Sheth U, Simunovic N, Klein G, et al. Efficacy of autologous platelet-rich plasma use for orthopaedic indications: a meta-analysis. J Bone Joint Surg Am 2012;94:298-307.

4. Emerson GB, Warme WJ, Wolf FM, Heckman JD, Brand RA, Leopold SS. Testing for the presence of positive-outcome bias in peer review: a randomized controlled trial. Arch Intern Med 2010;170:1934-9.

5. Sato D, Takahara M, Narita A, et al. Effect of platelet-rich plasma with fibrin matrix on healing of intrasynovial flexor tendons. J Hand Surg Am 2012;37:1356-63.

Case Report: Salvage of a Collapsed Avascular Talus with a Fresh Talar Body Allograft

Michael E. Brage, MD

Treatment of displaced talus fractures remains challenging when the talar dome collapses due to necrosis and/or nonunion. Typically such patients had been treated with a hindfoot fusion with expected resulting permanent functional impairment. Joint function-preserving surgery combining current arthroplasty techniques with traditional reconstruction surgery may offer a chance for improved outcomes.

Background

Avascular necrosis of the talus is a dreaded complication in 30% or more of patients sustaining displaced talar neck fractures (2,3). Despite best attempts at early reduction and rigid anatomic fracture fixation using vascularity preserving approaches a fair number of these fractures fail to heal in desirable fashion. Unfortunately, the only conventional treatment for advanced avascular necrosis of the talar head in combination with or separate from unstable nonunion of the talar neck traditionally has consisted of a pan talar fusion, often in combination with other reconstructive foot procedures. In a young and active population, which is disproportionally frequently affected by talar neck fractures, this frequently leads to functionally undesirable outcomes. In this case report we demonstrate the option of combining an arthroplasty based reconstruction of the tibiotalar joint in conjunction with hindfoot reconstruction to produce a functionally satisfactory ankle joint.

Case Description

This 22-year-old female presented with right ankle pain two years after sustaining multiple injuries in a high-speed car crash. Among many other injuries the patient had been diagnosed with a displaced talar neck fracture and was expediently treated with open reduction and internal fixation. Over time she sadly developed daily activity related pain, which was getting worse with the passage of time. Her quality of life was significantly adversely affected.

Physical Findings

We found an otherwise healthy patient with antalgic gait pattern and swollen diffusely tender ankle and hindfoot without deformity and absent of erythema or draining sinus. She was left with a painful jog of ankle motion of approximately 10-degree excursion in the sagittal plane. We could not elicit any hindfoot motion. The patient identified moderate pain on a daily basis. Her foot and ankle scores showed a very reduced quality of life. The patient had failed in a trial of modified flat roomy shoes and even an ankle brace.

Imaging

Radiographs showed an avascular talar dome with severe collapse and likely nonunion of the fracture status post open reduction, internal fixation with retained implants. This resulted in misalignment of the tibiotalar and subtalar joints and resultant advanced posttraumatic tibio-talar and subtalararthritis (Figure 1).

Results

After extensive counseling of surgical and nonsurgical options the patient requested an ankle joint preserving option and declined a fusion procedure. For joint reconstruction surgery we discussed debridement of her deficient talus and replacement with a fresh

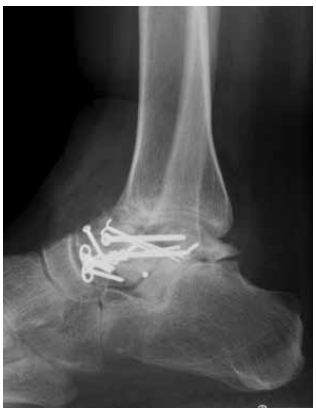


Figure 1: Lateral ankle radiograph demonstrates complete collapse of the talus, nonunion, and displaced hardware.

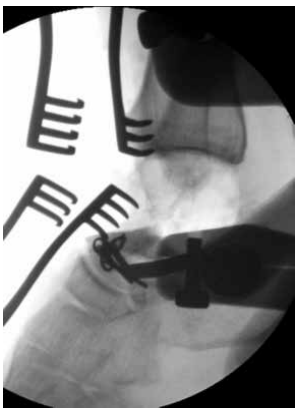


Figure 2: Intra-operative radiograph showing the extent of the talar debridement.

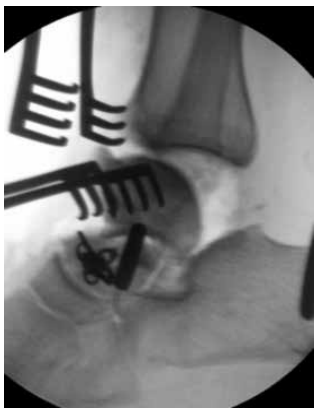


Figure 3: Reduction of the fresh talus graft into the ankle.

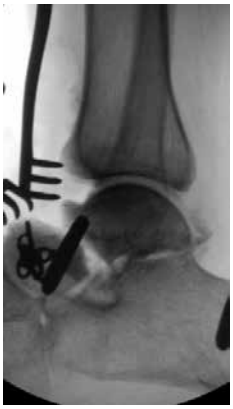
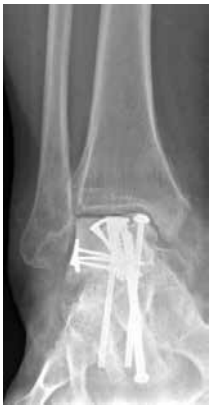


Figure 4: The fresh talus graft successfully reduced.



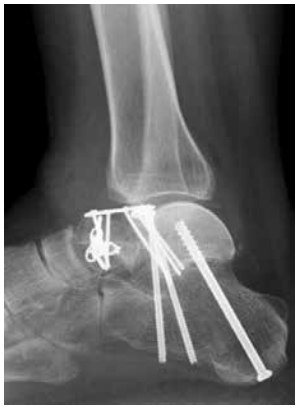


Figure 5: Radiographs at 3 months postoperative that show early union of the host – graft interfaces.

osseocartilaginous allograft. We also discussed total ankle replacement for which this patient was not a candidate due to her youth and because of her talus being completely collapsed and avascular. After discussion of risks and complications under specific discussion that we didn't know of the long-term survival of the graft the patient requested we proceed.

Surgical Technique

The reconstruction procedure involved debridement of the dead talar head and some of the talar neck, along with the entire talar body, which had been found to be devitalized as well (Figure 2). We used an external fixator to distract and realign the ankle. Following this, a matched fresh allograft was shaped to anatomically fill the deficit (Figure 3 and 4). We denuded the cartilage from the calcaneal facet of the patient and removed the cartilage from the posterior facet of the talar allograft in an attempt to achieve a fusion there. We fused the subtalar joint using a screw construct and then used small screws and plates to further stabilize the talar head and neck to the graft.

The patient was kept nonweight bearing for three months, followed by a gradual incremental weight bearing return over the next 2 months (Figure 5).

Outcome

At 16 month follow-up the patient was found to be subjectively quite happy, she reported 1/ 10 pain, and was fully functional gait-wise. Radiographs at one year show the graft to be solidly healed to the subtalar joint and the talar neck. We noted some slight wear and tear and arthritic change of the tibiotalar and medial ankle joints area (Figure 6 and 7).

Discussion

This case exemplifies that an avascular talus can be salvaged with an allograft under appropriate circumstances. We do not anticipate that the allograft will last the patient's lifetime but it hopefully will provide her 5 to 10 years of good quality of life before further surgical intervention will be needed. Should this graft fail, further surgery could be considered with either another allograft, an ankle and/ or extended hindfoot fusion (1), or a prosthetic ankle replacement. Certainly no bridges have been burned by using this technique.

References

1. Faillace JJ, Leopold SS, Brage ME: Exended Hindfoot Fusions and Pantalar Fusions- History, Biomechanics, and Clinical Results: Foot & Ankle Clinics: 5(4); 777-798. 2000.

2. Vallier HA, Nork SE, Benirschke SK, Sangeorzan BJ: Surgical Treatment of Talar Body Fractures. J Bone Joint Surg AM. 2004 Aug; 86A(8): 1616-24.

3. Kopp L, Oruba P, Riegl J, Meluzinova P, Edlemann K: Surgical Management of Talus Fractures: Mid-term functional and Radiographic Outcomes. Acta Chir Orthop Traumatol Cech. 2013; 80(2): 165-70.

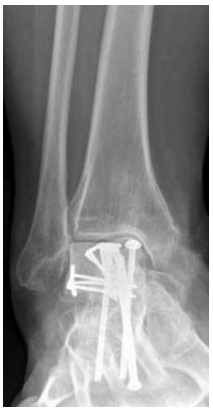


Figure 6: AP ankle radiograph at one year. There is some mild arthritic changes occurring, but the graft is otherwise intact and healed.

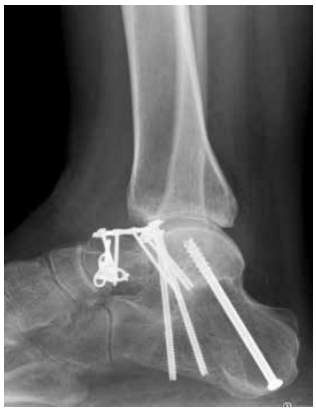


Figure 7: Lateral radiograph at one year. The graft union appears well healed.

Harborview Medical Center Orthopaedics

Teaching

Harborview is the busiest of the four major teaching hospitals in the orthopaedic department at the University of Washington. It is home to five chief residents, two fourth year residents, one third-year resident, and six second-year residents. These residents are primarily assigned to the trauma service but also cover spine, and foot and ankle. In addition there are five trauma fellows, three spine fellows, four hand fellows, and three foot and ankle fellows. First-year residents also spend time on the trauma service. Teaching conferences occur Monday mornings, Monday evenings, Wednesday mornings, Thursday mornings and Friday mornings. Teaching also occurs during clinical care in the outpatient departments and in the operating room.

Clinical Care

Harborview Orthopaedic clinical care is divided into four divisions - two of which include clinical interaction with other departments. The Trauma Division is responsible for fractures and dislocations of the musculoskeletal system and work closely with the general surgery department with the severely injured in the WWAMI region. Harborview is widely regarded as one of the world's best trauma centers and is highly dependent on the interaction of the orthopaedic trauma division with general surgery, neurosurgery, anesthesia and other clinical services. It also provides educational outreach to medical centers in the WWAMI region. Clinical care grew 2% during the last year. We welcomed Reza Firoozabadi, MD as the newest member of the trauma division. Dr. Firoozabadi joined the red trauma team in September 2012 and has quickly become a busy clinician and teacher. The trauma division also said good-bye to Milton "Chip" Routt, MD who returned to his native Texas where he will share his expertise in pelvic and acetabular fracture surgery with a new generation of Texas orthopaedic surgeons.

The Spine division shares its clinical research and teaching skills with surgeons from the Department of Neurological Surgery. Over the years spine care at Harborview has grown into a dominant player in the Pacific Northwest. Spine service provides complex spine instrumentation for trauma, tumors, degenerative and congenital spine deformities as well as more straightforward spine services. It is closely paired with the Department of Rehabilitation Medicine in the Harborview Sports and Spine Center. As in the past the spine division is extremely busy clinically and manages to teach orthopaedic residents, neurosurgical residents, and fellows while contributing to primary research. This division shares service coverage with the University of Washington Medical Center.

The adult Foot and Ankle division also provides clinical care for musculoskeletal disorders of the foot and ankle and provides podiatric services for diabetic foot care and limbs at risk. The foot and ankle division has grown about 9% during the last year and has improved from a three month wait to less than a week long wait for new patient visits.

Research

The Harborview faculty are involved in local regional and national research both in clinical care and basic science. Drs. Ted Gross, Steve Bain and Sundar Srinivasan provide the basic science core based at HMC Research and Training building. The group is funded through the the National Institutes of Health (NIH) and Dr. Bain recently received funding through the Peer Reviewed Orthopaedic Research Program (PRORP) of the Congressionally Directed Medical Research Programs (CDMRP) with Cecilia M. Giachelli, PhD in the department of Bioengineering, and Bruce Sangeorzan, MD, to study engineered osteoclasts in heterotopic bone. In addition the Harborview division continues to participate in the Major Extremity Trauma Research Consortium (METRC) program as a principal site for studies involving long bone fractures with bone loss, contamination of open wounds in high-energy injuries, prevention of surgical site infections, outcomes in severely injured limbs and traumatic amputations and pain management in the postoperative period. Multiple investigators on the trauma division are involved in the studies including Drs. Barei, Beingessner, Dunbar, Henley, Firoozabadi, Nork, Taitsman, Benirschke and Sangeorzan, the site Principal Investigator. Finally, Harborview faculty are running a national trial funded by the NIH comparing Ankle Replacement to Ankle Fusion in adult patients with End Stage Ankle Arthritis.

Bruce J. Sangeorzan, MD
Professor
Harborview Medical Center

Seattle Children's Hospital Orthopaedics

Education

Seattle Children's Hospital (SCH) Pediatric Orthopaedics department provides training and education in Pediatric Orthopaedics for the University of Washington Department of Orthopaedics and Sports Medicine, Madigan Army Medical Center, and Kingsbrook Jewish Hospital of Long Island. Currently four R3 UW residents and one R1 UW resident, and two visiting R3/R4 residents from Madigan and Kingsbrook represent the full-time house staff at Children's. A pediatric orthopaedic fellow assists with resident staff education and supervision. Other residents and fellows attending part-time include orthopaedic oncology, spine and hand. Education is well-defined by the pediatric orthopaedic resident curriculum and the ACGME milestone curriculum, and teaching conferences occur 4 days per week with 6 hours of teaching conferences per week. A comprehensive redesign of Pediatric Orthopaedic M&M conferences has also contributed significantly to resident education, because of a conference structure that emphasizes a literature search for all reported diagnostic topics.

Clinical Programs

All Pediatric Orthopaedic faculty at Children's care for both specialty areas and pediatric trauma. Clinical programs are managed by CPI and a musculoskeletal value stream. Included in this value stream assessment are pediatric spine, pediatric upper extremity, pediatric sports, and skeletal dysplasia, in addition to pediatric tumor, pediatric trauma and pediatric foot. Dedicated value stream work began in 2007 with a focus on standard work eliminating waste and maximizing patient access. Access improved from 35 days to 5-6 days, as a result of that work, with an increase in clinic visits from 15 to 40,000 per year.

The SCH Pediatric Orthopaedics department has been a leader in providing expanded access at all sites of practice that include Laurelhurst, Bellevue, Mill Creek, and Federal Way. Sports coverage for Seattle Public Schools has been provided by the SCH Orthopaedic Sports Medicine athletic trainers for 10 Seattle schools in the Seattle School District, plus Inglemoor, Bellevue Christian, Mercer Island, Bainbridge, Foster, Issaquah, Holy Names, Woodinville, and Marysville Pilchuck. SCH clinical outreach programs have expanded access and coverage in Wenatchee, Anchorage, Tri Cities, Olympia, and a new clinic in Missoula, Montana. Dr. Cammy Mowery continues to provide care and supervision for the Orthopaedic Outreach program.

Seattle Children's Hospital Main Campus

Research and Clinical Pathways

The department continues extensive research efforts with both clinical research programs and clinical pathways at SCH now established for supracondylar fractures, forearm fractures, femur fractures, and scoliosis. Those pathways have implemented AAOS guidelines for pediatric femur fractures and now routinely collect outcomes metrics for review. Dr. Michael Goldberg has pioneered this work, which includes both resident and faculty attestations for each treated patient.

The department has also participated in several national prospective protocols for scoliosis (Dr. Krengel) and club foot bracing (Dr. Mosca), in addition to an international Biomarin study in the evaluation of Mucopolysaccharidoses (Dr. White). Significant other projects include several studies in spine patients (Dr. Krengel), intraoperative navigation for orthopaedic oncology (Dr. Conrad), the re-evaluation of the incidence of pediatric trauma via the pediatric trauma registry (Dr. Dales/Dr. Lindberg), the multiple concussion studies in the high school athletes (Dr. Jinguji), and the effectiveness of post-operative physical therapy (Dr. Schmale). The first clinical pathway data regarding the treatment of supracondylar elbow fractures was presented by Doctors Steinman and Goldberg at this year's POSNA Speciality Meeting. The department also hosted its first Orthopaedic Research Guild fundraising event on June 7, 2013. This new guild, led by Kim Dales and Amy White, is dedicated to fundraising for pediatric orthopaedic research via the SCH Foundation office.

Ernest U. Conrad III, MD
Director of Pediatric Orthopaedics
Seattle Children's Hospital

University of Washington Medical Center Orthopaedics

State of the Union:
University of Washington Medical Center and Northwest Hospital

The University is a busy center for tertiary and quaternary orthopaedic care. We continue to have robust programs in spine, upper extremity, sports, tumor and adult reconstruction. In each of these programs the focus is on complex cases that are best handled in an academic center. In fact, our orthopaedic case mix index, a national measure of complexity of care, has continued to increase. We have one of the highest measures of complexity in the University Health Systems Consortium, a group consisting of the nation's premier academic medical centers. Despite this high case mix index our infection and mortality rates remain lower than expected. These achievements are in no small part due to the hard work of Sue Theiler, the nursing director of 6SE and Michael Lee, MD, the medical director of 6SE and Chief of the UWMC spine service.

University of Washington Medical Center Surgery Pavilion

When adjusted on a per faculty basis, our surgical and clinic volumes have increased while our total volumes are comparable to 2012. Though he has not yet operated at the UWMC, Bruce Twaddle, BHB, FRACS has just joined our faculty to lead our sports program in the new sports medicine clinic at Husky Stadium. Bruce has completed several fellowships including sports medicine and trauma surgery and will be working primarily at the UWMC but will also be helping with the multiligament injuries at HMC. Bruce will be working closely with Carol Teitz, MD, who is also the Dean of Admissions for the UW School of Medicine, to learn the ins and outs of caring for our Husky intercollegiate athletic teams. We have had one new faculty recruit in the past year who has directly contributed to our surgical volumes. We were fortunate to have secured the services of Dr. Albert Gee who did his residency at the University of Pennsylvania and his sports medicine fellowship at The Hospital for Special Surgery. Dr. Gee's surgical expertise, teaching skills and research acumen have made an immediate mark on our program and the care of our athletes. He has boundless energy and enthusiasm and this has translated into making him a departmental and hospital resource. Albert's wife, Jennifer Gardner, MD joined the Dermatology section of the Department of Medicine. Our orthopaedic family recently increased by one as Jenny and Albert had their first child, Benjamin Yoo-Ahn Gee. Rounding out the expansion of our sports medicine faculty, Jason Maris has joined our PA faculty and will be helping Dr. Gee in clinic and in surgery. Jason brings a unique perspective to orthopaedics as he has spent the past 15 years as a family practitioner. John "Trey" Green, MD continues to anchor our sports medicine program with a very busy general sports practice. Nicole Patrick, PAC is a keystone of the sports service and she has also been mentoring Jason Maris as he learns the intricacies of sports medicine. Finally, no report of our sports section could be complete without mention of Dr. Roger Larson whose name has been synonymous with our sports program over the past 30 years.

Darin Davidson, MD, in partnership with Ernest "Chappie" Conrad, MD, has continued to expand our orthopaedic oncology volumes at the UWMC. These increased volumes also result in more collaborative cases with our general and plastic surgery colleagues. This substantial increase in workload

UW Medicine Eastside Specialty Center

UW Medicine established the UW Medicine Cares Award in 2013, a program to formally recognize and celebrate the accomplishments and excellence of those in the UW Medicine community who consistently exemplify the UW Medicine Service Culture Guidelines. This Spring, Winston J. Warme, MD received this prestigious award. He is pictured above with his wife Jeanne.

has resulted in the approval to recruit a second musculoskeletal tumor fellow. For the first time our tumor program will have two tumor fellows shared between the SCCA, CHMC and the UWMC. It also represents the first time we have partnered with the SCCA in recruiting and supporting our tumor fellows. Jenny Hamilton, PAC assists Drs. Davidson and Conrad and she plays a key role in organizing such a complex service.

We have busy outpatient surgical practices at the Roosevelt Bone and Joint Center. Jerry Huang, MD is the Chief of the UWMC Hand Service and works at both the Bone and Joint Center and the Eastside Specialty Center. Dr. Huang has boundless energy and somehow also manages to cover trauma at Harborview. We were fortunate to recently recruit Stephen Kennedy, MD who also covers the UWMC and along with Jason Ko, MD, will focus on building a busy hand practice at Northwest Hospital. Dr. Kennedy also covers hand trauma call at Harborview Medical Center. In the first three months of his practice Dr. Kennedy is off to a running start under the mentorship of Dr. Huang.

Seth Leopold, MD and Paul Manner, MD perform total joints at Northwest Hospital. Alongside his busy clinical practice, Dr. Manner maintains several productive research collaborations. He has funding from the Department of Defense to study the regeneration of articular cartilage and along with Peter R. Cavanagh, PhD, DSc has formed a company to develop and market a unique device to remotely monitor orthopaedic outcomes. Dr. Leopold also had substantial "extra-clinical" activities and has become the Chief Editor of the journal Clinical Orthopaedics and Related Research, a job that has been described as being the equivalent of an orthopaedic Supreme Court Justice with a chance to affect the national practice of orthoapedics for years to come. Tim Coglon, Pete Hall and Connie Ly are the hard working PACs that help care for the total joint and general orthopaedic patients. Connie also works closely with our spine surgeons.

We are very fortunate to have Bahaa Wanly, MHA take over for the legendary Pat Maxwell, RN as the Manager of the Eastside Clinic. Bahaa oversaw a remarkable transformation of the Vascular Surgery clinic at HMC and we anticipate great things from him at the current and future expanded ESC. Claudia Happe, RN, BSN brings years of experience to managing the Sports Medicine Clinic and we are very excited that Claudia has been named the Nurse Manager of the future Sports Medicine Clinic at Husky Stadium. In turn we also have new clinic manager for the Bone and Joint Center. Jim Arteaga, MBA will assume the title of UWMC Health Services Manager of Bone and Joint/Sports and Spine at our Roosevelt Clinic. Jim brings a wealth of experience and has already brought new insights and energy to our clinic.

Drs. Rick Matsen and Winston Warme constitute our shoulder and elbow service with the assistance of Alex Bertelsen, PAC and Jill Eggers-Knight, PAC. In addition to maintaining his busy referral-based shoulder practice, Dr. Matsen has spearheaded the practice of blogging about clinical topics on our departmental website. His blog generates many "hits" and as much discussion. Dr. Warme has a busy general shoulder surgery practice and was a recent recipient of a prestigious UW Medicine Cares Award. Sarah Jackins, PT continues to lead our Exercise Training Center collaborates closely with the Shoulder & Elbow Service. The entire department was proud of Sarah when she was named a recipient of a 2013 UW Distinguished Staff Award. Each year this honor is awarded to only a handful of UW staff, making it one of the UW's highest forms of recognition.

Our Spine Service remains in the capable hands of Michael Lee, MD, Jens Chapman, MD, Ted Wagner, MD, EChing Voon Bertelsen, PAC and Connie Ly, PAC. Dr. Lee was recently promoted to Associate Professor, a decision made very easy by his prolific output of practice-changing clinical papers, excellence in clinical care and teaching, and his accelerating national reputation.

There are a wide variety of research interests at the University of Washington

Sarah Jackins, PT. Winner of a 2013 University of Washington Distinguished Staff Award. Allow us to quote Frederick A. Matsen III, MD from his nominating letter: "There are many high-profile candidates, I am sure, for the University of Washington Distinguished Staff Award. By contrast, Sarah is truly an unsung heroine, with a 36-year record of daily contributions to the missions of the University of Washington and UW Medicine. All she does is accomplished with the greatest possible humility. The most common words from Ms. Jackins' mouth are "how can I help?" She is a role model for all who see her in action, making us wish we could be more like her. Having been active at the UW since 1971, I have met no one as deserving of the University of Washington Distinguished Staff Award as Sarah Jackins, PT."

Medical Center and these are strongly reflected in the publication of this year's research report. The Orthopaedic Robotics Laboratory is under the directorship of Peter R. Cavanagh, PhD, DSc who is also our Vice Chair of Research. This laboratory is the first of its kind in our department and the lab has become a hub of activity as several faculty members and residents have started collaborative projects with Dr. Cavanagh. Clinical projects under the tutelage of Dr. Cavanagh include fixation of wrist fractures, ACL injuries and tibial anatomy, remote monitoring of total join clinical outcomes and the biomechanics of total knee replacement.

Under our Chairman, Jens Chapman, MD, we are continuing to recruit additional surgeons to the University of Washington Medical Center and Northwest Hospital. Patty O'Leary-Crutcher in her role as the Service Line leader for Sports and Spine and Musculoskeletal Medicine has been instrumental in our recruitment of new physician and PA faculty. She has also helped integrate these new faculty into their respective sites of practice. As the practices of our newer faculty mature, we anticipate a further increase in the number of inpatient procedures, as well as outpatient clinic visits. We hope to further increase access to our clinics in keeping with the principles of the UW "Patients First Initiative". At this time next year we anticipate being able to report new and exciting developments including the further expansion of our surgical faculty and the opening of our newest facility – the Sports Medicine Clinic at Husky Stadium.

Howard A. Chansky, MD
Professor, Vice Chair and Chief

VA Puget Sound Orthopaedics

State of the Union:
The Puget Sound Veteran's Administration Medical Center

The Puget Sound Veteran's Administration Medical Center (VA) is a popular rotation for the orthopaedic residents. We have a general tertiary care orthopaedic practice at the VA and we see patients from the entire northwest including Alaska as well as states outside of the Northwest. We see a wide variety of both general orthopaedics, as well as complex or revision orthopaedic problems. Despite changes in VA national and local policy that has led to decreased OR efficiency and block time, we remain one of the busiest VA orthopaedic programs in the country.

Our University of Washington residents continue to rate the VA rotation as one of their favorites. While closely supervised, they are given graduated responsibility throughout their training at the VA. Our goal at the VA is to train residents that are dedicated to caring for patients with potentially difficult socioeconomic issues as well as residents who, if they so choose, would be able to graduate from our residency and achieve a high level of expertise in a general orthopaedic practice.

Dr. Albert Gee is our newest faculty member to join the orthopaedic staff at the VA. Dr. Gee has an infectious enthusiasm for clinical orthopaedics as well as for orthopaedic research. Having trained on the East Coast at the University of Pennsylvania and The Hospital for Special Surgery, the veterans and we have also benefitted from Albert's unique perspective on orthopaedic problems. He has quickly become a favorite of the residents and it would be fair to say that when not focusing on the work at hand, he keeps Dr. Chansky entertained with his humor and unique turn of a phrase.

Partnering with Dr. Gee in running the Thursday clinics, Ted Greenlee, MD continues to lend us his invaluable experience, particularly his knowledge of the natural history of conservatively treated fractures and his great sense for what is best for the patient and when not to operate. These skills are becoming increasingly rare as orthopaedic surgery and training continue to evolve in a more technical direction.

VA Puget Sound Health Care System

Cindy Lostoski provides our administrative support and has many roles in assisting patients as well as the physicians. Our physician's assistants Steve Casowitz and Dustin Higbee are the backbone of our surgical service. Nobody is more pleased than the Ortho residents and patients that we now have an ARNP, Michael Ramos, to help cover the inpatient orthopaedic floor. Michael also helps out in clinic and we are all glad that he joined us. Amy Katzenmeyer, ARNP has been a great addition to our service; she is a quick study and we have all appreciated her skills and patience in dealing with complex patients. Monette Manio, RN and Katherine German, RN manage all of our surgical scheduling and with a large population of very ill patients of limited means for travel, this can be extremely challenging. They have perhaps the most difficult jobs in the entire VA! Annette Testa, LPN, assists in our outpatient clinics and is an accomplished casting technician. Fred Huang, MD, a former UW resident has a busy practice at Valley Hospital but still finds time to maintain a presence at the VA. At the VA, Dr. Huang focuses on sports problems while also assisting with general orthopaedics. The orthopaedic service at the Puget Sound VA could not function without Anne Dinsmore, RN. Anne is the head orthopaedic nurse and helps the attendings and residents navigate the elaborate system of rules governing equipment procurement, setting up for cases and sterile processing. Our trusted scrub technicians and friends, Leo Cruz, Adrian Sisson and Amy Arce round out our surgical service.

There is an active orthopaedic research program at the VA with extramurally funded programs for Drs. Chansky and Yang in sarcoma and cartilage biology as well as a state-of-the-art orthopaedic biomechanics laboratory run by Bruce Sangeorzan, MD who is assisted by William Ledoux, PhD. Orthopaedic residents participate in research in both laboratories and in fact one of our residents, Jacques Hacquebord, was just awarded a grant by AO North America to study fracture healing with Dr. Yang and Dr. Chansky. Dr. Gee has also established collaborations with both laboratories and we anticipate that great things will result from these collaborations.

The Puget Sound VA Medical Center itself is undergoing a major expansion with the addition of a new research building and starting this fall the addition of above and below ground parking facilities. All of this should improve the quality and quantity of care for our veterans, their families and the quality of the work environment.

Howard A. Chansky, MD
Professor, Vice Chair and Chief

A Brief Overview of the Educational Mission of UW Orthopaedics

Jens R. Chapman, MD

Of course it is virtually impossible to do the educational efforts of the members of UW Orthopaedics justice through a simple update. It is safe to say that some form of teaching activity permeates virtually all facets of our departmental work lives and all members of our department either directly or indirectly support our educational mission.

With this article I wanted to highlight some of our current educational activities in this year's Discoveries 2013 edition and celebrate the spirit of education as well as innovation so proudly carried out by members of our department. This review will hopefully also be of interest to those not familiar with the everyday activities of our UW Orthopaedics colleagues as the amount of ongoing education may be underappreciated and while it remains a privilege to teach, it is also a largely 'unfunded mandate'.

Teaching is one of the core elements in the UW Medicine mission to improve the health of the public, as well as one of the clearly apparent foundational values for all members of our department regardless of level or job category. It remains a source of considerable inspiration and gratification for those who provide it, as it hopefully benefits its recipients, such as members of the public, health care administrators, or the students, residents, fellows and fellow surgical colleagues and other care providers engaged in musculoskeletal care. It is the thought that through our educational efforts we can help to improve the quality of care and make things better in our field of musculoskeletal medicine in our present and for future generations, which drives our UW Orthopaedics colleagues to excel in this domain. I hope you find this brief overview compelling and interesting as well.

(Left to right) Drs. Paul Manner, Peter Cavanagh, Patrick Shelby, and Rachael Tanner lecturing at the May 1, 2013 Grand Rounds on the topic of commercialization resources.

Teaching Resources

Over the years we have expanded our delivery modalities away from relying on the formal auditorium and printed materials towards also offering web-based and televised content and providing interactive engagement opportunities to enhance orthopaedic learning opportunities. Our target groups have expanded as well – extending beyond the traditional focus groups of students, residents and fellows to increasingly include patients, members of allied health specialties and fellow surgical colleagues.

In order to accomplish this goal we now routinely use technology to broadcast our high value formal educational sessions, such as Grand Rounds and conferences to the public (www.orthop.washington.edu/?q=patient-care/videos-for-patients.html). Beyond our own website, our informational materials can be accessed through a wide variety of tools and repositories, such as UWTV.org, YouTube and many search engines and social media. More recently some of our service lines have also added blogs to enable interested patients to better engage on an informal basis with some of our experts. An exceedingly popular example is the blog of Dr. Matsen in shoulder disorders (shoulderarthritis.blogspot.com). How popular are these access points? As of May 2013 the Shoulder Arthritis blog has averaged over 12,000 unique visitors per month with a total of over 218,000 views as of this publication. Judging from the targeted pages two-thirds or more of hits on our pages come from potential health care consumers.

In the content management and

Resident education is undergoing a transformation. Unprecedented knowledge expectations have to be paired with demonstrable skills and judgment capabilities.

delivery sphere our computer support group headed by Arien Cherones deserves tremendous kudos for having enabled these internet-based changes in a compliant and secure, yet dynamic as well as interesting fashion. With our increasing technological capabilities we have also decided to enhance the way in which we provide educational materials to our residents. Where in the past our residents were weighted down by a few heavy textbooks and tree loads full of photocopies largely provided by our book fund, we have now been able to execute a big leap forward by switching to an all electronic knowledge delivery format through the close collaboration of a number of dedicated members of our department.

Funded through the generosity of our Alumni and its official fundraising entity Friends of Orthopaedic Research and Education (FORE), we are now proud to be one of the very first residencies in North America that provides iPad tablets filled with a cornucopia of all imaginable major orthopaedic educational resources to all of our fabulous residents. Not only has this step afforded us with the potential for significant cost savings (with all materials properly purchased and licensed), this step is also increasing efficiencies for our residents as these devices are configured to serve as mobile access points for their daily electronic medical record interactions. Moving out of the clumsy book age into the efficiencies and expansion capabilities afforded by the information age has been a welcome boost for our residents in many ways, but perhaps most importantly it has been a great source of pride for all involved as we can consider ourselves on the forefront of redefining integrated orthopaedic learning.

Several individuals deserve tremendous credit for their efforts in turning this dream into reality: aside from our Departmental IT group headed by Arien Cherones, our Program Director Doug Hanel deserves our gratitude for his vision and project organization; our Alumni chair Lyle Sorensen was instrumental in procuring the needed funding through our generous Alumni organization while working closely with our Director of Financial Affairs Karl Engdahl; our residents Dayne Mickelsen and Kenneth Gundle who followed the pioneering ideas of the orthopaedic residency of the University of Hawaii in assembling content and structure. I invite you to read more about this major advancement for our residents and its potential for positive impact by looking through the pertinent articles, which are after this introduction.

Teaching Formats

Sometimes the most successful teaching formats are not provided in regular formal course settings encased by 'goals and objectives' curricular goals, but emerge out of the dedication of specially engaged individuals who intend to meet a perceived need on behalf of underserved recipients. An example of such effective grass-roots teaching can be seen in the efforts of our residents by enhancing musculoskeletal education for our UW medical students through informal teaching and education sessions through Orthopaedic Surgery and Sports Medicine Interest Group

UW Orthopaedics Monthly Conference Schedule

Monday	Tuesday	Wednesday	Thursday	Friday
Every Hand Fellows Journal Club 6:30 - 7:30 AM HMC 6NJB Conference Room HMC All Residents Conference 7:00 - 9:50 AM HMC NJB ISIS Lab (3rd floor) ACEs Meeting 7:30 - 8:30 AM HMC MB606 VA All Residents Conference 7:30 - 8:30 AM VA Cavanagh Research Group Meeting 10:00 - 11:00 AM UWMC BB1065 HMC Trauma X-Ray Conference (for Residents/ACEs/Faculty) 4:00 - 6:00 PM HMC R&T 117 HMC Foot and Ankle X-Ray Conference (for Residents/ACEs/Faculty) 5:00 - 6:00 PM HMC R&T 117	**Every** Hand Faculty Lectures 6:00 - 7:00 AM HMC 6NJB Conference Room UWMC Spine Indications 7:00 - 8:00 AM RR213 Eyre Research Group Meeting 9:00 - 10:00 AM UWMC BB1065	**Every** PreOp Conference / Journal Club 7:30 - 8:30 AM VA	**Every** SCH Resident Teaching Conference 6:30 - 7:30 AM SCH Conf Rm A7932 Spine Research Meeting (for Spine Faculty/ACEs) 7:30 - 8:30 AM HMC DKC Library Rm 6EH77 HMC Spine X-Ray Conference (for Residents/ACEs/Faculty) 8:30 - 9:30 AM HMC 1WH247	**Every** Hand Service Journal Club 6:30 - 7:30 AM HMC 6NJB Conference Room PGY2 Trauma Conference (every other Friday) 6:45 - 7:30 AM HMC DKC Library Room 6EH77 HMC Spine PreOp Conference 7:15 - 8:00 AM 3WH Room 310 HMC Spine Indications Conference 7:15 - 8:15 AM HMC 3WH108 PGY2 Hand Conference 7:30 - 9:00 AM ISIS Lab, NJB Building
1st All Faculty Meeting 6:00 - 7:00 AM Quarterly (March, June, September, December) HMC R&T 121 All Residents Meeting 7:00 - 8:00 AM HMC R&T 121 Spine Journal Club 7:00 - 9:00 PM Ted Wagner's House	**1st**	**1st** Orthopaedic Grand Rounds 6:45 - 7:45 AM UW Foege Auditorium	**1st** UWMC M&M Conference 6:30 - 7:30 AM UWMC RR134	**1st** SCH PreOp/PostOp Care Conference 6:30 - 8:00 AM SCH Conf Room W7712
2nd Ortho IT Forum Quarterly 6:30 - 7:30 AM UWMC BB1065 UWMC Sarcoma Protocol Meeting 11:00 - 12:00 Noon UWMC NE 100-K	**2nd**	**2nd** SCH Faculty/Provider Meeting 6:00 - 7:00 AM SCH W3747B Montlake Faculty Meeting 6:30 - 8:00 AM UWMC BB1065 HMC M&M Meeting (for Residents/ACEs) 7:00 - 9:00 AM HMC R&T 121 VA M&M Meeting 7:30 - 8:30 AM VA	**2nd** UWMC OITE Review (Residents only) 6:30 - 7:30 AM UWMC BB134	**2nd** SCH Resident Lecture 6:30 - 7:30 AM SCH Conf Room W7738
3rd	**3rd** Sports Medicine Provider Meeting 6:45 - 8:00 AM Graves Annex, 3rd Floor Conference Room	**3rd** SCH Faculty/Provider Meeting 6:00 - 7:00 AM SCH W3747B UWMC Ortho/Radiology/MRI/ Arthroscopic Correlation (for Residents/ACEs) 6:30 - 8:00 AM R2, 2nd Floor Conference Room Spine Grand Rounds 7:00 - 8:00 AM HMC R&T Auditorium	**3rd** UWMC Indications Conference (for Residents/Faculty) 6:30 - 7:30 AM UWMC RR134	**3rd** SCH Resident Lecture 6:30 - 7:30 AM SCH Conf Room W7738
4th	**4th**	**4th** SCH Faculty/Provider Meeting 6:00 - 7:00 AM SCH W3747B UWMC Upper Extremity Indications or Journal Club (for Residents/ACEs) 6:30 - 8:00 AM R2, 2nd Floor Conference Room HMC Faculty Meeting 6:45 - 8:30 AM HMC 1WC113 HMC Journal Club (for Residents/ACEs) 7:00 - 9:00 AM HMC R&T 121 Sports Academic Conference 7:30 - 9:00 AM Rose Aud, Conibear Shellhouse	**4th** UWMC Evidence-Based Ortho Journal Club (for Residents/Faculty) 6:30 - 7:30 AM UWMC RR134	**4th** SCH Resident Lecture 6:30 - 7:30 AM SCH Conf Room W7738
5th	**5th**	**5th** SCH Faculty/Provider Meeting 6:00 - 7:00 AM SCH W3747B HMC Journal Club 7:00 - 9:00 AM HMC R&T 121 (only takes place when there are five Wednesdays in a month)	In addition, Monday through Sunday is the HMC Spine Check Out Conf from 7:00 - 7:15 AM in the HMC Radiology Conf Rm and Saturday through Sunday is the HMC Trauma Check Out Conference from 7:30 - 8:00 am in HMC DKC 6EC07.	

Our Grand Rounds lectures enjoy widespread appeal through top lecturers and current hot-button topics. Seen here is Dr. Bas Masri, UBC, Vancouver, BC, showing ways to improve operating room efficiency.

(OSSMIG). Through OSSMIG our volunteer residents and students organized themselves to have after hour sessions on topics such as splinting and casting, how to provide first aid for injured individuals in a wilderness setting and how to examine the knee and other joints. A real life orthopaedic exposure opportunity titled 'Weekend Call at Harborview' has also been very successful among interested medical students and has necessitated creation of a waiting list.

On the more formal side of teaching we have a rich regular conference schedule (page 90), which includes weekly indications conferences for all specialties at all sites and regular formal quality improvement meetings. There are also a number of informal meetings, such as Journal Clubs, currently organized by Hand, Joints, Spine and Trauma specialties, that have had a long tradition and usually take place after hours with volunteer attendees including community colleagues.

On a formal CME basis we now feature a rich and regular educational portfolio for a wide variety of interested groups: We have three types of monthly Grand Rounds (Orthopaedics, Research and Spine), two major annual regional/national conferences (coming up July 25-27, 2013: Summit in Seattle 4: Focus on Knee Disorders and the Spine Summit in Seattle XI: The Value of Spine Care, October 25-26, 2013) as well as our annual UW Hand Course for occupational, PT, and massage therapists. These events have proven to be very popular through their high quality presentations, featuring not only our faculty but also interdisciplinary collaborations across departmental lines.

In our Orthopaedic Grand Rounds series we continue to feature our popular mini-symposium format along with interdisciplinary presentations and headliner lecturers from North America. Just take a look at the program bandwidth over the last few months: we had the privilege of experiencing an instructional course lecture caliber presentation on physeal fractures by Drs. Dales, Lindberg and Sousa, an overview on commercialization opportunities within the UW by Drs. Peter Cavanagh, Paul Manner, Patrick Shelby and Rachael Tanner and headline lectures by visiting professors such as Dr. Steven Kates from the University of Rochester, NY who together with Carrie Bradt, PA, discussed what it takes to organize a modern Fragility Fracture Service and Dr. Bas Masri, Professor and Chairman from the University of British Columbia, who spoke on ways to increase operating room efficiency. Spine Grand Rounds continue to be a focal combined learning opportunity for our orthopaedic, neurosurgical, rehabilitation and neurology communities with very compelling speakers organized from the orthopaedic side by Dr. Rick Bransford. Research Grand Rounds have become a valuable platform to facilitate translational research for our basic scientists and clinicians, while allowing our residents to receive regular

UW Orthopaedics Grand Rounds Schedule 2013-2014

Date	Topic	Speakers (alphabetical)
7/3/2013	Preoperative Lab Testing - an Opportunity for Streamlined Care and Cost Savings	Drs Chansky, Mallette and van Norman
8/7/2013	Stress Fractures: When to Immobilize, When to Operate, and the Potential of Biologics	Drs Patton, Teitz and Twaddle
9/4/2013	Hallux Valgus: An Assessment and Treatment Primer	Drs Brage, Hansen and Kim
10/2/2013	Visiting Professor, on Osteogenesis Imperfecta	Dr Michelle Caird, from University of Michigan Department of Orthopaedic Surgery
11/6/2013	Hamstring Injuries: Therapy Protocols and Emerging Surgical Indications	Drs Green, Holtzman and Twaddle
12/4/2013	Were Are We in Collagen Repair?	Dr Eyre
1/8/14	Navigation at UW Orthopaedics - Practicalities, Promise, and Peril	Drs Gundle, Ching and Conrad
2/5/14	Checklists and Clinical Pathways in the OR, ED, and on the Ward	Drs Dellinger, Goldberg and Roof
3/5/14	Living Local, Acting Global - UW's Role in Global Orthopaedics	Drs O'Donnell, King, and an Orthopaedics Faculty Panel
4/2/14	Balancing AO Principles - Focus on Calcaneus Fractures	Drs Benirschke, Sangeorzan and Stoll
5/7/14	Finger Transplantation: When, Where, and What to Expect	Drs Allan, Kennedy, Kwon and Stoll
6/4/14	End of Life in Orthopaedics - A Roundtable Discussion	Drs Hurd, McCormick, and an Orthopaedics Faculty Panel

Please note that Grand Rounds in July, August, and September are in HSB D209 Turner Auditorium. In October we will return to Foege Auditorium. Participants and topics subject to change. Please refer to final program in monthly email reminders.

feedback for their research milestones.

We finally have four wonderful lectureships annually which allow us to benefit from the insights of our eminent visiting professors. The John F. LeCocq lectureship with its focus on providing our Seattle orthopaedic community a combined platform for medical education is the oldest of these lectureships, celebrating its 50th anniversary in 2014. Please note that we are planning a very special event in light of this anniversary. Our Resident Research Day has been held for 25 years now and has recently been shifted to coincide with our resident graduation in order to enhance the celebration of accomplishment of our residents. Next year will also mark the tenth anniversary of the Jim Garrick lectureship in UW Sports Medicine, another occasion for a celebration. The most recent addition has been the Lynn Staheli Conference. The Lynn Staheli Conference reviews a popular topic in pediatric orthopaedics with an invited visiting lecturer from other children's hospitals in North America. The Staheli Conference has traditionally been an annual conference with the highest attendance at Seattle Children's Hospital. All of these lectureships allow us to benefit from our visitors' wisdom while affording members of our Department the opportunity to feature their work in front of the distinguished leaders of our specialty.

For GME education our weekly schedule may provide you with a perspective of the ongoing learning opportunities for all of our trainees. A particular point of pride is our Resident Bootcamp with which we have traditionally started out our academic year for our incoming class. We now have a number of outside residency programs, such as Madigan Orthopaedics, OHSU Orthopaedics and UW Emergency Medicine with other programs having expressed interest as well. Yet again, I view this as an expression of the deep commitment to quality education delivered by our faculty.

Conclusions

As stated initially this is just a small overview of many facets of educational events with some noteworthy new developments that are taking place at UW Orthopaedics day-in/ day-out. In looking at the many educationally related activities in our Department I think that it is safe to conclude this brief update and overview with two points:

1. There appears to be a palpable and apparently still growing interest in the quality of education provided by members of UW Orthopaedics;

2. There is a deeply engrained culture of education passed through all ranks and specialties of our faculty through our many trainees, which gives us great cause for optimism as we hope to positively affect future generations.

Increasing Medical Student Exposure to Orthopaedics: Developing an Orthopaedic Surgery and Sports Medicine Interest Group

Dayne Mickelson, MD, Philip Louie, BS, Alex Farnand, BA, Lauren Meyer, BS, Brian Gilmer, MD, and Jens R. Chapman, MD

The University of Washington (UW) Orthopaedic Surgery and Sports Medicine Interest Group, launched in 2008, is a student run and department supported group that provides medical students with opportunities to explore Orthopaedic Surgery in an organized, supportive, and motivational environment. The group's goal is to expose students to the orthopaedic field, promote mentorship, as well as help teach basic musculoskeletal anatomy and orthopaedic concepts. The group's inception, successful initiatives, impact on medical students' interest in orthopaedic surgery and the university's subsequent improved matching rates into this specialty are discussed.

Background

The University of Washington School of Medicine (UWSOM) has been ranked by US News & World Report as the No. 1 medical school in the nation for primary-care training for 19 consecutive years, during which the average number of medical students entering primary-care specialties has been close to 50% annually (1). Although the curriculum at the UWSOM is always evolving, it requires a core set of clinical rotations that provide limited exposure to surgical specialty fields such as Orthopaedics. The literature proposes that medical students are more likely to pursue a career in surgery when offered early academic opportunities, clinical exposure and positive role models (2). Surgical specialty support within the UWSOM was deficient in 2008 as the school's orthopaedic interest group had become nonoperational. To rectify this situation, especially in light of the literature, a new Orthopaedic Surgery and Sports Medicine Interest Group (OSSMIG) was established.

Process

The UWSOM OSSMIG was instituted in August 2008 by a group of junior medical students supported by UW Orthopaedic surgery residents. Upon becoming a sanctioned interest group, OSSMIG was assigned a secure email address, listserv (membership email service) and website.

The OSSMIG constitution outlined five core objectives:

1. Present students with opportunities to explore the field of Orthopaedic Surgery through discussions, preceptorships and clinical experiences.
2. Give students additional instruction through lectures and workshops about musculoskeletal anatomy and orthopaedic concepts.
3. Offer students research and networking opportunities within the Department of Orthopaedics and Sports Medicine.
4. Support students applying to Orthopaedic Surgery by advising them about sub-internships, residency

Figure 1: OSSMIG Shoulder Examination Workshop

applications, and the interview process.

5. Promote mentoring among medical students, residents, and the attending surgeons within the Department of Orthopaedics and Sports Medicine.

To improve the quality of mentorship and experiences for its members, OSSMIG cultivated a relationship with the University of Washington Department of Orthopaedics and Sports Medicine. This relationship was established with a faculty advisor as well as UW Orthopaedic residents who acted as liaisons between the department and OSSMIG officers, and who helped coordinate activities and lead events.

Since its inception, OSSMIG has continued to develop a diverse offering of monthly activities that allow students to explore the field of Orthopaedics. Annual conferences and events include:

- OSSMIG Annual Planning Meeting
- Introduction to Orthopaedics
- Orthopaedic Applicant Optimization
- Workshop: Basic Knee, Shoulder & Neurologic Examination (Figure 1)
- Workshop: Basic Splinting (Figure 2)
- Sub-Internships / Application Counseling
- Detailed Medical Student Guides
- Mock Interviews
- Match Celebration Dinner
- Socials and Networking Events

The Medical Student Trauma Call Initiative was developed to allow UWSOM medical students to take call with the orthopaedic trauma team at Harborview Medical Center, the region's busiest Level I trauma center. This initiative started in the winter of 2010, with the assistance of the UW Orthopaedic Department and its residents. The OSSMIG provides an initial information session and packet for those interested. Once assigned a call position, the student assists the orthopaedic resident on call, learning to evaluate and manage acute injuries in the ER and obtaining a firsthand understanding of practical orthopaedics.

To communicate the wealth of knowledge and information available to students interested in surgical orthopaedics, the OSSMIG planning team and members use email (member listserv) and the OSSMIG website, which is hosted on the UW Department of Orthopaedics and Sports Medicine website (www.orthop.washington.edu/?q=ossmig.html). This full-featured site includes announcements, event assets, a resident spotlight, and other member resources.

Outcomes

Over the past 5 years, OSSMIG has developed quickly to become one of the most popular UWSOM interest groups. Its membership has swelled to include more than 130 students. Meeting attendance varies based on its target audience, but averages 50 students for Trauma Call information sessions and examination workshops (Figures 1 & 2).

The popular Medical Student Trauma Call has grown steadily. In total, students have taken over 100 call shifts

Figure 2: OSSMIG Splinting Workshop

Year	Applied	Matched	Unmatched
2009	16	8*	8**
2010	7	5	2**
2011	5	5	0
2012	6	6	0
2013	8	Pending	Pending

Table 1: University of Washington School of Medicine Orthopaedic Match Data. *Three were OSSMIG members. **Candidates were not OSSMIG members.

and there are now waitlists to participate in weekend call. First and second year students volunteer for the majority of these opportunities and the average participant completes 4 call shifts.

In 2008 only 50% of UWSOM students applying to Orthopaedic Surgery matched into accredited residency programs. Since the inception of the OSSMIG the match rate has improved annually to 100% in the past two years (Table 1). Five students applied to Orthopaedics in 2011 and six in 2012. Eight fourth year students are currently applying for the 2013 match cycle, and fifteen third year students are setting up sub-internships.

Discussion

The UW OSSMIG is still evolving in terms of the breadth of educational activities and its membership. The program has been very well received by the UWSOM and has become a great asset to the medical students. It allows them to experience practical orthopaedics and offers a supportive environment in which to pursue their interests. Such efforts are supported by the literature, which reports that providing students with supplementary experiences and positive clinical role models enhances their selection of orthopaedic surgery as a career (3). In addition, interest group workshops and educational events have been demonstrated to increase student competence and confidence (4).

Integration with the University of Washington Department of Orthopaedics and Sports Medicine has been pivotal to OSSMIG's success. Students are impressed with the Orthopaedic resident advocates who are actively involved in planning and participating in events. The student leadership will continue to leverage departmental assets to build on the group's peer mentoring capacity. One direction for future enhancement is the establishment of student-faculty preceptorships.

For many medical students, the Internet is their primary source of information. Being responsive to this need, the OSSMIG website has evolved in step with the growth and success of the program. In fact, a Google search for 'orthopaedic interest group' yields OSSMIG as the top hit (accessed March, 2013). To foster even more student interest, the website will be enhanced with additional resources and assets from a centralized repository. Also, the popular workshop series will expand to include topics such as musculoskeletal radiology and fracture fundamentals.

The Medical Student Trauma Call initiative has become the most popular aspect of OSSMIG. Students have limited opportunities outside the classroom in their 1st and 2nd years, and have been very enthusiastic about this unique early experience. Their participation on call has become jointly beneficial: for the students, it offers a true insight into orthopaedics and for the resident, it provides another team member to assist during a busy call shift.

To objectively investigate the success of OSSMIG, the officer team continually monitors membership and participation. The team is beginning to assess the group's strengths and limitations via student surveys, as well as analyzing the UWSOM residency match outcomes. The goal of increasing interest in orthopaedics should translate into an increased number of students applying into Orthopaedics as exhibited over the last three years when the group became more influential (Table 1). Additionally, enhanced mentorship and guidance should translate into better match outcomes. Over the past four years, the UWSOM orthopaedic match results have improved (Table 1). Of note, not every student who applied into Orthopaedic Surgery was a member of OSSMIG – most notably the unmatched students in 2009 and 2010. Since 2009, every OSSMIG member desiring to do so has obtained an Orthopaedic residency position.

The future of OSSMIG is bright, and its outcomes should encourage the development of similar orthopaedic interest groups in medical schools across the nation. To date, the group has achieved its goal of inspiring students to develop an interest in the exciting field of orthopaedic surgery and plans to build upon its success.

References

1. Ramsey, Paul. University of Washington School of Medicine 2012 report to the Washington State Medical Association. Accessed February 2013. (http://engage.washington.edu/site/DocServer/2012_WSMA_Report_7-26_final.pdf?docID=1541).

2. Erzurum VZ, et al. What influences medical students' choice of surgical careers. Surgery. 2000 Aug;128(2):253-6.

3. Johnson AL, et al. Why do medical students choose orthopaedics as a career? J Bone Joint Surg Am. 2012 Jun 6;94(11):e78.

4. Li R, et al. Assessment of the role of a student-led surgical interest group in surgical education. J Surg Educ. 2013 Jan;70(1):55-8.

Digital Curriculum Initiative - Developing an iPad-based Platform for Orthopaedic Surgery Resident Education and Collaboration

Dayne Mickelson, MD, Kenneth R. Gundle, MD, and Douglas P. Hanel, MD

In response to the increasing interest in mobile computing and rich digital content among residents, The University of Washington Orthopaedic Surgery Residency program developed the 'Digital Curriculum Initiative' (DCI). Launched in 2012 as a collaboration between designated faculty and residents, the DCI's goal is to construct a robust digital environment of high quality orthopaedic content. In addition, the initiative provides a preconfigured Apple iPad to each resident to optimize their learning experience and clinical efficacy by providing a mobile access point for education, collaboration, and patient care. The successful development, implementation, and future direction of the DCI are discussed.

Introduction

Mobile tablet computers are quickly becoming commonplace throughout personal life, business, and education. They are also redefining the healthcare field in clinical and educational realms by igniting a transformation in the ability for providers to connect, disseminate knowledge, and access patient care information (1, 2). However, there is limited literature substantiating an orthopaedic residency program implementing tablet devices in a comprehensive fashion. The University of Washington (UW) Orthopaedic Surgery Residency program has instituted a novel 'Digital Curriculum Initiative' (DCI). This places an iPad in residents' hands, with expansive departmental support structures in place to optimize the residents' learning experience and clinical efficacy by providing a mobile access point for education, collaboration, and patient care.

Background

In addition to a literature review on mobile technologies' use in medical fields (1, 2, 3, 4), a 2012 survey of UW Orthopaedic residents demonstrated that 87% were "very interested" and 9% were "fairly interested" in instituting iPads within the residency program. At the time, 66% of residents did not own or use an iPad (Table 1).

The residents indicated their preferred method for studying was through computers (52%) and tablets (28%), as compared to physical books or journals (20%) (Table 1), and their preferred sources for studying were digital textbooks (75%) and digital journals (69%) (Table 1). The ability to carry multiple textbooks and journal articles, access the internet for answers to clinical questions, as well as the ability to take digital notes and share them with peers were the primary reasons behind these preferences.

Using this data, a comprehensive resident-driven proposal suggested the UW Orthopaedic Surgery Residency program leverage the iPad as the foundation of a standardized electronic educational program for orthopaedic knowledge, resident collaboration and, ultimately, clinical efficacy.

Interested in iPad and Digital Curriculum Initiative:	
Very Interested	87%
Somewhat Interested	9%
Neutral	3%
Not Interested	0%

Currently own a tablet computer:	
Yes	34%
No	66%

Preferred method of studying:	
Physical Books / Articles	20%
Tablet / iPad	28%
Computer	52%

Primary sources of self study orthopaedic information:	
Digital Textbooks	75%
Digital Journals	69%
Physical Textbooks	43%
Physical Journals	14%
Internet Resources	43%

Table 1: Proposal for UW Orthopaedic Resident iPad Initiative – August, 2012. Total participants: 35 UW Residents (PGY1 - PGY5).

Process

The department accepted the proposal in early fall 2012, giving immediate birth to the Digital Curriculum Initiative. Following are the processes for inaugurating this program:

1. Selected a Digital Curriculum Committee (DCC)
   This group is comprised of the Program Director, a department information technology (IT) member, and a representative from each residency year to ensure continuity and sustainability. The DCC was tasked to develop, maintain, and disseminate a set of core resources including textbooks, articles, and electronic media. Their choice of resources was aided by input from residents and faculty within each subspecialty. The electronic assets selected and purchased are iteratively reviewed and

Figure 1: Preconfigured iPads ready for distribution to orthopaedic residents.

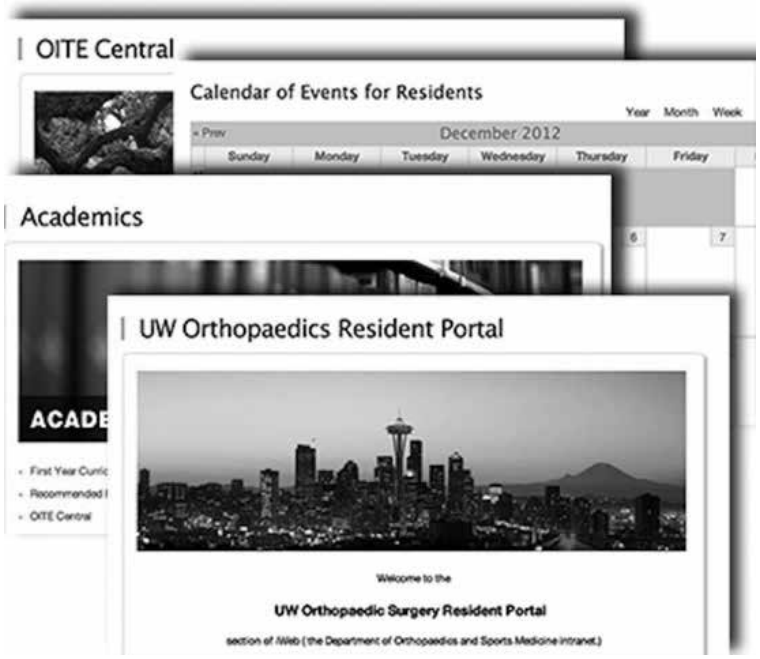


Figure 2: UW Orthopaedic Surgery Resident Portal.

updated by the DCC based on user feedback and disseminated electronically.

2. Selected Orthopaedic Assets for iPad

   Electronic textbooks (ebooks) were selected for general orthopaedics and each of the nine subspecialties (hand, shoulder and elbow, arthroplasty, pediatrics, foot and ankle, spine, musculoskeletal oncology, sports medicine, and trauma).

   Having a full range of evolving applications (or apps) is a powerful feature of the mobile platforms in resident education (3). Therefore, ebook readers, productivity tools and medical applications were selected by the DCC. To date, additional apps include digital note-taking and storage via Evernote; the drug reference program Epocrates; the paging application iPage; and orthopaedic trauma applications AO Surgery and Traumapedia. Utility applications were installed to allow completely secure access for input and retrieval within the hospital electronic medical records system (Citrix Receiver) and radiology (Centricity).

3. Department Approval and Funding

   A business proposal for the DCI and resident iPad implementation was generated to address the limitations and obstacles of the project as well as its promising potential. The proposal addressed the DCC-developed benchmarks to meet academic goals, selected ebooks, articles and apps. It also encompassed the future direction, support infrastructure, and the possible barriers and their solutions. This proposal and its cost analysis was then provided to the department for review. After faculty and department approval, capital was made available through a resident enrichment fund and financial support from alumni.

4. Rebuilt the UW Orthopaedic

Figure 3: UW Orthopaedic residents using iPads during instructional session.

Surgery Resident Digital Portal
The Resident Digital Portal, accessible only to UW orthopaedic surgery residents, is the centralized repository of the digital content managed by the DCC (Figure 2). Each iPad has a direct link to the Portal, which houses lecture slides, conference assets, Orthopaedic In Training Exam (OITE) study materials, curriculum media, suggested readings and a central residency calendar. Within the next year, these resources will be fully integrated with the resident Monday Morning Curriculum, and the R1 Curriculum (www.orthop.washington.edu/?q=education/residency/current-residents-portal.html).

5. Deployed iPads and Provided Training
In January 2013, the DCI deployed the iPads (4th generation, black, 16 gigabyte) to all 40 orthopaedic residents (Figure 1) after they electronically signed an online agreement. The tablets will be provided to each incoming intern class annually and returned to the department upon graduation. Each iPad comes preloaded with the high-yield set of electronic textbooks, articles, and applications selected by the DCC.
Department IT professionals have set up device security, which includes a mandatory password at startup and unlocking, secure access to the hospital's wireless network, and protected access to the department's virtual private network and email server.
Upon deployment, a quickstart guide was provided to each resident. Weekly 'tutorial emails' were sent for the first month, and in-person training and help sessions were provided at monthly all-resident meetings (Figure 3). DCC resident members were available at weekly educational conferences to provide additional technical support.

Discussion

Use of hardcover journals and books is declining as more doctors transition to digital media - especially when that media is digested using a multipurpose portable device such as the iPad.

In response to this paradigm shift, the UW Department of Orthopaedics and Sports Medicine accepted a resident-driven proposal to provide iPads to each resident. The devices were rolled out in January of 2013. To make full use of this investment, there is an ongoing effort to create a digital platform for the orthopaedic surgery residency program and its educational curriculum. One risk in the digital age is an overabundance of information, with a lack of insight on how to distinguish key sources. With the ongoing help of residents and faculty, the Digital Curriculum Committee will continue to select key foundational texts as well as key journal articles for each subspecialty and to ensure these are digitally available.

To organize this effort, the UW Orthopaedic Surgery Resident Portal will continue to evolve as the digital home of the educational initiative. By integrating a calendar, curriculum, rotation-specific information, and associated digital resources, residents will have a window through which to study, share and build orthopaedic knowledge wherever they are.

Although the primary focus is on resident education, the growing landscape of iPad apps will bring other opportunities. For example,

residents can easily bring visual aids regarding injuries or treatment options directly to patients' bedsides via access to injury films and interactive anatomy applications such as Draw MD: Orthopaedics. Additional apps are available that can provide animations or media regarding topics such as surgical steps and how orthopaedic implants work. Patients have reacted positively to tablet computers when used to enhance their care (4). These bedside visual aids improve patient understanding of their injuries and care, and facilitate shared decision-making.

UW orthopaedic surgical residents have responded favorably to the introduction of the iPad-based curriculum. We will continue to track data to objectively capture and demonstrate the magnitude of impact. Based on user responses, we expect to make iterative changes and improvements. The iPad provides endless opportunities in a resident's hand and can improve the didactic process, resident collaboration, and patient education. With the development of interactive and informative apps, alongside an ability to easily carry an entire library of textbooks and papers, the tablet computer has become a mobile repository of orthopaedic knowledge and a powerful tool for surgeons in training. The Digital Curriculum Initiative takes the UW Orthopaedic Surgery Residency Program to the cutting edge of residency education, enhancing our program and ultimately improving patient care.

References

1. Sclafani J, et al. Mobile Tablet Use among Academic Physicians and Trainees. J Med Syst. 2013 Feb;37.

2. Mark J. Lobo, et al. (2013) Pilot Study of iPad Incorporation Into Graduate Medical Education. J Grad Med Educ. In-Press.

3. Franko OI, Tirrell TF. Smartphone app use among medical providers in ACGME training programs. J Med Syst. 2012 Oct;36(5):3135-9.

4. Strayer SM, et al. Patient attitudes toward physician use of tablet computers in the exam room. Fam Med. 2010 Oct;42(9):643–647

Reconciling Resident Education with MOC in the New Practice Environment

Gregory A. Schmale, MD and Michael J. Goldberg, MD

"Best Practice", "Standard Work", "Safety-first", "Plan-Do-Check-Act", "Pay for Performance", "Identify Value" - can these approaches to the delivery of health care meld with a residency training program and lead to future maintenance of competence for our residents?

At Seattle Children's Hospital, we have adopted new approaches to the delivery of health care: Lean methodology and Clinical Standard Work. The Toyota *Lean* method emphasizes the importance of *value* first to eliminate waste and optimize safety, and recognizes the contributions of all workers on "the line", from the lowest rank on up. Clinical Standard Work focuses on standardizing processes, reducing variability, and promoting patient safety. Evidence based clinical pathways capture "best practices", foreseeable events, and actionable metrics. We have worked to include our residents in the processes of *Lean* thinking and Clinical Standard Work, emphasizing routine communication and careful reevaluation to assure quality and optimize care. Given our cyclical cadre of resident trainees, with their single 6-month rotation at Seattle Children's, we have enlisted the help of nurse practitioners during the day and hospitalists during the nights to standardize our in-patient care. This has allowed us to preserve valuable resident work hours from an excess of service, providing the residents their *own* version of *Lean*, a version that, for them, typically places education over service. "Front line workers", including our residents, have been the key to the development of clinical pathways that are of value to the end user. By incorporating checklists into the Electronic Medical Record we have been able to collect metrics in real time which not only serves to improve care, but becomes a robust reservoir for outcomes research. When combined with our systematic collection and compilation of M&M complications, the residents have a unique research opportunity.

The American Board of Medical Specialties and the American Board of Orthopaedic Surgery mandate a 10-year cycle of Maintenance of Certification (MOC) for all practicing orthopaedic surgeons, the teachers in this residency. The four major elements of this MOC include maintaining a license and professional standing, lifelong-learning and self-assessment, cognitive expertise, and assessment of practice performance.[1] How do these goals for MOC compare to our goals for our residents? They seem to be pretty similar to us. The ACGME has identified six core competencies which we must help our residents attain, including medical knowledge, patient care and procedural skills, practice based learning and improvement, systems based practice, professionalism, and interpersonal skills and communication. We believe that what we do from week to week with our residents will lead them on a road of lifelong learning and improvement, and encouraging maintenance of competency.

With the limitations of the 80-hour work week, and requirements for residents to work only a few hours post-call, we have had to shift our focus from that of a solely in-patient care paradigm managed by residents, to a more consistent model of routine and regular floor care provided by two full-time nurse practitioners (ARNP's) during the day, and a cadre of hospitalists at night (soon to be expanded to day-time collaboration and consultation). Residents still round daily on all in-patients, but much of the day to day management of the in-patient stay is now left to our non-resident care-team. Though our residents are still involved in the in-patient care process, the addition of this team of ARNP's and hospitalists frees residents to concentrate more on out-patient clinical, surgical, and

emergency department care of patients. Regular weekly indications conferences, didactic teaching sessions, and non-operative fracture management reviews round out a rich educational program. The reality is that those who practice pediatric orthopaedics as a career will be Fellowship trained. While we hope to attract more of our residents into this speciality, we must present a balanced pediatric education to those who will go into general orthopaedic practice or another orthopaedic specialty. During their one 6-month rotation we strive to tailor our education to meet broad needs and instill lasting competency and interest in children. How does pediatric hip disease inform the future adult arthroplasty surgeon; how does physeal growth inform the adult traumatologist?

We have changed the tone and intent of our monthly morbidity and mortality conferences from quality assurance to care review and quality improvement; from identifying individual missteps to one of a "just culture"[2] in which reporting is not punitive, and that there is a difference between honest mistakes and culpability. These concepts have been embraced by both faculty and residents. The resident is assigned a group of similar complications; performs background research; frames the complication in light of the published literature; and attempts to identify ways to improve practice. Patient, system, normative and technical errors are all considered. Since in most instances the resident presenting has not been directly involved in the cases under discussion, blame and acrimony are replaced with education and understanding. Bimonthly journal club for critical review of the recent literature helps keep both residents and attendings up to date on the latest evidenced-based reports in pediatric orthopaedics.

We have also focused on practice-based learning and improvement and systems-based practice through the adoption (in August 2011) of clinical pathways developed from two AAOS evidence-based guidelines. Standardized order sets incorporated into the Electronic Medical Record for patients admitted with the diagnoses of supracondylar humerus fracture or femur fracture include a series of required safety check-lists that must be completed by the ordering physician – primarily our R3 orthopaedic residents. The check-lists once completed must be attested to by the Attending physician, indicating the Attending's agreement (or lack thereof) with the reported safety elements. The first check–list is completed at the time of admission prior to the patient's leaving the ED and includes elements related to neurovascular status; radiology evaluations; risk of child abuse; and appropriate splinting. A second check-list is completed after surgery confirms that the selected procedure fits our guideline of care, and that limb perfusion is documented. The third check-list is completed at the time of discharge to insure neurovascular status is intact; brace and cast education has been provided, and safe transportation home arranged. Every three months the data generated by these checklists are reviewed with faculty and residents to assess practice trends, checklist compliance and attending/resident discrepancies.

In December 2012 a similar clinical pathway for care of idiopathic Adolescent Scoliosis was introduced into the Electronic Medical Record. This pathway begins in the outpatient clinic and includes patient safety checklists for appropriate surgical indications, pre-operative assessment, intraoperative care, and discharge management. This major quality improvement initiative is now being expanded to include all patients admitted for spine surgery regardless of diagnosis (from scoliosis to spondylolisthesis). The safety lynchpin for this pathway is a Nurse-MD Patient Safety Huddle in the outpatient clinic, not unlike team rounds on the inpatient floor; and like inpatient rounds, our residents will be included.

"Best Practice", "Standard Work", "Safety-first", "Plan-Do-Check-Act", "Pay for Performance", "Identify Value" - we believe that adopting these practices in the academic health care setting that is training residents can be done in an effective and successful manner, for patients, for the faculty and for the resident trainees, as they prepare for careers as knowledgeable, communicative, safe and reflective physicians.

References

1. Inglehart JK, Baron RB: Ensuring Physicians' Competence - Is Maintenance of Certification the Answer? NEJM. 367(26):2543-2547, 2012.

2. Dekker, Sidney: Just Culture: Balancing Safety and Accountability. Ashgate Publishing Burlington VT USA 2007.

Musculoskeletal Medicine and Medical Student Education

Gregory A. Schmale, MD and Barry Goldstein, MD, PhD[1]

The Musculoskeletal System Core Course is one of the most important courses in a medical student's education. The class teaches gross, surface, applied, and radiographic anatomy, clinical manifestations in the musculoskeletal system and pathophysiology of trauma, aging, infection, and inflammation, as well as congenital and metabolic disorders. The class includes dissections, physical examinations, and problem- based learning.

One of the most enjoyable and rewarding annual experiences as a physician is, without a doubt, the three months spent with enthusiastic first year medical students, community physicians, university faculty, and fellows teaching in the Musculoskeletal System core course. The course has changed much over the years, as we have worked to hone the experience into a better product, making it an exciting, interactive, and stimulating initiation for our doctors-to-be into the world of musculoskeletal medicine.

The course has moved from its long-standing home in the middle of the second year to the first year, a time when students have just begun to meet patients in clinics, to learn how to hold and use a reflex hammer, and to practice thinking like a physician. Initially we worried that this course, full of clinical correlations, references to organ systems, and common pathologies, would be too much for the first year student. But no! We have been surprised by the students' ability to learn basic musculoskeletal anatomy and integrate clinical correlations; we are encouraged by the positive feedback about integrating clinical material and the introduction of musculoskeletal health and disease to this group of early trainees. The result is an experience that is much more applied than learning anatomy in isolation.

As the medical school is pushing to "modernize instruction", limiting large group lectures, encouraging instructors to adopt more team-based and other active learning approaches, we have worked to emphasize small-group team-based-learning in our course as well. Weekly cadaver dissections with four students to a table are supplemented with "Living Anatomy "small group sessions every other week, where students identify anatomic landmarks on one another with washable markers, learning basic elements of the musculoskeletal exam. Many university faculty and community physicians participate in the course allowing us to keep groups small. To this we have added Team Based Learning activities: students begin each class with a quiz pertaining to the day's clinical correlations done first individually, then as a group. The group answer sheet is a scratch-out form that provides immediate feedback to the students. The more questions they miss, the lower their group score. This leads to lively discussions, it provides opportunities for students to practice realistic cases and to actively participate in their learning.

Changes in the two 50-minute lectures in the first half of each week of the 9-week course are also intended to engage the students so they have greater involvement in their learning. The students complete on-line a pre-class quiz relating to that day's presentations requiring preparation and mastery of the day's material. In addition, we are creating short 10-12 minute concise video presentations for the students to view specific topics or concepts. They can view these videos at their convenience any time before they come to class. These then provide a base of information for the day that frees us from filling each moment of "lecture" with passive lectures-listening, allowing us to present questions, problems, and cases. To ask questions in real time during the lectures, we use a simple audience response system and the students respond on their phones, laptops, or tablets.

We encourage our residents and fellows to participate in the course. Kenny Gundle, one of our Orthopaedics Residents writes: "Helping to teach first year medical students in the musculoskeletal anatomy course was a fantastic experience for me. I was impressed with the students' preparations for labs, their eagerness to learn, and the way that each table really worked as a team. The faculty were always energetic and helpful, like when Dr. Schmale brought in surgical instruments to help with spine dissections. Understanding anatomy is so fundamental to medicine and surgery, and the chance to participate in teaching helped me see the topics I knew well enough to explain it to the medical students, as well as the questions that we could explore more deeply together. Experiencing how rewarding it was for the faculty to introduce anatomy to the students, and how much fun I had, made me eager for a career in academic orthopaedic surgery. I hope to be involved with well-organized classes such as this musculoskeletal anatomy course."

[1]Department of Rehabilitation Medicine, University of Washington, Seattle, WA.

Who is Responsible for Shaping the Future of Orthopaedics in the Pacific Northwest?

Brian Gilmer, MD, Sarah E. Timmons, BA, and Theodore A. Wagner, MD

The Orthopaedic residency program at the University of Washington has dramatically shaped the impact of musculoskeletal care in the WWAMI region: Washington, Wyoming, Alaska, Montana, and Idaho. Physician recruitment in the region typically begins during residency, as approximately 50% of UW Orthopaedic resident alumni have remained in the state of Washington to practice where they were trained (years 1962-2011). Dr. Douglas Hanel and his team of assistants have been responsible for selecting and retaining residents at the University for the past sixteen years. Dr. Hanel deserves recognition for his continued dedication to recruiting and mentoring Orthopaedists who are committed to delivering the best Orthopaedic care in the country.

Introduction

On a misty fall evening at a high school football game, the star running back of the local team is tackled awkwardly and falls to the ground clutching his leg. As he is carted off the field the concerned fans, players, coaches, and family worry about the outcome and treatment of his injury.

If this injury occurred in the Pacific Northwest, the odds are good that the football player will be treated by a board certified Orthopaedic surgeon trained at the University of Washington. And if the player was injured in the past 16 years, there is a good chance that his surgeon was recruited and trained by one individual. This individual oversees a team that is dedicated to assembling the best and brightest applicants in the nation to fulfill the roll of exemplary Orthopaedic Surgeon Resident.

Discussion

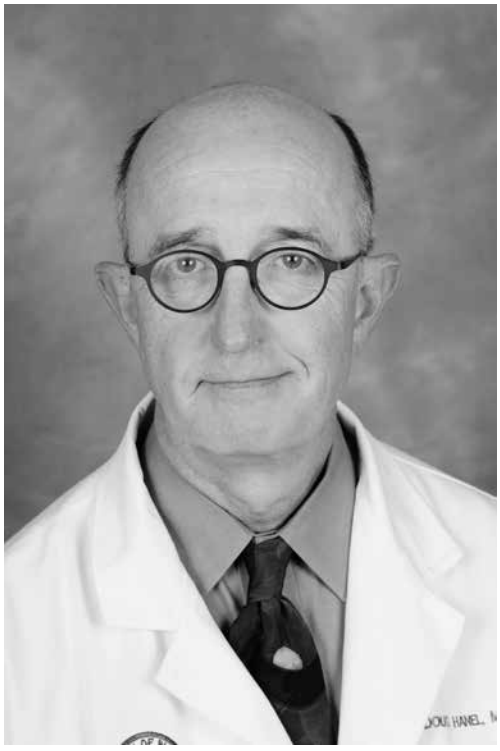
Currently there are 626 practicing Orthopaedic surgeons in the state of Washington.[1] Of those, 18% completed residency training at the University of Washington. In fact, of the 229 UW alumni between years 1962 and 2011, approximately 50% have remained in the state of Washington to practice medicine where they were trained.[2] This suggests that physician recruitment in our region begins during residency training.

The numbers are similarly impressive if expanded to the WWAMI region. This area, composed of Washington, Wyoming, Alaska, Montana, and Idaho, composes one-quarter of the landmass of the United States and is served by a single civilian institution for graduate medical education. In this large area there are approximately 1,000 practicing Orthopaedic surgeons. Of those, 129 trained at the University of Washington.[3] The University of Washington, therefore, has dramatically shaped the impact of musculoskeletal care in the entire WWAMI region for the last 50 years.

Over the past 16 years, the burden of selecting and retaining Orthopaedic residents at the University has been carried out by Dr. Douglas Hanel and his team.

Dr. Hanel received his medical degree from St. Louis University School of Medicine, followed by an Orthopaedic Residency at St. Louis University Health Center and then a Fellowship in Hand and Microsurgery at the University of Louisville in 1984. As a trainee, colleagues frequently noted him to be an ever-present entity in the library dedicating long hours to studying and perfecting his skills.

After a series of professional appointments, Dr. Hanel was hired by Dr. Frederick Matsen to join the Hand Surgery Service at the University of



Dr. Douglas Hanel, UW Professor of Orthopaedics & Sports Medicine, Residency Program Director

Washington as an Associate Professor in 1992. Clinically, his areas of expertise include Hand Trauma at Harborview Medical Center and the Pediatric Hand Surgery Program at Seattle Children's Hospital. In addition to being featured periodically in local media, he was recently listed in Seattle Magazine's 2011 'Top Doctors Hall of Fame' for having been named a top doctor for 10 consecutive years. These clinical accolades however do not attest to his additional efforts in teaching and mentoring young physicians.

Dr. Hanel assumed the role of Residency Program Director in 1996. By his own account, he annually contributes 112 hours over approximately 12 days to resident recruitment alone.

In 2012 there were 578 applicants to the University of Washington Orthopaedic Surgery Residency Program. These applications were hand-screened individually by Dr. Hanel with the assistance of Dr. Lisa Taitsman and Dr. Darin Davidson, with administrative assistance from Residency Coordinator Angela Weiss. Of these applicants, a total of 60 interviews are granted to the nation's very best applications.

Dr. Hanel invites the interviewees to his home the night before their interview appointment where Mrs. Peggy Hanel and a selection of current residents graciously host them. Mrs. Hanel's hospitality is legendary among residents, who are also treated to an annual Christmas party at the Hanel's home during their Residency.

The interviewees then complete a rigorous day of both individual and panel interviews. Dr. Hanel and the Chair of the Department, Dr. Jens Chapman, meet individually with each applicant during their visit to Seattle. Of this final pool, 8 residents are selected annually based in part on the results of the National Resident Matching Program.

After selection to the program, Dr. Hanel manages each resident as they progress through their education. He meets individually with each of the 40 residents (PGY1-5) on a biannual basis to review evaluations and ensure they are reaching their educational benchmarks. Furthermore, Dr. Hanel must balance any issues related to remediation for the residents and manage the sometimes complex interactions between attending surgeons and trainees. Finally, he must continually keep abreast of existing and emerging requirements for maintaining the program's accreditation with the ACGME.

Mrs. Peggy Hanel and Dr. Douglas Hanel

The recent rising costs of medical education are often cited with the increasing financial burden of student loans and the costs of residency applications, travel, and lodging when applying to a program.[4] In addition to the costs to individuals, there is a real cost to programs, faculty, residents, and staff that is more difficult to enumerate.

In the future, technology may refine the application and selection process, as well as the managing of residents. However in the meantime, the demands of this increasingly complex system fall on a single individual and his team to be responsible for issues related to managing a renowned Orthopaedic residency program. Unfortunately, there is not enough recognition afforded to this critical task.

So the next time a family member involved in a motor vehicle collision is taken to Harborview Medical Center or the star running back of the high school football team is taken off the field and into an operating room in our community, there is a good chance he will be cared for by an Orthopaedist recruited and mentored by Dr. Douglas Hanel.

Dr. Hanel is very influential in the selection, retention, and graduation of surgeons who care for patients in the Northwest community. In many cases, he is instrumental in bringing the right surgeon to the right place at the right time in order to provide the best Orthopaedic care available in the Northwest or anywhere in the country. We should take a moment to thank all of our faculty mentors and Dr. Hanel in particular.

Thank you Dr. Hanel.

And thank you to Peggy Hanel for her support of the UW Residency Program as well.

References

1. AAOS Orthopaedic Surgeon Census, AAOS Department of Research and Scientific Affairs January 2013.

2. "Alumni." UW Orthopaedics and Sports Medicine, Seattle.

3. "Alumni." UW Orthopaedics and Sports Medicine, Seattle. N.p.. Web. 8 Apr 2013.

4. Kesler, RW, G Nowacek, and JA Lohr. "The High Cost of Recruiting Residents." Southern Medical Journal. 73.11 (1980): 1521-3.

The University of Washington Sports Medicine Clinic: Back to the Future

Carol C. Teitz, MD

The UW Sports Medicine Clinic began in the fall of 1967 with a call from Dr. John Hogness, Dean of the School of Medicine, to Dr. D. Kay Clawson, founding chairman of the Department of Orthopaedics (now the Department of Orthopaedics and Sports Medicine). Dr. Hogness noted that University of Washington president Charles Odegaard had terminated the relationship with the team physician (a community physician) and trainer and wanted the Department of Orthopaedics to take over as team physicians for UW Intercollegiate Athletics. When Dr. Clawson asked for a timeline to begin caring for the Husky athletes, he was told "Be on the football field at 2 p.m. this afternoon!" Practice would be followed by a 5 p.m. meeting with Head Football Coach Jim Owens and the Athletic Director, Joe Kearney.

During Dr. Clawson's first foray onto the field he was dismayed to find that the stock medicines consisted of "uppers and downers, codeine and seconal." Although the coaches were grumbling about working with someone they had never heard of, Dr. Clawson managed to ingratiate himself with the coaches through successful treatment of an injured male gymnast.

"The gymnastics coach had a spectacular athlete that could do the horse and more particularly the rings and he had an act of flipping himself from the rings. They were preparing for an international event over in Czechoslovakia and the kid dislocated his elbow. The trainer popped it back in but the description was a clearly dislocated elbow. I didn't know much about anything and I asked him what the problem is. Well, you have to be able to extend or maybe even hyper-extend a little bit to be up on the rings and that's how he dislocated it. I traditionally popped dislocated elbows in, put them in a sling and a splint and let the swelling cool down. But they said, 'we have internationals in 6 weeks and he has to be able to participate.' My mind says he's got to be able to be in full extension. I've never seen anybody that couldn't be in flexion but most dislocated elbows never got full extension so I said he has to go in the hospital. Joe Kearney backed me up to put him in the hospital. I put him in full extension, wrapped it, elevated it and iced it and kept him in the hospital for 2 or 3 days until I redressed it. The swelling was virtually down and I kept him in extension for about a week to 10 days and then I started him flexing. He competed but did not win. He competed in internationals as a gymnast and that blew the minds of the coaches. After that I had back-up from the wrestling coach, the gymnastics coach, the basketball coach, all of them."

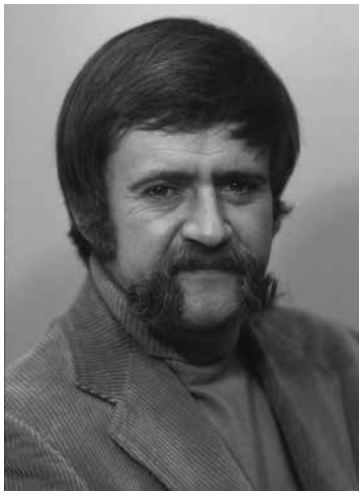
Dr. Clawson was a reconstructive surgeon, but the president really wanted him as the team physician so he needed to get up to speed on taking care of athletes. As an orthopaedic resident at Stanford he had assisted Dr. Frank Cox with the care of the San Francisco 49ers. He called Dr. Fred Reynolds, Chairman of Orthopedics at Washington University and team physician for the St. Louis Cardinals, and created his own traveling fellowship, visiting training rooms in Ann Arbor, Baltimore, St. Louis and Los Angeles. Dr. Clawson reminisces,

"By 1968-69, a lot of people were bitching that I wasn't doing my job as chairman because I was so involved, travelling with the team, all this sort of stuff. At the time, I required all the residents to do a paper like a fellowship year. They did different things. Some of the residents went to Sweden. Al Blue did a hand fellowship. Ted Greenlee got his Masters here in Pathology. Al Roser came to me and said 'I'd like to do mine in Sports Medicine. I can be a fellow, and I can relieve you of a lot of the day to day stuff' which was a big sell. If it hadn't been for Joe Kearney it would never have happened because Jim Owens wanted to be sure it was yours truly showing up at two o'clock for each practice. I set it up so Roser could appear and I would come around later to follow up on things before I went home."

About this time sports medicine was being born as a specialty in the U.S. with significant resistance from the American Academy of Orthopaedic Surgeons. In addition to realizing that being the team physician was all consuming, Dr. Clawson wanted to be the first to have an academic sports medicine program. As Dr. Clawson recalls,

"I met with President Odegaard and thanked him for everything and I said 'but you know if we are going to pull this off I want sports medicine to be a division of the department.' He said 'fine, go ahead we'll call it that' and I said 'no, I want it to go through the Board of Regents so I get publicity on it' and he agreed."

Dr. Clawson requested funds to recruit a faculty member who would dedicate his time to Sports Medicine and the Husky athletes. The funding for the position and that of a half-time secretary would be shared between the Department of Orthopaedics and the Department of Intercollegiate Athletics. A departmental Division of



James Garrick, MD

Ralph Requa

Sports Medicine soon followed. After a year in which Dr. Roser actively assisted with the care of the athletes, Dr. Clawson began recruiting for a sports medicine faculty member. At the time Dr. James G. Garrick was doing ski injury prevention research on a grant at the Mayo Foundation. He had been planning to go into practice in Wyoming when Dr. Clawson called him. Dr. Garrick recalls,

"Kay called me. Well the Academy meeting is next week, he said. 'Are you going to the Academy meeting?' and I was presenting something there and I said 'Yeah, we'll get together there'. And I thought to myself, Academy meeting, 5,000 orthopaedic surgeons, there is no way I'm ever gonna see him at the Academy Meeting and we can go off to Casper, Wyoming and I can forget about this. I was registered and I had just put my badge on and I turned around and this guy looks at me and says 'Jim Garrick? My god! You know we were going to meet here!' It's Kay Clawson and he said 'Why don't you and your wife come up to the room and chat with me?' So we went upstairs. Took him about 45 minutes and there was no more Casper, Wyoming. You know what his final argument was? He told me about all this sports medicine stuff and, you know, I was too young to understand any of that sort of thing and he looked at the badge I had on and I don't know what colors they are now, but you know if you had a green badge it meant you were a speaker and if you had a yellow badge you were just an attendee and after we'd gone through all this he looked at this and he said, 'You know, if you want to keep wearing a green badge, you better take this job'. So I came up twice and the two times I came up I was here 3 or 4 days and there was not a cloud in the sky. I mean, he was the salesman of all time."

Dr. Garrick came to the UW in 1970 as the first Chief of the Sports Medicine Division and brought his research coordinator, Ralph Requa.

Dr. Clawson recalls that part of what made the establishment of the Division and the clinic possible was a combination of a vision and the personal relationships forged.

"Joe Kearney talked to me at length about a month before he died. We kept that relationship going all the time and the reason I could get things done was totally personal relations with Charlie Odegaard, with Joe Kearney, later with Jim Owens. It was all social. Roy Rambeck was the hospital administrator that gave me so many opportunities to do so many things, like intra-medullary nailing, and bit the bullet and paid for it. We shared a golfing locker together up at Sand Point. We were pals supporting each other and trusted each other. I trusted Joe Kearney too. If my thought was different than his I would take his advice because I thought he was better prepared to make that decision and we respected each other. We lost all of that along the way."

The original UW Sports Medicine Clinic was housed in Clinic 16, the Orthopaedic clinic at the time. It was in the hallway which currently houses the EEG lab on the 2nd floor of the University of Washington Medical Center between the ED and the OR. Dr. Garrick saw patients in Clinic 16 and also had an office in the training room. Gradually he started bringing non-Husky patients to the training room. All patients, Husky or not, had physical therapy by the Huskies' physical therapist/trainer group in the training room. Although the regular clinic patients loved doing their rehabilitation alongside the Husky athletes, the Department of Intercollegiate Athletics, and especially the coaches, were concerned that word would get out about which of the Huskies were injured.

In 1974 space in the front of the south end of the Hec Edmundson pavilion was made available by Joe Kearney. Dr. Garrick recalls,

"He controlled the space and we went to him and said 'we're seeing all these patients down in the training room'. We were using the trainers as physical therapists too and they didn't have a very good way to bill for it. When I first came, we started taking care of all the athletes. Women's gymnastics, for example, had never had anybody that took care of them. So, all the trainers, even the head trainer, had multiple sport responsibilities. There weren't many women's sports at the time. There was half-court basketball, track and gymnastics. At the time you also couldn't bring the women down into the men's training room. Soon all the athletes got pre-season physicals and so when we wanted to put the clinic there, we said it's gonna be easier to take care of everybody because we'll have enough room now and we'll have an x-ray machine and we won't have to truck people across to the hospital. So we put one in the clinic and that was a big expense which again was borne in part by the athletic department and the orthopaedic department. Previously, if someone got hurt on the field and needed an x-ray, it took forever, and of course the coaches were not very excited about that. Joe Kearney was in favor of having a clinic in the same building as the training room because it would enhance the ability to take care of the athletes. He and Coach Jim Owens got on well and so they said 'Well this is just the way it is going to be.'"

Colleen Johnson, RN had been a nurse on 4 South, the orthopaedic patient care floor at the time, and was spending a year working on a Consumer Product Safety Grant for Dr. Garrick. Colleen also helped him see the athletes in Clinic 16 until construction of the official Sports Medicine Clinic in the Hec Ed Pavilion was completed. When the clinic moved to the Hec Ed Pavilion in fall of 1974, Colleen was the clinic nurse. Within the next 2 years, she was appointed to the

First clinic waiting room in the SE corner of Hec Edmundson Pavilion.

orthopaedics faculty and then became nurse-manager shortly thereafter, a position she held until her retirement in 2009. Dr. Garrick attributes the lack of significant problems to both Dr. Clawson's leg work in setting up the clinic in the first place and to Colleen Johnson for keeping it running.

"The reason things went well was because Kay had spent, I suspect, a huge amount of time laying the right groundwork and he got Joe Kearney on board. I mean, you go across the country and there isn't anybody in the country who managed to get the athletic director on board for anything medical, at least certainly nothing that wasn't going to make the athletic department money. Kay had done the same thing with Dean Robert Van Citters because Van Citters was a basketball player and he was a jock guy and so those three people were all trying to get it done."

"Colleen dealt with all the day to day stuff that physicians shouldn't deal with anyway and weren't able to and she always kept a good running relationship with the department and the medical school. Everybody respected her."

Colleen recalls

"There was a visionary. People had a vision and worked toward their vision and I think that makes a difference. If the bottom line is 'let's make a lot of money,' you lose your way with the vision. I really think you do. There needs to be a passion."

At the time Dr. Garrick arrived, the Department of Intercollegiate Athletics was watering the Astroturf and Dr. Garrick quickly got caught up in the Astroturf controversy as well as clearing the sidelines of random observers. Astroturf came out in 1967 and the department started looking at its association with injuries and whether or not it should be wet. Dr. Garrick recalls,

"When I came they had just installed the synthetic turf. I came just before the spring game and the anointed quarterback for the next year trashed his ankle on the turf in the spring game. In the previous year they put it in Seattle at the Metropolitan Stadium for all the high schools. Anyway, I read all this stuff from Monsanto and all this stuff said well, it's safer. But I was kinda looking around and I was seeing that there seemed to be a lot of injuries on this 'safe stuff' but there wasn't enough data. Nobody really kept that information prior to that so we didn't know how many injuries we were supposed to see. We ran a study of all the high schools in Seattle. Home games were all played on the synthetic turf field and the rest were played someplace else in mud. We compared the two and then we got some grant money and we expanded it to Oregon because a municipal stadium in Oregon had artificial turf and found that when it was dry it was more dangerous than grass, at least at the high school level. We published our findings. One day I find out that I've gotten a call from Jack Houston because the synthetic turf study hit some national wire service or something and in

Colleen Johnson, RN

those days it was unconscionable for anybody dealing in sports medicine to mention it in the news media. The people who started the American Orthopaedic Society for Sports Medicine were publicity-phobic not publicity-philic like they are now. Monsanto mounted this giant campaign to disprove it, so that was kind of an interesting time. The UW was the first to examine this issue and no subsequent study has contradicted the results."

Based on the evidence, Dr. Garrick instructed the groundskeeper to wet the field. That lasted for 2 weeks. It didn't drain well, but it was a lot safer. Dr. Clawson and Dr. Garrick reminisce,

Dr. Clawson: *"USC was willing to put us out of business over wetting the field before they had to play on it."*

Dr. Garrick: *"Well our coaches were the ones that stopped it though. They sent this memo down to the groundskeeper to not wet the field anymore and we ignored it. We would have student trainers go out there and wet down the field. Then they came to us and said 'you can't do this anymore.' We said 'you know, it's safer' and they said 'It doesn't matter. We can't practice plays and what not because everybody is falling down and the play is too sloppy.'*

The next year, Ralph Requa and I looked at hundreds of game films and found that the covered side of the stadium cast a big shadow during football season so the other side of the field was dry and the covered side was wet and we were going to look to see if there were differences right there. As you remember, the turf was laid in alternating 5 yard strips with the grain going one way for 5 yards and then the other way for the next 5 yards. If you look back at pictures of it they are different colors and that's because of the sun hitting it. Well the thing we discovered inadvertently was on the wet side of the field when players would cross the 5 yard line, if they were making a turn into the grain of the turf, they were fine. One step over the yard marker and they fell on their ass. We had 60 or 70 instances of that which showed it was absolutely predictable and it was always on that side of the field that was always wet. So we went to the coaches and we were really pretty proud of ourselves to show them these numbers and said, 'you know, when it comes to running plays, if you're going to run a sweep and someone is going to make a sharp turn, you do not want them making the sharp turn from one direction into the other direction of the field because they are going to fall down.' It was too much for the coaches to handle. And we said 'this could give you an advantage' and they just rolled their eyes and shook their heads and said 'oh no.'

Additional research projects at that time looked at ski bindings and boots, sports shoes and cleats, and techniques of ankle strapping with a goal of preventing injuries. Dr. Garrick and Ralph Requa developed stellar reputations for epidemiologic research looking at patterns of injuries in numerous sports. Anterior cruciate ligament reconstruction was controversial at that time. Semitendinosus graft was used for openly reconstructing the ACL but nationally, people were questioning whether you needed to do anything at all. Dr. Clawson recalls,

"You'd horrify anybody now, but if you tore your anterior cruciate ligament (ACL) I'd aspirate it, wrap it, brace it and they played the season out and then repairs were always done at the end of the season."

An additional controversy of the time was fluid replacement for football players. Coach Owens liked his players "dry." Coach Shula in Baltimore liked to "water" his players. Gatorade© appeared on the scene. Jim Owens was interested in this because it wasn't water, but it was too expensive. Dr. Clawson and Dr. Garrick devised their own solution (figuratively and literally). Dr. Clawson recalls:

"We had a consultant pediatrician, Dr. Nate Smith, who came to clinic twice a week and who was also very interested in nutrition. We went to Mrs. Rogge (wife of orthopaedic surgeon Lee Rogge) who owned Sunny Jim Peanut Butter© and we propositioned her to make this formula much like replacement IV fluid and made it up as Husky-ade. I still have a packet of Husky-ade as a memento. Jim Owens was thrilled and allowed the kids for the first time to have fluids. But it never materialized commercially like I expected it was going to."

Dr. Garrick adds, *"It was purple and it was the greatest hangover preventative on the face of the earth!"*

Once Dr. Garrick was in charge, the athletic department wanted him to find another head trainer. That trainer also had to have a PhD and be a registered physical therapist. The first trainer hired was Sayers "Bud" Miller from Ball State in Indiana. He had his PhD in Exercise Physiology. There weren't many athletic trainers with those credentials at that time and he was superb. Dr. Garrick recalls:

"When I came the coaches kind of wanted to hire their own trainers and the qualifications didn't really mean anything. I mean, you brought your trainer with you if you were a coach and had been recruited, it didn't matter anything about what they knew. So, when we started, we demanded that the trainers all be registered physical therapists because one of the problems was licensing. At that time the athletic trainers weren't licensed in Washington and they had less stature than beauticians because beauticians were licensed. So we said all the trainers had to be PTs, because the trainers and I were paid by the Medical School and the Athletic Department. It was a jointly funded program and the Medical School wasn't terribly excited about having non-professional people on their payroll. I learned more from Bud than I have from anyone I was ever associated with."

Dr. Garrick developed a 12-week sports medicine rotation for third year UW Orthopaedic residents. Dr. Nate Smith and Dr. Stan Newell also saw patients in the clinic and Dr. Garrick was also assisted in team care by Chris Meyers, an internist (Table 1). Dr. Garrick travelled with the football team and Dr. Meyers travelled with the basketball team.

Although it is hard to imagine, arthroscopy didn't arrive at UW until 1975. Prior to that time, when meniscal and intra-articular ligament injuries were suspected clinically, additional information was obtained via double contrast arthrography, developed at UW by Dr. Mel Figley, chair of Department of

1970-1976	James G. Garrick	Chris Meyers (Internal medicine) Stanley Newell (Podiatry) Nathan Smith (Pediatrics)
1977-1981	Steven T. Bramwell	Letha Hunter (Griffin) & Carol Teitz (Orthopaedic Surgery)
1981-1983	John E. Olerud	Carol Teitz, Roger Larson, Ted Greenlee, Harry Kretzler, Rick Matsen, James B. Smith, & Dan Spengler (Orthopaedic Surgery) Karen Nilson (Internal Medicine) Steve Rice & Steve Anderson (Pediatrics) Stan Herring (Physical Medicine and Rehabilitation) Janet Edlefson (Counselor for eating disorders)
1983-1987	Roger V. Larson (clinical) Carol C. Teitz (research & education)	Harry Kretzler & James B Smith (Orthopaedic Surgery) John Olerud (Internal Medicine) Steve Rice (Pediatrics)
1987-1989	Carol C. Teitz	Roger Larson
1989-1998	Roger V. Larson	John O'Kane (Family Medicine) Peter Simonian & Carol Teitz (Orthopaedic Surgery)
1998-2002	Peter T. Simonian	Roger Larson & Carol Teitz (Orthopaedic Surgery) John O'Kane (Family Medicine) Mark Harrast (Rehabilitation Medicine)
2002-2011	John R. (Trey) Green	Roger Larson, Carol Teitz, & Chris Wahl (Orthopaedic Surgery) John O'Kane (Family Medicine) Mark Harrast & Brian Krabak (Rehabilitation Medicine)
2011-2013	Carol C. Teitz	Albert Gee, Trey Green, Roger Larson, & Chris Wahl (Orthopaedic Surgery) John O'Kane (Family Medicine) Brian Krabak (Rehabilitation Medicine)
2013-	Mark A. Harrast	Albert Gee, Trey Green, Roger Larson, Carol Teitz (Orthopaedic Surgery) John Drezner, Kim Harmon, John O'Kane, & Ashwin Rao (Family Medicine) Alfred Gelhorn, Nelson Hager, Mark Harrast, & Brian Krabak (Rehabilitation Medicine)

Table 1: History of Providers at the University of Washington Sports Medicine Clinic. Division Chiefs and clinic directors on the left, providers listed on the right.

Radiology. Dr. Clawson recalls:

"Mel Figley was an absolutely superb radiologist who was developing and publishing on this double contrast arthrography. He agreed that if we would send acute knee injuries over during a game he would even leave his seat in the stadium and do an immediate double contrast arthrography. By the end of the game, I would have the report on whether a meniscus or ACL was torn because he was so interested. About the same time there was an orthopaedic surgeon in Michigan named Lanny Johnson who was putting these funny scopes into knees. He tried to convince me that that was the way to go. Figley would not approve our getting an arthroscope at the UW so the first one we got was through the VA." Dr. Garrick adds, "The scope quickly moved to the University and we got to start using it. When we started using the arthroscope what you did is you put the patient to sleep and you put the arthroscope in and looked around and then you opened the knee and operated. In the operating room, everybody except the surgeon hated the arthroscope because no one else could see what the surgeon was seeing. There was no camera attached to the scope. You were in there going 'Wow! That's really neat!' but everyone is looking at the ceiling. And then you contaminate everything because you put your head on the table to get your eye on the lens."

Cameras that attached to the arthroscope became available in 1980 and markedly changed surgical efficiency, the operating room environment, and the ability to teach residents arthroscopic skills. For the first time, everyone in the room could see the inside of the knee at the same time.

In 1975 Coach Don James replaced Jim Owens. With him came Gary Derscheid, RPT, as the head athletic trainer. Victor Frankel became Orthopaedic department chair in 1975 and the Athletic Department hired a new athletic director, Mike Lude. Dr. Garrick, Dr. Frankel, and Mr. Lude disagreed about which athletes should get care and thought the annual budget for sports medicine could be markedly reduced if care was provided only for the revenue-generating sports. Dr. Garrick resigned in 1976 and moved to San Francisco. Colleen Johnson was left to pull together a new group of team physicians.

Dr. Steve Bramwell had been a star running back for the Huskies and completed his orthopaedic surgery residency at the UW in 1976. He was the chief of the Sports Medicine Division from 1977-1981 with Dr. Letha (Hunter) Griffin from Michigan as his orthopaedic side-kick. Dr. Carol Teitz completed her orthopaedic residency at UW and joined the group in 1980. Dr. Bramwell left the UW in 1981 taking with him into a community practice the contract for care of the Huskies. Colleen Johnson notes *"So he took 'the children', the department kept 'the house'."* Dr. John Olerud became chief of the Division in 1981 and served until 1983. Dr. Griffin left and Dr. Roger Larson arrived in 1982. Even without team care, the Sports Medicine Clinic continued as it always had, seeing patients with sports and activity related problems. (See Table 1 for additional providers.)

Before the Huskies left the UW, the clinic had shared Physical Therapists and Athletic Trainers with the Athletic Department. They were paid in part by the Athletic Department and in part by the Orthopaedic Department, and they answered to the team doctors. It was a win-win situation. The RPT/ATCs improved their clinical acumen by following the doctors around in clinic. The doctors won because they had experts to explain exercise regimens at the end of the clinic visit. The patients were thrilled and inspired because they received their exercise instruction from the Husky trainers. When Husky care

left the UW the clinic created its own physical therapy space in old squash courts above the clinic and hired its own physical therapists, some of whom worked in the training room as well. At that time Dennis Sealey was the head trainer and Howard Roth and Kim Green were assistant trainers. They also treated our patients along with our physical therapists Kathy Rockefeller and subsequently Diane Cook, and Jan Rotkis.

From 1983-1987 the Directorship of the Division was split between Drs. Larson and Teitz. The clinic continued to run successfully as an Orthopaedic Departmental clinic which prided itself on delivering excellent patient care much like a private practice. For many years the staff was limited to a receptionist, a medical transcriptionist, (Marcia Patten) an LPN, (Wanda Kinion) and an administrative assistant (Betty Condon). Colleen assisted with rooming patients, x-rays, and drawing blood while she continued to manage the clinic. Everyone who met the patients knew the patients and cared about them. Phone calls were answered instantly and with a personal touch, charts were on site, up to date, and never lost. X-rays were taken, read by the orthopaedic surgeons, and filed on site.

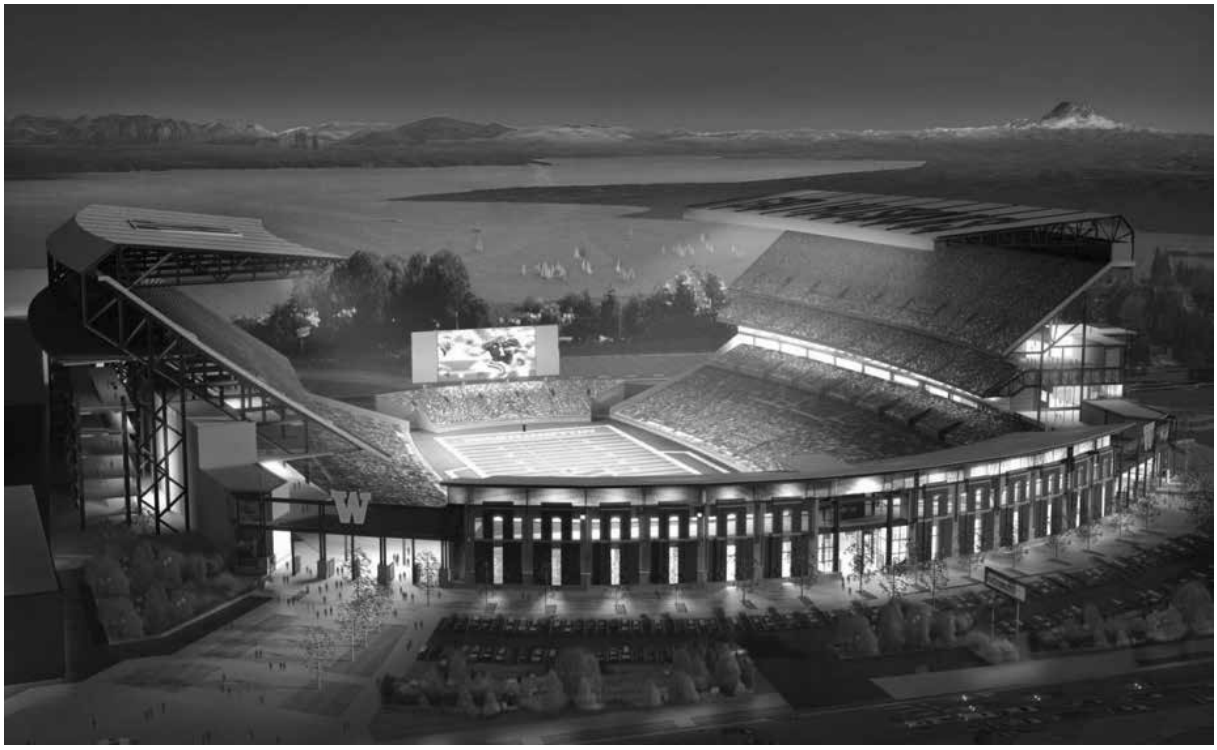
The Department of Intercollegiate Athletics decided to remodel the Hec Edmundson Pavilion in 1998 and the clinic functioned in a basement space in the Electrical Engineering building, which was slated for destruction (site of the current Paul Allen center for Computer Science and Engineering). During that time our physician group was joined by Drs. John O'Kane and Peter Simonian. Dr. Simonian had completed his orthopaedic training at UW in 1996 and became the Sports Medicine Division Chief in 1998 after a sports medicine fellowship at Hospital for Special Surgery. He remained in that position until 2002. Dr. O'Kane had completed his family medicine residency at UW and was the first primary care sports medicine fellow at the UW. Dr. O'Kane received permission from Orthopaedics Department Chair Frederick A. Matsen to create his own fellowship. At that time, there was no subspecialty certification in sports medicine (either via primary care or orthopaedics). As part of Dr. O'Kane's fellowship, he spent time with Dr. Bramwell, assisting him with the care of the Huskies. That ultimately opened the door for the UW group to regain care of the Husky athletes in 1997. Colleen recalls,

1975 University of Washington Department of Orthopaedics. Sports Medicine providers: First row, center: Dr. D. Kay Clawson, far right: Dr. Frederick Matsen. Second row, 3rd from right, Dr. James Garrick. Third row, center: Dr. Carol Teitz, second from right: Dr. Steve Bramwell.

Current clinic in NE corner of Hec Edmundson Pavilion.

> *"Rick Matsen and I met with Barbara Hedges (Athletic Director 1991-2004) and Jim Collier (Vice President for University Relations). I looked Barbara Hedges in the eye and I said 'Barbara, there will be a time at this University when you and I will have been departed and I think it's our responsibility to bring the care of the athletes to where they belong, here at the University.' When I got back to my office Jim Collier called me up and said 'That was the most powerful thing you could have said to Barbara because she wants to do the right thing'. So Jim Collier really worked hard lobbying to get the athletes back, Barbara Hedges was instrumental in what happened, and John O'Kane was in the right place at the right time and everybody liked him."*

In 2000, the Sports Medicine Clinic



New Sports Medicine Clinic on the south side of New Husky Stadium.

moved into its current space in the northeast corner of the newly remodeled Hec Ed Pavilion. The previous clinic in the southwest corner became the Hall of Fame. Clinic, office, and physical therapy space was provided by ICA in exchange for providing care for the athletes. No money changed hands. Originally, the Athletic Department also provided clinic and office space with no rent. (Office space was above the current Husky shop.) However, team care was reimbursed. Colleen Johnson and Dr. Garrick recall that Dr. G's last budget submitted to Mike Lude in 1976 was in the ballpark of $122,000. That was the budget to buy the tape, provide the care, run the clinic, pay part of Dr. Garrick's salary, and pay the trainers.

During Dr. Simonian's tenure, we trained 2 orthopaedic sports medicine fellows, Dr. Ed Tingstad (now the team doc for Washington State University) and Dr. David Belfie (now at Virginia Mason). When Dr. Simonian left in 2002, Dr. John R. (Trey) Green came from LSU and became the next head of the Sports Medicine Clinic, the position he held until November of 2011. Colleen Johnson retired in 2009 and Claudia Happe became the clinic manager. Dr. Mark Harrast was part of the team from 2001-2006. In 2007 Drs. Brian Krabak and Chris Wahl joined the group, with Dr. Wahl leaving in 2012. Dr. Teitz resumed leadership of the clinic in November 2011. Our recent orthopaedic sports medicine colleague additions are Dr. Albert Gee and Dr. Bruce Twaddle.

In fall of 2013 our clinic will no longer be an Orthopaedic Department clinic, but will become an official multidisciplinary University of Washington Medical Center clinic incorporating doctors from Orthopaedics and Sports Medicine, Family Medicine and Rehabilitation Medicine with subspecialty sports medicine training. We will have a new area devoted to performance enhancement and physical therapy, and will have a musculoskeletal radiologist on site. The clinic will be run by a leadership group including Dr. Kim Harmon (Family Medicine), Dr. Mark Harrast (Rehabilitation Medicine) Dr. Carol Teitz (Orthopaedics and Sports Medicine), Claudia Happe (clinic manager), and the head of the Performance Training Center. The Medical Director position will rotate every 2 years among the physicians, with Dr. Harrast as our first Director. We're excited about the next chapter in this story, moving into the new Husky Stadium, and invite our readers to celebrate the opening with us. Watch the Orthopaedics and Sports Medicine website for details.

Acknowledgment

Special thanks to Drs. Clawson and Garrick and to Colleen Johnson for their vision, leadership, guidance, friendship, and willingness to be interviewed for this article. Thank you to Ralph Requa for sharing photos from his private collection.

References

1. Roser, LA, Clawson, DK. Football Injuries in the Very Young Athletes: Clin Ortho and Rel Res 69:219-223. Mar-April, 1970.

2. Cho KO. Reconstruction of the anterior cruciate ligament by semitendinosus tenodesis. Bone joint Surg 57A:608-612, 1975.

Thomas M. Green, MD Retirement

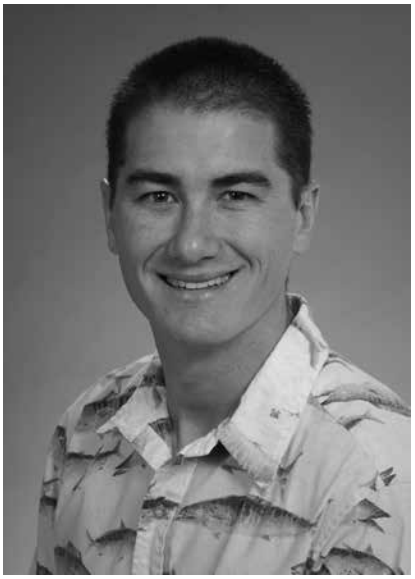
Thomas M. Green, MD - one of our senior clinical faculty members and respected major figures in the regional orthopaedic community and beyond - retired from a most successful 38 year long practice this past April.

As a graduate of the '75 UW Orthopaedics class Tom witnessed the beginning of the Trauma era, which vaulted Harborview with its Medic One program and advanced fracture fixation methods to a leadership role in orthopaedics. He brought these experiences to his first and only place of employment – Virginia Mason Medical Center, where he paired his insights in fracture fixation with his growing interest in adult reconstructive orthopaedics and subsequently built a well-regarded joint replacement service. Inspired by his innate disdain for clutter, Dr. Green was also was an early proponent of efficiency by standardizing implant systems well before the 'Lean' methodology became fashionable. For our residency, Dr. Green was instrumental in making Virginia Mason a regular and very popular rotation for our UW Orthopaedics residents by teaching them the 'Virginia Mason way' in hip and knee replacement surgery. Dr. Green was promoted to the rank of Clinical Professor in our Department in 1989 for his tremendous dedication to our resident education. In light of his leadership qualities Dr. Green was appointed Section Chief of Orthopaedics at Virginia Mason Medical Center, a position he held with distinction for 25 years. With his reputation of fairness and professional calm held in high regard, Dr. Green was also asked to serve as a long-time member of the Ethics Committee at Virginia Mason and as a special consultant for the Committee on Standards of Professionalism at the American Academy of Orthopaedic Surgeons. He was appointed by our State Governor to the Medical Quality Assurance Committee of Washington State, and is past president of both the King County Medical Association and Washington State Orthopaedic Association.

Dr. Green has always known the importance of giving back. He and his wife are well-known humanitarians, supporting countless children and orphanages in other countries and welcoming 10 foster kids into their home over the years. For decades he has worked overseas with NPH (Nuestros Pequeños Hermanos - "Our Little Brothers and Sisters"), which provides housing and care to abandoned and orphaned children all over the world. In 2010 he answered the call for help in Haiti after the devastating magnitude 7.0 earthquake, traveling with a small team from Virginia Mason to perform dozens of orthopedic surgeries. For all of his accomplishments, leadership, and generosity with his time and talents, Dr. Green received Virginia Mason's James Tate Mason Award in 2010. Dr. Green remains a welcome regular participant in meetings and educational events around Puget Sound including programs held by our Department while enjoying his many other pastimes such as boating and the outdoors with his friends and family. Our Department is much indebted to his years of dedication to our resident education and for helping define standards of care in our specialty.

Jens R. Chapman, M.D.
Lyle Sorensen, M.D.

Graduating Residents



Kyle Chun, MD

After residency, Kyle will move with his family to Los Angeles to complete a sports medicine fellowship at the Southern California Orthopaedic Institute (SCOI). Upon completion, he will return home with his family to Hawaii as a partner in the Orthopaedic Associates group based in Honolulu on O'ahu.

Andrew Ghatan, MD

After graduation, Andrew will complete a fellowship in Hand and Upper Extremity surgery at Hospital for Special Surgery in New York. Though he is unsure where he wants to settle, he is planning to pursue an academic career.

Elizabeth Dailey, MD

Following residency, Liz and her husband, Dave, will return to Chicago, where she will complete a fellowship in Adult Joint Reconstruction with Midwest Orthopaedics at Rush University. Upon completion of fellowship, Liz plans to practice adult reconstruction in the Pacific Northwest or the Midwest.

Brian Gilmer, MD

After residency, Brian and his wife Kira, will move to Taos, New Mexico to pursue a fellowship in Sports Medicine at the Taos Orthopedic Institute. Upon completion of fellowship, he plans to seek a general orthopaedic practice in the West.

Graduating Residents

Jennifer Hagen, MD

Following residency, Jenn will be completing a trauma fellowship at the R. A. Cowley Shock Trauma Center in Baltimore, MD. She and her husband will then be traveling to Davos, Switzerland where she will be a research fellow at the AO Research Institute.

David Patterson, MD

After residency, David will pursue a fellowship in Sports Medicine at the University of Iowa. His wife Jessica is applying this fall for residency herself as she completes the MD-PhD program at UW. David will seek out a practice where she matches next spring.

Mark Miller, MD

Following residency, Mark will complete a fellowship in Pediatric Orthopaedics at Washington University in St. Louis. He and his wife Rebecca are considering career locations in the Northwest and the upper Midwest.

Emily Squyer, MD

After graduation, Em and her husband will move to Indianapolis to complete fellowship training in Orthopedic Trauma at Ortho Indy. From there they plan to venture back to the Mountain West.

Incoming Residents

Ahmad Bayomy, MD

Ahmad Bayomy is from Moscow, Idaho. He attended college at Washington State University and medical school at the University of Washington. He is interested in pediatric orthopaedics, foot & ankle, upper extremity, biomechanics, and tissue engineering. He also enjoys traveling, good food with friends and family, cycling, swimming, snowboarding, and football.

Kevin Hug, MD

Kevin Hug is from Tampa, Florida. For his undergraduate education, he attended Brown University and for medical school, he attended Duke University. He is interested in all areas of orthopaedics, and clinical and translational research. He likes to spend his spare time swimming, working out, watching lots of sports, traveling, and getting out and seeing the Pacific Northwest, and photography.

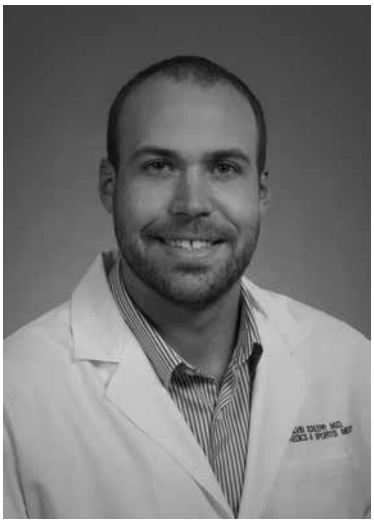
Christopher Domes, MD

Christopher Domes, from Hillsboro, Oregon, attended Linfield College. He attended medical school at Oregon Health and Science University. Sports medicine, upper extremity, and trauma are his areas of interest. In his free time, he enjoys long distance hiking, mountaineering, climbing, endurance sports, and drumming.

Alexander Lauder, MD

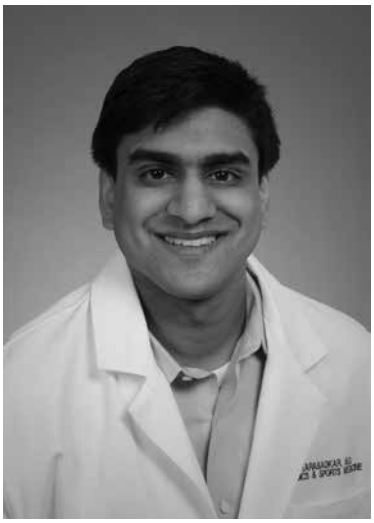
Alexander Lauder, from Libertyville, Illinois, attended college at Northwestern University and medical school at Boston University School of Medicine. His areas of interest are trauma, sports medicine and physiology, and epidemiology. When away from the hospital, he enjoys rock climbing, cycling, camping, guitar, and traveling.

Incoming Residents



Calvin Schlepp, MD

Calvin Schlepp is from Conrad, Montana. He completed his undergraduate education at Carroll College and his medical training at the University of Washington. He is most interested in rural and international orthopaedics, trauma, sports medicine, and wilderness medicine. Outside of medical interests, he enjoys international travel, fly fishing, backpacking, and exploring Seattle's music and restaurant scene.



Neil Tarabadkar, MD

Neil Tarabadkar, from Macon, Georgia, attended Bates College. He attended medical school at the Medical College of Georgia. Hand, upper extremity, and international orthopaedics are his areas of interest. In his free time, he enjoys soccer, tennis, good food and wine, international travel, and exploring the Northwest.

Sara Shippee, MD

Sara Shippee is from Chicago, Illinois. She attended Colgate University and University of Illinois Chicago for her undergraduate education and MPH. She attended medical school at University of Illinois at Chicago. She's interested in all areas of orthopaedics. In her spare time, she enjoys bikram yoga, running, swimming, skiing, restaurants, food and wine, and hanging out with her dog, Tater.

Shawn Werner, MD

Shawn Werner is from Tillamook, Oregon. She attended Oregon State University for her undergraduate degree and Oregon Health & Science University for medical school. Her main area of interest is trauma. When away from work, she likes watching and playing sports, cooking, the outdoors, and international travel.

ACEs and Fellows

Karim Bakri, MD
Hand Service

Mathias W. Daniels, MD
Spine

Michael D. Johnson, MD
Foot & Ankle

Mitchell Bernstein, MD
Trauma

Jennifer Hsu, MD
Hand Service

Nathan J. Kiewiet, MD
Foot & Ankle

Zachary A. Child, MD
Spine

Calvin T. Hu, MD
Foot & Ankle

Mark W. Manoso, MD
Spine

ACEs and Fellows

Geoffrey S. Marecek, MD
Trauma

Joan D. Miles, MD
Oncology

John A. Scolaro, MD
Trauma

Matthew D. McElvany, MD
Shoulder & Elbow

Shelley Noland, MD
Hand Service

Joshua B. Shatsky, MD
Trauma

Erik T. McGoldrick, MD
Shoulder & Elbow

Brett Peterson, MD
Hand Service

Edward R. Westrick, MD
Trauma

Research Grants

National Institutes of Health

Aging-Related Degradation in Bone Mechanotransduction
Sundar Srinivasan, PhD
Ted S. Gross, PhD

Brief Rest Intervals Amplify the Response of Bone Mechanical Loading
Ted S. Gross, PhD
Steven D. Bain, PhD
Sundar Srinivasan, PhD

Collagen Assembly in Tissue-Engineered Cartilage
Russell J. Fernandes, PhD
Jiann-Jiu Wu, PhD

Collagen Cross-Linking in Skeletal Aging and Diseases
David R. Eyre, PhD
Jiann-Jiu Wu, PhD

Collagen Diversity and Pathobiology in Skeletal Tissues
David R. Eyre, PhD
Jiann-Jiu Wu, PhD

Comparing Ankle Arthrodesis to Ankle Arthroplasty
Bruce J. Sangeorzan, MD

Neuronal Modulation of Focal Bone Homeostasis
Ted S. Gross, PhD
Steven D. Bain, PhD
Edith M. Gardiner, PhD

T-Cell Mediation of Focal Bone Loss Induced by Transient Muscle Paralysis
Ted S. Gross, PhD
Brandon J. Ausk, MS

National Aeronautics and Space Administration

Activity Monitoring During Parabolic Flight
Peter R. Cavanagh, PhD, DSc

National Space Biomedical Research Institute

Monitoring Bone Health by Daily Load Stimulus Measurement During Lunar Missions
Peter R. Cavanagh, PhD, DSc

An Integrated Musculoskeletal Countermeasure Battery for Long-Duration Lunar Missions
Peter R. Cavanagh, PhD, DSc

Extent, Causes, and Countermeasures of Impaired Fracture Healing in Hypogravity
Peter R. Cavanagh, PhD, DSc

Veterans Affairs Rehabilitation Research and Development Service

Dynamic Foot Bone Motion: Evaluation of Reconstructive Procedures
Bruce J. Sangeorzan, MD

Foot Bone Motion in End Stage Ankle Arthritis Patients
Bruce J. Sangeorzan, MD

Reducing Internal Stresses in Deformed Diabetic Feet
Bruce J. Sangeorzan, MD
Peter R. Cavanagh, PhD, DSc

VA Center of Excellence in Amputation Prevention and Prosthetic Engineering
Bruce J. Sangeorzan, MD

VA Merit Review Functional Analysis of EWS/FLI-1
Howard A. Chansky, MD
Liu Yang, PhD

A.O. Foundation

Quality Of Fracture Reduction And Its Influence On Functional Outcome In Patients With Pilon Fractures
Sean E. Nork, MD

A.O. North America

An Observational Study Comparing Surgical to Conservative Management in the Treatment of Type II Odontoid Fractures Among the Elderly
Jens R. Chapman, MD

Effect of Riluzole in Patients with Cervical Spondylotic Myelopathy Undergoing Surgical Treatment
Jens R. Chapman, MD

AO North America Orthopaedic Trauma Fellowship
David P. Barei, MD

AO Spine North America Fellowship
Carlo Bellabarba, MD

AO Spine North America Young Investigators Research Grant
Michael J. Lee, MD
Randal P. Ching, PhD

A.O. Spine International

AO Spine Injury Case Collection
Carlo Bellabarba, MD

American Society for Surgery of the Hand

The Use of Vibration Anesthesia
Jerry I. Huang, MD
Jason H. Ko, MD

Research Grants

Baylor College of Medicine

Pathogenesis of Novel Forms
of Osteogenesis Imperfecta
David R. Eyre, PhD

Boston Medical Center

Intramedullary Nails versus Plate Fixation
Re-Evaluation Study in Proximal Tibia Fractures
a Multi-Center Randomized Trial Comparing Nails
and Plate Fixation
Robert P. Dunbar, MD

DIApedia LLC

Advanced Military Footwear System With Composite
Orthotic
Peter R. Cavanagh, PhD, DSc

Johns Hopkins University

The Major Extremity Trauma Research Consortium
Bruce J. Sangeorzan, MD

National Science Foundation Extreme Science and Engineering Development Environment (XSEDE)

Agent-Based Models Of Bone Mechanotransduction
Sundar Srinivasan, PhD

OMeGA Medical Grants Association, LLC

OMeGA Shoulder and Elbow Fellowship
Program Grant
Winston J. Warme, MD

OMeGA Spine Fellowship
Carlo Bellabarba, MD

OMeGA Trauma Fellowship
David P. Barei, MD

Orthopaedic Research and Education Foundation

OREF Residency Enhancement Grant
Douglas P. Hanel, MD

OREF Spine Fellowship
Carlo Bellabarba, MD

OREF Trauma Fellowship Grant
David P. Barei, MD

Orthopaedic Trauma Association

A Multi-Center Prospective Cohort Study Of Sacral
Fractures Using Patient Based And Objective
Outcomes
Jens R. Chapman, M.D.

Development of Fracture Specific MFA
M. Bradford Henley, MD

COTA Trauma Fellowship
David P. Barei, MD

Skeletal Accumulation Of Bisphosphonate Impairs
Bone Healing
Sean E. Nork, MD

Ostex International, Inc.

Molecular Markers of Connective Tissue Degradation
David R. Eyre, PhD

Paradigm Spine LLC

A Multi-Center, Prospective, Randomized, Clinical Trial
Comparing Stabilization With Coflex Vs. Pedicle Screw
Fixation And Fusion After Decompression For At Least
Moderate Lumbar Spinal Stenosis
Jens R. Chapman, MD

Synthes USA

PRODISC-C Versus Anterior Cervical Discectomy and
Fusion (ACDF)
Jens R. Chapman, MD

Spine End-Results Research Fund
Jens R. Chapman, MD

Synthes Grand Rounds
Richard J. Bransford, MD

Synthes Request for Basic AO Course R2s
Douglas P. Hanel, MD

The Boeing Company

Randomized Clinical Trial Of Open Versus Endoscopic
Carpal Tunnel Release And Hand Therapy Comparing
Patient Satisfaction. Functional Outcome And Cost
Effectiveness
Jerry I. Huang, MD

US Army Research Office

Exogenous Blastema Delivery To Injured Human Digits
Christopher H. Allan, MD

UW Department of Bioengineering

Remote Monitoring Of Knee Function After Total Joint
Replacement (Coulter Grant)
Peter R. Cavanagh, PhD, DSc
Paul A. Manner, MD

Department Publications 2012-2013

A list of publications authored by our faculty from January 2012 through June 2013. Our faculty members names are in **bold type**.

1. Aliprantis AO, Stolina M, Kostenuik PJ, Poliachik SL, Warner SE, **Bain SD**, **Gross TS**. Transient muscle paralysis degrades bone via rapid osteoclastogenesis. FASEB journal : official publication of the Federation of American Societies for Experimental Biology. 2012 Mar;26(3):1110-8.

2. Amman S, Cizik A, **Leopold SS**, **Manner PA**. Two-incision minimally invasive vs standard total hip arthroplasty: comparison of component position and hospital costs. The Journal of arthroplasty. 2012 Sep;27(8):1569-74 e1.

3. Apostle KL, **Sangeorzan BJ**. Anatomy of the varus foot and ankle. Foot and ankle clinics. 2012 Mar;17(1):1-11.

4. Ausk BJ, Huber P, Poliachik SL, **Bain SD**, **Srinivasan S**, **Gross TS**. Cortical bone resorption following muscle paralysis is spatially heterogeneous. Bone. 2012 Jan;50(1):14-22.

5. Baker GA, Cizik AM, **Bransford RJ**, **Bellabarba C**, Konodi MA, **Chapman JR**, **Lee MJ**. Risk factors for unintended durotomy during spine surgery: a multivariate analysis. The spine journal : official journal of the North American Spine Society. 2012 Feb;12(2):121-6.

6. **Barei DP**, **Beingessner DM**. Open distal femur fractures treated with lateral locked implants: union, secondary bone grafting, and predictive parameters. Orthopedics. 2012 Jun;35(6):e843-6.

7. Barnes AM, Cabral WA, Weis M, Makareeva E, Mertz EL, Leikin S, **Eyre D**, Trujillo C, Marini JC. Absence of FKBP10 in recessive type XI osteogenesis imperfecta leads to diminished collagen cross-linking and reduced collagen deposition in extracellular matrix. Human mutation. 2012;33(11):1589-98.

8. **Bellabarba C**, Zhang F, **Wagner T**. [Controversies in TL classifications. What are we actually treating? Some perspectives on the evolution of spine fracture classification systems]. Der Unfallchirurg. 2012 Dec;115(12):1056-60.

9. **Benirschke SK**, Kramer PA. Update on the Management of Calcaneal Fractures: American Academy of Orthopaedic Surgeons; 2012. Available from: http://orthoportal.aaos.org/oko/article.aspx?article=OKO_TRA038#abstract.

10. **Benirschke SK**, Kramer PA. Open reduction and internal fixation of the tibial plafond. Atlas of essential orthopaedic procedures: AAOS; 2013. p. 437-42.

11. **Benirschke SK**, Meinberg E, Anderson SA, Jones CB, Cole PA. Fractures and dislocations of the midfoot: Lisfranc and Chopart injuries. The Journal of bone and joint surgery American volume. 2012 Jul 18;94(14):1325-37.

12. **Benirschke SK**, Rush SM, Reddix RN, Jr., Zwipp H, Carpenter B, Schuberth JM. Locking plates. Foot & ankle specialist. 2012 Feb;5(1):54-9.

13. Biau DJ, Weiss KR, Bhumbra RS, **Davidson D**, Brown C, Wunder JS, Ferguson PC. Using the CUSUM test to control the proportion of inadequate open biopsies of musculoskeletal tumors. Clin Orthop Relat Res. 2013 Mar;471(3):905-14.

14. Bishop J, Agel J, **Dunbar R**. Predictive factors for knee stiffness after periarticular fracture: a case-control study. J Bone Joint Surg Am. 2012 Oct 17;94(20):1833-8.

15. Bishop JA, Dikos GD, Mickelson D, **Barei DP**. Open reduction and intramedullary nail fixation of closed tibial fractures. Orthopedics. 2012 Nov;35(11):e1631-4.

16. Bozic KJ, Browne J, Dangles CJ, **Manner PA**, Yates AJ, Jr., Weber KL, Boyer KM, Zemaitis P, Woznica A, Turkelson CM, Wies JL. Modern metal-on-metal hip implants. The Journal of the American Academy of Orthopaedic Surgeons. 2012 Jun;20(6):402-6.

17. **Bransford RJ**, Dekutoski M. [Does MIS in thoracolumbar fracture care really improve outcome?]. Der Unfallchirurg. 2012 Dec;115(12):1061-5.

18. **Bransford RJ**, Freeborn MA, Russo AJ, Nguyen QT, **Lee MJ**, **Chapman JR**, **Bellabarba C**. Accuracy and complications associated with posterior C1 screw fixation techniques: a radiographic and clinical assessment. The spine journal : official journal of the North American Spine Society. 2012 Mar;12(3):231-8.

19. **Bransford RJ**, Koller H, Caron T, Zenner J, Hitzl W, Tomasino A, Mayer M. Cervical Spine Trauma in Diffuse Idiopathic Skeletal Hyperostosis (DISH) - Injury Characteristics and Outcome with Surgical Treatment. Spine. 2012 Apr 24.

20. Bugbee WD, Khanna G, Cavallo M, McCauley JC, Gortz S, **Brage ME**. Bipolar Fresh Osteochondral Allografting of the Tibiotalar Joint. The Journal of Bone & Joint Surgery. 2013;95(5):426-32.

21. Carofino BC, **Leopold SS**. Classifications in brief: the Neer classification for proximal humerus fractures. Clinical orthopaedics and related research. 2013 Jan;471(1):39-43.

22. **Cavanagh P**, Attinger C, Abbas Z, Bal A, Rojas N, Xu ZR. Cost of treating diabetic foot ulcers in five different countries. Diabetes/metabolism research and reviews. 2012 Feb;28 Suppl 1:107-11.

23. **Chapman J**, Smith JS, Kopjar B, Vaccaro AR, Arnold P, Shaffrey CI, Fehlings MG. The AOSpine North America Geriatric Odontoid Fracture Mortality Study: A Retrospective Review of Mortality Outcomes for Operative versus Non-Operative Treatment in 322 Patients with Long-Term Follow-Up. Spine. 2013 Jan 24.

24. **Chapman JR**. [Perspectives of spine trauma]. Der Unfallchirurg. 2012 Dec;115(12):1054-5.

25. **Chapman JR**, Riew D. Cervical artificial disc replacement: still experimental? Introduction and perspectives on cervical artificial disc replacement. Evidence-based spine-care journal. 2012 Feb;3(S1):5-8.

26. Chrisman SP, **O'Kane JW**, Polissar NL, **Tencer AF**, Mack CD, Levy MR, Schiff MA. Strength and jump biomechanics of elite and recreational female youth soccer players. Journal of athletic training. 2012;47(6):609-15.

27. Cizik AM, **Lee MJ**, Martin BI, **Bransford RJ**, **Bellabarba C**, **Chapman JR**, Mirza SK. Using the spine surgical invasiveness index to identify risk of surgical site infection: a multivariate analysis. The Journal of bone and joint surgery American volume. 2012 Feb 15;94(4):335-42.

28. Dombroski D, Scolaro JA, Pulos N, **Beingessner DM**, **Dunbar RP**, Mehta S. Fibula Fracture Stabilization With a Guide Wire as Supplementary Fixation in Tibia Fractures. Am J Orthopedics. 2012;41(5):157-60.

29. **Dunbar RP, Jr**. The realities of relationship failure. Journal of orthopaedic trauma. 2012 Sep;26 Suppl 1:S32-3.

30. Ehsan A, **Hanel DP**. Recurrent or Persistent Cubital Tunnel Syndrome. The Journal of hand surgery. 2012 Feb 1.

31. Ehsan A, **Huang JI**, Lyons M, **Hanel DP**. Surgical management of posttraumatic elbow arthrofibrosis. The journal of trauma and acute care surgery. 2012 May;72(5):1399-403.

32. Evans AR, **Routt ML, Jr**., **Nork SE**, Krieg JC. Oblique distraction external pelvic fixation. Journal of orthopaedic trauma. 2012 May;26(5):322-6.

33. **Eyre DR**, Weis MA. Bone Collagen: New Clues to Its Mineralization Mechanism from Recessive Osteogenesis Imperfecta. Calcified tissue international. 2013 Mar 19.

34. Fehlings MG, Smith JS, Kopjar B, Arnold PM, Yoon ST, Vaccaro AR, Brodke DS, Janssen ME, **Chapman JR**, Sasso RC, Woodard EJ, Banco RJ, Massicotte EM, Dekutoski MB, Gokaslan ZL, Bono CM, Shaffrey CI. Perioperative and delayed complications associated with the surgical treatment of cervical spondylotic myelopathy based on 302 patients from the AOSpine North America Cervical Spondylotic Myelopathy Study. Journal of neurosurgery Spine. 2012 May;16(5):425-32.

35. **Firoozabadi R**, Kramer PA, **Benirschke SK**. Technical Trick: Kirschner wire (K-wire) bending. J Orthop Trauma. 2013 Mar 19.

36. **Firoozabadi R**, Kramer PA, **Benirschke SK**. Plantar Medial Wounds Associated With Calcaneal Fractures. Foot & ankle international / American Orthopaedic Foot and Ankle Society [and] Swiss Foot and Ankle Society. 2013 Mar 11.

37. **Gee AO**, Kinsella S, Huffman GR, Sennett BJ, Tjoumakaris FP. Anterior cruciate ligament reconstruction in patients aged > 40 years: a case-control study. The Physician and sportsmedicine. 2013 Feb;41(1):30-4.

38. Gilmer BB, Comstock BA, Jette JL, **Warme WJ**, Jackins SE, **Matsen FA**. The prognosis for improvement in comfort and function after the ream-and-run arthroplasty for glenohumeral arthritis: an analysis of 176 consecutive cases. The Journal of bone and joint surgery American volume. 2012 Jul 18;94(14):e102.

39. Hacquebord J, Cizik AM, Malempati SH, Konodi MA, **Bransford RJ**, **Bellabarba C**, **Chapman J**, **Lee MJ**. Medicaid Status is Associated With Higher Complication Rates After Spine Surgery. Spine. 2013 Apr 15.

40. Hacquebord JH, **Leopold SS**. In Brief: The Risser Classification: A Classic Tool for the Clinician Treating Adolescent Idiopathic Scoliosis. Clinical orthopaedics and related research. 2012 Apr 27.

41. Hahn ME, Wright ES, Segal AD, Orendurff MS, Ledoux WR, **Sangeorzan BJ**. Comparative gait analysis of ankle arthrodesis and arthroplasty: initial findings of a prospective study. Foot & ankle international / American Orthopaedic Foot and Ankle Society [and] Swiss Foot and Ankle Society. 2012 Apr;33(4):282-9.

42. Harvey E, Audige L, Herscovici D, Jr., Agel J, Madsen JE, Babst R, **Nork S**, Kellam J. Development and validation of the new international classification for scapula fractures. Journal of orthopaedic trauma. 2012 Jun;26(6):364-9.

43. **Henley MB**. Finding your ideal job and negotiating your contract: where to get the information and numbers you need to know. Journal of orthopaedic trauma. 2012 Sep;26 Suppl 1:S9-S13.

44. **Huang JI**, **Hanel DP**. Anatomy and Biomechanics of the Distal Radioulnar Joint. Hand Clinics. 2012 May 2012;28(2):157-63.

45. Hudson DM, Kim LS, Weis M, Cohn DH, **Eyre DR**. Peptidyl 3-hydroxyproline binding properties of type I collagen suggest a function in fibril supramolecular assembly. Biochemistry. 2012 Mar 27;51(12):2417-24.

46. Iannuzzi NP, **Leopold SS**. In Brief: The Mason Classification of Radial Head Fractures. Clinical orthopaedics and related research. 2012 Mar 17.

47. Isvilanonda V, Dengler E, Iaquinto JM, **Sangeorzan BJ**, Ledoux WR. Finite element analysis of the foot: model validation and comparison between two common treatments of the clawed hallux deformity. Clinical biomechanics. 2012 Oct;27(8):837-44.

48. Jacobs CR, Huang H, **Kwon RY**. Introduction to Cell Mechanics and Mechanobiology. 1st edition ed: Garland Science; 2012.

49. Karandikar M, Mirza SK, Song K, Yang T, **Krengel WF, 3rd**, Spratt KF, Avellino AM. Complex pediatric cervical spine surgery using smaller nonspinal screws and plates and intraoperative computed tomography. Journal of neurosurgery Pediatrics. 2012 Jun;9(6):594-601.

50. Kazemi N, **Bellabarba C**, **Bransford R**, Vilela M. Incidence of blunt cerebrovascular injuries associated with craniocervical distraction injuries. Evidence-based spine-care journal. 2012 Nov;3(4):63-4.

51. **Kennedy SA**, **Allan CH**. In brief: Mayfield et al. Classification: carpal dislocations and progressive perilunar instability. Clinical orthopaedics and related research. 2012 Apr;470(4):1243-5.

52. **Kennedy SA**, **Hanel DP**. Complex distal radius fractures. The Orthopedic clinics of North America. 2013 Jan;44(1):81-92.

53. Kim PH, **Leopold SS**. In brief: Gustilo-Anderson classification. [corrected]. Clinical orthopaedics and related research. 2012 Nov;470(11):3270-4.

54. Koehler RJ, Amsdell S, Arendt EA, Bisson LJ, Bramen JP, Butler A, Cosgarea AJ, Harner CD, Garrett WE, Olson T, **Warme WJ**, Nicandri GT. The Arthroscopic Surgical Skill Evaluation Tool (ASSET). The American journal of sports medicine. 2013 Apr 2.

55. Kollitz KM, **Gee AO**. Classifications in Brief: The Herring Lateral Pillar Classification for Legg-Calve-Perthes Disease. Clin Orthop Relat Res. 2013 Apr 20.

56. **Krengel WF, 3rd**, Wiater BP, Pace JL, Jinguji TM, Bompadre V, Stults JK, **Schmale GA**. Does using the medial or lateral humeral line improve reliability of Baumann angle measurement on plain x-ray? The effect of humeral length visualized on the x-ray. Journal of pediatric orthopedics. 2012 Jun;32(4):373-7.

57. Kumar A, Donley B, **Cavanagh PR**. Design of an implant for first metatarsophalangeal hemi-arthroplasty. Computer methods in biomechanics and biomedical engineering. 2013 Mar 12.

58. **Kwon RY**, Meays DR, Meilan AS, Jones J, Miramontes R, Kardos N, Yeh J-C, Frangos JA. Skeletal Adaptation to Intramedullary Pressure-Induced Interstitial Fluid Flow Is Enhanced in Mice Subjected to Targeted Osteocyte Ablation. PloS one. 2012;7(3):e33336.

59. **Lee MJ**, Dettori JR, Standaert CJ, Brodt ED, **Chapman JR**. The natural history of degeneration of the lumbar and cervical spines: a systematic review. Spine. 2012 Oct 15;37(22 Suppl):S18-30.

60. **Lee MJ**, Dettori JR, Standaert CJ, Ely CG, **Chapman JR**. Indication for spinal fusion and the risk of adjacent segment pathology: does reason for fusion affect risk? A systematic review. Spine. 2012 Oct 15;37(22 Suppl):S40-51.

61. **Lee MJ**, Konodi MA, Cizik AM, **Bransford RJ**, **Bellabarba C**, **Chapman JR**. Risk factors for medical complication after spine surgery: a multivariate analysis of 1,591 patients. The spine journal : official journal of the North American Spine Society. 2012 Mar;12(3):197-206.

62. **Lee MJ**, Konodi MA, Cizik AM, Weinreich MA, **Bransford RJ**, **Bellabarba C**, Chapman J. Risk factors for medical complication after cervical spine surgery: a multivariate analysis of 582 patients. Spine. 2013 Feb 1;38(3):223-8.

63. **Leopold SS**. Association or causation? A common misunderstanding in clinical research. J Bone Joint Surg Am. 2012 May 2;94(9):1-3.

64. **Leopold SS**. Words and Images (Editorial). J Bone Joint Surg Am. 2012;94(1-3).

65. **Leopold SS**. AAOS or ACCP? Still No Answers on Thromboprophylaxis. J Bone Joint Surg Am. 2012;94(1-3).

66. **Leopold SS**. Seek Pseudotumors and You Shall Find. JBJS Orthopaedic Highlights: Hip Surgery. 2012 Jul 03;7(5):e3.

67. **Leopold SS**. Losing Our Bearings. JBJS Orthopaedic Highlights: Hip Surgery. 2012 Jun 20;2(6):e9-e.

68. **Leopold SS**. Big surgery – knowing when to say when. J Bone Joint Surg Am. 2012;94(1-3).

69. **Leopold SS**. Editor's Spotlight/Take 5: A Review of Current Fixation Usage and Registry Outcomes in Total Hip Arthroplasty: The Uncemented Paradox [DOI 10.1007/s11999-013-2941-7]. Clinical orthopaedics and related research. 2013 Apr 30.

70. **Leopold SS**. Editor's Spotlight/Take 5: Orthopaedic Surgeons Frequently Underestimate the Cost of Orthopaedic Implants (DOI 10.1007/s11999-012-2757-x). Clinical orthopaedics and related research. 2013 Jun;471(6):1741-3.

71. **Leopold SS**. Editorial: duplicate submission and dual publication: what is so wrong with them? Clinical orthopaedics and related research. 2013 May;471(5):1401-2.

72. **Leopold SS**. Editor's Spotlight/Take 5: Comparative Responsiveness and Minimal Clinically Important Differences for Idiopathic Ulnar Impaction Syndrome (DOI 10.1007/s11999-013-2843-8). Clinical orthopaedics and related research. 2013 May;471(5):1403-5.

73. **Leopold SS**. Editorial: orthopaedics, advocacy, action. Clinical orthopaedics and related research. 2013 Apr;471(4):1067-8.

74. **Leopold SS**. Editorial: Research is a team sport: updated authorship guidelines for CORR. Clinical orthopaedics and related research. 2013 Mar;471(3):701-2.

75. **Leopold SS**. Editor's Spotlight/Take 5: Novel 3-D quantification and classification of cam lesions in patients with femoroacetabular impingement (DOI 10.1007/s11999-012-2693-9). Clinical orthopaedics and related research. 2013 Feb;471(2):355-7.

76. **Leopold SS**. Editor's Spotlight/Take 5: Prophylactic stabilization for bone metastases, myeloma, or lymphoma: do we need to protect the entire bone? (DOI 10.1007/s11999-012-2656-1). Clinical orthopaedics and related research. 2013 Mar;471(3):703-5.

77. **Leopold SS**. Editorial: Let's talk about Level IV: The bones of a good retrospective case series. Clinical orthopaedics and related research. 2013 Feb;471(2):353-4.

78. **Leopold SS**. Editorial: Sharing Some Goals. Clin Orthop. 2013;471(2-3).

79. **Leopold SS**. Editor's Spotlight/Take 5: Have Bilateral Total Knee Arthroplasties Become Safer? A Population-Based Trend Analysis. Clin Orthop. 2013;471(13-16).

80. **Leopold SS**. Editor's Spotlight/Take 5: Orthopaedic surgeons' knowledge and misconceptions in the identification of intimate partner violence against women. Clin Orthop. 2013(471):1069-73.

81. **Leopold SS**. Growth plate alteration precedes cam-type deformity in elite basketball players. Clin Orthop. 2013(471):1081-3.

82. **Leopold SS**, Porcher R. Editorial: reporting statistics in abstracts in clinical orthopaedics and related research ((R)). Clinical orthopaedics and related research. 2013 Jun;471(6):1739-40.

83. Lewis R, Ravindran S, Wirthlin L, Traeger G, **Fernandes RJ**, McAlinden A. Disruption of the developmentally-regulated Col2a1 pre-mRNA alternative splicing switch in a transgenic knock-in mouse model. Matrix biology : journal of the International Society for Matrix Biology. 2012 Apr;31(3):214-26.

84. Lohse GR, **Leopold SS**, Theiler S, Sayre C, Cizik A, **Lee MJ**. Systems-based safety intervention: reducing falls with injury and total falls on an orthopaedic ward. The Journal of bone and joint surgery American volume. 2012 Jul 3;94(13):1217-22.

85. Loraas EK, **Schmale GA**. Endoscopically aided physeal bar takedown and guided growth for the treatment of angular limb deformity. Journal of pediatric orthopedics Part B. 2012 Jul;21(4):348-51.

86. Malone KJ, Martineau PA, **Hanel DP**. Ulnar collateral ligament reconstruction in posttraumatic elbow release. Orthopedics. 2012;35(2):e202-7.

87. **Manner P**. Minimally invasive hip surgery. Orthopedics. 2012 Feb 1;35(2):123-4.

88. **Manner P**. Twist and turn: commentary on an article by Robert S. Namba, MD, et al.: "risk of revision for fixed versus mobile-bearing primary total knee replacements". The Journal of bone and joint surgery American volume. 2012 Nov 7;94(21):e162.

89. **Manner P**. A spoonful of sugar: commentary on an article by Annette L. Adams, PhD, MPH, et al.: "surgical outcomes of total knee replacement according to diabetes status and glycemic control, 2001 to 2009". The Journal of bone and joint surgery American volume. 2013 Mar 20;95(6):e371-2.

90. Martinez-Glez V, Valencia M, Caparros-Martin JA, Aglan M, Temtamy S, Tenorio J, Pulido V, Lindert U, Rohrbach M, **Eyre D**, Giunta C, Lapunzina P, Ruiz-Perez VL. Identification of a mutation causing deficient BMP1/mTLD proteolytic activity in autosomal recessive osteogenesis imperfecta. Human mutation. 2012 Feb;33(2):343-50.

91. **Matsen FA, 3rd**, Jette JL, Neradilek MB. Demographics of disclosure of conflicts of interest at the 2011 annual meeting of the American Academy of Orthopaedic Surgeons. The Journal of bone and joint surgery American volume. 2013 Mar 6;95(5):e29.

92. **Matsen FA, 3rd**, Stephens L, Jette JL, **Warme WJ**, Posner KL. Lessons regarding the safety of orthopaedic patient care: an analysis of four hundred and sixty-four closed malpractice claims. The Journal of bone and joint surgery American volume. 2013 Feb 20;95(4):e201-8.

93. Miller AN, **Barei DP**, Iaquinto JM, Ledoux WR, **Beingessner DM**. Iatrogenic Syndesmosis Malreduction Via Clamp and Screw Placement. Journal of orthopaedic trauma. 2012 Apr 28.

94. Miller AN, **Beingessner DM**. Intra-articular distal humerus fractures. The Orthopedic clinics of North America. 2013 Jan;44(1):35-45.

95. **Mosca VS**, Bevan WP. Talocalcaneal tarsal coalitions and the calcaneal lengthening osteotomy: the role of deformity correction. The Journal of bone and joint surgery American volume. 2012 Sep 5;94(17):1584-94.

96. Moustafa A, Sugiyama T, Prasad J, Zaman G, **Gross TS**, Lanyon LE, Price JS. Mechanical loading-related changes in osteocyte sclerostin expression in mice are more closely associated with the subsequent osteogenic response than the peak strains engendered. Osteoporosis international : a journal established as result of cooperation between the European Foundation for Osteoporosis and the National Osteoporosis Foundation of the USA. 2012 Apr;23(4):1225-34.

97. Pace JL, Wiater B, **Schmale G**, Jinguji T, Bompadre V, **Krengel W, 3rd**. Baumann angle and radial-ulnar overlap: a radiographic study to control for the angle of the x-ray beam. Journal of pediatric orthopedics. 2012 Jul-Aug;32(5):467-72.

98. Papadonikolakis A, McKenna M, **Warme WJ**, **Matsen FA**. Intramedullary fibular and impaction allografting in revision total elbow arthroplasty with endosteal deficiency. Techniques in hand & upper extremity surgery. 2012 Mar;16(1):5-11.

99. Parada SA, Krieg JC, **Benirschke SK**, **Nork SE**. Bicortical fixation of medial malleolar fractures. American journal of orthopedics. 2013 Feb;42(2):90-2.

100. Pollack SM, Li Y, Blaisdell MJ, Farrar EA, Chou J, Hoch BL, Loggers ET, Rodler E, Eary JF, **Conrad EU, 3rd**, Jones RL, Yee C. NYESO-1/LAGE-1s and PRAME are targets for antigen specific T cells in chondrosarcoma following treatment with 5-Aza-2-deoxycitabine. PloS one. 2012;7(2):e32165.

101. Pottinger P, Butler-Wu S, Neradilek MB, Merritt A, Bertelsen A, Jette JL, **Warme WJ**, **Matsen FA, 3rd**. Prognostic factors for bacterial cultures positive for Propionibacterium acnes and other organisms in a large series of revision shoulder arthroplasties performed for stiffness, pain, or loosening. The Journal of bone and joint surgery American volume. 2012 Nov 21;94(22):2075-83.

102. Richard MJ, Katolik LI, **Hanel DP**, Wartinbee DA, Ruch DS. Distraction plating for the treatment of highly comminuted distal radius fractures in elderly patients. The Journal of hand surgery. 2012 May;37(5):948-56.

103. Roberts TT, Prummer CM, Papaliodis DN, Uhl RL, **Wagner TA**. History of the orthopedic screw. Orthopedics. 2013 Jan;36(1):12-4.

104. Rubin CT, Seeherman H, Qin Y-X, **Gross TS**. The mechanical consequences of load bearing in the equine third metacarpal across speed and gait: the nonuniform distributions of normal strain, shear strain, and strain energy density. The FASEB Journal. 2013;27(5):1887-94.

105. Sanders JO, Jevsevar DS, **Goldberg MJ**, Weber KL. Quality guidelines need evidence, not opinion: commentary on an article by Robert A. Pedowitz, MD, PhD, et al.: "American Academy of Orthopaedic Surgeons Clinical Practice Guideline on optimizing the management of rotator cuff problems". The Journal of bone and joint surgery American volume. 2012 Jan 18;94(2):e14.

106. Sanyal A, Gupta A, Bayraktar HH, **Kwon RY**, Keaveny TM. Shear strength behavior of human trabecular bone. Journal of biomechanics. 2012;45(15):2513-19.

107. Schaefer KL, **Sangeorzan BJ**, Fassbind MJ, Ledoux WR. The comparative morphology of idiopathic ankle osteoarthritis. The Journal of bone and joint surgery American volume. 2012 Dec 19;94(24):e181.

108. Schwarze U, Cundy T, Pyott SM, Christiansen HE, Hegde MR, Bank RA, Pals G, Ankala A, Conneely K, Seaver L, Yandow SM, Raney E, Babovic-Vuksanovic D, Stoler J, Ben-Neriah Z, Segel R, Lieberman S, Siderius L, Al-Aqeel A, Hannibal M, Hudgins L, McPherson E, Clemens M, Sussman MD, Steiner RD, Mahan J, Smith R, Anyane-Yeboa K, Wynn J, Chong K, Uster T, Aftimos S, Sutton VR, Davis EC, Kim LS, Weis MA, **Eyre D**, Byers PH. Mutations in FKBP10, which result in Bruck syndrome and recessive forms of osteogenesis imperfecta, inhibit the hydroxylation of telopeptide lysines in bone collagen. Human molecular genetics. 2013 Jan 1;22(1):1-17.

109. Segal AD, Shofer J, Hahn ME, Orendurff MS, Ledoux WR, **Sangeorzan BJ**. Functional limitations associated with end-stage ankle arthritis. The Journal of bone and joint surgery American volume. 2012 May 2;94(9):777-83.

110. Sheiko M, Bjornson K, Lisle J, Song K, Eary JF, **Conrad EU, 3rd**. Physical activity assessment in adolescents with limb salvage. The Journal of pediatrics. 2012 Dec;161(6):1138-41.

111. Solanki GA, Alden TD, Burton BK, Giugliani R, Horovitz DD, Jones SA, Lampe C, Martin KW, Ryan ME, Schaefer MK, Siddiqui A, **White KK**, Harmatz P. A multinational, multidisciplinary consensus for the diagnosis and management of spinal cord compression among patients with mucopolysaccharidosis VI. Molecular genetics and metabolism. 2012 Sep;107(1-2):15-24.

112. Squyer E, Stamper DL, Hamilton DT, Sabin JA, **Leopold SS**. Unloader knee braces for osteoarthritis: do patients actually wear them? Clinical orthopaedics and related research. 2013 Jun;471(6):1982-91.

113. **Srinivasan S**, **Gross TS**, **Bain SD**. Bone mechanotransduction may require augmentation in order to strengthen the senescent skeleton. Ageing research reviews. 2012 Jan 5.

114. Stern PJ, Albanese S, Bostrom M, Day CS, Frick SL, Hopkinson W, Hurwitz S, Kenter K, Kirkpatrick JS, Marsh J, Murthi A, **Taitsman L**, Toolan B, Weber K, Wright R, Derstine P, Edgar L. Orthopaedic Surgery Milestones. Journal of Graduate Medical Education. 2013 March 2013;5(1s1):36-58.

115. Takagi M, Ishii T, Barnes AM, Weis M, Amano N, Tanaka M, Fukuzawa R, Nishimura G, **Eyre DR**, Marini JC. A Novel Mutation in LEPRE1 That Eliminates Only the KDEL ER-Retrieval Sequence Causes Non-Lethal Osteogenesis Imperfecta. PloS one. 2012;7(5):e36809.

116. Tsai TL, **Manner PA**, Li WJ. Regulation of mesenchymal stem cell chondrogenesis by glucose through protein kinase C/transforming growth factor signaling. Osteoarthritis and cartilage / OARS, Osteoarthritis Research Society. 2013 Feb;21(2):368-76.

117. Vining NC, Done S, Glass IA, Parnell SE, Sternen DL, Leppig KA, **Mosca VS**, **Goldberg MJ**. EXT2-positive multiple hereditary osteochondromas with some features suggestive of metachondromatosis. Skeletal radiology. 2012 May;41(5):607-10.

118. Vining NC, **Warme WJ**, **Mosca VS**. Comparison of structural bone autografts and allografts in pediatric foot surgery. Journal of pediatric orthopedics. 2012 Oct-Nov;32(7):719-23.

119. Wang VM, Bell RM, Thakore R, **Eyre DR**, Galante JO, Li J, Sandy JD, Plaas A. Murine tendon function is adversely affected by aggrecan accumulation due to the knockout of ADAMTS5. Journal of orthopaedic research : official publication of the Orthopaedic Research Society. 2012 Apr;30(4):620-6.

120. **Warme WJ**. CORR Insights: Surgical Technique: Results of Stabilization of Sternoclavicular Joint Luxations Using a Polydioxanone Envelope Plasty. Clinical orthopaedics and related research. 2013 Apr 9.

121. Weber JR, Aubin PM, Ledoux WR, **Sangeorzan BJ**. Second metatarsal length is positively correlated with increased pressure and medial deviation of the second toe in a robotic cadaveric simulation of gait. Foot & ankle international / American Orthopaedic Foot and Ankle Society [and] Swiss Foot and Ankle Society. 2012 Apr;33(4):312-9.

122. Wein MN, Jones DC, Shim JH, Aliprantis AO, Sulyanto R, Lazarevic V, Poliachik SL, **Gross TS**, Glimcher LH. Control of bone resorption in mice by Schnurri-3. Proc Natl Acad Sci U S A. 2012 May 22;109(21):8173-8.

123. **White KK**. Mucopolysaccharidoses: Etiology, Classification, Deformities Unique to Each Type, Treatment Modalities and Indications for Surgical Intervention. Spine Deformity. 2012.

124. **White KK**. Orthopaedic Surgery for Mucopolysaccharidosis. Current Orthopaedic Practice. 2012;23(5):394-9.

125. **White KK**, Sousa T. Mucopolysaccharide disorders in orthopaedic surgery. J Am Acad Orthop Surg. 2013 Jan;21(1):12-22.

126. Wood WG, Guy TS, Arora K, **Firoozabadi R**, Kim H, Kukreja J, Pagel PS, Brzezinski M. Posterior glenohumeral joint dislocation: a rare complication of central venous cannulation in a patient undergoing coronary artery surgery. J Cardiothorac Vasc Anesth. 2013 Feb;27(1):101-2.

127. Yang L, Lawson KA, Teteak CJ, Zou J, Hacquebord J, Patterson D, Ghatan AC, Mei Q, Zielinska-Kwiatkowska A, **Bain SD**, **Fernandes RJ**, **Chansky HA**. ESET histone methyltransferase is essential to hypertrophic differentiation of growth plate chondrocytes and formation of epiphyseal plates. Dev Biol. 2013 May 4.

128. Zeltser DW, **Leopold SS**. Classifications in brief: Schatzker classification of tibial plateau fractures. Clinical orthopaedics and related research. 2013 Feb;471(2):371-4.

Alumni

In appreciation of the generous support over the years from the University of Washington Orthopaedic Alumni to fund Orthopaedic Resident Research and Education

1952
Park W. Gloyd, MD ★

1954
Trygve Forland, MD ★

1955
Robert W. Florence, MD

1956
J. Michael Egglin, MD ★
John E. Goeckler, MD
Robert L. Romano, MD ★

1957
John H. Aberle, MD ★★
John R. Beebe, MD

1958
Harry H. Kretzler, Jr., MD ★
James R. Friend, MD ★
Kenneth L. Martin, MD ★
Samuel L. Clifford, MD

1959
James W. Tupper, MD

1960
Irving Tobin, MD ★
William V. Smith, MD ★

1961
Robert C. Colburn, MD ★

1962
Arthur Ratcliffe, MD
Marr P. Mullen, MD ★★★★

1963
Alfred I. Blue, MD
Robert A. Kraft, MD

1964
David E. Karges, MD ★★★★★
Harold J. Forney, MD ★★
Theodore K. Greenlee II, MD ★★★★★★★
Thomas E. Soderberg, MD

1966
F. Richard Convery, MD ★
Joseph S. Mezistrano, MD ★
William A. Reilly, Jr., MD

1967
Ivar W. Birkeland, MD ★★
J. Conrad Clifford, MD ★
Robert F. Smith, MD ★★★★★

1968
Lynn T. Staheli, MD ★
Stewart M. Scham, MD ★★
William T. Thieme, MD ★★

1969
Edward E. Almquist, MD ★★★
Edward L. Lester, MD ★
Hugh E. Toomey, MD ★★★
Sigvard T. Hansen, Jr., MD ★★★★★★

1970
John C. Brown, MD ★★
John M. Coletti, Jr., MD ★
Malcolm B. Madenwald, MD ★★
Michael T. Phillips, MD ★
Robert D Schrock, Jr., MD

1971
Bruce E. Bradley, Jr., MD ★
Franklin G. Alvine, MD ★★★★★
Jerome H. Zechmann, MD
Louis A. Roser, MD ★
Nils Fauchald, Jr., MD

1972
David J. LaGasse, MD ★
David R. Nank, MD ★★
Donald D. Hubbard, MD ★
John A. Neufeld, MD ★★
Thomas L. Gritzka, MD ★

1973
Frederick J. Davis, MD ★
Larry D. Hull, MD ★
Robert P. Watkins, Jr., MD ★
Theodore A. Wagner, MD ★★★★★★

1974
Richard A. Dimond, MD ★★
Ronald B.H. Sandler, MD ★★★★
Samuel R. Baker, MD ★★★★
Robert A. Winquist, MD ★★★★★★★

1975
Donald L. Plowman, MD ★★★★
Frederick A. Matsen III, MD ★★★★★★★
Gunter Knittel, MD
Larry R. Pedegana, MD ★★
Thomas M. Green, MD ★★★★★★
William M. Backlund, MD, P.S. ★

1976
Douglas K. Kehl, MD
Douglas T. Davidson III, MD ★
John F. Burns, MD ★
Peter Melcher, MD
Richard A. Zorn, MD ★

1977
Carl A. Andrews, MD ★
Geoffrey W. Sheridan, MD ★★★
Larry D. Iversen, MD ★
Mark C. Olson, MD ★★
Steven T. Bramwell, MD

1978
Arnold G. Peterson, MD ★★★★
Gary J. Clancey, MD ★★★★★
John W. Brantigan, MD
Richard S. Westbrook, MD ★★
Robert J. Strukel, MD
William Oppenheim, MD ★★

1979
Allan W. Bach, MD ★★★★★★
Gregory M. Engel, MD ★★
Jonathan L. Knight, MD ★★
Richard L. Semon, MD ★★★★

1980
Carol C. Teitz, MD ★★★★
Douglas G. Norquist, MD ★
John M. Hendrickson, MD ★★★
Michael A. Sousa, MD ★★★★★★
Stuart R. Hutchinson, MD ★

1981
Dennis J. Kvidera, MD ★
John M. Clark, Jr., MD, PhD ★★★
Martin S. Tullus, MD ★★★★★★
Robert G. Veith, MD ★★★★★★★

1982
John L. Thayer, MD ★★
Richard M. Kirby, MD ★★★★★★★
Steven S. Ratcliffe, MD ★★★★
William D. Burman, MD

1983
Elizabeth Anne Ouellette, MD ★★
Edward L. Farrar III, MD ★★★★★
Henry K. Yee, MD ★
Joseph D. Zuckerman, MD ★★★★★
Keith A. Mayo, MD ★★★★★★
Robert M. Berry, MD ★★★★

1984
Jeffrey C. Parker, MD ★
Jeffrey W. Akeson, MD ★★★★
Kevin P. Schoenfelder, MD ★
Marc F. Swiontkowski, MD
★★★★★★★
Thomas J. Fischer, MD ★★★★★★

1985
Daniel L. Flugstad, MD ★★★★★
Jeffrey N. Hansen, MD ★★★
Paul J. Abbott, MD ★★★★
Richard J. Barry, MD ★★
William P. Barrett, MD ★★★★★

1986
Carleton A. Keck, Jr., MD ★★★
Gary Bergman, MD ★★★★★★
Lawrence E. Holland, MD ★★
Michael E. Morris, MD ★★★★

1987
Craig T. Arntz, MD ★★★
Herbert R. Clark, MD ★★
Michael K. Gannon, MD ★★
Steven L. Reed, MD ★

1988
Jonathan L. Franklin, MD ★★★★★
Michael A. Thorpe, MD ★★★★★★
Richard V. Williamson, MD ★

1989
James P. Crutcher, MD ★★★★★
Lawrence V. Page, DO ★★★
Martin G. Mankey, MD ★★★★★
Nancy J. Ensley, MD
Steve C. Thomas, MD ★★★★★

1990
David M. Kieras, MD ★
J. Roberto R. Carreon, MD
Jay A. Winzenried, MD ★★★
Ken Fujii, MD ★
Walter F. Krengel III, MD ★★★★

1991
David H. Bishop, MD ★★
Kit M. Song, MD
Mark Remington, MD ★★★★
Mark E. Murphy, MD, Ph.D. ★★
Tim P. Lovell, MD ★★

1992
Curt Rodin, MD
Don Striplin, MD ★★
Eli Powell, MD ★
Jeff Stickney, MD ★★
John D. West, MD ★★
Michael Sailer, MD ★★★★

1993
J. Eric Vanderhooft, MD ★★★★★
Lyle S. Sorensen, MD ★★★★★★★
Philip J. Kregor, MD ★★
Susan R. Cero, MD ★★★★★★

1994
Brodie Wood, MD ★★★★★★★
Eric Bowton, MD ★★
Jim Vahey, MD ★
Sohail K. Mirza, MD ★
William Obremskey, MD ★★★★★★

1995
Ron Kristensen, MD ★★★★
Scott Hormel, MD ★★★
Timothy Beals, MD ★★
Todd Clarke, MD ★★★★
William J. Mills III, MD ★★★

1996
David Deneka, MD ★★★
Peter Mitchell, MD ★★★★
Peter T. Simonian, MD ★★★★
Vernon Cooley, MD ★★
William Wagner, MD ★★★★

1997
Daniel Stechschulte, Jr., MD
★★★★★★★
David Levinsohn, MD ★★
L. Anthony Agtarap, MD ★
Mohammad Diab, MD
Randall W. Viola, MD

1998
Colin Poole, MD ★
David Belfie, MD ★
Don Ericksen, MD ★★★★★
Jay Crary, MD ★★★★★★
Oriente DiTano, MD ★

1999
Craig Boatright, MD
Jeffrey Garr, MD ★
John Michelotti, MD ★★
Julie A. Switzer, MD ★
Thomas D. Chi, MD ★

2000
Brett Quigley, MD ★
Cara Beth Lee, MD ★
Daniel Jones, MD ★
Joel Hoekema, MD ★★★
Patrick McNair, MD

2001
Eric Novack, MD
Frederick Huang, MD ★★★★
Matthew Camuso, MD
Michael Metcalf, MD ★★★★★
Richard Bransford, MD ★

2002
Timothy DuMontier, MD ★★
Scott Hacker, MD ★
Timothy Rapp, MD ★
William Sims, MD ★
Carla Smith, MD ★★

2003
Ben DuBois, MD ★★★
Andy Howlett, MD ★
Guy Schmidt, MD ★★
Brian Shafer, MD ★
Emma Woodhouse, MD ★

2004
Jon Braman, MD ★
Alexis Falicov, MD ★
Mike McAdam, MD ★
Jason H. Thompson, MD ★★
Thea W. Khan-Farooqi, MD

2005
Anthony Buoncristiani, MD ★
Waqqar Khan-Farooqi, MD
Wren McCallister, MD
Timothy O'Mara, MD ★★
David W. Stevens, MD ★

2006
Heidi Shors, MD ★
Stacey Donion, MD
Eric Klineberg, MD ★
Bill Montgomery, MD ★
Mel Wahl, MD ★
Burt Yaszay, MD ★

2007
Jamie Antoine, MD ★
Jeremiah Clinton, MD ★
Mary Cunningham, MD ★
Evan Ellis, MD ★
Joseph Lynch, MD ★
Allison MacLennan, MD ★

2008
Drew Fehsenfeld, MD ★
Mark Freeborn, MD ★★
Christopher Howe, MD ★
John Howlett, MD ★
Michael Lee, MD ★
Gregg Nicandri, MD ★

2009
Rajshri Maheshwari Bolson, MD ★
Jason King, MD ★
Annie Links, MD ★
Soren Olson, MD ★
Karen Perser, MD ★
Scott Ruhlman, MD ★
Addison Stone, MD ★
Jason Wilcox, MD ★

2010
Sean Amann, MD ★
Jeremy Bauer, MD ★
Aric Christal, MD ★
Wendy Emerson, MD ★
Michael Hwang, MD ★
Lee Pace, MD ★
Christopher Wolf, MD ★
Vinko Zlomislic, MD ★

2011
Aaron Chamberlain, MD ★
Brian Daines, MD ★
Cory Lamblin, MD ★
Edward Moon, MD ★
Derek Rains, MD ★
Peter Scheffel, MD ★
Christian Sybrowsky, MD ★
Brett Wiater, MD ★

2012
Benjamin Amis, MD ★
Adam Bakker, MD ★
Gregory Blaisdell, MD ★
Joshua Lindsey, MD ★
Grant Lohse, MD ★
Matthew Lyons, MD ★
Andrew Merritt, MD ★
Nels Sampatacos, MD ★

2013
Kyle Chun, MD
Elizabeth Dailey, MD
Andrew Ghatan, MD
Brian Gilmer, MD
Jennifer Hagen, MD
Mark Miller, MD
David Patterson, MD
Emily Squyer, MD

STARS INDICATE TOTAL DONATIONS IN SUPPORT OF THE RESIDENCY

★★★★★★★ = $20,000 and above
★★★★★★ = $15,000 - $19,999
★★★★★ = $10,000 - $14,999
★★★★ = $7,500 - $9,999
★★★ = $5,000 - $7,499
★★ = $2,500 - $4,999
★ = $1 - $2,499

Endowments

We express our appreciation to all who have contributed to the endowments of the Department of Orthopaedics and Sports Medicine. This assistance makes possible special research activities, educational programs, and other projects that we could not offer without this extra support from our alumni, faculty, and friends in the community. In this day and age of funding cutbacks and decreased returns on investment, an endowment in the University of Washington continues to provide above market returns and is a crucial way to support advancement of musculoskeletal medicine. If you have any questions, please contact our Chairman, Jens R. Chapman, MD (jenschap@uw.edu), or our Director, Ken Karbowski (kkarb@uw.edu). Thank You!

Hansjörg Wyss Endowed Chair - Jens R. Chapman, MD

Hansjörg Wyss Endowed Chairs for the Advancement of Hip & Pelvis Surgery

Ernest M. Burgess Endowed Chair for Orthopaedics Investigation - David R. Eyre, PhD

Sigvard T. Hansen Jr. Endowed Chair in Orthopaedic Traumatology - Ted S. Gross, PhD

Jerome H. Debs II Endowed Chair in Orthopaedic Traumatology - Stephen K. Benirschke, MD

Bob and Sally Behnke Endowed Chair for the Health of the Student Athlete - John W. O'Kane, MD

Endowed Chair for Women's Sports Medicine and Lifetime Fitness - Peter R. Cavanagh, PhD, DSc

Surgical Dynamics Endowed Chair for Spine Research

Douglas T. Harryman II/DePuy Endowed Chair for Shoulder Research - Frederick A. Matsen III, MD

Synthes Spinal Surgery Outcomes Research Endowed Fund

Fracture Fixation Biology Endowed Professorship

Ostex Bone and Joint Research Endowment

Orthopaedic Traumatology Endowed Lectureship

John F. LeCocq Lectureship in Orthopaedic Surgery

Don and Carol James Research Fund in Sports Medicine and Fitness

Victor H. Frankel Endowed Award

Esther Whiting Award

Edwin L. Laurnen, M.D. Award

Spine Research Endowment

James G. Garrick Endowed Lectureship in Sports Medicine

Allan Treuer - Ted Wagner, M.D. Endowed Chair in Regenerative Spine Surgery

Richard and Betsy Kirby Orthopaedic Resident Endowed Fund

Huang-Biu Orthopaedic Resident Endowed Support Fund

Orthopaedic Resident Endowed Support Fund

Josh and Max Myers Endowed Orthopaedic Fellowship Fund

Sarcoma Oncology Endowed Fund

Clawson Family Orthopaedic Library Endowed Fund

Index

A

Agnew, Sam
16
Allan, Christopher
3,9,86,114,116
Amendola, Ned
15,26

B

Bain, Steven
9,76,113,115,119
Barei, David
9,62,76,113,114,115,118
Behnke, Bob and Sally
123
Beingessner, Daphne
9,76,115,116,118
Bellabarba, Carlo
9,16,113,114,115,116,117
Benirschke, Stephen
9,62,73,76,86,115,116,118,123
Bergholz, Theresa
3
Bigos, Stanley
12
Bompadre, Viviana
64,117,118
Bradt, Carrie
15,85
Brage, Michael
9,74,75,86,115
Bransford, Richard
9,85,114,115,116,117,121
Burgess, Ernest
123
Bykov, Alexandre
46

C

Cavanagh, Peter
3,9,46,48,79,80,82,85,113,
114,115,117,123
Chamberlain, Aaron
34,122
Chansky, Howard
9,16,18,36,38,49,58,61,80,81,
86,113,119
Chapman, Jens
3,9,16,68,79,80,82,87,98,
106,113,114,115,116,117,123
Chen, Michael
36,58
Chun, Kyle
107,122
Cizik, Amy
3,68,69,115,116,117,118
Clawson, D. Kay
99,100,101,102,103,104,105,
123
Coleman, Nathan
17,34
Comstock, Bryan
34,116
Conrad, Ernest
9,77,78,79,86,118,119

D

Dailey, Elizabeth
107,122
Dales, Mark
9,77,85
Davidson, Darin
9,78,79,98,115
Debs, Jerome
117
Downey, Leah
70,71
Dunbar, Robert
10,16,62,76,114,115,116

E

Eastman, Jonathan
62
Eyre, David
10,29,30,33,55,57,86,113,114,
115,116,118,119,123

F

Farnand, Alex
87
Farrar, Edward
5,121
Fernandes, Russell
10,27,29,33,113,117,119
Firoozabadi, Reza
6,10,39,52,62,76,116,119
Frankel, Victor
103,123

G

Garrick, James
15,86,99,100,101,102,103,
104,105,123
Gee, Albert
6,10,17,55,78,81,103,105,116,
117
Ghatan, Andrew
38,61,107,119,122
Gilkey, Kelly
46
Gilmer, Brian
34,87,97,107,116,122
Glauberman, Molly
46
Goldberg, Michael
3,10,16,67,77,86,94,118,119
Goldstein, Barry
96
Green, John
10,78,86,103,105
Greenlee, Theodore
12,81,99,103,120
Green, Thomas
106,120
Gross, Ted
3,10,70,76,113,115,118,119,
123
Gundle, Kenneth
83,86,90,96

H

Haas, Norbert
1,18
Hacquebord, Heero
3
Hacquebord, Jacques
3,38,61,81,116,119
Hagen, Jennifer
108,122
Hamilton, Deven
23,26,119
Hanel, Douglas
3,10,83,90,97,98,114,116,
118
Hanel, Peggy
98
Hansen, Sigvard
10,63,71,120,123
Hanson, Andrea
46
Harnden, Emily
39
Harryman, Douglas
4,123
Henley, M. Bradford
10,76,116
Herring, Stanley
17,103
Huang, Jerry
10,72,79,113,114,116
Hudson, David
33,55,57,116

J

Jackins, Sarah
79,80,116
James, Carol
123
James, Don
103,123
Jegapragasan, Katie Kollitz
72
Jette, Jocelyn
19,43,114,118
Johnson, Colleen
15,100,101,103,105

K

Kates, Steven
15,85
Kennedy, Stephen
7,10,79,86,116
Kirby, Betsy
123
Kirby, Richard
121,123
Krengel, Walter
10,77,116,117,118,121
Krieg, James
1,39,116,118
Kwon, Ronald
3,7,11,70,86,116,117,118

L

Larson, Roger
11,78,103,104
Laurnen, Edwin
123
Lawson, Kevin
36,38,49,58,59,119
LeCocq
13,86,123
Ledoux, William
81,116,118,119
Lee, Michael J.
3,11,68,69,78,79,113,115,116,
117,118
Leopold, Seth
11,21,22,23,26,34,73,75,79,
115,116,117,118,119
Louie, Philip
87

M

Manner, Paul
11,16,79,82,85,114,115,116,
119
Masri, Bas
85
Matsen, Anne
4
Matsen, Frederick
4,11,17,19,34,43,79,80,82,
97,103,104,116,118,120,123
McAlinden, Audrey
27,29,117
McCornack, John
36,49
Meyer, Lauren
87
Meyer, Leslie
26
Mickelson, Dayne
87,90,115
Miller, Mark
108,122
Montgomery, Alexander
17
Mosca, Vincent
11,77,118,119
Mowery, Carol
11,77
Myers, Josh and Max
123

N

Neradilek, Moni Blazej
19,118
Nork, Sean
11,75,76,113,114,116,118

O

O'Kane, John
11,103,104,116,123

P

Parsons, Erin
72
Patterson, David
38,61,108,119,122
Posner, Karen
43,118

R

Requa, Ralph
100,102,105
Rice, Andrea
46,48
Routt, Milton
1,52,76,116
Rubash, Harry
13,16

S

Sabin, Janice
23,26,119
Sangeorzan, Bruce
11,16,75,76,81,86,113,114,
115,116,118,119
Schmale, Gregory
3,11,77,94,96,117,118
Shelby, Patrick
82,85
Sidles, John
11
Smith, Douglas
11
Sousa, Ted
70,85,119
Squyer, Emily
23,26,108,119,122
Srinivasan, Sundar
12,76,113,114,115,119
Staheli, Lynn
12,86,120
Stamper, Daniel
23,26,119
Steinman, Suzanne
12,77
Stephens, Linda
43,118
Swenson, Alan
52

T

Taitsman, Lisa
12,15,16,76,98,119
Tanner, Rachael
82,85
Teitz, Carol
12,15,78,86,99,103,104,105,
120
Tencer, Allan
12,116
Teteak, Colin
36,38,49,58,119
Timmons, Sarah
97
Twaddle, Bruce
8,12,18,78,86,105

W

Wagner, Theodore
5,12,79,97,115,118,120,123
Warme, Jeanne
79
Warme, Winston
12,17,22,34,43,73,79,114,116,
117,118,119
Weber, Kristy
14
Weis, Mary Ann
30,33,57,115,116,118,119
White, Klane
12,64,77,119
Whiting, Esther
123
Wilt, Grier
46
Wu, Jiann-Jiu
12,33,57,113
Wyss, HansJörg
3,123

Y

Yang, Liu
36,38,49,58,61,81,113,116,119